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Review Article

Associations between sleep bruxism and other sleep-related disorders in adults: a systematic review

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ABSTRACT

Objective: Systematic reviews on sleep bruxism (SB) as a comorbid condition of other sleep-related disorders are lacking. Such reviews would contribute to the insight of sleep clinicians into the occurrence of SB in patients with other sleep-related disorders, and into the underlying mechanisms of such comorbid associations. This systematic review aimed: 1. to determine the prevalence of SB in adults with other sleep-related disorders; and 2. to determine the associations between SB and other sleep-related disorders, and to explain the underlying mechanisms of these associations.

Methods: A systematic search on SB and sleep-related disorders was performed in PubMed, Embase, Cochrane Library, and Web of Science to identify eligible studies published until May 15, 2020. Quality assessment was performed using the Risk of Bias Assessment tool for Non-randomized Studies.

Results: Of the 1539 unique retrieved studies, 37 articles were included in this systematic review. The prevalence of SB in adult patients with obstructive sleep apnea, restless leg syndrome, periodic limb movement during sleep, sleep-related gastroesophageal reflux disease, REM behavior disorder (RBD), and sleep-related epilepsy was higher than that in the general population. The specific mechanisms behind these positive associations could not be identified.

Conclusions: SB is more prevalent in patients with the previously mentioned disorders than in the general population. Sleep arousal may be a common factor with which all the identified disorders are associated, except RBD and Parkinson's disease. The associations between SB and these identified sleep-related disorders call for more SB screening in patients with the abovementioned sleep-related disorders.

1. Introduction

Sleep bruxism (SB) is a repetitive jaw-muscle activity characterized by clenching or grinding of the teeth and/or by bracing or thrusting of the mandible [1]. The criteria for the assessment of definite SB are so far based on polysomnographic (PSG) recordings, which allow the identification of rhythmic masticatory muscle activity (RMMA) on electromyographic (EMG) traces [1,2]. Based on a systematic review on the epidemiology of SB, the prevalence of self-reported SB in adults is 12.8 ± 3.1% [3].

The etiology of SB is multifactorial [4,5], including biological factors, psychosocial factors, and lifestyle factors. According to observations among family members [6] and gene analysis studies [7,8], the occurrence of SB may partially be explained by both environmental and genetic factors [9,10]. Moreover, an imbalance in centrally acting neurotransmitters (eg, dopamine, serotonin) may play a role in the genesis of RMMA and SB [11,12]. Many

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psychosocial factors, such as anxiety, depression, stress, and maladaptive coping strategies, have been suggested to increase risk for SB [13,14]. Lifestyle factors like smoking, alcohol and caffeine intake have also been suggested to increase the risk of SB [15,16].

The potential negative consequences of SB described in literature are, amongst others, headache upon awakening, temporomandibular pain complaints [17,18], severe mechanical tooth wear, and tooth/dental restoration/implant fractures/failures [19,20]. Interestingly, nowadays, also some positive consequences of SB are suggested, for example, the condition having a protective role in maintaining airway patency in patients with obstructive sleep apnea (OSA) [21,22], promoting saliva secretion by mechanical salivary (parotid) gland stimulation for esophageal acid clearance [23,24], and even preventing cognitive decline when aging [25]. Although SB management should be based on the negative clinical consequence of SB [26], evidence-based recommendations at the individual level are not available at this moment [27]. So, it is still recommended that SB management is provided with caution within the framework of a conservative "multiple-P″ approach (ie, pep talk (counseling), plates (occlusal appliances), physiotherapy, psychotherapy, and pills (pharmacotherapy)) [26].

The genesis of most RMMA episodes seems to be preceded by a cascade of events in relation to sleep arousals, such as an increase in the autonomic sympathetic–cardiac activity, in the frequency of electroencephalographic (EEG) activity, in heart rate, in EMG activity of jaw-opening muscles, in breathing amplitude, and in blood pressure [28,29]. Several studies [28,30–32] further suggested that sleep arousals could be regarded as a permissive window for the initiation of RMMA episodes. The occurrence of arousals may therefore explain the correlations found in previous studies between SB and other sleep-related disorders that are associated with arousals (eg, OSA, periodic limb movements during sleep (PLMS), and epilepsy) [33–37]. However, systematic reviews on SB as a comorbid condition of other sleep-related disorders are lacking so far. Such reviews would contribute to the insight of sleep clinicians into the occurrence of SB in patients suffering from other sleep-related disorders, as well as into the underlying mechanisms of such comorbid associations. Therefore, the aims of this systematic review were: 1. to determine the prevalence of SB in adult patients with other sleep-related disorders; and 2. to determine the associations between SB and other sleep-related disorders, and to explain the underlying mechanisms of these associations. This systematic review will finalize with recommendations for sleep clinicians on how to proceed with the further prevention, assessment, and management of the possible negative consequences of SB as a comorbid condition in their patients with sleep disorders.

2. Materials and methods

The entire review process was conducted in accordance with the Preferred Reporting Items for Systematic Review and Meta-Analyses (PRISMA) statement [38]. This systematic review is registered at PROSPERO (registration number: CRD42020186555).

2.1. Search strategy

To identify relevant publications, systematic searches were conducted in the bibliographic databases PubMed (https://pubmed.ncbi.nlm.nih.gov/), Embase (https://www.embase.com), Cochrane Library (https://www.cochranelibrary.com/advanced-search), and Web of Science (https://www.webofknowledge.com/) from inception up to May 15, 2020, in collaboration with a medical information specialist (R.V.). In all four databases, the names and synonyms of sleep bruxism and all sleep-related disorders (including sleep wake disorders, parkinsonian disorders, epilepsy, gastroesophageal reflux, and REM behavior disorder) have been searched using text word to identify articles that used these names in their title or abstract. Moreover, in PubMed and Embase, where the index term (MeSH term and Emtree term, respectively) is available, the index term for sleep bruxism (appeared in PubMed as "Sleep Bruxism" [Mesh]; in Embase as 'sleep'/exp AND 'bruxism'/exp), as well as the index term containing all the sleep disorders (appeared in PubMed as "Sleep Wake Disorders"[Mesh]"; in Embase as 'sleep disorder'/exp) were further added into the search strategy to identify relevant articles. The reference lists of the identified articles were manually searched for relevant publications. Duplicate articles were excluded. All languages were accepted. The full search strategies for all databases can be found in Supplement A.

2.2. Article screening

The article screening included two phases: title and abstracts screening, and full-text review. Firstly, all identified titles and abstracts were independently screened by two reviewers (B.K. and D.L.). The inclusion criteria were: (1) studies on adult human subjects (age over 18 years old); (2) studies dealing with sleep-related disorders, diagnosed based on self-report (questionnaire or interview), clinical inspection, or PSG/polygraph; (3) studies dealing with SB, diagnosed by self-report (eg, reporting of teeth grinding sound by questionnaire or interview), clinical inspection (eg, tooth wear, masseter hypertrophy, and masticatory muscle fatigue or pain), and/or instrumental assessment (eg, scoring of SB episodes based on PSG, polygraphy, or EMG) [2]; and (4) studies having the following designs: observational studies, controlled clinical trials, or randomized controlled clinical trials. The exclusion criteria were: (1) studies on animals; (2) studies on children; and (3) certain publication types: editorials, letters, legal cases, interviews, and conference abstracts.

Secondly, for all potentially eligible studies identified after the first phase, the two reviewers (B.K. and D.L.) read the full texts independently to check if they fulfilled the eligibility criteria. Studies without accessible full text were excluded.

For both title and abstract screening and full-text review, differences in judgment were resolved through a consensus procedure between the two reviewers. If the differences remained, the issue was resolved by discussion with a third reviewer (G.A.).

Data concerning study design, methods, and results of the final selected studies were extracted by the above-identified two reviewers (B.K. and D.L.).

2.3. Quality assessment

Two reviewers (B.K. and D.L.) independently evaluated the methodological quality of the full-text papers, using an adapted version of "Risk of Bias Assessment Tool for Nonrandomized Studies (RoBANS)" (See Supplement B for the adapted version, and the study by Kim et al. [39] for the original version). The checklist assesses the potential risk of bias in the following aspects: (1) selection of participants, (2) confounding variables, (3) measurement of exposure, (4) blinding of outcome assessments, (5) incomplete outcome data, and (6) selective outcome reporting.

Different diagnostic methods have different advantages and disadvantages. Self-report could reflect the condition of SB over a prolonged period of time and reach a very large population. The clinical inspection could elevate the objectiveness of SB measurement by documenting related clinical signs and symptoms of SB, such as tooth wear, masticatory muscle hypertrophy, while it is still difficult to acquire objective sleep information and to determine the possible mechanisms for SB. PSG could offer objective and detailed parameters of sleep and jaw-muscle contraction

during the night. However, PSG only records jaw-muscle activity over a limited period of time and is less accessible for a large sample because its availability is limited and due to the high costs associated with this approach. During the adaptation of the RoBANS, the different characteristics of the diagnostic methods were taken into consideration. Specifically, when evaluating articles regarding our aim concerning prevalence, it was further required that sample size, based on empirical knowledge, should not be smaller than 100 for self-report studies, and 30 for PSG studies, in order to be categorized as low risk of bias for the 'selection of participants' section. When evaluating articles regarding our aim concerning mechanism, as long as the sample size was justified in the article, a low risk of bias was given for the 'selection of participants' section. In addition, studies using either self-report or PSG would be scored as low risk of bias for the 'measurement of exposure' section when evaluating articles regarding our aim concerning prevalence. In contrast, when evaluating articles regarding our aim concerning mechanism, only studies that employed PSG to diagnose SB would be regarded as low risk for the 'measurement of exposure' section.

3. Results

3.1. Literature search results

The literature search process and results are presented in Fig. 1. The employed search strategy identified 2635 articles in total, and the manual search identified one more. After duplicates were eliminated, 1593 articles remained for the title and abstract screening. According to the inclusion and exclusion criteria mentioned above, 1556 articles were excluded, and 37 articles thus qualified for the full-text reading phase. After full-text reading, all 37 articles qualified for this systematic review.

Among the 37 articles, 14 articles were related to SB and OSA, 7 articles to SB and restless legs syndrome (RLS)/PLMS, and 6 articles to SB and sleep-related gastroesophageal reflux disease (GERD). Another 5 articles were related to SB and insomnia, 3 articles to SB and Parkinson's Disease (PD), 1 article to SB and REM behavior disorders (RBD), and 3 articles to SB and sleep-related epilepsy. Further, there was one article studying SB with sleep talking and sleepwalking. Finally, there was one article that investigated the associations between SB and nightmares. Some of the articles included were involved in multiple associations, and subsequently counted more than once.

3.2. Sleep bruxism and obstructive sleep apnea

Fourteen articles [36,40–52] investigated the associations between SB and OSA. The characteristics of these articles are shown in Table 1.

Among these articles, six articles [41,42,46–48,50] reported the prevalence of SB in adult patients with OSA. One study [48] used a questionnaire to assess SB, while the other five studies [41,42,46,47,50] used PSG. Due to the limited number of participants and lack of representativeness of patients with OSA, four articles [41,46–48] were regarded as having high risks of bias in the "selection of participants" section. Besides, five articles [41,42,46,47,50] have high risks of bias in the "blinding of outcome assessment" section. Detailed quality assessment results of the articles that reported the prevalence of SB in adult patients with OSA are shown in Table 2. Based on the quality assessment, four articles [41,42,48,50] are regarded to have relatively higher quality than the other two articles [46,47], with only two sections of high risks of bias. The mean ages of the participants with OSA in the above four studies [41,42,48,50] ranged from 44.6 to 54.3 years. In addition, among these four studies, only Hesselbacher et al. [48] recruited

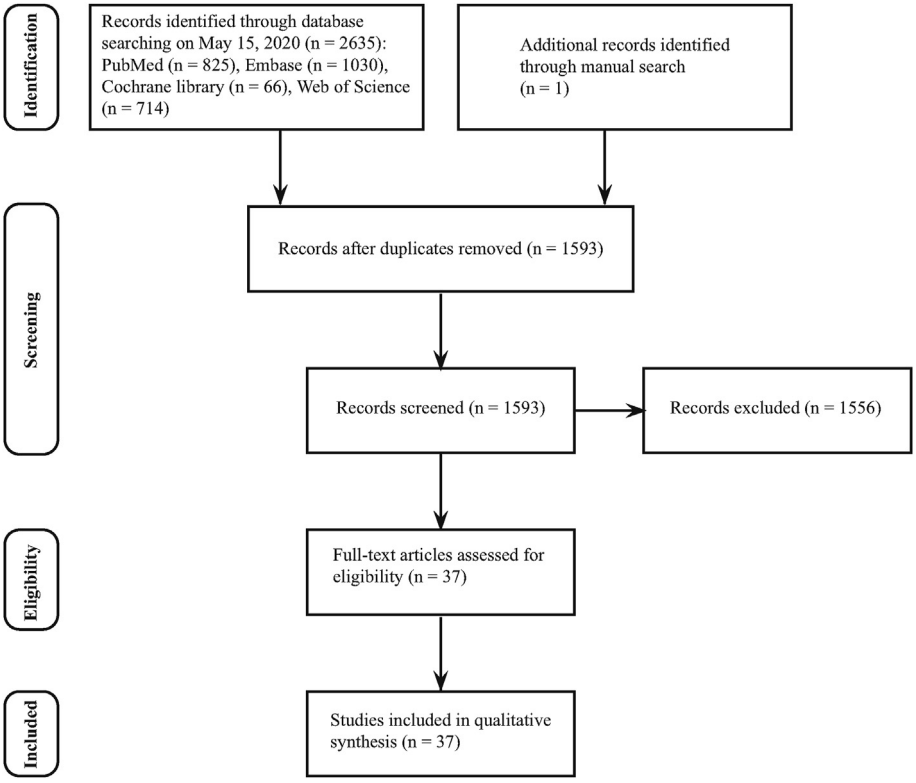

Fig. 1. Flow diagram of search strategy. Greyscale should be fine for this figure in print.

Table 1
Characteristics of studies on sleep bruxism (SB) and obstructive sleep apnea (OSA).

References (authors, year)	Study type	Sample	Match	Age (years)	Gender (M, %)	Country/Race	OSA/SDB diagnosis	SB diagnostic methods	SB scoring/ diagnosis criteria	SB Prevalence (%)	Associations/ mechanisms
Phillips et al., 1986 [40]	prospective	SDB 14; control 10	NR	SDB 52.0 ± 15.9; Control 50.2 ± 16.4	SDB 85.7; Control 90.0	USA/NR	PSG: AHI ≥10	PSG (masseter)	NR	N/A	The clench index and the AHI are positively correlated.
Okeson et al., 1991 [45]	prospective	SDB 12; control 12	age, gender	SDB 57.0 ± 11.5; Control 57.0 ± 11.7	SDB 100; Control 100	USA/NR	PSG: AHI≥10	PSG (masseter)	NR	N/A	Bruxing events are closely associated with sleep arousals.
Sjöholm et al., 2000 [46]	prospective	OSA 21	N/A	40.0 ± 9.2	90.5	Canada/NR	diagnosed by sleep Physician	SR; CA; PSG (submentalis)	Fulfill 2 out of 3: (1) SR (positive); (2) CA (positive);(3) RJM >2.5/h	47.6	SB is rarely directly associated with apneic or hypopneic events but is rather related to the disturbed sleep of patients with OSA.
Maluly et al., 2013 [47]	prospective	general population 1019	N/A	NR	NR	Brazil/NR	NR	QNR + PSG (masseter)	1) QNR (positive) 2) PSG (AASM [117]; RMMA ≥2)	9.0	The presence of SB was not associated with the presence of OSA.
Hosoya et al., 2014 [50]	prospective	OSA 67; control 16	NR	OSA 54.3 ± 13.2; Control 23.9 ± 5.5	OSA 73.1; Control 50.0	Japan/NR	PSG: AHI ≥5	PSG + AV (masseter)	Lavigne et al. [118]; RMMA index >4;	47.8	SB is a sequential event secondary to an arousal event that result from an obstructive apneic event;
Saito et al., 2014 [49]	prospective	OSA and SB 10	N/A	46.7 ± 11.5	100.0	Japan/NR	PSG: AHI>5	PSG + AV (masseter)	ICSD-2 [119]; Lavigne et al. [118]; RMMA index >4	N/A	SB events occurring close to sleep apneic hypopneic events are a secondary form of sleep bruxism.
Hesselbacher et al., 2014 [48]	Retrospective	OSA 300	N/A	M 46.8 ± 10.8; F 51.7 ± 9.5	50.0	USA /Caucasian, African American, Hispanic	PSG: AHI>5	QNR	QNR (positive)	26	The mean AHI and SpO_2 nadir were similar between OSA patients with and without sleep bruxism.
Saito et al., 2016 [36]	prospective	possible SB with suspicious OSA 59	N/A	44.8 ± 10.8	79.0	Japan/Japanese	PSG: AHI ≥5	PSG + AV (masseter)	ICSD-3 [86]; Rompré et al. [120]; RMMA index >2; and/or RMMA bursts >25/h	N/A	RMMA was moderately correlated with arousal, but not apneic or hypopneic events. SB genesis and OSA activity are probably influenced by different mechanisms.
Winck et al., 2017 [51]	prospective	OSA 9	N/A	46.3 ± 11.3	55.6	Portugal/NR	polygraph: AHI ≥15; or AHI ≥5 +symptoms	QNR + EMG (masseters)	AASM criteria [117]; RMMA index ≥2	N/A	No significant correlation was found between AHI and bruxism.

Tsujisaka et al., 2018 [52]	prospective	SB 16; SB + OSA 6	gender, BMI	SB 23.6 ± 1.9; SB + OSA 25.5 ± 1.2	SB 56.3; SB/OSA 83.3	Japan/NR	PSG: AHI≥5	QNR + PSG + AV (masseter and temporalis)	Rompré et al. [120] RMMA index ≥4;	N/A	RMMA after respiratory events was followed to arousals while those before respiratory events were mostly associated with central apnea;
Tan et al., 2019 [41]	retrospective	OSA 147	N/A	44.6 ± 12.8	68.0	Singapore/NR	PSG: AHI ≥5	PSG + AV (masseter)	AASM criteria [121]; Lavigne et al. [118]; RMMA index >4;	33.3	Patients with SB had more respiratory events and arousals than non-bruxers. A phenotypic subtype of OSA patients may present with SB as a physiologic response to a respiratory-related event.
Martynowicz et al., 2019 [42]	prospective	suspicious OSA 110	N/A	51.0 ± 14.2	60.0	Poland/NR	PSG: AHI ≥5	PSG + AV (masseter)	Lavigne et al. [118]; RMMA index ≥2	53.7	The relationship between OSA and SB depends on the degree of severity of OSA.
Smardz et al., 2020 [44]	prospective	probable SB; bruxer: 58, control: 19	NR′	total: 34.8 ± 10.8 /subgroup NR	total 27.3 (subgroup NR)	Poland/Caucasian	NR	PSG + AV (masseter)	ICSD-3 [86]: NR; RMMA index ≥2	N/A	The occurrence of tonic RMMA may be the key to understanding the causality between SB and sleep-disordered breathing.
De Holanda et al., 2020 [43]	prospective	bruxer: 58 Nonbruxer:58	Sex, age	bruxers 42.2 ± 14.5; control 42.6 ± 14.8	bruxers 43.1; control 43.1.	Brazil/NR	NR	PSG + AV (masseter)	20% MVC; Carra et al. [122]; RMMA index >2	N/A	Arousals index, respiratory disturbance index, and AHI were lower in bruxers than in non-bruxers.

Abbreviations: AASM = American Academy of Sleep Medicine; AHI = apnea-hypopnea index; AV = audio and video; BMI = body mass index; CA = clinical assessment; EMG = electromyography; ICSD = International Classification of Sleep Disorders; M = male; F = female; MVC = maximum voluntary contraction; N/A = not applicable; NR = not reported; PSG = polysomnography; QNR = questionnaire; RJM = rhythmic jaw movement; RMMA = rhythmic masticatory muscle activity; SDB = sleep-disordered breathing; SR = self-report.

Table 2
Quality assessment of the studies reporting the prevalence of sleep bruxism (SB) in patients with other sleep-related disorders using the Risk of Bias Assessment tool for Non-Randomized Studies (RoBANS).

Disorders	References (authors, year)	Risk of bias					
		Selection of participants	Confounding variables	Exposure measurement	Blinding of outcome assessment	Incomplete outcome data	Selective outcome reporting
OSA	Sjöholm et al., 2000 [46]	high	high	low	high	low	low
OSA, RLS, Insomnia	Maluly et al., 2013 [47]	high	low	low	high	low	high
OSA	Hesselbacher et al., 2014 [48]	high	high	low	low	low	low
OSA	Hosoya et al., 2014 [50]	low	high	low	high	low	low
OSA	Tan et al., 2019 [41]	high	low	low	high	low	low
OSA	Martynowicz et al., 2019 [42]	low	low	low	high	low	high
RLS/PLMS	Lavigne et al., 1994 [53]	high	low	low	low	low	low
Sleep-related GERD	Mengatto et al., 2013 [60]	high	low	low	low	low	low
Insomnia	de Campos et al., 2006 [63]	high	high	high	low	low	high
Insomnia	Blanken et al., 2019 [65]	low	high	high	low	low	low
RBD or PD	Abe et al., 2013 [66]	high	high	low	low	low	low
PD	Ylikoski et al., 2014 [67]	low	low	high	low	low	low
Sleep-related epilepsy	Khatami et al., 2006 [69]	high	high	high	low	low	low
Sleep-related epilepsy	Bisulli et al., 2010 [70]	low	high	low	low	low	low
Sleep-related epilepsy	Khachatryan et al., 2020 [37]	low	low	low	low	low	low
Nightmare	Serra-Negra et al., 2019 [71]	high	high	high	low	low	low

Abbreviations: GERD = gastroesophageal reflux disease; OSA = obstructive sleep apnea; PD = Parkinson's disease; PLMS = periodic limb movement during sleep; RBD = rapid eye movement sleep behavior disorder; RLS = restless legs syndrome.

the same number of females and males, while the other three studies [41,42,50] enrolled more males than females. Based on these four articles, the prevalence of self-reported SB in adult patients with OSA is 26.0% [48], while PSG-confirmed SB prevalence in adults with OSA ranges from 33.3% to 53.7% [41,42,50].

There were thirteen [36,40–46,48–52] out of the fourteen articles that studied the association between SB and OSA. Based on RoBANS, all these articles have high risks of bias in the "blinding of outcome assessment" section, because they fail to describe whether the investigators were blinded to the patients' information during the scoring of the PSG recordings. All but two articles [42,43] have high risks of bias in the "selection of participants" section because of a limited number of participants. Due to a lack of appropriate matching between the control group and the patients group, seven articles [36,40,44,48–50,52] were deemed to have high risks of bias in the "confounding variables" section. Detailed quality assessment results of articles that reported the association between SB and OSA are shown in Table 3. Overall, five PSG studies [41–43,45,46] only have two sections of high risks of bias, and were thus regarded as having high quality. Okeson et al. [45] reported that bruxing events are closely associated with sleep arousals. Sjöholm et al. [46] showed that sleep bruxism is rarely directly associated with respiratory events but is related to disturbed sleep in OSA. Martynowicz et al. [42] displayed that there is a positive correlation between SB events and sleep arousal in the entire group, while the association between SB and AHI was observed only in participants with mild and moderate OSA. Tan et al. [41] indicated that OSA patients with SB demonstrated a significantly higher respiratory arousal index ($P = 0.001$), AHI ($P = 0.003$) and oxygen desaturation index ($P = 0.005$) than OSA patients without SB. In addition, De Holanda et al. [43] reported that the AHI and arousal index were lower in bruxers than in non-bruxers.

3.3. Sleep bruxism and restless legs syndrome/periodic limb movement during sleep

Seven articles [34,48,53–57] investigated SB and RLS/PLMS. The extracted data are shown in Table 4.

Only Lavigne et al. [53] reported that, based on questionnaires, the prevalence of SB was 17.3% in adults who reported RLS, and 14.5% in those who reported unpleasant leg sensation during sleep. There were roughly equal male and female participants in the study. The age distribution of the participants had also been controlled for this study to have a similar number of participants in every age range. This article only has a high risk of bias in the "selection of participants" section, and was thus regarded as having high quality. Detailed quality assessment results of this article are shown in Table 2.

The other six articles [34,48,54–57] studied the association between SB and RLS/PLMS. Five of these articles [34,48,54,56,57] have high risks of bias in the "selection of participants" section because of small sample sizes and the samples' lack of representativeness. Two articles [34,55] were regarded as relatively high quality articles, with only one high risk of bias section. Detailed quality assessment results of articles that reported the association between SB and RLS/PLMS are shown in Table 3. Saletu et al. [55] performed a case–control study with PSG and demonstrated that the PLM index was higher in SB patients than in controls ($P < 0.05$). Moreover, in another PSG study, van der Zaag et al. [34] found that the combined SB/PLMS events are more likely to be linked with arousal than without arousal in SB patients ($P < 0.001$).

3.4. Sleep bruxism and sleep-related gastroesophageal reflux disease

Six articles [48,58–62] were identified that studied SB and sleep-related GERD (extracted data in Table 4).

Mengatto et al. [60] reported the occurrence of probable SB (validated by self-report and clinical inspection) in adult patients with sleep-related GERD to be 73.7%. The majority of the selected participants of this study were females (71.7%). The mean age of these participants was 44 years old. According to the assessment tool, this article was regarded as having high quality, with only the "selection of participants" section being scored as high risk of bias. Detailed quality assessment results of this article are shown in Table 2.

All six articles [48,58–62] reported the association between SB and sleep-related GERD. Five articles [48,58,59,61,62] have high risks of bias for the "selection of participants" section because of low validity of the selection mechanism and limited number of

Table 3
Quality assessment of the studies investigating the association between sleep bruxism (SB) and other sleep-related disorders using the Risk of Bias Assessment tool for Non-Randomized Studies (RoBANS).

Disorders	References (authors, year)	Risk of bias					
		Selection of participants	Confounding variables	Exposure measurement	Blinding of outcome assessment	Incomplete outcome data	Selective outcome reporting
OSA	Phillips et al., 1986 [40]	High	high	low	high	low	low
OSA	Okeson et al., 1991 [45]	High	low	low	high	low	low
OSA	Sjöholm et al., 2000 [46]	High	low	low	high	low	low
OSA	Hesselbacher et al., 2014 [48]	high	high	high	high	low	high
OSA	Hosoya et al., 2014 [50]	high	high	low	high	low	low
OSA	Saito et al., 2014 [49]	high	high	low	high	low	low
OSA	Winck et al., 2017 [51]	high	low	high	high	low	low
OSA	Saito et al., 2016 [36]	high	high	low	high	low	low
OSA	Tsujisaka et al., 2018 [52]	high	high	low	high	low	low
OSA	Tan et al., 2019 [41]	high	low	low	high	low	low
OSA	Martynowicz et al., 2019 [42]	low	low	low	high	low	high
OSA	Smardz et al., 2020 [44]	high	high	low	high	low	high
OSA	De Holanda et al., 2020 [43]	low	low	low	high	low	low
RLS/PLMS	Ahlberg et al., 2005 [54]	high	high	high	low	low	low
Insomnia, RLS/PLMS	Saletu et al., 2010 [55]	low	low	low	high	low	low
RLS/PLMS	van der Zaag et al., 2014 [34]	high	low	low	low	low	low
RLS/PLMS	Han et al., 2019 [56]	high	low	low	high	low	low
RLS/PLMS	Miki et al., 2020 [57]	high	high	low	low	low	low
Sleep-related GERD	Miyawaki et al., 2003 [58]	high	low	low	high	low	low
Sleep-related GERD	Miyawaki et al., 2004 [59]	high	high	low	low	low	low
Sleep-related GERD	Mengatto et al., 2013 [60]	low	low	high	low	low	low
Sleep-related GERD	Li et al., 2018 [61]	high	low	low	low	low	low
Sleep-related GERD	Li et al., 2018 [62]	high	low	low	low	low	low
Insomnia	Ahlberg et al., 2008 [64]	high	high	high	low	low	low
Sleep-related RBD/PD	Abe et al., 2013 [66]	high	high	low	low	low	low
Sleep-related PD	Verhoeff et al., 2018 [68]	high	high	high	low	low	high
Sleep talking, Sleepwalking, Nightmare	Hublin et al., 2001 [72]	high	low	high	low	low	low

Abbreviations: GERD = gastroesophageal reflux disease; OSA = obstructive sleep apnea; PD = Parkinson's disease; PLMS = periodic limb movement during sleep; RBD = rapid eye movement sleep behavior disorder; RLS = restless legs syndrome.

participants. Three articles [60–62] have a relatively higher quality than the other three articles [48,58,59], with only one section scored as high risk of bias. Detailed quality assessment results of articles that reported the association between SB and sleep-related GERD are shown in Table 3. In the three articles of high quality [60–62], SB was diagnosed based on self-report and clinical inspection, but not on PSG. Mengatto et al. [60] demonstrated that sleep-related GERD was associated with SB ($P = 0.017$; OR = 6.58). In addition, age [60], gender [61], and the duration of GERD episodes [62] influence the association between SB and sleep-related GERD.

3.5. Sleep bruxism and insomnia

There are five articles [47,55,63–65] that studied SB and insomnia (extracted data in Table 5).

Three [47,63,65] out of the five articles reported the prevalence of SB in adult patients with insomnia. All of these three articles have high risks of bias in the "selection of participants" section. De Campos et al. [63] only included postmenopausal females, and the other two articles [47,65] only reported insomnia-related findings for a part of their included participants. Among the portion of participants, on which Maluly et al. [47] based their insomnia findings, 54% of them were female, but the age distribution was not reported for these participants. Two articles [63,65] did not describe their criteria for SB diagnosis. Detailed quality assessment results of articles that reported the prevalence of SB in patients with insomnia are shown in Table 2. Taking the above-indicated methodological shortcomings into account, the prevalence of PSG-confirmed SB in patients with insomnia was determined to be 16.5% [47].

Two [55,64] of the five articles reported the possible mechanism and the association between SB and insomnia. The article by Saletu et al. [55] has a higher quality than the article by Ahlberg et al. [64], with only one high risk of bias. Detailed quality assessment results of articles that reported the association between SB and insomnia are shown in Table 3. Based on PSG outcomes, it suggested that compared with controls, sleep bruxers showed no significant difference in sleep initiation but did show significantly deteriorated sleep maintenance [55].

3.6. Sleep bruxism and REM behavior disorder, and Parkinson's disease

Three articles [66–68] investigated the association between SB and PD. One article [66] investigated SB and RBD (extracted data in Table 5).

The questionnaire-based study by Ylikoski et al. [67] included a large number of participants (n = 661) and reported an SB prevalence of 4.7% in patients with PD. Fifty-three percent of the participants in the study by Ylikoski et al. [67] were male. The mean age of the participants was 68 years old. Abe et al. [66], a PSG and medical RBD diagnosis study, reported an SB occurrence of 25% among the 25 included patients with RBD. Seventy-eight percent of the participants in the study by Abe et al. [66] were male. The mean age of those participants was 66 years old. Both of the two studies had high quality, and the detailed quality assessment results of these two articles are shown in Table 2.

Two articles [66,68] reported the association between SB and PD, and one article [66] investigated the association between SB and RBD. Detailed quality assessment results of articles that reported the association between SB and PD as well as the association

Table 4
Characteristics of studies on restless legs syndrome/periodic leg movement during sleep (RLS/PLMS) and sleep-related gastroesophageal reflux disease (GERD).

References (authors, year)	Study type	Sample	Match	Age (years)	Gender (M, %)	Country/Race	Diagnostic criteria for sleep-related disorders	SB diagnostic methods	SB scoring/ diagnosis criteria	SB Prevalence/ occurrence (%)	Associations/ mechanisms
Lavigne et al., 1994 [53]	prospective	general population 2019	N/A	18-29: 29%, 30-44: 31%, 45-59: 20%, >60: 20%	49.0	Canada/ Francophone, Anglophone Canadian	RLS: QNR	QNR	SR (positive)	In patients with RLS: 10.9	NR
Ahlberg et al., 2005 [54]	prospective	regular shift 257; irregular shift 617	N/A	regular shift: M 45.0 ± 10.6, F 42.6 ± 10.7; irregular shift: M 47.4 ± 9.7, F 45.5 ± 10.1	regular shift: 46.7; irregular shift: 56.6	Finland/NR	RLS: SR based on NIH workshop report	QNR	SR (positive)	NR	Self-reported bruxism was positively associated with RLS (OR: 2.0; $P = 0.036$)
Saletu et al., 2010 [55]	prospective	SB 21; control 21	age, gender	SB 45.1 ± 12.6; control 45.0 ± 12.7	SB: 47.6; control: 47.6	Austria/NR	PLMS: ASDA Atlas Task Force	PSG	Lavigne et al. [118]	NR	PLMs were significantly elevated in bruxers as compared with control.
van der Zaag et al., 2014 [34]	prospective	SB 17; control 11	age, gender	SB: 32.1 ± 6.5; control:34.5 ± 12.8	SB 29.4; control 36.4	Netherlands/NR	PLMS: ICSD, revised [123] criteria	PSG	Automatic analyzing tool; 10% MVC	NR	The combined SB/ PLMS index is larger than isolated SB index or isolated PLM index; the combined SB/PLMS with arousal events are larger than combined SB/PLMS without arousal.
Han et al., 2019 [56]	prospective	SB 8; control 9	NR	SB: 21.4 ± 1.9; control:21.8 ± 1.8	SB: 37.5; control: 22.2	China/NR	Leg movement: ICSD-3 [86] criteria	SR; CA; PSG + AV	SR (positive); CA (positive) PSG: ICSD-3 [86]	NR	In SB patients, most RMMAs and LMs are associated with each other. In normal subjects, most RMMAs are associated with LMs, while most LMs are isolated.
Miki et al., 2020 [57]	prospective	Subjects 14	N/A	31.5 ± 5.7	71.4	Japan/NR	Leg movement: video + EMG (tibialis)	PSG + AV	Automatic analyzing tool; 10% MVC	NR	Lower leg movement was observed more frequently in concomitance with arousal and SB than in arousal without SB ($P < 0.01$).
Miyawaki et al., 2003 [58]	prospective	SB 10; control 10	age, gender height, weight	SB: 27.0 ± 7.0; Control: 26.4 ± 4.7	SB: 50; control: 60	Japan/NR	GERD: Esophageal PH-metric and manometric system	AV + polygraph (EMG: temporalis)	10% MVC; RMMA index≥4, bursts index ≥25, more than 2 tooth-grinding sounds per night.	NR	Around 60% of the RMMAs occurred during GER episodes; frequency of RMMA is lower after PPI intake compared with after placebo intake

Miyawaki et al., 2004 [59]	prospective	volunteers 12	N/A	24.0 ± 2.1	33.3	Japan/NR	GERD: Kahrilas et al. [124]	AV + polygraph (EMG: temporalis)	10% MVC, confirmed by AV	NR	The frequency of RMMA was significantly higher during periods of decreased esophageal pH than during other times.
Mengatto et al., 2013 [60]	prospective	GERD 19; Non-GERD 26	NR	44.6 ± 14.0	GERD:36.9 Non-GERD: 23.1	Brazil/NR	GERD: Montreal criteria [125]	SR + CA	SR (positive); CA (positive)	In patients with GERD: 73.7	GERD was associated with SB ($P = 0.001$).
Hesselbacher et al., 2014 [48]	retrospective	300 OSA	N/A	M 46.8 ± 10.8; F 51.7 ± 9.5	50	USA/Caucasian, African American, Hispanic	GERD/RLS: QNR	QNR	QNR (positive)	NR	Bruxism was associated with nocturnal GERD ($P = 0.008$) and with RLS ($P = 0.01$).
Li et al., 2018 [61]	prospective	SB 887; control 887	age, gender	Median (Q1-Q3) SB: 27 (22–37); control: 28 (22–37)	SB: 39.7 control: 39.7	China/NR	GERD: Montreal criteria [125]	QNR + CA	ICSD-3 [86]	NR	GERD was significantly associated with bruxism (OR = 7.95, $P < 0.001$).
Li et al., 2018 [62]	prospective	SB 398; control 398	age, gender	Median (Q1-Q3) SB: 28 (22–38); control: 28 (23–39)	SB: 40.5 control: 40.5	China/NR	GERD: Montreal criteria [125]	QNR + CA	ICSD-3 [86]	NR	GERD was associated with bruxism; Patients with a longer duration of GERD symptoms have a higher OR for bruxism than those with a shorter duration.

Abbreviations: AASM = American Academy of Sleep Medicine; ASDA = American Sleep Disorders Association; AV = audio and video; CA = clinical assessment; F = female; GERD = gastroesophageal reflux disease; ICSD-3 = International Classification of Sleep Disorders; M = male; MVC = maximum voluntary contraction; N/A = not applicable; NR = not reported; OR = odd ratio; PH = potential of hydrogen; PLMS = periodic limb movement during sleep; PPI = proton pump inhibitor; PSG = polysomnography; Q1: first quartile; Q3: third quartile; QNR = questionnaire; RLS = restless legs syndrome; SB = sleep bruxism; SR = self-report.

Table 5
Characteristics of studies on insomnia, REM behavior disorder (RBD), Parkinson's Disease (PD), epilepsy, and other sleep-related disorders.

References (authors, year)	Study type	Sample	Match	Age (years)	Gender (M, %)	Country/ Race	Diagnostic criteria for sleep-related disorders	SB diagnostic methods	SB scoring/ diagnosis criteria	SB Prevalence/ occurrence (%)	Associations/ mechanism
Hachul de Campos et al., 2006 [63]	prospective	38 females with insomnia complaints	N/A	55.0 ± 4.0	0	Brazil/NR	Insomnia: SR & PSG	QNR	NR	in patients with insomnia 2.6	NR
Ahlberg et al., 2008 [64]	prospective	regular shift: 257; irregular shift: 617	N/A	irregular shift: M 45.0 ± 10.6, F 42.6 ± 10.7; regular shift: M 47.4 ± 9.7, F 45.5 ± 10.1	irregular shift: 56.6; regular shift: 46.7	Finland/NR	Insomnia: QNR based on ICSD [123]	QNR	NR	NR	Frequent SB was associated with DIS ($P = 0.019$) and DS ($P = 0.021$)
Saletu et al., 2010 [55]	prospective	SB 21; Controls 21	age, gender	SB: 45.1 ± 12.6; control: 45.0 ± 12.7	SB: 47.6; control: 47.6	Austria/NR	Insomnia: ICD-10 [126]	PSG	Lavigne et al. [118]	NR	Sleep bruxers showed no significant difference in sleep initiation but significantly deteriorated sleep maintenance.
Maluly et al., 2013 [47]	prospective	1019 (partially reported)	N/A	20-29 (22.72%); 30-39 (24.48%); 40-49 (23.04%); 50-59 (15.84%); 60-80 (13.92%)	45.8 (partially reported)	Brazil/NR	Insomnia: QNR	PSG	AASM criteria [117]	in patients with insomnia 16.5	An association between SB and insomnia was detected ($\chi^2 = 5.69$, $P < 0.01$).
Blanken et al., 2019 [65]	retrospective	126 (partially reported)	NR	NR	NR	Netherlands/ NR	Insomnia: QNR	QNR	NR	in patients with insomnia 6.6	NR
Abe et al., 2013 [66]	prospective	iRBD 13; RBD-PD 13; control 9	age	iRBD: 65.3 ± 3.1; RBD-PD: 67.1 ± 2.6; control: 65.1 ± 4.0	iRBD: 76.9; RBD-PD: 80; control: 55.6	Canada/NR	(1) RBD based on ICSD [119] (2) PD diagnosed by specialist	PSG + AV (EMG: chin/masseter)	Two of the three were met: RMMA index≥4, bursts index ≥25, more than 2 tooth-grinding sounds per night	in patients with RBD 25.0	1) iRBD patients had significantly higher RMMA index during REM than controls; 2) iRBD and RBD-PD patients had higher RMMA index during sleep than controls
Ylikoski et al., 2014 [67]	prospective	PD 661	N/A	68.8 ± 8.5	53.0	Finland/NR	PD: diagnosis by neurologist	QNR	NR	in patients with PD 4.7	NR
Verhoeff et al., 2018 [68]	prospective	PD or PR 368; control 340	NR	PD or PR: 67 ± 9.3; Control: 65 ± 9.3	PD or PR: 49; control: 37.	Netherlands/ NR	PD: NR	QNR	Lobbezoo et al. [1]	NR	A significant association between possible SB and PD ($P = 0.001$).
Khatami et al., 2006 [69]	prospective	Epilepsy 100; non-epilepsy 90	age	epilepsy: 47 (mean); non-epilepsy:44 (mean)	epilepsy: 63; non-epilepsy: 46	Switzerland/ NR	Epilepsy: QNR International League Against Epilepsy	QNR	NR	in patients with epilepsy 10	NR
Bisulli et al., 2010 [70]	mixed	NFLE 33; Control 31	age, gender	NFLE: 31.9 ± 12.4; control: 31.3 ± 11.8	NFLE: 54.5 control: 51.6	Italy/NR	Epilepsy: PSG + video: ≥ 1 major epileptic episode or ≥ 2 minor stereotyped episodes	Interview	NR	in patients with NFLE 12	Bruxism occurred more frequently in the proband versus the control group (OR = 5.4; $P = 0.017$)

Khachatryan et al., 2020 [37]	prospective	Epilepsy 175; Controls 130	age, gender	epilepsy: 35.4 ± 13.7; control: 33.6 ± 11.3	epilepsy: 52.6; control: 52.3	Armenia/ NR	Epilepsy: Fisher et al. [127] + neuroimaging and EEG		[86]	in patients with epilepsy 23.7	SB occurred more frequently in epilepsy group than in control ($\chi^2 = 18.7$; $P < 0.05$).
Serra-Negra et al., 2019 [71]	prospective	119 adults	N/A	24.8 ± 2.6	43.9	Brazil/NR	Nightmare: QNR used by Serra et al. [128]	QNR	Lobbezoo et al. [1]	in patients with nightmare 38.3	Possible SB is associated with nightmares at least once a week ($P = 0.008$).
Hublin et al., 2001 [72]	prospective	11,220 twins (8567 responded in adult)	N/A	NR	NR	Finland/NR	Parasomnia: NR	QNR	QNR	NR	There is significant correlation between SB and sleepwalking, sleep talking, and nightmare.

Abbreviations: AASM = American Academy of Sleep Medicine; ASDA = American Sleep Disorders Association; AV = audio and video; DIS = difficulty initiating sleep; DS = disturbed sleep; EEG: electroencephalograph; EMG = electromyography; F = female; ICD= International Statistical Classification of Diseases and Related Health Problems; ICSD = International Classification of Sleep Disorders; (i)RBD = (idiopathic) REM behavior disorder; M = male; N/A = not applicable; NFLE = nocturnal frontal lobe epilepsy; NR = not reported; PD = Parkinson's disease; PR = parkinsonism; PSG = polysomnography; QNR = questionnaire; SB = sleep bruxism; SR = self-report; AV = audio and video.

between SB and RBD are shown in Table 3. Even though both articles suggested an association between SB and PD, and between SB and RBD, no underlying mechanism has been reported.

3.7. Sleep bruxism and sleep-related epilepsy

There are three articles [37,69,70] that investigated SB and sleep-related epilepsy (extracted data in Table 5).

All three articles reported the prevalence of SB in adult patients with sleep-related epilepsy. Detailed quality assessment results of these articles are shown in Table 2. The study by Khachatryan et al. [37] enrolled a relatively large number of participants (175 patients with epilepsy and 130 controls) and was regarded as the best quality article among the three identified articles [37,69,70]. 23.7% of the patients with epilepsy reported SB by an interview, compared with 5.4% of controls. The age and gender of the epilepsy and control group were matched. The mean ages of the participants of the two groups were 33 and 35 years old. Fifty-two percent of the participants of both groups were males. No underlying mechanism was reported in any of the three studies.

3.8. Sleep bruxism and other sleep-related disorders

One article [71], based on a questionnaire, reported the prevalence of possible SB in patients with nightmare. Also, another questionnaire study [72] suggested a correlation between SB and sleepwalking, as well as SB and sleep talking. The characteristics of these two articles are shown in Table 5. Due to the relatively high risk of bias of these two articles (details in Table 2andTable 3), no reliable results could be extracted.

4. Discussion

This systematic review was conducted: 1. to determine the prevalence of SB in patients with other sleep-related disorders; and 2. to determine the associations between SB and other sleep-related disorders, and to explain the underlying mechanisms for the associations found. As such, several disorders have been identified, including OSA, RLS/PLMS, sleep-related GERD, insomnia, Parkinson's disease, RBD, and sleep-related epilepsy. Below, we will discuss these findings in relation to the clinical practice of sleep physicians as well as that of dental practitioners to promote better cooperation. Further, we provide recommendations for future studies [73,74].

4.1. Sleep bruxism and obstructive sleep apnea

Four articles [41,42,48,50] reported that the prevalence of SB in adult patients with OSA ranges from 26.0% to 53.7%, which is much higher than that in the general population (12.8%) [3]. Nonetheless, there is a significant discrepancy in the prevalence among studies. Since the diagnosis of definite SB should be confirmed by PSG [2], a prevalence of 26.0% from one questionnaire study [48] was considered biased. Although the other three PSG studies employed the American Academy of Sleep Medicine (AASM) manual for SB scoring, two of them [41,50] set the cutoff value of RMMA index at four episodes/hour for SB diagnosis while the third one [42] set the cutoff at two episodes/hour. This might partially explain the lower prevalence in the first two PSG studies than that in the last one (33.3%, 47.6% vs. 53.7%). Apart from this, previous studies have reported that the prevalence of SB differs among age groups, genders, and races [3,48,75]. Thus, the diversity of these confounders among studies may also contribute to the variation in SB prevalence in patients with OSA.

Concerning the mechanism, as OSA is characterized by repetitive apneic or hypopneic events that often result in sleep arousals, most studies identified in this review investigated the relationship between SB and apneic hypopneic events, and the relationship between SB and sleep arousals. A PSG study composed of 14 patients with OSA [40] reported that the clenching index was positively correlated with the AHI. However, later PSG studies [36,41] with larger sample sizes found no association between the RMMA index and AHI. Besides, some studies [46,49] showed that while part of SB episodes occurred after apneic hypopneic events, a large number of SB episodes were unrelated to the termination of apneic hypopneic events. The abovementioned evidence suggests a weak association between SB and apneic hypopneic events.

It is also possible that, as suggested by some PSG studies, only a subtype of RMMA (phasic or tonic) was associated with apneic hypopneic events [44,50]. Hosoya et al. [50] selected OSA patients for whom phasic type was dominant to assess such an association, and concluded that phasic RMMA positively correlated with apnea-hypopnea index. On the contrary, Smardz et al. [44] selected individuals from a dental specialty clinic (prosthodontics) with probable SB and no clear OSA diagnosis who presented a dominance of the tonic type, and concluded that tonic RMMA could be associated with the formation of respiratory events. Based on these contrasting findings, we could speculate that the predominant subtype of RMMA may vary between the OSA population and SB population, and that different phenotypes of RMMA may have different causal relationships with respiratory events. Also, as Smardz et al. [44] enrolled participants with relatively younger age (18–63 years with a mean of 35 years) than Hosoya et al. [50] (mean age = 54 years), age might be a factor that affects the dominant pattern of jaw-muscle activity as well. In addition, another high quality PSG study suggested that the close association between SB and OSA is only present in mild to moderate OSA [42]. Taken together, these results suggest that the association between RMMA and respiratory events may be present only at a subtype or subgroup level.

Despite the above, another PSG study [76] indicated that the occurrence of masseter muscle contractions time-linked to apneic hypopneic events in patients with OSA is related to sleep arousals that result from apneic hypopneic events rather than to the apneic hypopneic events *per se*. Also, one PSG study [52] reported that after apneic hypopneic events, SB episodes occurred more frequently when sleep arousals were present than when sleep arousals were absent. In line with this, three studies [41,45,46] of higher quality included in this review denoted that SB is positively associated with sleep arousals in patients with OSA. This finding agrees with some other studies [32,77,78] that stated that SB is an oromotor activity secondary to sleep arousals. Taking all the evidence into consideration, we therefore postulate that the association between SB and OSA may depend on the presence of sleep arousals in patients with OSA.

It is noteworthy that a recent study [43] of high quality showed that PSG-confirmed bruxers had lower AHI and arousal indices than non-bruxers and that OSA decreased the risk for SB (odds ratio = 0.55, P = 0.173). The results suggested an inverse association between OSA and SB. As explained in that study, partially obstructed breathing that is not classified as OSA could explain the higher frequency of SB in patients without OSA [43]. Besides, as discussed above, the occurrence of SB might be related to arousals. In the OSA population, most arousals result from respiratory events, while in bruxers, arousal may be triggered by different stimuli. Thus, the difference in the composition of samples among studies may contribute to contrary conclusions concerning the association between OSA and SB.

In addition, OSA has been reported to be more prevalent in males than in females, and in older patients than in the younger ones [79]. On the other hand, based on currently available evidence, SB prevalence seems to be equal between genders [47,53], and decreases with aging [53,75]. Given the fact that age and gender have different influences on SB and OSA, we can speculate that the prevalence of SB in patients with OSA, as well as the association between SB and OSA, may vary among age groups and genders. However, none of the included studies have investigated the effect of age and gender on the association between SB and OSA.

Considering the high prevalence of SB in patients with OSA (33.3%–53.7%), sleep physicians are urged to consider SB as a common comorbidity of OSA. As SB is suggested to be related to sleep arousals, we speculate that SB episodes related to respiratory arousals would decrease significantly by effective OSA treatment. This has been proved by a previous PSG study [76]. Also, some studies [80–83] have reported that, to some extent, OSA therapies (such as oral appliances and continuous positive airway pressure) can reduce the frequency of SB episodes as well as the signs and symptoms of SB. Thus, for patients with concomitant OSA and SB, OSA should be treated first. The sleep physicians should then check if the negative consequences of SB, as summarized in the introduction, are still severe enough that the patients need collaborative management by sleep physicians and dental practitioners.

Although SB is more prevalent in OSA individuals, which may suggest a close association between the two conditions, solid evidence in the mechanism to support this association is still limited. Current evidence indicated that the close association between SB and OSA might be related to the presence of arousal. Due to the limited samples of previous studies, large-scale PSG studies in patients with OSA are still needed to confirm the role of arousals in the relationship between SB and OSA, as well as the prevalence rate of SB in adult patients with OSA. Age and gender should also be considered in future studies on SB prevalence and the underlying mechanism of the association between OSA and SB.

4.2. Sleep bruxism and restless legs syndrome/periodic limb movement during sleep

Even though one article has reported the SB prevalence in patients with RLS [53], both SB and RLS were diagnosed based on a questionnaire. What could be concluded from this article is that the prevalence of SB in adult patients with RLS (17.3%) is relatively higher than that in the general population (12.8%) [3,53]. In addition, van der Zaag et al. [34] reported that the combined SB/PLMS index is significantly higher than the isolated ones in SB patients, which further indicates the positive association between these disorders.

In terms of mechanism, both SB and RLS/PLMS were found to be associated with arousal events [34,55,57]. Although the three identified studies [34,55,57] employed PSG to assess SB and RLS/PLMS, they included too small sample sizes, and their methods leave room for improvement. Van der Zaag et al. [34] and Miki et al. [57] both used 10% maximum voluntary contraction (MVC) as the cutoff to score SB episodes. Saletu et al. [55] used 20% MVC as the cutoff to score SB episodes. The use of MVC to score SB episodes does not take the real-time fluctuation of the EMG signal, due to, eg, sweating [84] and body movement [85], into consideration. However, despite these limitations, the positive correlations between SB and arousals as well as RLS/PLMS and arousals are the same for all the identified studies, which strengthens the validity of the reported findings.

According to Lavigne et al. [53], the prevalence of SB decreased, while the prevalence of RLS increased as the age of the participants

increased, based on a large population survey in Canada. However, how the age differences could influence the association between SB and RLS/PLMS, is still to be investigated in future studies.

The common co-occurrence of SB and RLS/PLMS may suggest that when screening and treating patients with RLS/PLMS, sleep physicians should also take the probable SB signs and symptoms into consideration. Due to the relative scarcity of isolated SB or RLS/PLMS episodes [34], it could be speculated that successful treatment of RLS/PLMS by the sleep specialist could result in a decrease of SB as well. On the other hand, if treatment of RLS/PLMS does not decrease the severity of the symptoms of SB, and SB is causing obvious negative consequences (summarized in the introduction), the physician should seek collaboration with dental practitioners.

Future large-scale population studies, using higher validity methods, are needed to acquire a precise SB prevalence rate in patients with RLS/PLMS and further elucidate the role of sleep arousal in the association between SB and RLS/PLMS. Finally, large sample studies focusing on the effect of the RLS/PLMS treatment on SB are needed to further examine the finding that RLS/PLMS and SB episodes are more often combined than isolated in clinical settings.

4.3. Sleep bruxism and sleep-related gastroesophageal reflux disease

Only one article [60] reported the occurrence of SB in adult patients with sleep-related GERD (73.7%), which is much higher than that in the general population (12.8%) [3]. However, SB diagnosis was established based on self-report/partner-report and a clinical examination, without performing PSG. Thus, the specific prevalence rates based on PSG still need to be determined in future studies.

Based on the results of the identified articles, it is suggested that SB and sleep-related GERD are associated. The three articles of higher quality [60–62] all measured SB using self-reports, because PSG is not the standard of care for patients with GERD [86]. However, we still can speculate, based on Miyawaki et al. [58], that SB episodes, together with swallowing, could be a response towards acid reflux episodes. Furthermore, it has been reported that nocturnal gastroesophageal reflux episodes were often associated with arousals [23,87,88]. At the same time, SB has long been associated with arousals [28,30–32]. So, it could be summarized that SB and sleep-related GERD are associated, and that arousal seems to be the bridging factor between both conditions.

It has been speculated that masticatory muscle activity has a positive role in stimulating the salivary gland secretion, accelerating esophageal acid clearance [23]. Subsequently, a collaboration between sleep physicians and dental practitioners is recommended to manage GERD as well as the possible negative consequences of SB (summarized in the introduction).

Based on the current identified literature, SB and sleep-related GERD seem to be associated with each other, yet the specific SB prevalence in patients with sleep-related GERD needs further investigation. Moreover, no EEG monitoring was implemented in sleep-related GERD studies. Consequently, it is impossible to objectively determine whether the muscle contraction detected by EMG was during sleep or wake. Thus, we recommend future studies to use PSG to measure SB as well as arousal to study the association between SB and sleep-related GERD. We also recommend the enrollment of more participants to establish the prevalence of SB in patients with sleep-related GERD as to achieve higher statistical power. At the same time, esophageal pH monitoring is recommended to objectively measure individual sleep-related gastroesophageal reflux events.

4.4. Sleep bruxism and insomnia

A newly published PSG study by Maluly et al. [89] found that the prevalence of SB in adults with insomnia complaints is 17.7%. This is higher than the SB prevalence in the general population, which is around 12.8%, as reported by a review study [3]. However, it is necessary to point out that the gold standard for diagnosing insomnia requires not only the patients' self-reports about their insomnia complaints, but also the exclusion by sleep physicians that other sleep disorders may be causing the sleep/wake difficulty [86]. Many studies suggested that insomnia is also associated with OSA [90,91], RLS [92,93], and sleep-related GERD [94,95]. Thus, if there is no physicians' diagnosis, the reported sleep complaints may be secondary to OSA, RLS, or sleep-related GERD. Subsequently, there could very likely be an overestimation of insomnia based on questionnaires as compared to insomnia based on physician diagnosis. In the study by Maluly et al. [89], the diagnosis of insomnia was based on patients' self-reports (Diagnostic and Statistical Manual of Mental Disorders-IV criteria) and interviews. Thus, the SB prevalence rate of 17.7% in patients with insomnia reported by Maluly et al. [89] should be taken with caution. Future studies on the prevalence of SB in patients with insomnia should take the diagnosis of insomnia from physicians into account.

Regarding the mechanism, Saletu et al. [55] found that PSG-confirmed sleep bruxers showed no significant difference in sleep initiation but significantly deteriorated sleep maintenance compared with controls. Chronic insomnia, the symptoms of which include deteriorated sleep maintenance, was found to be associated with elevated physiological arousal [96]. At the same time, SB has also been found to be related to arousal [28,30–32]. Thus, we could speculate that SB is associated with insomnia via arousal. Again, the analysis in the study by Saletu et al. [55] was not based on a definite insomnia diagnosis by physicians.

To summarize, within the scope of the identified articles about SB and insomnia, we could only determine that there is a possible association between SB and the symptom of difficulty maintaining sleep. Future studies should investigate SB prevalence in insomniac patients diagnosed by physicians to avoid the subjective reporting bias caused by questionnaire usage. Moreover, when sleep physicians treat patients with major complaints related to sleep maintenance issues, it could be a good idea to ask further questions about symptoms related to SB. If the patients report obvious negative consequences related to SB (summarized in the introduction), the sleep physician should seek collaboration with dental practitioners to further evaluate the condition of the patients.

4.5. Sleep bruxism and REM behavior disorder, and Parkinson's disease

RBD is an abnormal condition consisting of REM sleep without atonia in conjunction with a history of recurrent nocturnal dream enactment behavior [97,98]. According to a review by Dauvilliers et al. [99], RBD has been considered as a potential precursor of later development of neurodegenerative disorders, such as PD. At the same time, Sixel-Doring et al. [100] reported that among sleep-disturbed patients with PD, 46% of them were identified with RBD.

One article [66] reported the prevalence of SB in adult patients with RBD. Abe et al. [66] reported, using PSG to confirm, an SB occurrence of 25% in patients with RBD, which is significantly higher than self-reported SB prevalence in the general population (12.8% [3]). At the same time, it should be noted that the diagnostic methods of SB in these two studies were different. While the PSG could offer objective data, Abe et al. [66] only enrolled 28 patients

with RBD. Manfredini et al. [3] acquired the prevalence rate by summarizing three large sample studies with a total of more than 2000 participants, albeit using questionnaires. Ylikoski et al. [67] reported, using only questionnaire, that SB occurrence in patients with PD was 4.7%, which is significantly lower than that in the general population (12.8% [3]). Even though the sample size was large, questionnaire was used to identify SB. When considering the mental and cognition status of patients with PD [101–103], their results may not be reliable. A strong point of both studies [66,67], however, is that their RBD and PD diagnoses were determined by medical specialists. The big difference in SB prevalence between in patients with either RBD or PD and in the general population suggests potential associations between SB and RBD as well as between SB and PD.

Although Abe et al. [66] mentioned that the RMMA index was higher in patients with RBD than in the healthy control group, no specific mechanism has been reported yet since associations do not provide direct support for SB causality. Thus, no specific mechanism has been found in these papers.

Since one of the main pathological findings in PD is the loss of dopaminergic neurons [104], studies that reported the association between SB and dopamine/dopaminergic neurons could shed some light on the association between SB and PD. Lobbezoo et al. [11] reported that the side imbalance of striatal dopaminergic receptors could be associated with SB. Lobbezoo et al. [105] also reported that short-term usage of levodopa, a dopamine precursor which is the most effective medication for the treatment of the motor symptoms of PD [106], has an attenuating effect on SB. However, a randomized crossover study by Cahlin et al. [107] found that short-term use of pramipexole, a dopamine agonist, does not affect SB. Also, a double-blind, crossover, placebo-controlled trial showed that short-term use of bromocriptine, a dopamine D2 receptor agonist, has no effect on the severity of SB [108]. However, long-term usage of levodopa could be a disruptor of the striatal dopaminergic balance, thus possibly be an SB-inducing factor. More studies are needed to further elucidate the possible roles of neurotransmitters and medications in the association between SB and PD [109].

One intriguing phenomenon suggested by Abe et al. [66] is that in patients with RBD, the RMMA burst index during REM sleep is significantly higher than in controls, while it is widely recognized that most RMMA episodes occur in sleep stages N1 and N2 in otherwise healthy individuals [110,111]. So, elevated RMMA activity during REM could serve as a red flag for sleep specialists for a possible presence of RBD. Sleep physicians have to keep in mind that idiopathic RBD, a clinical manifestation in the absence of PD of multiple system atrophy, is a condition with a high risk of neurodegenerative disease conversion at 12 years post diagnosis [99].

To summarize, studies on the association between SB and RBD, and on the association between SB and PD are limited. SB occurrence in patients with RBD may be higher than that in the general population, while SB prevalence in patients with PD may be lower than that in the general population. Further, increased RMMA activity during REM could be an important sign for sleep specialists to further screen their patients for RBD. Future studies should enroll more patients, utilize PSG to evaluate SB, and diagnose RBD and PD by medical specialists to offer objective information on the prevalence of SB in patients with either RBD or PD, and on the mechanism of the association between SB and RBD as well as the association between SB and PD.

4.6. Sleep bruxism and sleep-related epilepsy

Khachatryan et al. [37] suggested that the SB prevalence in adult patients with epilepsy was 23.7%, which was significantly higher than that in healthy controls (5.4%). Despite the relatively large number of participants (175 patients with epilepsy and 130 healthy controls), SB was diagnosed based on self-report, which could potentially overestimate the prevalence of SB [47]. However, since questionnaires were used for both groups, the use of this tool does not affect the positive correlation between SB and epilepsy. Giuliano et al. [112] published an article after the search date of this review, suggesting that SB is significantly more frequent in the epilepsy group than healthy controls using PSG with audio and video recordings in a relatively large sample (100 patients with epilepsy and 62 healthy controls). The significantly higher prevalence of SB in patients with epilepsy calls for more SB-related screening of patients suspected of epilepsy. Besides, it has been speculated in a case report [33] that epileptic discharge could present a direct inductive effect on SB. So, there is a possibility that the treatment of epilepsy could improve the SB condition for patients with epilepsy.

There is no mention of the mechanism that could explain the association between SB and sleep-related epilepsy. However, a detailed review on epilepsy and motor events during sleep [113] found that major episodes of epilepsy, especially nocturnal frontal lobe epilepsy between 10 and 60 s, were preceded by a prolonged cyclic alternating pattern sequence which reflects a condition of sustained arousal instability. As previously mentioned, arousal was found to be associated with SB. Consequently, it could be speculated that the association between SB and sleep-related epilepsy is mediated by arousal.

To summarize, SB is positively associated with epilepsy. However, the precise SB prevalence in patients with epilepsy is yet to be determined in a larger population using a method of high validity. Based on the evidence available, the association between SB and epilepsy could be explained by the common association with arousals. Sleep physicians, when treating patients with epilepsy, should be more aware of the possible negative consequences caused by SB and seek collaboration with dental practitioners in their treatment planning.

4.7. Sleep bruxism and other sleep-related disorders

The identified two articles on SB and other sleep-related disorders were questionnaire studies regarding SB and nightmare [71], as well as SB and sleep talking and sleepwalking [72]. More studies are needed to further understand the relationship between SB and nightmare, sleepwalking, and sleep talking. At the same time, sleep specialists need to be aware of these possible associations and seek collaborations with dentists when needed.

4.8. Strengths and limitations

One of this review's strengths is that our article search was performed in four different databases. Two reviewers independently did the title and abstract screening and full-text reading to minimize the potential personal bias. On the other hand, despite the effort to select articles of higher quality, those selected articles still have one or two section(s) of high risks of bias, including a limited number of participants and relatively low validity of the diagnostic tools. Thus, the conclusions that were reached based on these studies should be considered with caution.

Different methods were used to assess SB as well as the other identified disorders. Some studies used questionnaires or interviews, and some other studies used clinical inspection. Even among those articles that used PSG to assess SB, different scoring methods were used to score sleep as well as SB. Some articles used the RMMA index of 4 as the cutoff between SB positive and negative, while others used the RMMA index of 2. All the articles related to PD or RBD had their participants' diagnoses by physicians, while

none of the insomnia related articles involved the diagnosis by physicians. These differences in the assessment of all sleep-related disorders could potentially lead to different prevalence rates and associations. As each method has its own merits and demerits, standardized approaches for assessing SB and other sleep disorders with a global valuation of biopsychosocial and clinical data of a given individual are essential [114,115]. Future studies based on standardized and validated approaches to assess sleep-related disorders would provide more reliable evidence on the prevalence rates and associations between SB and other sleep-related disorders.

Despite these shortcomings, the review has identified and summarized all the sleep-related disorders in association with SB that are currently reported in the literature. Further, recommendations are made to medical specialists to raise awareness of SB as a potential indicator for these associated sleep-related disorders and to advocate the closer collaboration between medical specialists and dental practitioners.

Even though there are a lot of studies supporting the association between SB and sleep arousal, few studies, if any, could prove the causal relationship. Carra et al. [32], using a cyclic alternating pattern, which is another marker of sleep instability [116] and part of the sleep microstructure, reported that cyclic alternating pattern phase A3 is a permissive window rather than a generator of RMMA/SB activity. Thus, SB generation could be influenced by other factors yet to be identified.

5. Conclusion

The systematic review identified sleep-related disorders that are possibly associated with SB, including OSA, RLS/PLMS, sleep-related GERD, insomnia, PD, RBD, and sleep-related epilepsy. Within the main limitation of this review (ie, large methodological differences between the included studies in the assessment of SB and of other sleep disorders), the prevalence of SB in patients with OSA, RLS/PLMS, sleep-related GERD, RBD, and sleep-related epilepsy is higher than that in the general population, which sheds more light on the importance of routine SB screening in patients with aforementioned sleep-related disorders. Even though the specific mechanisms behind the associations between SB and other sleep-related disorders have not been identified yet, considering all the available evidence, sleep arousals could be a common factor with which all the identified disorders are associated, except RBD and PD.

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Conflict of interest

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The ICMJE Uniform Disclosure Form for Potential Conflicts of Interest associated with this article can be viewed by clicking on the following link: https://doi.org/10.1016/j.sleep.2021.11.008.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.sleep.2021.11.008.

References

[1] Lobbezoo F, Ahlberg J, Glaros AG, et al. Bruxism defined and graded: an international consensus. J Oral Rehabil 2013;40:2–4. https://doi.org/10.1111/joor.12011.
[2] Lobbezoo F, Ahlberg J, Raphael KG, et al. International consensus on the assessment of bruxism: report of a work in progress. J Oral Rehabil 2018;45:837–44. https://doi.org/10.1111/joor.12663.
[3] Manfredini D, Winocur E, Guarda-Nardini L, et al. Epidemiology of bruxism in adults: a systematic review of the literature. J Orofac Pain 2013;27:99–110. https://doi.org/10.11607/jop.921.
[4] Lobbezoo F, Naeije M. Bruxism is mainly regulated centrally, not peripherally. J Oral Rehabil 2001;28:1085–91. https://doi.org/10.1046/j.1365-2842.2001.00839.x.
[5] Lobbezoo F, Van Der Zaag J, Naeije M. Bruxism: its multiple causes and its effects on dental implants - an updated review. J Oral Rehabil 2006;33:293–300. https://doi.org/10.1111/j.1365-2842.2006.01609.x.
[6] Rintakoski K, Hublin C, Lobbezoo F, et al. Genetic factors account for half of the phenotypic variance in liability to sleep-related bruxism in young adults: a nationwide Finnish twin cohort study. Twin Res Hum Genet 2012;15:714–9. https://doi.org/10.1017/thg.2012.54.
[7] Abe Y, Suganuma T, Ishii M, et al. Association of genetic, psychological and behavioral factors with sleep bruxism in a Japanese population. J Sleep Res 2012;21:289–96. https://doi.org/10.1111/j.1365-2869.2011.00961.x.
[8] Oporto GH, Bornhardt T, Iturriaga V, et al. Single nucleotide polymorphisms in genes of dopaminergic pathways are associated with bruxism. Clin Oral Invest 2018;22:331–7. https://doi.org/10.1007/s00784-017-2117-z.
[9] Ahlberg J, Piirtola M, Lobbezoo F, et al. Correlates and genetics of self-reported sleep and awake bruxism in a nationwide twin cohort. J Oral Rehabil 2020;47:1110–9. https://doi.org/10.1111/joor.13042.
[10] Lobbezoo F, Visscher CM, Koutris M, et al. Bruxism in dentists' families. J Oral Rehabil 2018;45:657–8. https://doi.org/10.1111/joor.12648.
[11] Lobbezoo F, Soucy JP, Montplaisir JY, et al. Striatal D2 receptor binding in sleep bruxism: a controlled study with iodine-123-iodobenzamide and single-photon-emission computed tomography. J Dent Res 1996;75:1804–10. https://doi.org/10.1177/00220345960750101401.
[12] Lobbezoo F, Soucy JP, Hartman NG, et al. Effects of the D2 receptor agonist bromocriptine on sleep bruxism: report of two single-patient clinical trials. J Dent Res 1997;76:1610–4. https://doi.org/10.1177/00220345970760091401.
[13] Ahlberg J, Lobbezoo F, Ahlberg K, et al. Self-reported bruxism mirrors anxiety and stress in adults. Med Oral Patol Oral Cir Bucal 2013;18:e7–11. https://doi.org/10.4317/medoral.18232.
[14] Manfredini D, Lobbezoo F. Role of psychosocial factors in the etiology of bruxism. J Orofac Pain 2009;23:153–66.
[15] Lavigne GJ, Lobbezoo F, Rompré PH, et al. Cigarette smoking as a risk factor or an exacerbating factor for restless legs syndrome and sleep bruxism. Sleep 1997;20:290–3. https://doi.org/10.1093/sleep/20.4.290.
[16] Bertazzo-Silveira E, Kruger CM, Porto De Toledo I, et al. Association between sleep bruxism and alcohol, caffeine, tobacco, and drug abuse: a systematic review. J Am Dent Assoc 2016;147:859–66. https://doi.org/10.1016/j.adaj.2016.06.014. e4.
[17] Manfredini D, Lobbezoo F. Relationship between bruxism and temporomandibular disorders: a systematic review of literature from 1998 to 2008. Oral Surgery. Oral Med Oral Pathol Oral Radiol Endodontology 2010;109:e26–50. https://doi.org/10.1016/j.tripleo.2010.02.013.
[18] Lobbezoo F, Lavigne GJ. Do bruxism and temporomandibular disorders have a cause-and-effect relationship? J Orofac Pain 1997;11:15–23.
[19] Svensson P, Arima T, Lavigne G, et al. Principles and practice of sleep medicine. Princ. Pract. Sleep med. 6th ed. Philadelphia, PA: Elsevier; 2017. p. 1423–6. https://doi.org/10.1016/c2012-0-03543-0.
[20] Manfredini D, Poggio CE, Lobbezoo F. Is bruxism a risk factor for dental implants? A systematic review of the literature. Clin Implant Dent Relat Res 2014;16:460–9. https://doi.org/10.1111/cid.12015.
[21] Lavigne GJ, Kato T, Kolta A, et al. Neurobiologicalmechanisms involved in sleep bruxism. Crit Rev Oral Biol Med 2003;14:30–46. https://doi.org/10.1177/154411130301400104.
[22] Manfredini D, Guarda-Nardini L, Marchese-Ragona R, et al. Theories on possible temporal relationships between sleep bruxism and obstructive

sleep apnea events. An expert opinion. Sleep Breath 2015;19:1459–65. https://doi.org/10.1007/s11325-015-1163-5.
[23] Ohmure H, Oikawa K, Kanematsu K, et al. Influence of experimental esophageal acidification on sleep bruxism: a randomized trial. J Dent Res 2011;90:665–71. https://doi.org/10.1177/0022034510393516.
[24] Lobbezoo F, Aarab G, Wetselaar P, et al. A new definition of dental sleep medicine. J Oral Rehabil 2016;43:786–90. https://doi.org/10.1111/joor.12421.
[25] Lobbezoo F. Chewing on bruxism. Diagnosis, imaging, epidemiology and aetiology. Ned Tijdschr Tandheelkd 2017;124:309–16. https://doi.org/10.5177/ntvt.2017.06.16194.
[26] Manfredini D, Ahlberg J, Winocur E, et al. Management of sleep bruxism in adults: a qualitative systematic literature review. J Oral Rehabil 2015;42:862–74. https://doi.org/10.1111/joor.12322.
[27] Lobbezoo F, van der Zaag J, Van Selms MKA, et al. Principles for the management of bruxism. J Oral Rehabil 2008;35:509–23. https://doi.org/10.1111/j.1365-2842.2008.01853.x.
[28] Lavigne GJ, Huynh N, Kato T, et al. Genesis of sleep bruxism: motor and autonomic-cardiac interactions. Arch Oral Biol 2007;52:381–4. https://doi.org/10.1016/j.archoralbio.2006.11.017.
[29] Carra MC, Huynh N, Lavigne G. Sleep bruxism: a comprehensive overview for the dental clinician interested in sleep medicine. Dent Clin 2012;56:387–413. https://doi.org/10.1016/j.cden.2012.01.003.
[30] Lavigne GJ, Khoury S, Abe S, et al. Bruxism physiology and pathology: an overview for clinicians. J Oral Rehabil 2008;35:476–94. https://doi.org/10.1111/j.1365-2842.2008.01881.x.
[31] Huynh N, Kato T, Rompré PH, et al. Sleep bruxism is associated to micro-arousals and an increase in cardiac sympathetic activity. J Sleep Res 2006;15:339–46. https://doi.org/10.1111/j.1365-2869.2006.00536.x.
[32] Carra MC, Rompré PH, Kato T, et al. Sleep bruxism and sleep arousal: an experimental challenge to assess the role of cyclic alternating pattern. J Oral Rehabil 2011;38:635–42. https://doi.org/10.1111/j.1365-2842.2011.02203.x.
[33] Meletti S, Cantalupo G, Volpi L, et al. Rhythmic teeth grinding induced by temporal lobe seizures. Neurology 2004;62:2306–9. https://doi.org/10.1212/WNL.62.12.2306.
[34] van der Zaag J, Naeije M, Wicks DJ, et al. Time-linked concurrence of sleep bruxism, periodic limb movements, and EEG arousals in sleep bruxers and healthy controls. Clin Oral Invest 2014;18:507–13. https://doi.org/10.1007/s00784-013-0994-3.
[35] Srivastava M, Srivastava T, Ahuja M, et al. Bruxism as disabling clinical feature of elderly Parkinson's disease. Int Psychogeriatr 2003;15:317–8. https://doi.org/10.1017/S1041610203009426.
[36] Saito M, Yamaguchi T, Mikami S, et al. Weak association between sleep bruxism and obstructive sleep apnea. A sleep laboratory study. Sleep Breath 2016;20:703–9. https://doi.org/10.1007/s11325-015-1284-x.
[37] Khachatryan SG, Ghahramanyan L, Tavadyan Z, et al. Sleep-related movement disorders in a population of patients with epilepsy: prevalence and impact of restless legs syndrome and sleep bruxism. J Clin Sleep Med 2020;16:409–14. https://doi.org/10.5664/JCSM.8218.
[38] Moher D, Liberati A, Tetzlaff J, et al. Preferred reporting items for systematic reviews and meta-analyses: the PRISMA statement. PLoS Med 2009;6:1685–91. https://doi.org/10.1371/journal.pmed.1000097.
[39] Kim SY, Park JE, Lee YJ, et al. Testing a tool for assessing the risk of bias for nonrandomized studies showed moderate reliability and promising validity. J Clin Epidemiol 2013;66:408–14. https://doi.org/10.1016/j.jclinepi.2012.09.016.
[40] Phillips BA, Okeson J, Paesani D, et al. Effect of sleep position on sleep apnea and parafunctional activity. Chest 1986;90:424–9. https://doi.org/10.1378/chest.90.3.424.
[41] Tan M, Yap A, Chua A, 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;33:269–77. https://doi.org/10.11607/ofph.2068.
[42] Martynowicz H, Gac P, Brzecka A, et al. The relationship between sleep bruxism and obstructive sleep apnea based on polysomnographic findings. J Clin Med 2019;8:1653. https://doi.org/10.3390/jcm8101653.
[43] de Holanda TA, Castagno CD, Barbon FJ, 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. https://doi.org/10.1016/j.archoralbio.2020.104685.
[44] Smardz J, Martynowicz H, Wojakowska A, 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;68:131–7. https://doi.org/10.1016/j.sleep.2019.08.025.
[45] Okeson JP, Phillips BA, Berry DT, et al. Nocturnal bruxing events in subjects with sleep-disordered breathing and control subjects. J Craniomandib Disord 1991;5:258–64.
[46] Sjöholm TT, Lowe AA, Miyamoto K, et al. Sleep bruxism in patients with sleep-disordered breathing. Arch Oral Biol 2000;45:889–96. https://doi.org/10.1016/S0003-9969(00)00044-3.
[47] Maluly M, Andersen ML, Dal-Fabbro C, et al. Polysomnographic study of the prevalence of sleep bruxism in a population sample. J Dent Res 2013;92:S97–103. https://doi.org/10.1177/0022034513484328.
[48] Hesselbacher S, Subramanian S, Rao S, et al. Self-reported sleep bruxism and nocturnal gastroesophageal reflux disease in patients with obstructive sleep apnea: relationship to gender and ethnicity. Open Respir Med J 2014;8:34–40. https://doi.org/10.2174/1874306401408010034.
[49] Saito M, Yamaguchi T, Mikami S, et al. Temporal association between sleep apnea-hypopnea and sleep bruxism events. J Sleep Res 2014;23:196–203. https://doi.org/10.1111/jsr.12099.
[50] Hosoya H, Kitaura H, Hashimoto T, et al. Relationship between sleep bruxism and sleep respiratory events in patients with obstructive sleep apnea syndrome. Sleep Breath 2014;18:837–44. https://doi.org/10.1007/s11325-014-0953-5.
[51] Winck M, Drummond M, Viana P, et al. Sleep bruxism associated with obstructive sleep apnoea syndrome - a pilot study using a new portable device. Rev Port Pneumol (English 2017;23:22–6. https://doi.org/10.1016/j.rppnen.2016.07.001.
[52] Tsujisaka A, Haraki S, Nonoue S, et al. The occurrence of respiratory events in young subjects with a frequent rhythmic masticatory muscle activity: a pilot study. J Prosthodont Res 2018;62:317–23. https://doi.org/10.1016/j.jpor.2017.12.004.
[53] Lavigne GJ, Montplaisir JY. Restless legs syndrome and sleep bruxism: prevalence and association among Canadians. Sleep 1994;17:739–43. https://doi.org/10.1093/sleep/17.8.739.
[54] Ahlberg K, Ahlberg J, Könönen M, et al. Reported bruxism and restless legs syndrome in media personnel with or without irregular shift work. Acta Odontol Scand 2005;63:94–8. https://doi.org/10.1080/00016350510019757.
[55] Saletu A, Parapatics S, Anderer P, et al. Controlled clinical, polysomnographic and psychometric studies on differences between sleep bruxers and controls and acute effects of clonazepam as compared with placebo. Eur Arch Psychiatr Clin Neurosci 2010;260:163–74. https://doi.org/10.1007/s00406-009-0034-0.
[56] Han K, Wang C, Zhong Z, et al. Characterisation of the relationships between rhythmic masticatory muscle activities and limb movements in patients with sleep bruxism. J Oral Rehabil 2019;46:399–408. https://doi.org/10.1111/joor.12760.
[57] Miki H, Minakuchi H, Miyagi M, et al. Association of masticatory muscle activity with sleep arousal and other concomitant movements during sleep. J Oral Rehabil 2020;47:281–8. https://doi.org/10.1111/joor.12913.
[58] Miyawaki S, Tanimoto Y, Araki Y, et al. Association between nocturnal bruxism and gastroesophageal reflux. Sleep 2003;26:888–92. https://doi.org/10.1093/sleep/26.7.888.
[59] Miyawaki S, Tanimoto Y, Araki Y, et al. Relationships among nocturnal jaw muscle activities, decreased esophageal pH, and sleep positions. Am J Orthod Dentofacial Orthop 2004;126:615–9. https://doi.org/10.1016/j.ajodo.2004.02.007.
[60] Mengatto CM, Dalberto CDS, Scheeren B, et al. Association between sleep bruxism and gastroesophageal reflux disease. J Prosthet Dent 2013;110:349–55. https://doi.org/10.1016/j.prosdent.2013.05.002.
[61] Li Y, Yu F, Niu L, et al. Association between bruxism and symptomatic gastroesophageal reflux disease: a case-control study. J Dent 2018;77:51–8. https://doi.org/10.1016/j.jdent.2018.07.005.
[62] Li Y, Yu F, Niu L, et al. Associations among bruxism, gastroesophageal reflux disease, and tooth wear. J Clin Med 2018;7:417. https://doi.org/10.3390/jcm7110417.
[63] Hachul De Campos H, Brandão L, D'Almeida V, et al. Sleep disturbances, oxidative stress and cardiovascular risk parameters in postmenopausal women complaining of insomnia. Climacteric 2006;9:312–9. https://doi.org/10.1080/13697130600871947.
[64] Ahlberg K, Jahkola A, Savolainen A, et al. Associations of reported bruxism with insomnia and insufficient sleep symptoms among media personnel with or without irregular shift work. Head Face Med 2008;4:1–6. https://doi.org/10.1186/1746-160X-4-4.
[65] Blanken TF, Benjamins JS, Borsboom D, et al. Insomnia disorder subtypes derived from life history and traits of affect and personality. The Lancet Psychiatry 2019;6:151–63. https://doi.org/10.1016/S2215-0366(18)30464-4.
[66] Abe S, Gagnon JF, Montplaisir JY, et al. Sleep bruxism and oromandibular myoclonus in rapid eye movement sleep behavior disorder: a preliminary report. Sleep Med 2013;14:1024–30. https://doi.org/10.1016/j.sleep.2013.04.021.
[67] Ylikoski A, Martikainen K, Partinen M. Parasomnias and isolated sleep symptoms in Parkinson's disease: a questionnaire study on 661 patients. J Neurol Sci 2014;346:204–8. https://doi.org/10.1016/j.jns.2014.08.025.
[68] Verhoeff MC, Lobbezoo F, Wetselaar P, et al. Parkinson's disease, temporomandibular disorders and bruxism: a pilot study. J Oral Rehabil 2018;45:854–63. https://doi.org/10.1111/joor.12697.
[69] Khatami R, Zutter D, Siegel A, et al. Sleep-wake habits and disorders in a series of 100 adult epilepsy patients-A prospective study. Seizure 2006;15:299–306. https://doi.org/10.1016/j.seizure.2006.02.018.
[70] Bisulli F, Vignatelli L, Naldi I, et al. Increased frequency of arousal parasomnias in families with nocturnal frontal lobe epilepsy: a common mechanism? Epilepsia 2010;51:1852–60. https://doi.org/10.1111/j.1528-1167.2010.02581.x.
[71] Serra-Negra JM, Lobbezoo F, Correa-Faria P, et al. Relationship of self-reported sleep bruxism and awake bruxism with chronotype profiles in Italian dental students. Cranio - J Craniomandib Pract 2019;37:147–52. https://doi.org/10.1080/08869634.2018.1431600.

[72] Hublin C, Kaprio J, Partinen M, et al. Parasomnias: Co-occurrence and genetics. Psychiatr Genet 2001;11:65–70. https://doi.org/10.1097/00041444-200106000-00002.
[73] Lobbezoo F, Aarab G. Increasing the visibility of dental sleep disorders. J Clin Sleep Med 2018;14:1827. https://doi.org/10.5664/jcsm.7424.
[74] Aarab G, Lobbezoo F. Dental sleep medicine redefined. Sleep Breath 2018;22:1233. https://doi.org/10.1007/s11325-018-1697-4.
[75] Wetselaar P, Vermaire EJH, Lobbezoo F, et al. The prevalence of awake bruxism and sleep bruxism in the Dutch adult population. J Oral Rehabil 2019;46:617–23. https://doi.org/10.1111/joor.12787.
[76] Aarab G, Arcache P, Lavigne GJ, et al. The effects of mandibular advancement appliance therapy on jaw-closing muscle activity during sleep in patients with obstructive sleep apnea: a 3-6 months follow-up. J Clin Sleep Med 2020;16:1545–53. https://doi.org/10.5664/jcsm.8612.
[77] Kato T, Rompré P, Montplaisir JY, et al. Sleep bruxism: an oromotor activity secondary to micro-arousal. J Dent Res 2001;80:1940–4. https://doi.org/10.1177/00220345010800101501.
[78] Macaluso GM, Guerra P, Di Giovanni G, et al. Sleep bruxism is a disorder related to periodic arousals during sleep. J Dent Res 1998;77:565–73. https://doi.org/10.1177/00220345980770040901.
[79] Senaratna CV, Perret JL, Lodge CJ, et al. Prevalence of obstructive sleep apnea in the general population: a systematic review. Sleep Med Rev 2017;34:70–81. https://doi.org/10.1016/j.smrv.2016.07.002.
[80] Landry M-L, Rompré PH, Manzini C, et al. Reduction of sleep bruxism using a mandibular advancement device: an experimental controlled study. Int J Prosthodont (IJP) 2006;19:549–56.
[81] Martinot JB, Borel JC, Le-Dong NN, et al. Bruxism relieved under CPAP treatment in a patient with OSA syndrome. Chest 2020;157:e59–62. https://doi.org/10.1016/j.chest.2019.07.032.
[82] Oksenberg A, Arons E. Sleep bruxism related to obstructive sleep apnea: the effect of continuous positive airway pressure. Sleep Med 2002;3:513–5. https://doi.org/10.1016/S1389-9457(02)00130-2.
[83] Jokubauskas L, Baltrušaitytė A, Pileičikienė G. Oral appliances for managing sleep bruxism in adults: a systematic review from 2007 to 2017. J Oral Rehabil 2018;45:81–95. https://doi.org/10.1111/joor.12558.
[84] Kalevo L, Miettinen T, Leino A, et al. Effect of sweating on electrode-skin contact impedances and artifacts in EEG recordings with various screen-printed Ag/agcl electrodes. IEEE Access 2020;8:50934–43. https://doi.org/10.1109/ACCESS.2020.2977172.
[85] De Luca CJ, Donald Gilmore L, Kuznetsov M, et al. Filtering the surface EMG signal: movement artifact and baseline noise contamination. J Biomech 2010;43:1573–9. https://doi.org/10.1016/j.jbiomech.2010.01.027.
[86] American Academy of Sleep Medicine. International classification of sleep disorders. 3rd ed. Darien, IL: American Academy of Sleep Medicine; 2014.
[87] Orr WC, Robinson MG, Johnson LF. The effect of esophageal acid volume on arousals from sleep and acid clearance. Chest 1991;99:351–4. https://doi.org/10.1378/chest.99.2.351.
[88] Ju G, Yoon IY, Lee SD, et al. Relationships between sleep disturbances and gastroesophageal reflux disease in Asian sleep clinic referrals. J Psychosom Res 2013;75:551–5. https://doi.org/10.1016/j.jpsychores.2013.10.004.
[89] Maluly M, Dal Fabbro C, Andersen ML, et al. Sleep bruxism and its associations with insomnia and OSA in the general population of Sao Paulo. Sleep Med 2020;75:141–8. https://doi.org/10.1016/j.sleep.2020.06.016.
[90] Cho YW, Kim KT, Moon H, et al. Comorbid insomnia with obstructive sleep apnea: clinical characteristics and risk factors. J Clin Sleep Med 2018;14:409–17. https://doi.org/10.5664/jcsm.6988.
[91] Hein M, Lanquart J-P, Loas G, et al. Prevalence and risk factors of moderate to severe obstructive sleep apnea syndrome in insomnia sufferers: a study on 1311 subjects. Respir Res 2017;18:135. https://doi.org/10.1186/s12931-017-0616-8.
[92] Chaiard J, Weaver TE. Update on research and practices in major sleep disorders: Part II—insomnia, willis-ekbom disease (restless leg syndrome), and narcolepsy. J Nurs Scholarsh 2019;51:624–33. https://doi.org/10.1111/jnu.12515.
[93] Townsend D, Kazaglis L, Savik K, et al. A brief tool to differentiate factors contributing to insomnia complaints. Health Psychol 2017;36:291–7. https://doi.org/10.1037/hea0000442.
[94] Jha LK, Fass R, Gadam R, et al. The effect of ramelteon on heartburn symptoms of patients with gastroesophageal reflux disease and chronic insomnia a pilot study. J Clin Gastroenterol 2016;50:e19–24. https://doi.org/10.1097/MCG.0000000000000322.
[95] Ballou S, Alhassan E, Hon E, et al. Sleep disturbances are commonly reported among patients presenting to a gastroenterology clinic. Dig Dis Sci 2018;63:2983–91. https://doi.org/10.1007/s10620-018-5237-7.
[96] Bonnet MH, Arand DL. Hyperarousal and insomnia: state of the science. Sleep Med Rev 2010;14:9–15. https://doi.org/10.1016/j.smrv.2009.05.002.
[97] Barone DA, Ebben MR, Samie A, et al. Autonomic dysfunction in isolated rapid eye movement sleep without atonia. Clin Neurophysiol 2015;126:731–5. https://doi.org/10.1016/j.clinph.2014.07.015.
[98] Schenck CH, Bundlie SR, Ettinger MG, et al. Chronic behavioral disorders of human REM sleep: a new category of parasomnia. Sleep 1986;9:293–308. https://doi.org/10.1093/sleep/9.2.293.
[99] Dauvilliers Y, Schenck CH, Postuma RB, et al. REM sleep behaviour disorder. Nat Rev Dis Prim 2018;4:19. https://doi.org/10.1038/s41572-018-0016-5.
[100] Sixel-Döring F, Trautmann E, Mollenhauer B, et al. Associated factors for REM sleep behavior disorder in Parkinson disease. Neurology 2011;77:1048–54. https://doi.org/10.1212/WNL.0b013e31822e560e.
[101] Lewis SJG, Dove A, Robbins TW, et al. Cognitive impairments in early Parkinson's disease are accompanied by reductions in activity in frontostriatal neural circuitry. J Neurosci 2003;23:6351–6. https://doi.org/10.1523/jneurosci.23-15-06351.2003.
[102] Arie L, Herman T, Shema-Shiratzky S, et al. Do cognition and other non-motor symptoms decline similarly among patients with Parkinson's disease motor subtypes? Findings from a 5-year prospective study. J Neurol 2017;264:2149–57. https://doi.org/10.1007/s00415-017-8605-x.
[103] Jankovic J. Parkinson's disease: clinical features and diagnosis. J Neurol Neurosurg Psychiatry 2008;79:368–76. https://doi.org/10.1136/jnnp.2007.131045.
[104] Fröhlich F. Parkinson's disease. Netw. Neurosci. Elsevier; 2016. p. 291–6. https://doi.org/10.1016/B978-0-12-801560-5.00023-9.
[105] Lobbezoo F, Lavigne GJ, Tanguay R, et al. The effect of the catecholamine precursor L-dopa on sleep bruxism: a controlled clinical trial. Mov Disord 1997;12:73–8. https://doi.org/10.1002/mds.870120113.
[106] Aradi SD, Hauser RA. Medical management and prevention of motor complications in Parkinson's disease. Neurotherapeutics 2020;17:1339–65. https://doi.org/10.1007/s13311-020-00889-4.
[107] Cahlin BJ, Hedner J, Dahlström L. A randomised, open-label, crossover study of the dopamine agonist, pramipexole, in patients with sleep bruxism. J Sleep Res 2017;26:64–72. https://doi.org/10.1111/jsr.12440.
[108] Lavigne GJ, Soucy J-P, Lobbezoo F, et al. Double-blind, crossover, placebo-controlled trial of bromocriptine in patients with sleep bruxism. Clin Neuropharmacol 2001;24:145–9. https://doi.org/10.1097/00002826-200105000-00005.
[109] de Baat C, Verhoeff M, Ahlberg J, et al. Medications and addictive substances potentially inducing or attenuating sleep bruxism and/or awake bruxism. J Oral Rehabil 2021;48:343–54. https://doi.org/10.1111/joor.13061. joor.13061.
[110] Kato T, Thie NM, Montplaisir JY, et al. Bruxism and orofacial movements during sleep. Dent Clin 2001;45:657–84.
[111] Carra MC, Huynh N, Lavigne GJ. Diagnostic accuracy of sleep bruxism scoring in absence of audio-video recording: a pilot study. Sleep Breath 2015;19:183–90. https://doi.org/10.1007/s11325-014-0986-9.
[112] Giuliano L, Mainieri G, Cicero CE, et al. Parasomnias, sleep-related movement disorders and physiological sleep variants in focal epilepsy: a polysomnographic study. Seizure 2020;81:84–90. https://doi.org/10.1016/j.seizure.2020.07.026.
[113] Parrino L, Halasz P, Tassinari CA, et al. CAP, epilepsy and motor events during sleep: the unifying role of arousal. Sleep Med Rev 2006;10:267–85. https://doi.org/10.1016/j.smrv.2005.12.004.
[114] Lavigne G, Kato T, Herrero Babiloni A, et al. Research routes on improved sleep bruxism metrics: toward a standardised approach. J Sleep Res 2021;30:1–12. https://doi.org/10.1111/jsr.13320.
[115] Pevernagie DA, Gnidovec-Strazisar B, Grote L, et al. On the rise and fall of the apnea–hypopnea index: a historical review and critical appraisal. J Sleep Res 2020;29:13066. https://doi.org/10.1111/jsr.13066.
[116] Parrino L, Ferri R, Bruni O, et al. Cyclic alternating pattern (CAP): the marker of sleep instability. Sleep Med Rev 2012;16:27–45. https://doi.org/10.1016/j.smrv.2011.02.003.
[117] AASM. The AASM manual for the scoring of sleep and associated events: rules, terminology and technical specifications. 1st ed. Westchester (IL): American Academy of Sleep Medicine; 2007.
[118] Lavigne GJ, Rompré PH, Montplaisir JY. Sleep bruxism: validity of clinical research diagnostic criteria in a controlled polysomnographic study. J Dent Res 1996;75:546–52. https://doi.org/10.1177/00220345960750010601.
[119] American Academy of Sleep Medicine. International classification of sleep disorders Westchester, IL. 2nd ed. 2005.
[120] Rompré PH, Daigle-Landry D, Guitara F, et al. Identification of a sleep bruxism subgroup with a higher risk of pain. J Dent Res 2007;86:837–42. https://doi.org/10.1177/154405910708600906.
[121] Berry R, Brooks R, Garnaldo C. The AASM manual for the scoring of sleep and associated events: rules, terminology and technical specifications. 2.3. IL: Darien; 2016.
[122] Carra MC, Huynh N, Fleury B, et al. Overview on sleep bruxism for sleep medicine clinicians. Sleep Med Clin 2015;10:375–84. https://doi.org/10.1016/j.jsmc.2015.05.005.
[123] American Academy of Sleep Medicine. International classification of sleep disorders, revised: diagnostic and coding manual. Chicago, IL. 2001.
[124] Kahrilas PJ, Quigley EM. Clinical esophageal pH recording: a technical review for practice guideline development. Gastroenterology 1996;110:1982–96. https://doi.org/10.1053/gast.1996.1101982.
[125] Vakil N, van Zanten SV, Kahrilas P, et al. The montreal definition and classification of gastroesophageal reflux disease: a global evidence-based consensus. Am J Gastroenterol 2006;101:1900–20. https://doi.org/10.1111/j.1572-0241.2006.00630.x.
[126] World Health Organization. International statistical classification of diseases and related Health Problems. 1992.
[127] Fisher RS, Acevedo C, Arzimanoglou A, et al. ILAE Official Report: a practical clinical definition of epilepsy. Epilepsia 2014;55:475–82. https://doi.org/10.1111/epi.12550.
[128] Serra-Negra JM, Scarpelli AC, Tirsa-Costa D, et al. Sleep bruxism, awake bruxism and sleep quality among Brazilian dental students: a cross-sectional study. Braz Dent J 2014;25:241–7. https://doi.org/10.1590/0103-6440201302429.