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Patrícia Pauletto

Associação entre bruxismo do sono e apneia obstrutiva do sono

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Orientadora: Prof.^a Dr.^a Graziela De Luca Canto

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Patrícia Pauletto

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O presente trabalho em nível de Doutorado foi avaliado e aprovado por banca examinadora composta pelos seguintes membros:

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Certificamos que esta é a **versão original e final** do trabalho de conclusão que foi julgado adequado para obtenção do título de Doutora em Odontologia.

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Orientadora

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Dedico esta tese aos meus pais Alcírio Pauletto e Donatila Trevisan Pauletto (*in memoriam*). A vocês, tudo que tenho, todo meu esforço, dedicação e amor.

“Se você for corajoso o suficiente para deixar para trás tudo que é familiar e reconfortante (que pode ser qualquer coisa, desde sua casa até seus ressentimentos antigos e amargos) e partir em uma jornada em busca da verdade (seja externamente ou internamente), e se você está realmente disposto a considerar tudo o que lhe acontece nessa jornada como uma pista, e se você aceita todos que encontra pelo caminho como professor, e se você está preparado – acima de tudo – para enfrentar (e esquecer) algumas realidades muito difíceis sobre você... então a verdade não será negada a você. Ou então eu vim a acreditar.”

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RESUMO

O bruxismo do sono é uma atividade da musculatura mastigatória durante o sono. A apneia obstrutiva do sono é um distúrbio respiratório do sono caracterizado pela cessação completa ou parcial do fluxo de ar. Alguns estudos têm sugerido uma associação entre as duas condições, no entanto, ainda há inúmeras inconsistências na literatura sobre o tema. Assim, os objetivos desta tese foram: 1) Mapear e analisar criticamente a literatura, encontrar as lacunas de conhecimento e recomendar pesquisas futuras sobre bruxismo do sono e apneia obstrutiva em adultos e crianças; 2) Avaliar os padrões de ocorrência entre bruxismo do sono e apneia obstrutiva. Foram realizados dois estudos: uma revisão de escopo e um estudo observacional descritivo. Na revisão de escopo realizou-se uma busca em seis bases de dados, bem como literatura cinzenta, os estudos foram selecionados por dois revisores independentes e uma análise narrativa dos resultados foi conduzida. O estudo observacional descritivo foi realizado em pacientes com suspeita de apneia encaminhados para o exame de polissonografia. A amostra inicial foi constituída por 95 pacientes, no entanto 33 pacientes foram excluídos porque não apresentaram boa qualidade da polissonografia para análise do bruxismo do sono. Dois padrões temporais foram analisados na polissonografia dentro de uma janela de tempo de dois minutos: (T1) = intervalo entre o término dos eventos de apneia-hipopneia e o início do episódio de bruxismo, e (T2) = intervalo entre o término do episódio de bruxismo e o início dos eventos de apneia-hipopneia. Cinco padrões de ocorrência de bruxismo do sono e apneia também foram analisados em uma janela de tempo de 2 minutos, tendo como referência um episódio de bruxismo: (1) Episódio de bruxismo isolado (sem eventos de apneia antes ou depois); (2) Evento de apneia-hipopneia ocorrido antes do episódio de bruxismo; (3) Evento de apneia-hipopneia ocorrido após o episódio de bruxismo; (4) Evento de apneia-hipopneia ocorridos antes e depois do episódio de bruxismo; e (5) Evento de apneia-hipopneia acontecendo concomitante ao episódio de bruxismo. A diferença de médias nos tempos de ocorrência de bruxismo e apneia obstrutiva foi verificada pelo teste t e a diferença entre os padrões de ocorrência das condições foi analisada pelo teste ANOVA de medidas repetidas. Foi adotado um nível de significância de 5%. A revisão de escopo não encontrou associação entre bruxismo e apneia em adultos. Em crianças essa associação parece estar presente, no entanto os estudos apresentaram variabilidade nos métodos de detecção das condições. No estudo observacional descritivo, dos 62 participantes, 61 (98,4%) apresentaram apneia obstrutiva. A prevalência de bruxismo do sono foi de 74,2%. Não houve diferença estatística entre a ocorrência dos episódios de bruxismo do sono após ($15,36 \pm 12,52$ segundos) ou antes dos eventos de apneia-hipopneia ($13,92 \pm 11,04$), $p=0,51$. A maioria dos episódios de bruxismo do sono aconteceram de forma isolada, ou seja, sem um evento de apneia prévio ou posterior. A revisão de escopo não encontrou associação entre bruxismo do sono e apneia obstrutiva em adultos. Em crianças essa associação parece estar presente, no entanto mais estudos são necessários. No estudo observacional, a prevalência de bruxismo do sono na população estudada foi alta. No entanto, não foi comprovado um padrão de ocorrência entre as duas condições. A maioria dos episódios de bruxismo do sono (47,7%) aconteceram de forma isolada no período de observação.

Palavras-chave: Bruxismo do sono; Apneia Obstrutiva do Sono; Revisão de Escopo; Associação; Prevalência.

ABSTRACT

Sleep bruxism is an activity of the masticatory muscles during sleep. Obstructive sleep apnea is a sleep-disordered breathing characterized by complete or partial cessation of airflow. Some studies have suggested an association between the two conditions, however, there are still numerous inconsistencies in the literature on the subject. Thus, the objectives of this thesis were: 1) To map and critically analyze the literature, find knowledge gaps and recommend future research on sleep bruxism and obstructive apnea in adults and children; 2) Evaluate the patterns of occurrence between sleep bruxism and obstructive apnea. Two studies were performed: a scoping review and a descriptive observational study. In the scoping review, a search was carried out in six databases, as well as gray literature, the studies were selected by two independent reviewers and a narrative analysis of the results was conducted. The descriptive observational study was carried out in patients with suspected apnea referred for polysomnography. The initial sample consisted of 95 patients, however 33 patients were excluded because they did not have good quality polysomnography for analysis of sleep bruxism. Two temporal patterns were analyzed on polysomnography within a two-minute time window: (T1) = interval between the end of the apnea-hypopnea events and the beginning of the bruxism episode, and (T2) = interval between the end of the episode of bruxism and the onset of apnea-hypopnea events. Five patterns of occurrence of sleep bruxism and apnea were also analyzed in a 2-minute time window, with reference to one bruxism episode: (1) Isolated bruxism episode (no apnea events before or after); (2) Apnea-hypopnea event occurred before the bruxism episode; (3) Apnea-hypopnea event occurring after the bruxism episode; (4) Apnea-hypopnea event occurring before and after the bruxism episode; and (5) Apnea-hypopnea event occurring concomitantly with the bruxism episode. The difference in means in the times of occurrence of bruxism and obstructive apnea was verified by the t test and the difference between the patterns of occurrence of the conditions was analyzed by the ANOVA test of repeated measures. A significance level of 5% was adopted. The scoping review found no association between bruxism and apnea in adults. In children this association seems to be present, however the studies showed variability in the methods of detecting the conditions. In the descriptive observational study, of the 62 participants, 61 (98.4%) had obstructive apnea. The prevalence of sleep bruxism was 74.2%. There was no statistical difference between the occurrence of sleep bruxism episodes after (15.36 ± 12.52 seconds) or before the apnea-hypopnea events (13.92 ± 11.04), $p=0.51$. Most episodes of sleep bruxism occurred in isolation, that is, without a previous or subsequent apnea event. The scoping review found no association between sleep bruxism and obstructive apnea in adults. In children this association seems to be present, however further studies are needed. In the observational study, the prevalence of sleep bruxism in the studied population was high. However, a pattern of occurrence between the two conditions has not been proven. Most episodes of sleep bruxism (47.7%) occurred in isolation during the observation period.

Keywords: Sleep bruxism; Obstructive Sleep Apnea; Scoping Review; Association; Prevalence.

LISTA DE ABREVIATURAS E SIGLAS

AASM	American Academy of Sleep Medicine
AAMS	Academia Americana de Medicina do Sono
AHI	Apnea-Hypopnea Index
AHE	Apnea-Hypopnea Event
AI	Arousal Index
AOS	Apneia Obstrutiva do Sono
BMI	Body mass index
BS	Bruxismo do Sono
CI	Confidence Interval
EMG	Electromyography
EEG	Eletroencefalogramms
EEO	Eletrooculography
minOSAT	Minimum oxygen saturation
NREM	Non- rapid eye moviment
ODI	Oxygen desaturation index
OSA	Obstructive Sleep Apnea
PSG	Polissonografia / Polysomnography
REM	Rapid eye moviment
RS	Revisão Sistemática
SB	Sleep Bruxism
SE	Sleep Efficiency
SR	Systematic Review

LISTA DE SÍMBOLOS

® Marca Registrada

SUMÁRIO

1	APRESENTAÇÃO.....	15
2	INTRODUÇÃO E REVISÃO DA LITERATURA.....	16
3	OBJETIVOS.....	22
3.1	OBJETIVO GERAL.....	22
3.2	OBJETIVOS ESPECÍFICOS.....	22
4	ARTIGO 1.....	23
5	ARTIGO 2.....	67
6	CONSIDERAÇÕES FINAIS.....	88
	REFERÊNCIAS.....	90
	APÊNDICE A.....	93
	APÊNDICE B.....	101
	APÊNDICE C.....	104
	APÊNDICE D.....	105
	APÊNDICE E.....	106
	APÊNDICE F.....	109
	APÊNDICE G.....	110
	APÊNDICE H.....	112
	ANEXO A.....	113
	ANEXO B.....	121
	ANEXO C.....	125

1 APRESENTAÇÃO

Esta tese é composta por duas pesquisas desenvolvidas durante o Doutorado. A primeira trata-se de um artigo de revisão de escopo registrado na plataforma *Open Science Framework* (ANEXO A). O segundo artigo traz resultados parciais de um macroprojeto intitulado “Associação entre bruxismo do sono e apneia obstrutiva do sono”. O referido macroprojeto passou por avaliação pelo Comitê de Ética em Pesquisa com Seres Humanos (CEPSH) da Universidade Federal de Santa Catarina (UFSC) e foi aprovado sob parecer número 84783518.6.0000.0121 (ANEXO B).

A tese é composta dos seguintes capítulos: (1) Apresentação da tese, (2) Introdução e revisão de literatura, (3) Objetivos (4) Artigo 1, (5) Artigo 2, (6) Considerações Finais, Apêndices (A – H) e Anexos (A – C). Os seguintes artigos são apresentados:

- **Artigo 1.** Revisão de escopo, já publicada no *Journal SLEEP*.

Referência: Pauletto P, Polmann H, Conti Réus J, Massignan C, de Souza BD, Gozal D, Lavigne G, Flores-Mir C, De Luca Canto G. Sleep bruxism and obstructive sleep apnea: association, causality or spurious finding? A scoping review. *Sleep*. 2022 Apr 20 (ANEXO C).

- **Artigo 2.** Estudo observacional descritivo, formatado nas normas do *Journal SLEEP*, para ser submetido após a defesa.

2 INTRODUÇÃO E REVISÃO DA LITERATURA

O bruxismo do sono (BS), de acordo com um consenso internacional, pode ser considerado uma atividade da musculatura mastigatória durante o sono, caracterizada como rítmica (fásica) ou não-rítmica (tônica). Atualmente, não é considerado uma desordem do movimento ou um distúrbio do sono (LOBBEZOO *et al.*, 2018).

A prevalência de BS encontrada em uma revisão sistemática (RS) (MANFREDINI *et al.*, 2013) foi de 12,8% (\pm 3,1%) na população adulta, sem diferenças entre os sexos. No entanto, em uma amostra populacional de indivíduos brasileiros entre 20 e 80 anos de idade, a prevalência de BS confirmada por polissonografia (PSG) foi de 7,4% (MALULY *et al.*, 2013).

O bruxismo pode ser classificado, com base na forma de sua detecção (LOBBEZOO *et al.*, 2018). Quando o bruxismo é detectado somente com o autorrelato do paciente, chama-se “bruxismo possível”. Nos casos em que a detecção é realizada com base em avaliação clínica (com ou sem autorrelato) é chamado “bruxismo provável”. Finalmente, há o “bruxismo definitivo”, quando a detecção acontece por meio de eletromiografia ou PSG somente. Segundo a literatura, a PSG ainda é considerada o método padrão-referência para alcançar uma detecção precisa de BS (CASETT *et al.*, 2017; KHOURY *et al.*, 2016; MALULY *et al.*, 2013).

Estudos mostram que uma atividade aumentada dos músculos mastigatórios podem trazer consequências à saúde como dor muscular, distúrbios temporomandibulares, dores de cabeça matinais, mialgia facial, desgaste extremo dos dentes, fraturas dentárias, complicações protéticas e complicações em implantes dentários (HOZ AIZPURUA *et al.*, 2011; JOHANSSON; OMAR; CARLSSON, 2011; MELO *et al.*, 2019; PALINKAS *et al.*, 2016).

A literatura também evidencia alguns fatores de risco associados ao BS, como o refluxo gastroesofágico, polimorfismos genéticos (CASTROFLORIO *et al.*, 2017), consumo de álcool, cafeína, tabaco e drogas (BERTAZZO-SILVEIRA *et al.*, 2016).

Autores também têm mencionado a hipótese de que o BS poderia atuar como um fator de proteção em pacientes com apneia obstrutiva (LOBBEZOO *et al.*, 2018; MANFREDINI *et al.*, 2015). De acordo com esta hipótese, o BS poderia funcionar como reflexo motor autonômico em resposta a uma excitação noturna (SJOHOLM *et al.*, 2000).

A apneia obstrutiva do sono (AOS) é um distúrbio respiratório do sono e pode ser classificada como leve, moderada ou grave. O diagnóstico clínico de apneia do sono é estabelecido quando a apneia (cessação completa do fluxo de ar) e a hipopneia (reduções significativas no fluxo de ar além de um ponto de corte específico, por exemplo, 50%) estão

presentes em conjunto com a sonolência diurna excessiva (ACADEMIA AMERICANA DE MEDICINA DO SONO, 2008). A prevalência de AOS varia de 9% a 38% em adultos (SENARATNA *et al.*, 2017). Similar ao BS, o exame de referência para o diagnóstico de apneia e hipopneia é a PSG noturna (ACADEMIA AMERICANA DE MEDICINA DO SONO, 2008). Apesar da AOS estar associada a graves comorbidades como obesidade, hipertensão e diabetes tipo II (DE SOUZA *et al.*, 2020) ela ainda é uma condição subdiagnosticada e, conseqüentemente, os pacientes com esta condição não recebem um tratamento adequado (MARTYNOWICZ *et al.*, 2019). Esta informação reforça a importância de identificar fatores que possam atuar como indicadores ou suspeita de AOS.

O dentista pode atuar como um dos profissionais a identificar possíveis fatores indicadores de AOS, por meio da identificação da idade, sexo, índice de massa corporal, má qualidade do sono e, principalmente, dor orofacial ou cefaleia matinal, retrognatia, palato profundo, amígdalas ou língua aumentadas (LAVIGNE *et al.*, 2020). Além disso, médicos e dentistas podem realizar a detecção de possível BS entrevistando os pacientes sobre a percepção de ranger ou apertar dos dentes durante o sono. Assim como, o dentista poderia detectar dor ou sensibilidade nos músculos da mandíbula e sinais relacionados aos dentes, em um exame clínico. Nesse sentido, é fundamental ressaltar que a avaliação clínica não deve ser feita isoladamente e se basear apenas no desgaste dentário, pois este último não é o sinal mais confiável de bruxismo ativo (LOBBEZOO *et al.*, 2018). A avaliação do desgaste dental fornece informações sobre a quantidade acumulada de perda da superfície do dente, mas não fornece informações sobre o momento da perda. Não é possível distinguir se o processo está em andamento ou é resultado de uma perda anterior devido ao desgaste vs. exacerbação por refluxo gástrico ou dieta ácida (WETSELAAR *et al.*, 2019).

Apesar da grande importância do tema, estudos conduzidos buscando testar tal associação, ainda não apresentam conclusões definitivas. O primeiro estudo a testar esta associação foi realizado em 1986 (PHILLIPS *et al.*, 1986) em uma amostra de 24 pacientes, 14 com AOS e 10 sem. Os autores relataram que o apertamento dentário noturno foi ligeiramente maior em pacientes com AOS do que nos pacientes sem AOS, com diferença estatística (12,2 episódios vs 7,6). Os resultados foram justificados em função dos microdespertares frequentes, que foram responsáveis por uma maximização da atividade do BS (PHILLIPS *et al.*, 1986).

Okeson e colaboradores, em 1991 (OKESON *et al.*, 1991), estudaram a frequência de episódios de bruxismo em 12 pacientes com AOS e 12 controles (sem AOS). Embora os resultados tenham mostrado uma forte associação entre BS e eventos de despertares em ambos

os grupos, a análise estatística não mostrou diferença no número de eventos de BS entre os dois grupos (grupo AOS = 16,1 eventos/noite) e (grupo controle = 26, 2/noite).

Hosoya e colaboradores (2014) (HOSOYA *et al.*, 2014) analisaram o efeito do BS na arquitetura do sono e verificaram a associação entre BS e eventos respiratórios do sono em pacientes com AOS. A amostra incluiu 67 pacientes com AOS e 16 controles saudáveis. Os resultados demonstraram um risco maior de BS no grupo com AOS do que no grupo controle (OR: 3,96; IC 95%, 1,03 – 15,20; $p < 0,05$).

Em 2016, Saito e colaboradores (SAITO *et al.*, 2016) realizaram um estudo para investigar se existe alguma associação entre eventos respiratórios específicos e BS. Os resultados confirmaram que a presença de BS e apneia obstrutiva eram coincidentes em 50,8% participantes. Os resultados sugeriram que os eventos de apneia-hipopneia parecem estar relacionados a uma maior frequência com outros tipos de atividade motora do sono, e não à atividade do BS.

Tan e colaboradores (TAN *et al.*, 2019) investigaram a prevalência de BS em 147 adultos com AOS. Os autores avaliaram a associação entre BS e AOS com base na macroestrutura do sono e nos parâmetros respiratórios. Eles descobriram que um terço dos pacientes com AOS apresentavam BS. Além disso, identificaram que os pacientes com BS apresentavam mais despertares relacionados à respiração e dessaturações de oxigênio.

Martynowicz e colaboradores (MARTYNOWICZ *et al.*, 2019) analisaram 110 pacientes adultos com suspeita de AOS. Os episódios de bruxismo foram pontuados na PSG, de acordo com as normas da Academia Americana de Medicina do Sono (AAMS). A taxa de bruxismo foi maior no grupo com AOS leve e moderada, se comparado ao grupo com AOS grave ($5,50 \pm 4,58$ vs. $1,62 \pm 1,28$, $p < 0,05$). A AOS leve a moderada foi associada ao BS.

Maluly e colaboradores em 2020 (MALULY *et al.*, 2020) avaliaram a força da associação entre BS e AOS em um estudo de base populacional em São Paulo, Brasil. Foram coletados dados de BS definitivo por meio de PSG e BS possível com questionários de autorrelato em 1.042 participantes. Foi encontrada uma maior relação de prevalência em pacientes com BS provável e apneia severa (>30 episódios) (Razão de prevalência: 2,81; CI: 1,22 – 6,44, $p = 0,02$). No entanto, em pacientes com BS definitivo não foi encontrada associação (Razão de prevalência: 2,03; CI: 0,49 – 8,36, $p = 0,33$).

De Holanda e colaboradores (2020) conduziram um estudo de caso-controle, que incluiu uma amostra de 58 pacientes com BS (grupo caso) e 58 sem BS (grupo controle), identificados com base no exame de PSG e pareados por sexo e idade. Os autores verificaram

que o índice de apneia-hipopneia foi menor nos pacientes com BS do que nos pacientes sem BS.

Resultados semelhantes foram encontrados por Kim e colaboradores (2020) (KIM; LEE; LEE, 2020). O objetivo do estudo foi identificar os fatores associados a episódios de BS em 100 pacientes com AOS utilizando exames de PSG em um laboratório do sono. Dentre os 100 pacientes com AOS, 10 tiveram episódios de BS e 90 não apresentaram BS. O índice de apneia-hipoapneia (eventos por hora) foi maior no grupo sem BS 45.1 (\pm 28.2) do que no grupo com BS 25.5 (\pm 16.6) ($p=0.03$).

Smardz e colaboradores em 2020 (SMARDZ *et al.*, 2020) investigaram a relação entre BS e distúrbios respiratórios do sono, entre eles a AOS. Pacientes com diagnóstico de BS provável foram avaliados pela PSG e divididos em dois grupos, com e sem BS. De 77 participantes incluídos, 58 apresentaram BS e 19 não apresentaram. Não foi encontrada diferença estatística na relação entre BS e AOS ($p=0,82$).

Um estudo recente, conduzido por uma equipe da Universidade de Minas Gerais, Brasil, verificou a associação entre BS e AOS. A amostra foi composta de 240 participantes que foram submetidos à PSG em uma noite de sono. O BS foi definido pela presença de mais de dois eventos de atividade rítmica dos músculos mastigatórios por hora de sono. O estudo demonstrou um maior índice de apneia-hipopneia (eventos por hora) nos participantes com BS ($n=103$) ($48,28 \pm 25,84$) do que o grupo sem BS ($n=137$) ($37,37 \pm 23,46$), $p=0.001$ (MASSAHUD *et al.*, 2022).

Como se pode perceber, ao longo dos anos, diversos estudos foram conduzidos demonstrando resultados controversos. Alguns estudos demonstraram associação positiva, ou seja, pacientes com AOS apresentam maior atividade muscular rítmica (HOSOYA *et al.*, 2014; MARTYNOWICZ *et al.*, 2019; MASSAHUD *et al.*, 2022; PHILLIPS *et al.*, 1986; TAN *et al.*, 2019). Outros estudos apresentam associação negativa, ou seja, pacientes com AOS apresentam menor atividade muscular mastigatória rítmica (DE HOLANDA *et al.*, 2020; KIM; LEE; LEE, 2020). E por fim, há os estudos que não encontraram essa associação (OKESON *et al.*, 1991; MALULY *et al.*, 2013; SAITO *et al.*, 2016; SMARDZ *et al.*, 2020).

Uma revisão sistemática conduzida em 2014, avaliou a provável associação entre o BS e as distúrbios respiratórios do sono, e concluiu que não existiam evidências suficientes para aceitar ou refutar tal associação, indicando a necessidade de mais estudos primários com essa finalidade (DE LUCA CANTO *et al.*, 2014). Posteriormente, em 2017, outra RS sobre a temática concluiu que ainda não havia dados científicos suficientes para definir uma ligação

causal clara entre AOS e BS (JOKUBAUSKAS; BALTRUSAITYTE, 2017). No ano de 2020 uma nova RS foi conduzida, com o objetivo de avaliar a associação entre as duas condições e apesar da inclusão de novos estudos a conclusão dos autores foi que não havia evidências científicas para suportar uma relação entre SB e AOS (DA COSTA LOPES *et al.*, 2020). Recentemente uma RS investigou a associação de BS e distúrbios do sono, e apesar do BS parecer estar mais prevalente na população com AOS do que na população em geral, a variabilidade dos estudos não permitiu uma conclusão definitiva sobre a associação das duas condições (KUANG *et al.*, 2022). Buscando entender a metodologia dos estudos, uma revisão de escopo foi conduzida para avaliar a evidência disponível sobre a possível relação entre BS e AOS. Os autores concluíram que com base na literatura existente, não é possível confirmar uma associação entre as duas condições em adultos. Em crianças essa associação parece plausível, no entanto a evidência disponível é insuficiente para confirmar tal associação. Os autores sugerem inúmeras melhorias para os futuros estudos a serem conduzidos sobre este tema, a fim de se alcançar resultados mais robustos (PAULETTO *et al.*, 2022).

Existem poucos estudos na literatura que testam a hipótese de o BS atuar como fator protetor da AOS. O primeiro a avaliar a relação de temporalidade entre as duas condições foi realizado em 2000, por Sjöholm e colaboradores (SJOHOLM *et al.*, 2000). Neste estudo 21 pacientes com OSA foram incluídos, 11 pacientes apresentaram AOS leve e destes seis apresentaram BS, 10 pacientes apresentaram AOS moderada, e quatro apresentaram BS. Os episódios de contração do músculo masseter foram associados ao término de episódios de apneia ou hipopneia em apenas 3,5% dos grupos AOS leve e 14,4% do grupo moderado. Os resultados demonstraram que o BS raramente está diretamente associado com eventos de apneia, mas poderia estar relacionado ao sono perturbado de pacientes com AOS.

Saito e colaboradores (SAITO *et al.*, 2014) investigaram a associação temporal entre AOS e eventos de BS em dez pacientes submetidos à PSG. A maioria dos eventos de BS ocorreram após os episódios de apneia-hipopneia. Esses achados sugerem que eventos de BS ocorrendo próximo aos eventos de apneia-hipopneia são uma forma secundária de bruxismo. No entanto, é importante destacar que nem todos os episódios de BS estavam relacionados aos eventos de apneia-hipopneia, sugerindo que outras influências podem estar presentes no relacionamento temporal entre as duas condições (SAITO *et al.*, 2014).

Recentemente (2022), um estudo piloto com avaliação de PSGs de 30 pacientes não encontrou correlação temporal entre índice de bruxismo do sono e episódios de apneia-

hipopneia. A maioria dos episódios de bruxismo do sono (66,8%) aconteceram sem uma relação temporal com a apneia obstrutiva do sono (COLONNA *et al.*, 2022).

Devido às inúmeras inconsistências da literatura e à alta relevância clínica do tema, esta tese buscou contribuir com evidências científicas atualizadas sobre a temática.

3 OBJETIVOS

3.1 OBJETIVO GERAL

Verificar a relação entre bruxismo do sono e apneia obstrutiva do sono.

3.2 OBJETIVOS ESPECÍFICOS

- Mapear a literatura disponível sobre a relação entre bruxismo do sono e de apneia obstrutiva em adultos e crianças, verificando a prevalência de ambas as condições, associação e relação temporal entre os episódios de bruxismo e eventos de apneia obstrutiva;
- Descrever a presença de bruxismo do sono definitivo em uma população com apneia obstrutiva do sono.
- Verificar a relação temporal e padrão de ocorrência entre os episódios de bruxismo do sono e apneia obstrutiva;

Para responder a este objetivo, as hipóteses são:

Hipóteses Nula (H0) = Não há um padrão de ocorrência específico entre os episódios de bruxismo de apneia obstrutiva do sono.

Hipótese Alternativa (H1) = Há um padrão de ocorrência específico entre os episódios de bruxismo e apneia obstrutiva.

4 ARTIGO 1

O presente artigo foi publicado no Journal SLEEP (Fator de Impacto: 6.313; Qualis CAPES A1), em 20 de abril de 2022, e encontra-se redigido conforme última submissão do manuscrito. A sua versão publicada encontra-se no Anexo C.

Sleep Bruxism and Obstructive Sleep Apnea: Association, Causality or Spurious

Finding? A Scoping Review

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Abstract

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

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

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

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

Keywords: Sleep bruxism, obstructive sleep apnea, scoping review, association, prevalence.

Statement of Significance

Studying a possible relationship between sleep bruxism (SB) and obstructive sleep apnea (OSA) is of interest to physicians and dentists as it will allow improved comprehensive

management in presence of either comorbidity. The present synthesis reveals that it is not possible to confirm a relation between SB and OSA in adult patients due to the large variability and lack of standardized methods. In pediatric patients, although this association might be possible, it is not conclusive from the current literature. Phenotyping of the overlap between these comorbidities may reveal specificity in a sub-group that will contribute to personalized optimal management of the complex interactions between these conditions.

Introduction

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

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

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

Nowadays, the literature has avoided using the expression “diagnosis of bruxism”, preferring the use of the term’s “assessment” or “detection” since in otherwise healthy individuals, SB is considered a physiological motor behavior [1, 6]. SB detection remains a challenge in clinical practice. In 2018, the International Consensus for the Assessment of Bruxism proposed the following classification: (1) possible SB, exclusively based on self-report; (2) probable SB, based on a positive clinical inspection result with or without a positive

self-report; and (3) definitive SB, based on a positive measurement tool assessment with or without positive self-report and/or positive clinical inspection [1]. Accordingly, definitive SB can be confirmed with the use of electromyography (EMG) recording masticatory muscle during sleep, although alternative methodologies have been recently proposed [21]. When the presence of comorbidities is suspected, EMG activity recordings of the masticatory muscles should ideally be part of a more comprehensive examination, i.e., PSG that includes respiratory variables and, when possible, audio and video recordings [3, 22-24].

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

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

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

Methods

Protocol and registration

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

Eligibility criteria

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

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

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

Exclusion Criteria

1. Studies that did not evaluate the relationship between SB and OSA;
2. Studies in adults that did not detect the SB and OSA by PSG;
3. Reviews, letters, books, conference abstracts, case reports, case series, opinion articles, technique articles, posters, guidelines, short paper, pilot studies, mechanism evaluation studies;
4. Full-text or data not available, even after three attempts to contact the corresponding authors over three weeks.

Information sources

A search strategy developed with the help of an experienced health science librarian was applied on six databases: Embase, LILACS, Livivo, PubMed, Web of Science, and Scopus.

Grey literature was searched on Proquest Dissertation and Theses, OpenGrey and Google Scholar. Additionally, experts were contacted for the additional indication of studies for inclusion. Hand searches of bibliographies from included studies were also conducted. The search was carried out on March 17, 2021, and it was updated on August 9, 2021. The references were imported into a reference software manager (EndNote X9 ®; Thomson Reuters, Philadelphia, PA, United States), and the duplicate documents were excluded.

Search

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

Selection of sources of evidence

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

Data charting process

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

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

Data items

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

Synthesis of results

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

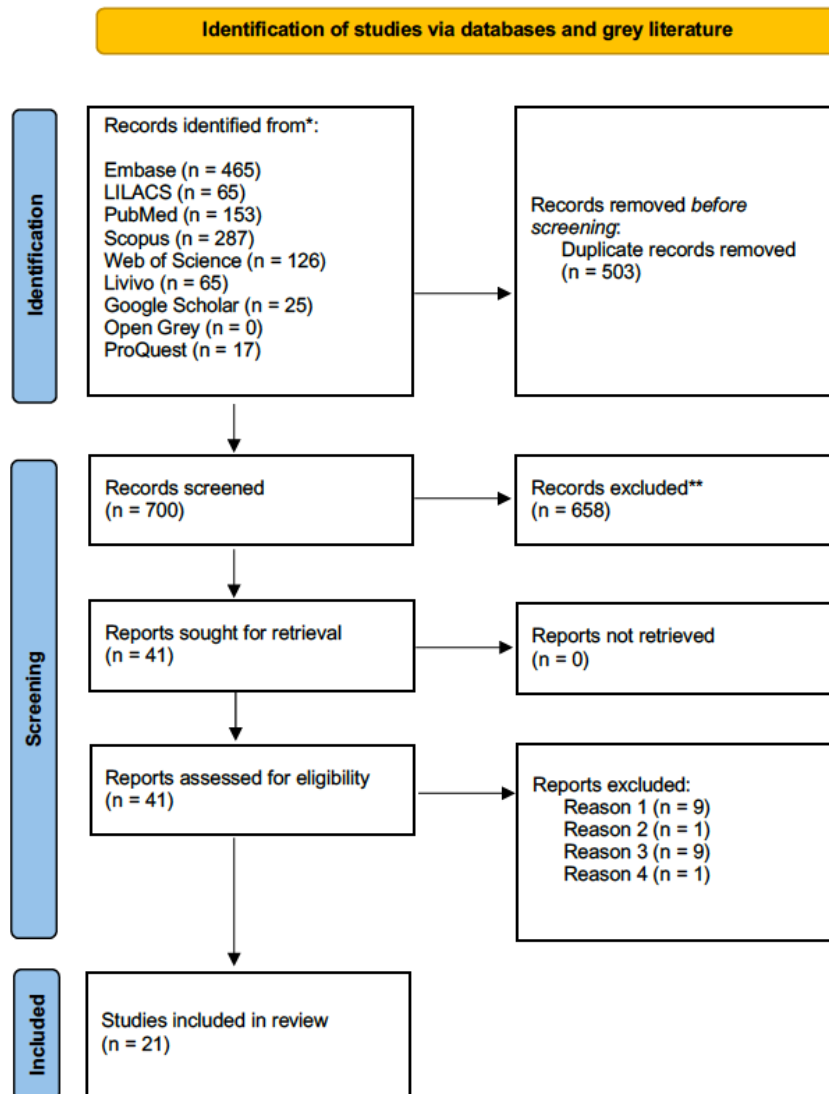
A narrative summary was drafted to synthesize the findings and describe the evidence identified concerning the review objective.

Results

Selection of sources of evidence

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

Figure 1. PRISMA 2020 flow diagram for new systematic reviews which included searches of databases, registers and other sources.



Source: prepared by the author.

Characteristics of sources of evidence

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

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

Results of individual sources of evidence

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

Sleep Bruxism and Obstructive Sleep Apnea Assessment

Adults

SB and OSA conditions were assessed by using PSG.

Children

Sleep Bruxism detection

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

Obstructive Sleep Apnea diagnosis

Three studies used PSG [50, 54, 55], three studies used only questionnaires answered by the caretakers [48, 51, 53], and two studies used a questionnaire plus clinical evaluation to detect OSA [49, 52]. More details are presented in Table 3.

Obstructive Sleep Apnea Diagnostic Criteria

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

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

Sleep Bruxism Detection Criteria

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

score is described in Table 2. The criteria used by studies in children to detect bruxism are shown in Table 3.

Synthesis of results

Prevalence of concurrent Sleep Bruxism and Obstructive Sleep Apnea

Adults

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

Children

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

Association between Sleep Bruxism and Obstructive Sleep Apnea

Adults

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

Figure 2. Association between SB and OSA in adults with SB and OSA detection by PSG. Green cells indicate a positive association between the two conditions; yellow indicates the absence of association, and amber color represents the negative association. *In the Martynowicz et al., 2019 study, this association was found in mild and moderate OSA.

Study	Association SB and OSA
Holanda et al., 2020 [34]	(-)
Hosoya et al., 2014 [17]	(+)
Kim et al., 2020 [46]	(-)
Maluly et al., 2020 [33]	No
Martynowicz et al., 2019*[45]	(+)
Okeson et al., 1991 [47]	No
Phillips et al., 1986 [42]	(+)
Saito et al., 2016 [32]	No
Smardz et al., 2020 [43]	No
Tan et al., 2019 [16]	(+)

Source: prepared by the author.

Children

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

Figure 3. Association between SB and OSA in children. Green indicates a positive association between the two conditions, and yellow indicates the absence of association.

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

Source: prepared by the author.

Temporal relationship

Two studies evaluated the temporal relationship between SB and OSA. The study by Sjöholm et al., 2000 [35] showed that events between the two conditions seem to be rarely associated. Saito et al. (2014) [36] indicated that most bruxism events occurred after sleep apnea-hypopnea events and that some occurred before, reducing the strength of a causal, temporal sequence.

Respiratory variables in adult studies

Findings related to the respiratory variables analyzed (Oxygen Desaturation Index, Sleep Efficiency, Arousal Index, AHI, and Minimum Oxygen Saturation) are shown in Table 4. There were no statistical differences in sleep efficiency between the groups with and without SB [16, 17, 46]. Oxygen Desaturation Index was associated with patients with SB in two studies

[16, 17]. Higher Arousal Index and lower Minimum Oxygen Saturation were associated with SB patients in one study [16], but with a small statistically significant difference, albeit not clinically relevant. Regarding the AHI, there were contradictory results, and one study showed higher AHI in SB participants [17], another in non-SB participants [46] and another study reported that no differences were found [43].

Discussion

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

Although OSA is linked to serious morbidities, it is still underdiagnosed and consequently not adequately or timely managed in the general population [45]. This fact emphasizes the importance of identifying factors that could indicate an increased probability of having OSA. In addition to physicians, the dentist could serve as one of the primary care professionals to screen patients for risk factors for OSA such as age, gender, body mass index, poor sleep, and especially, orofacial pain or morning headache, retrognathia, high palate, and enlarged tonsils or tongue due to their field of practice [58]. Furthermore, physicians and dentists can carry out the detection of possible SB by interviewing the patients on awareness of tooth grinding or clenching in relation to sleep, and to a probable status through clinical examination of jaw muscle pain or tenderness, and tooth related signals. In this regard, it is essential to emphasize that the clinical evaluation should not be done in isolation and be based solely on tooth wear, as the latter is not the most reliable sign [1]. The assessment of tooth wear provides information on the cumulative amount of tooth surface loss, but does not provide information on the timing of the loss, i.e., whether the process is ongoing or is a result of a previous loss due to grinding *vs.* exacerbation by gastric reflux or acidic diet [5].

The studies conducted to assess the association between SB and OSA showed significant variability in their findings and conclusions. The studies were based on very different populations (i.e., SB only for which respiratory variables were analyzed, preferentially OSA with some SB overlap up to general population) and were designed with *a priori* intent to explore other objectives. It is also possible to observe gender and age differences in the test and control groups among the studies. It is known that the incidence of OSA is higher in men than

in women, and there is a higher prevalence of OSA with high body mass index (BMI) and ageing [26]. On the other hand, the incidence of self-reported SB decreases with age, and there appears to be no gender-related differences, although no difference was observed using PSG data [2, 3]. The included studies do not describe the population in detail, particularly concerning the presence of comorbidities such as gastroesophageal reflux disease, habits such as alcohol and coffee intake, smoking and use of antidepressant drugs, all of which are factors known to be associated with SB [59]. Due to their cross-sectional and descriptive study design, it is therefore impossible to establish a causal relationship even when an association is found [60]. Furthermore, only two studies evaluated the temporal relationship between OSA and SB events, which could be a better strategy for potentially explaining the putative causal relationship between the two conditions.

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

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

ideally sleep recording done in a natural environment over more than one night to take into account the night-to-night variability of both SB and OSA metrics. [24, 61]. Alternatively, should we have to rethink the methods considered as a reference standard to detect SB when a study design challenges causality? Whether the temporality of SB episodes is protective or not was also not clarified since only two studies evaluated the temporal association, and even such studies used different methodologies [35, 36].

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

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

Strengths, limitations

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

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

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

Suggestions for future research

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

Future studies should focus their analysis not only on identifying a relationship between SB and OSA, but to investigate the temporal association of the episodes (order of occurrence, spacing between episodes and duration) [35,36]. Furthermore, studies with larger population-based samples, participants who present the condition and do not present the condition (not just people with suspected OSA) [33] will be necessary. Population matching, based on sex, age and BMI [16, 77], should also be needed which can act as important confounders [78]. Along with the inclusion of accurate information on issues which can induce or attenuating SB such as tobacco, alcohol, caffeine consumption [59, 79], use of medications and addictive substances [80], or previous treatment with positive airway pressure [81].

Information should be collected based on the medical history and behavior of the patient like hypertension, orofacial pain, tooth grinding, clenching [82], sleepiness, insomnia, fatigue, snoring choking, with validated questionnaire-based tools (e.g., Stop Bang or similar) [83].

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

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

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

For adults, it doesn't seem to be possible to confirm a relationship between OSA and SB, nor confirm a protective effect of bruxism in patients with OSA based on current literature. Although a relationship with OSA seems plausible for SB children, the identified scientific evidence is scarce and present limitations on the SB detection methods. At this point, it is important to study appropriate metrics, considering the possible existence of a subgroup for

whom an association of SB and OSA may be present, i.e., a distinct phenotype that still needs to be identified.

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References

1. Lobbezoo F, Ahlberg J, Raphael KG, Wetselaar P, Glaros AG, Kato T, et al. International consensus on the assessment of bruxism: Report of a work in progress. *J Oral Rehabil.* 2018;45(11):837-44. Epub 2018/06/22. doi: 10.1111/joor.12663.
2. Manfredini D, Winocur E, Guarda-Nardini L, Paesani D, Lobbezoo F. Epidemiology of Bruxism in Adults: A Systematic Review of the Literature. *J Orofac Pain.* 2013;27(2):99-110. doi: 10.11607/jop.921.
3. Maluly M, Andersen ML, Dal-Fabbro C, Garbuio S, Bittencourt L, de Siqueira JT, et al. Polysomnographic study of the prevalence of sleep bruxism in a population sample. *J Dent Res.* 2013;92(7 Suppl):97S-103S. Epub 2013/05/22. doi: 10.1177/0022034513484328.
4. Manfredini D, Restrepo C, Diaz-Serrano K, Winocur E, Lobbezoo F. Prevalence of sleep bruxism in children: a systematic review of the literature. *J Oral Rehabil.* 2013;40(8):631-42. Epub 2013/05/25. doi: 10.1111/joor.12069.
5. Wetselaar P, Manfredini D, Ahlberg J, Johansson A, Aarab G, Papagianni CE, et al. Associations between tooth wear and dental sleep disorders: A narrative overview. *J Oral Rehabil.* 2019;46(8):765-75. Epub 2019/05/01. doi: 10.1111/joor.12807.
6. Stuginski-Barbosa J, Porporatti AL, Costa YM, Svensson P, Conti PC. Agreement of the International Classification of Sleep Disorders Criteria with polysomnography for sleep bruxism diagnosis: A preliminary study. *J Prosthet Dent.* 2017;117(1):61-6. Epub 2016/07/28. doi: 10.1016/j.prosdent.2016.01.035.

7. Palinkas M, Bataglioni C, de Luca Canto G, Machado Camolezi N, Theodoro GT, Siessere S, et al. Impact of sleep bruxism on masseter and temporalis muscles and bite force. *Cranio*. 2016;34(5):309-15. Epub 2016/04/15. doi: 10.1080/08869634.2015.1106811.
8. Zhou Y, Gao J, Luo L, Wang Y. Does Bruxism Contribute to Dental Implant Failure? A Systematic Review and Meta-Analysis. *Clin Implant Dent Relat Res*. 2016;18(2):410-20. Epub 2015/03/03. doi: 10.1111/cid.12300.
9. Chrcanovic BR, Kisch J, Albrektsson T, Wennerberg A. Factors influencing the fracture of dental implants. *Clin Implant Dent Relat Res*. 2018;20(1):58-67. Epub 2017/12/07. doi: 10.1111/cid.12572.
10. Chrcanovic BR, Kisch J, Larsson C. Analysis of technical complications and risk factors for failure of combined tooth-implant-supported fixed dental prostheses. *Clin Implant Dent Relat Res*. 2020;22(4):523-32. Epub 2020/06/12. doi: 10.1111/cid.12927.
11. Neu D, Baniyadi N, Newell J, Styczen D, Glineur R, Mairesse O. Effect of sleep bruxism duration on perceived sleep quality in middle-aged subjects. *Eur J Oral Sci*. 2018;126(5):411-6. Epub 2018/08/17. doi: 10.1111/eos.12564.
12. Camara-Souza MB, de Figueredo OMC, Rodrigues Garcia RCM. Association of sleep bruxism with oral health-related quality of life and sleep quality. *Clin Oral Investig*. 2019;23(1):245-51. Epub 2018/03/29. doi: 10.1007/s00784-018-2431-0.
13. Duarte J, Pauletto P, Massignan C, Bolan M, Domingos FL, Hallal ALC, et al. Association Between Sleep Bruxism and Quality of Life: A Systematic Review. *J Oral Facial Pain Headache*. 2020;34(4):341-52. Epub 2020/12/09. doi: 10.11607/ofph.2687.
14. Feu D, Catharino F, Quintao CC, Almeida MA. A systematic review of etiological and risk factors associated with bruxism. *J Orthod*. 2013;40(2):163-71. Epub 2013/06/26. doi: 10.1179/1465313312Y.0000000021.
15. Castroflorio T, Bargellini A, Rossini G, Cugliari G, Deregibus A. Sleep bruxism and related risk factors in adults: A systematic literature review. *Arch Oral Biol*. 2017;83:25-32. Epub 2017/07/12. doi: 10.1016/j.archoralbio.2017.07.002.
16. Tan MWY, Yap AU, Chua AP, Wong JCM, Parot MVJ, Tan KBC. Prevalence of Sleep Bruxism and Its Association with Obstructive Sleep Apnea in Adult Patients: A Retrospective Polysomnographic Investigation. *J Oral Facial Pain Headache*. 2019;33(3):269-77. Epub 2018/10/30. doi: 10.11607/ofph.2068.
17. Hosoya H, Kitaura H, Hashimoto T, Ito M, Kinbara M, Deguchi T, et al. Relationship between sleep bruxism and sleep respiratory events in patients with obstructive sleep apnea

syndrome. *Sleep Breath.* 2014;18(4):837-44. Epub 2014/02/15. doi: 10.1007/s11325-014-0953-5.

18. Hollowell DE. Mandible position and activation of submental and masseter muscles during sleep. *J Appl Physiol.* 1991;71(6):2267-73.

19. Lavigne GJ KT, Kolta A, Sessle BJ. Neurobiological Mechanisms Involved in Sleep bruxism. *Crit Rev Oral Biol Med.* 2013;14(1):30-46.

20. Hollowell DE, Suratt PM. Activation of masseter muscles with inspiratory resistance loading. *J Appl Physiol.* 1989;67(1):270-5.

21. Martinot J-B, Le-Dong N-N, Cuthbert V, Denison S, Gozal D, Lavigne G, et al. Artificial Intelligence Analysis of Mandibular Movements Enables Accurate Detection of Phasic Sleep Bruxism in OSA Patients: A Pilot Study. *Nat and Sci Sleep.* 2021;Volume 13:1449-59. doi: 10.2147/nss.S320664.

22. Lavigne GJ, Rompre PH, Montplaisir JY. Sleep bruxism: validity of clinical research diagnostic criteria in a controlled polysomnographic study. *J Dent Res.* 1996;75(1):546-52. Epub 1996/01/01. doi: 10.1177/00220345960750010601.

23. Casett E, Reus JC, Stuginski-Barbosa J, Porporatti AL, Carra MC, Peres MA, et al. Validity of different tools to assess sleep bruxism: a meta-analysis. *J Oral Rehabil.* 2017;44(9):722-34. Epub 2017/05/10. doi: 10.1111/joor.12520.

24. Lavigne G, Kato T, Herrero Babiloni A, Huynh N, Dal Fabbro C, Svensson P, et al. Research routes on improved sleep bruxism metrics: Toward a standardised approach. *J Sleep Res.* 2021:e13320. Epub 2021/03/07. doi: 10.1111/jsr.13320.

25. Guilleminault C, Tilkian A, Dement WC. The sleep apnea syndromes. *Annu Rev Med.* 1976;27(1):465-84.

26. Senaratna CV, Perret JL, Lodge CJ, Lowe AJ, Campbell BE, Matheson MC, et al. Prevalence of obstructive sleep apnea in the general population: a systematic review. *Sleep Med Rev.* 2017;34:70-81.

27. Tan HL, Gozal D, Kheirandish-Gozal L. Obstructive sleep apnea in children: a critical update. *Nat Sci Sleep.* 2013;5:109-23. Epub 2013/10/11. doi: 10.2147/NSS.S51907.

28. Park JG, Ramar K, Olson EJ. Updates on definition, consequences, and management of obstructive sleep apnea. *Mayo Clin Proc.* 2011;86(6):549-54; quiz 54-5. Epub 2011/06/02. doi: 10.4065/mcp.2010.0810.

29. Leger D, Bayon V, Laaban JP, Philip P. Impact of sleep apnea on economics. *Sleep Med Rev.* 2012;16(5):455-62. Epub 2012/01/17. doi: 10.1016/j.smr.2011.10.001.

30. AlGhanim N, Comondore VR, Fleetham J, Marra CA, Ayas NT. The economic impact of obstructive sleep apnea. *Lung*. 2008;186(1):7-12. Epub 2007/12/11. doi: 10.1007/s00408-007-9055-5.
31. Manfredini D, Guarda-Nardini L, Marchese-Ragona R, Lobbezoo F. Theories on possible temporal relationships between sleep bruxism and obstructive sleep apnea events. An expert opinion. *Sleep Breath*. 2015;19(4):1459-65. Epub 2015/03/22. doi: 10.1007/s11325-015-1163-5.
32. Saito M, Yamaguchi T, Mikami S, Watanabe K, Gotouda A, Okada K, et al. Weak association between sleep bruxism and obstructive sleep apnea. A sleep laboratory study. *Sleep Breath*. 2016;20(2):703-9. Epub 2015/11/14. doi: 10.1007/s11325-015-1284-x.
33. Maluly M, Dal Fabbro C, Andersen ML, Herrero Babiloni A, Lavigne GJ, Tufik S. Sleep bruxism and its associations with insomnia and OSA in the general population of Sao Paulo. *Sleep Med*. 2020;75:141-8. Epub 2020/08/29. doi: 10.1016/j.sleep.2020.06.016.
34. de Holanda TA, Castagno CD, Barbon FJ, Costa YM, Goettems ML, Boscato N. Sleep architecture and factors associated with sleep bruxism diagnosis scored by polysomnography recordings: A case-control study. *Arch Oral Biol*. 2020;112:104685. Epub 2020/02/26. doi: 10.1016/j.archoralbio.2020.104685.
35. Sjöholm TT, Lowe AA, Miyamoto K, Fleetham JA, Ryan CF. Sleep bruxism in patients with sleep-disordered breathing. *Arch Oral Biol*. 2000;45:889-96.
36. Saito M, Yamaguchi T, Mikami S, Watanabe K, Gotouda A, Okada K, et al. Temporal association between sleep apnea-hypopnea and sleep bruxism events. *J Sleep Res*. 2014. Epub 2014/03/19. doi: 10.1111/jsr.12099.
37. De Luca Canto G, Singh V, Gozal D, Major PW, Flores-Mir C. Sleep bruxism and sleep-disordered breathing: a systematic review. *J Oral Facial Pain Headache*. 2014;28(4):299-305. Epub 2014/10/28. doi: 10.11607/ofph.1294.
38. da Costa Lopes AJ, Cunha TCA, Monteiro MCM, Serra-Negra JM, Cabral LC, Junior PCS. Is there an association between sleep bruxism and obstructive sleep apnea syndrome? A systematic review. *Sleep Breath*. 2020;24(3):913-21. Epub 2019/10/20. doi: 10.1007/s11325-019-01919-y.
39. Jokubauskas L, Baltrusaityte A. Relationship between obstructive sleep apnoea syndrome and sleep bruxism: a systematic review. *J Oral Rehabil*. 2017;44(2):144-53. Epub 2016/12/16. doi: 10.1111/joor.12468.

40. Munn Z, Peters MDJ, Stern C, Tufanaru C, McArthur A, Aromataris E. Systematic review or scoping review? Guidance for authors when choosing between a systematic or scoping review approach. *BMC Med Res Methodol.* 2018;18(1):143. Epub 2018/11/21. doi: 10.1186/s12874-018-0611-x.
41. Tricco AC, Lillie E, Zarin W, O'Brien KK, Colquhoun H, Levac D, et al. PRISMA Extension for Scoping Reviews (PRISMA-ScR): Checklist and Explanation. *Ann Intern Med.* 2018;169(7):467-73. Epub 2018/09/05. doi: 10.7326/M18-0850.
42. Phillips BA, Okeson J, Paesani D, Gilmore R. Effect of sleep position on sleep apnea and parafunctional activity. *Chest.* 1986;90(3):424-9. Epub 1986/09/01. doi: 10.1378/chest.90.3.424.
43. Smardz J, Martynowicz H, Wojakowska A, Michalek-Zrabkowska M, Mazur G, Wieczorek T, 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. Epub 2020/02/09. doi: 10.1016/j.sleep.2019.08.025.
44. Coelho PR, Curcio WB, EspíRito Santo RPd, Pereira AB, Leite FPP, Chaves MdGA. Prevalência da Comorbidade entre o Bruxismo do Sono e a Síndrome de Apneia - Hipopneia Obstrutiva do Sono: Um Estudo Polissonográfico. *Pesquisa Brasileira em Odontopediatria e Clínica Integrada.* 2012;12(4):491-6. doi: 10.4034/pboci.2012.124.07.
45. Martynowicz H, Gac P, Brzecka A, Poreba R, Wojakowska A, Mazur G, et al. The Relationship between Sleep Bruxism and Obstructive Sleep Apnea Based on Polysomnographic Findings. *J Clin Med.* 2019;8(10). Epub 2019/10/17. doi: 10.3390/jcm8101653.
46. Kim DH, Lee SH, Lee SH. Sleep Bruxism Episodes in Patients with Obstructive Sleep Apnea Syndrome Determined by In-Laboratory Polysomnography. *Appl Sci.* 2020;10(23). doi: 10.3390/app10238587.
47. Jeffrey P, Okeson BAP, David T.R. Berry, Yvette R. Cook, John F. Cabelka,. Nocturnal Bruxing Events in Subjects With Sleep-Disordered Breathing and Control Subjects. *J Craniomand Disord.* 1991;5:258-64.
48. Castilho RL, Matsumoto LH, Castilho GL, Weber SAT. The interface between dentistry and respiratory sleep disorders in children. *Sleep Sci.* 2020;13(4):220-3. Epub 2021/02/11. doi: 10.5935/1984-0063.20200004.

49. Ferreira NMR, dos Santos JFF, dos Santos MBF, Marchini L. Sleep bruxism associated with obstructive sleep apnea syndrome in children. *Cranio®*. 2016;33(4):251-5. doi: 10.1179/2151090314y.0000000025.
50. Paloma Baiardi Gregório RAA, Almir Galvão Vieira Bitencourt,, Flávia Branco Cerqueira Serra Neves RT, Francisco Hora. Symptoms of obstructive sleep apnea-hypopnea syndrome in children. *J Bras Pneumol*. 2008;34(6):356-61.
51. Abhishek Goyal APP, Girish C Bhatt, Bharat Choudhary, Rajesh Patil. Association of pediatric obstructive sleep apnea with poor academic performance: A school-based study from India. *Lung*. 2018;35(2):5. doi: 10.4103/lungindia.lungindia_218_17.
52. Segu M, Pollis M, Santagostini A, Meola F, Manfredini D. Correlation between Parental-Reported Tooth Grinding and Sleep Disorders: Investigation in a Cohort of 741 Consecutive Children. *Pain Res Manag*. 2020;2020:3408928. Epub 2020/08/18. doi: 10.1155/2020/3408928.
53. Tachibana M, Kato T, Kato-Nishimura K, Matsuzawa S, Mohri I, Taniike M. Associations of sleep bruxism with age, sleep apnea, and daytime problematic behaviors in children. *Oral Dis*. 2016;22(6):557-65. Epub 2016/04/19. doi: 10.1111/odi.12492.
54. Sheldon SH. Obstructive Sleep Apnea and Bruxism in Children. *Sleep Medicine Clinics*. 2010;5(1):163-8. doi: 10.1016/j.jsmc.2009.10.004.
55. Singh N. Sleep Bruxism Related Tooth Wear as a Clinical Marker for Obstructive Sleep Apnea/Hypopnea Syndrome in Children: Tufts University School of Dental Medicine; 2011.
56. The AASM Manual for the Scoring of Sleep and Associated Events - Rules, Terminology and Technical Specifications. Scoring Manual Version 2.2, (2015).
57. Tan HL, Kheirandish-Gozal L, Gozal D. Pediatric Home Sleep Apnea Testing: Slowly Getting There! *Chest*. 2015;148(6):1382-95. Epub 2015/08/14. doi: 10.1378/chest.15-1365.
58. Lavigne GJ, Babiloni AH, Beetz G, Dal Fabbro C, Sutherland K, Huynh N, et al. Critical Issues in Dental and Medical Management of Obstructive Sleep Apnea. *Crit Rev Oral Biol Med*. 2020;99:26-35.
59. Melo G, Duarte J, Pauletto P, Porporatti AL, Stuginski-Barbosa J, Winocur E, et al. Bruxism: An umbrella review of systematic reviews. *J Oral Rehabil*. 2019;46(7):666-90. Epub 2019/04/18. doi: 10.1111/joor.12801.
60. Grimes DA, Schulz KF. Descriptive studies: what they can and cannot do. *The Lancet*. 2002;359(9301):145-9. doi: 10.1016/s0140-6736(02)07373-7.

61. Pevernagie DA, Gnidovec-Strazisar B, Grote L, Heinzer R, McNicholas WT, Penzel T, et al. On the rise and fall of the apnea-hypopnea index: A historical review and critical appraisal. *J Sleep Res.* 2020;29(4):e13066. Epub 2020/05/15. doi: 10.1111/jsr.13066.
62. Osman AM, Carter SG, Carberry JC, Eckert DJ. Obstructive sleep apnea: current perspectives. *Nat Sci Sleep.* 2018;10:21-34. Epub 2018/02/09. doi: 10.2147/NSS.S124657.
63. Carberry JC, Amatoury J, Eckert DJ. Personalized Management Approach for OSA. *Chest.* 2018;153(3):744-55. Epub 2017/06/21. doi: 10.1016/j.chest.2017.06.011.
64. Thymi M, Lobbezoo F, Aarab G, Ahlberg J, Baba K, Carra MC, et al. Signal acquisition and analysis of ambulatory electromyographic recordings for the assessment of sleep bruxism: A scoping review. *J Oral Rehabil.* 2021;48(7):846-71.
65. Penzel T, Conradt R. Computer based sleep recording and analysis. *Sleep Med Rev.* 2000;4(2):131-48. Epub 2003/01/18. doi: 10.1053/smr.1999.0087.
66. Estevez PA, Held CM, Holzmann CA, Perez CA, Perez JP, Heiss J, et al. Polysomnographic pattern recognition for automated classification of sleep-waking states in infants. *Med Biol Eng Comput.* 2002;40(1):105-13. Epub 2002/04/17. doi: 10.1007/BF02347703.
67. Dumais I, Lavigne G, Carra M, Rompré P, Huynh N. Could transient hypoxia be associated with rhythmic masticatory muscle activity in sleep bruxism in the absence of sleep-disordered breathing? A preliminary report. *J Oral Rehabil.* 2015;42(11):810-8.
68. Chowdhury T, Bindu B, Singh GP, Schaller B. Sleep disorders: is the trigemino-cardiac reflex a missing link? *Front Neurol.* 2017;8:63.
69. Suzuki Y, Rompre P, Mayer P, Kato T, Okura K, Lavigne GJ. Changes in oxygen and carbon dioxide in the genesis of sleep bruxism: a mechanism study. *J Prosthodont Res.* 2020;64(1):43-7. Epub 2019/06/05. doi: 10.1016/j.jpor.2019.04.012.
70. Kadmon G, Shapiro CM, Chung SA, Gozal D. Validation of a pediatric obstructive sleep apnea screening tool. *Int J Pediatr Otorhinolaryngol.* 2013;77(9):1461-4. Epub 2013/07/11. doi: 10.1016/j.ijporl.2013.06.009.
71. Hasukic B. OSA-18 survey in evaluation of sleep-disordered breathing in children with adenotonsillar hypertrophy. *Med Arch.* 2013;67(2):111-4. Epub 2013/12/18. doi: 10.5455/medarh.2013.67.111-114.
72. Kuwada A, Mohri I, Asano R, Matsuzawa S, Kato-Nishimura K, Hirata I, et al. Japanese Sleep Questionnaire for Elementary Schoolers (JSQ-ES): validation and population-based score distribution. *Sleep Med.* 2018;41:69-77. Epub 2018/02/10. doi: 10.1016/j.sleep.2017.07.025.

73. Chang L, Wu J, Cao L. Combination of symptoms and oxygen desaturation index in predicting childhood obstructive sleep apnea. *Int J Pediatr Otorhinolaryngol*. 2013;77(3):365-71. Epub 2012/12/19. doi: 10.1016/j.ijporl.2012.11.028.
74. Machado E, Dal-Fabbro C, Cunali PA, Kaizer OB. Prevalence of sleep bruxism in children: a systematic review. *Dental Press J Orthod*. 2014;19(6):54-61. Epub 2015/01/30. doi: 10.1590/2176-9451.19.6.054-061.oar.
75. Castroflorio T, Bargellini A, Rossini G, Cugliari G, Rainoldi A, Deregibus A. Risk factors related to sleep bruxism in children: A systematic literature review. *Arch Oral Biol*. 2015;60(11):1618-24. Epub 2015/09/10. doi: 10.1016/j.archoralbio.2015.08.014.
76. Arksey H, O'Malley L. Scoping studies: towards a methodological framework. *International Journal of Social Research Methodology*. 2005;8(1):19-32. doi: 10.1080/1364557032000119616.
77. Kuang B, Li D, Lobbezoo F, de Vries R, Hilgevoord A, de Vries N, et al. Associations between sleep bruxism and other sleep-related disorders in adults: a systematic review. *Sleep Med*. 2022;89:31-47. Epub 2021/12/09. doi: 10.1016/j.sleep.2021.11.008.
78. Pauletto P, Reus JC, Bolan M, Massignan C, Flores-Mir C, Maia I, et al. Association between obstructive sleep apnea and health-related quality of life in untreated adults: a systematic review. *Sleep Breath*. doi: 10.1007/s11325-021-02323-1.
79. Bertazzo-Silveira E, Kruger CM, Porto De Toledo I, Porporatti AL, Dick B, Flores-Mir C, et al. Association between sleep bruxism and alcohol, caffeine, tobacco, and drug abuse: A systematic review. *J Am Dent Assoc*. 2016;147(11):859-66 e4. Epub 2016/10/30. doi: 10.1016/j.adaj.2016.06.014.
80. de Baat C, Verhoeff M, Ahlberg J, Manfredini D, Winocur E, Zweers P, et al. Medications and addictive substances potentially inducing or attenuating sleep bruxism and/or awake bruxism. *J Oral Rehabil*. 2021;48(3):343-54. Epub 2020/07/28. doi: 10.1111/joor.13061.
81. Oksenberg A, Arons E. Sleep bruxism related to obstructive sleep apnea: the effect of continuous positive airway pressure. *Sleep Med*. 2002;3:513-5.
82. Carra MC, Huynh N, Fleury B, Lavigne G. Overview on sleep bruxism for sleep medicine clinicians. *Sleep Medicine Clin*. 2015;10(3):375-84.
83. Kline LR, Collop N, Finlay G. Clinical presentation and diagnosis of obstructive sleep apnea in adults. 2017. UpToDate [https://www.uptodate.com/contents/clinical-presentation-and-diagnosis-of-obstructive-sleep-apnea-in-adults?topicRef=7695&source=related_link] accessed Febr 06 2022.

84. Rundo JV, Downey III R. Polysomnography. *Handb Clinical Neurol.* 2019;160:381-92. doi: 10.1016/B978-0-444-64032-1.00025-4
85. Rompré P, Daigle-Landry D, Guitard F, Montplaisir J, Lavigne G. Identification of a sleep bruxism subgroup with a higher risk of pain. *J Dent Res.* 2007;86(9):837-42.
86. Eckert DJ. Phenotypic approaches to obstructive sleep apnoea - New pathways for targeted therapy. *Sleep Med Rev.* 2018;37:45-59. Epub 2017/01/24. doi: 10.1016/j.smrv.2016.12.003.

Table 1. Search strategy in PubMed Database.

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

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

Author, Year	Objective	Sample Sex (M/F) Mean age or orange	Inclusion Criteria OSA and SB Assessment	OSA Diagnoses Criteria	SB Criteria	Findings Statistical Analysis	Prevalence SB/OSA concomitant	Report on the sources of funding Conflict of Interest
Coelho et al., 2012 [44] Brazil Descriptive	To investigate the prevalence of comorbidity between the SB and OSA in the polysomnographic findings of patients with sleep disturbance.	N=909 (NR) NR	Medical records of patients of both genders, with suspected sleep disorders, in the period from March 2007 to June 2011.	Apnea plus hypopnea index per hour of sleep, with the occurrence of at least five apnea plus hypopnea per hour of sleep, added to clinical symptoms, the most important of which are loud snoring and excessive daytime sleepiness.	At least two episodes of rhythmic activity of masticatory muscles (ARMM) associated with the sound of “gnashing of teeth”; more than four ARMM episodes per hour of sleep, with no “gritting your teeth” sound; more than five electromyographic bursts per AMMR episodes; or more than 25 EMG bursts per hour of sleep.	The SB/OSA comorbidity was prevalent in the surveyed population. Descriptive	5.28% (48/909)	NR NR
Holanda et al., 2020 [34] Brazil Cross-Sectional	To evaluate the association between the diagnosis SB scored by PSG recordings, clinical conditions	N=116 SB: 58 (M=25, F=33) Non-SB: 58 (M=25, F: 33)	All PSG recordings and self-reported data obtained from patients (20 years or older) who	NR	RMMA index was greater than 2 episodes per hour of sleep.	SB was diagnosed more frequently in subjects who had fewer OSA events ($p = 0.005$). OSA decreased the chances (OR 0.55; 95 % CI: 0.23–	15.5% (58/116)	This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

	and sleep architecture.	SB: 42.20 ± 14.52 Non-SB: 42.55 ± 14.78	underwent PSG at the Pelotas Sleep Institute, a private medical outpatient clinic, from January 2015 to December 2017.			1.30; p =0.173) of an SB diagnosis. The AHI (p =0.002) were all lower in bruxers than in non-bruxers. Logistic Regression test.		None declared.
Hosoya et al., 2014 [17] Japan Cross-Sectional	To examine the relationships between SB and sleep respiratory events in patients with OSA and healthy volunteers.	N=83 OSA:67 Non-OSA:16 OSA: 54.3±13.2 Non-OSA: 23.9±5.5	Patients with suspected OSA who consulted the Respiratory Medicine Department of Tohoku University Hospital between May 2010 and August 2011.	Apnea: cessation of airflow lasting 10 s or more. Hypopnea: a greater than 50% decrease in the thoracoabdominal amplitude associated with a greater than 3% decline in the oxygen saturation from the preceding value.	Subjects were diagnosed with SB when they had more than four bruxism events per hour of sleep.	Comparison: Apnea/hypopnea and desaturation events occurred significantly more frequently in subjects with than without SB. There were no significant differences in sleep efficiency, micro-arousal event, between subjects with and without SB. Correlation: The frequency of SB events was positively correlated with frequencies of each of the following: apnea/hypopnea, OSA, micro-arousal	47.8% (32/67)	NR None to declare.

						and oxygen desaturation. Mann-Whitney U test and Spearman correlation test		
Kim et al., 2020 [46] South Korea Cross-Sectional	To identify the factors associated with SB episodes in patients with OSA using in-laboratory PSG records.	N=100 SB:10 (M) Non-SB: 90 (M=80, F=10) SB: 43.4 ± 16.1 Non-SB: 48.5 ± 13.9)	Patients who visited the hospital for snoring, OSA, or drowsiness during the day and underwent in-laboratory full-night PSG between March 2017 and February 2019.	Apnea: a drop in the peak signal excursion by $\geq 90\%$ of the pre-event baseline using a thermistor or as complete airflow cessation for at least 10 s. Hypopnea: a peak signal excursion drop by $\geq 30\%$ of the pre-event baseline using a pressure cannula and a $\geq 3\%$ oxygen desaturation from the pre-event baseline, whether or not it was associated with arousal. AHI: was defined as the total number of apnea and hypopnea events	A constant burst episode lasting longer than 2 s or three or more bursts that were 0.25–2 s long was considered to be a SB episode. Simultaneously, tooth grinding sounds and typical jaw movements were required to consider these EMG activities or bursts as SB episodes.	Lower AHI, and higher oxygen saturation were associated with SB episodes in the OSA patients. Mann–Whitney U	10 % (10/100)	This research received no external funding. The authors declare no conflict of interest.

				within 1 h of sleep.				
Maluly et al., 2020 [33] Brazil Cross-Sectional	To assess the strength of the associations between SB, insomnia and OSA in a general population.	N=620 SB: 56 (M=22, F=34). Non-SB: 564 (M=247, F=317). NR	General representative population of São Paulo (EPISONO study).	The apnea and AHI were classified as events per hour: normal from 0 to 5, mild from 5 to 15, moderate from 15 to 30, and severe if greater than 30.	Participants who responded positively to the questionnaire in respect of SB symptoms, and had ≥ 2 RMMA episodes per hour in PSG recordings.	No statistical difference was found. Logistic regression	39.3% (22/56)	NR None
Martynowicz et al., 2019 [45] Poland Cross-Sectional	To assess the relationship between SB and OSA.	N=110 (M=66, F=44) 51.02 \pm 14.19	Patients with age between 18 and 90 years with clinical suspicion of OSA who went to Department and Clinic of Internal Diseases, Occupational Diseases, Hypertension, and Clinical Oncology at the Wrocław Medical University between March 2017	Apnea: the absence of airflow for ≥ 10 s. Hypopnea: was defined as a reduction in the amplitude of breathing by $\geq 30\%$ for ≥ 10 s with a $\geq 3\%$ decline in blood oxygen saturation or arousal.	For the consideration of SB, EMG bursts should not be separated by >3 s to be considered part of the same episode, and EMG activity had to be at least twice the amplitude of the background EMG.	The relationship between OSA and SB depends on the degree of severity of OSA. From the results of the present study, mild-to-moderate OSA is associated with SB in the group of patients with increased risk of OSA. Mann-Whitney U test, chi-square test and correlation.	Mild OSA: 61.6% (20/33) Moderate: 64.3% (18/28) Severe: 35.3% (12/34)	This research received no external funding. The authors declare no conflict of interest.

			and March 2019.					
Okeson et al., 1991 [47]	To determine if OSA patients experience more bruxing events than a non-OSA patients.	N=24 (male) SDB = 57 ± 11.5 years Non-SDB = 57 ± 11.7 years	Subjects who were referred to the University of Kentucky Laboratory for evaluation of OSA.	Apnea: was defined as cessation of air flow at the nose and mouth for 10 s or longer during sleep. Hypopnea: was defined as a 50% reduction in the thermocouple signal amplitude associated with a 4% fall in oxygen saturation. The number of apneas and hypopneas were combined and recorded as SDB events.	A clench was defined as activity of the masseter muscle exceeding 40% of the maximum clench of the muscle and lasting for 2 seconds or longer.	The SDB group had a total of 193 bruxing events, an average of 16.1 per subject (range 0 to 43, SD 11.6). The control group had a total of 314 bruxing events. There was an average of 26.2 bruxing events per subject (range 0 to 57, SD 23.9). There were no statistical differences. Student's t-test		NR NR
Phillips et al., 1986 [42]	To determine the relationship between OSA and parafunctional activity.	N=24 (M=21, F=3). OSA:14 Non-OSA:10 OSA: 52 ± 15.9 years Non-OSA:	Patients referred to the University of Kentucky Sleep Apnea Laboratory during the four-month period between July and October, 1985 for	Apnea: was defined as cessation of airflow at the nose and mouth for 10 s or longer during sleep. Hypopnea: was defined as a reduction in airflow associated with a	A clench was defined as activity of the masseter muscle exceeding 40% of the maximum clench of that muscle and lasting for two seconds or longer.	Nocturnal clenching was slightly higher in patients with OSA than those without (12.2 vs 7.6 p=0.18), and there was a correlation between the clench index and AHI by linear regression (r = 0.49, p<0.05). There were		NR NR

		50.2 ± 16.4 years	evaluation of possible OSA.	4% fall in oxygen saturation.		significant falls in both the AHI (64.4 ± 28.8 vs 36.5 ± 36.7, p=0.02) and clench index (12.5 ± 12.1 vs 7.0 ± 8.6, p = 0.04) in the lateral decubitus vs supine sleeping positions. NR		
Saito et al., 2014 [36] Japan Descriptive	To investigate the temporal association between SB events and OSA events.	N=10 (male) 46.7 ± 11.5 years	Subjects with confirmed OSA and SB.	Apnea-hypopnea event: American Academy of Sleep Medicine	SB: Events were identified as RMMA. The amplitude was set twice the baseline activity.	In patients with concomitant OSA and SB, most SB events occurred after OSA events, suggesting that SB events occurring close to OSA events is a secondary form of SB.		NR No conflict of interest declared.
Saito et al., 2016 [32] Japan Cross-Sectional	To investigate, in a population reporting awareness of both OSA and SB, the associations between each specific breathing and jaw muscle event.	N=59 (M=47, F=12) 44.8 ± 10.8 years	Japanese patients reporting awareness of breathing cessation and tooth grinding as well as signs and symptoms of OSA and SB.	Apnea and hypopnea events were scored according to standard criteria, with an AHI threshold of 5 events/h or more.	RMMA/SB Episode/h>2 and/or RMMA/SB bursts/h>25.	AHI did not show a significant correlation with RMMA/SB episodes nor with RMMA/SB bursts. Sleep arousals in patients with concomitant SB and OSA are not strongly associated with onset of RMMA/SB.	50.84% (30/59)	No No

						Spearman correlation test.		
Sjoholm et al., 2000 [35]	To test the hypothesis of a direct association between SDB and SB.	N=21 (M=19, F=2) 40.0 ± 29.2 years	Patients with OSA.	Apnea: was defined as the cessation of airflow for at least 10 s. Hypopneas: were defined as a decrease of more than 50% in thoracoabdominal amplitude for at least 10 s.	(1) Subjective estimation: teeth grinding, or clenching was reported by the patient one to two nights or more per week. (2) Clinical: the number and extent of visible wear facets on tooth enamel (attrition), the presence of masticatory muscle fatigue, and/or discomfort of the temporomandibular joint. (3) Masseter EMG: If the arbitrary cut-off point of 2.5 rhythmic jaw-movement episodes per hour was exceeded, the participant was considered to be as a bruxer during that night.	Masseter contraction episodes were associated with the termination of apnea or hypopnea episodes in 3.5% of the mild group and 14.4% of the moderate group (p<0.05). It appears that SB is rarely directly associated with apneic events. Student's t-test	47.61% (10/21) Mild OSA 54% (6/11) Moderate OSA 40% (4/10)	NR NR
Canada								
Cross-Sectional								

Smardz et al., 2020 [43] Poland Cross- Sectional	To evaluate the relationship between SDB and SB.	N=77 (M=21, F=56) SB:58 Non- SB:19 34.8 ± 10.8 years	Patients above 18 years age, with a positive diagnosis of probable SB of the Clinic of Prosthetic Dentistry operating in the Department of Prosthetic Dentistry at Wrocław Medical University.	Apnea: defined as the absence of airflow through the airway for more than 10 s. Hypopnea: has been defined as a decrease in respiratory amplitude by more than 30% for more than 10 s, followed by subsequent blood desaturation of more than 3% or subsequent arousal.	Episodes qualified as bruxism if there was a RMMA, often accompanied by grinding sounds and characteristic movements in the mandible occurring after a minimum of 3s break from the last muscle activity. Episodes were classified as phasic (lasting 0.25 to 2s), tonic (lasting more than 2s), or mixed.	Both groups (SB and Non-SB) not differ statistically significantly in terms of oxygen desaturation index (U=540.0, p= 0.90) and AHI (U = 531.5, p = 0.82). Quantitative analysis showed a lack of a statistically significant relationship between AHI and ODI in the SB and Non-SB patients. Mann-Whitney U test		Authors have no competing interests to declare. This study was co-financed by financial resources for Young Researchers of Wrocław Medical University, Poland (STM.B022.17.011). The funding source was not involved in the study design, in the collection, analysis and interpretation of data, in the writing of the report, and in the decision to submit the article for publication.
Tan et al., 2019 [16] Singapore Cross- Sectional	To determine the prevalence of SB in adult OSA patients, to assess the association between SB and OSA in terms of sleep macrostructure and respiratory parameters and to determine possible OSA risk factors for SB.	N=147 (M=100, F=47) SB: 49 (M=37, F=12) Non-SB: 98 (M=63, F=35)	Patients diagnosed with mild, moderate, or severe OSA and aged 25 years and above who underwent a PSG at the Ng Teng Fong General Hospital sleep clinic from	Apnea: was determined as a cessation in airflow of 90% for a minimum period of 10 s. Hypopnea: was identified when the airflow dropped by 30% for a period of 10 s, accompanied by an oxygen desaturation of	SB episodes were established when masseter RMMA exhibited twice the background EMG amplitude and were preceded by a period of > 3 second of stable background EMG.	Of the 147 OSA patients, 49 (33.3%; 95% CI: 25.7 to 40.9%) were diagnosed with SB, while 98 (66.7; 95% CI: 59.1% to 74.3%) had no SB (descriptive analysis). An association was found between AHI and SB group. SB patients had	33.33 % (49/147)	NR No Conflict of interest.

			July 2015 to February 2016.	3%. AHI: indicated the severity of OSA through the representation of number of apnea/hypopnea events per hour of sleep. Mild OSA was defined as having an AHI of 5 or more and less than 14 respiratory events per hour, moderate OSA between 15 or more and less than 30 events per hour, and severe OSA as 30 or more events per hour.		significantly higher Respiratory <i>Arousal Index</i> and Oxygen Desaturation Index than non-SB patients. This study demonstrated that SB occurs in about one-third of OSA patients. Mann-Whitney U test.		
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Legend: AHI: Apnea-Hypopnea Index; CI: Confidence Interval; EMG: Electromyography; F: Female; M: Male; NR: Not Reported; OSA: Obstructive Sleep Apnea; PSG: Polysomnography; RMMA: Rhythmic Masticatory Muscle Activity; SB: Sleep Bruxism; SAHOS: Obstructive sleep apnea-hypopnea syndrome.

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

Author, Year	Objective	Sample Sex (M/F)	Inclusion Criteria	OSA Diagnoses Criteria	SB Criteria	Findings	Prevalence SB/OSA concomitant	Report on the sources of funding
Country		Mean age or orange	OSA and SB Assessment			Statistical Analysis		Conflict of Interest
Castilho et al., 2020 [48]	To study the incidence of mouth breathing and its association with sleep disorders, dental caries, malocclusion and deleterious oral habits, in children.	152 (M=80, F=72)	Children of three schools with different socioeconomic indicators, chosen by the Education Department of the Municipality of Botucatu/SP, Brazil during the period of May 2016 to November 2017. OSA: OSA-18 SB: Questionnaire and clinical evaluation	The result of the OSA-18 questionnaire is given by the sum of the values chosen by the person interviewed for the frequency with which the cited events occur. The numerical value can be translated as low (<60 points), moderate (≥60 points and <80 points) or high (≥80 points) risk for OSAS by the child.	NR	Children who had a low and moderate risk for OSAS (OSA-1 and OSA-2) had 39% of bruxism. In 29 patients with severe risk for OSAS (OSA-3), the proportions were 67%. A descriptive statistical analysis of the data with frequency and percentage for the qualitative variables. Chi-square or Fisher's exact test was performed when necessary.	40.78 % (62/152) OSA mild and moderate: 39% (54/138) OSA severe 67% (8/14)	NR NR
Brazil		6-9 years						
Cross-Sectional								
Ferreira et al., 2015 [49]	To evaluate the prevalence and to test for possible associations	496 (M= 249, F= 247)	Children of preschool age (3–6 years old) who live in the city of Taubate.	Those participants who had a Mallampati score of III or	Clinical examinations evaluated the presence of wear facets on	There is an association between SB and OSAS in children. An association	2.82 % (14/496)	The authors would like to thank CNPq (a Brazilian governmental
Brazil		4.49 ± 1.04 years						

Cross-Sectional	between SB and OSA.		<p>OSA: clinical examinations and application of a questionnaire to be answered by the parents, based on a modified version of the Mallampati questionnaire.</p> <p>SB: clinical examinations and questionnaire to be answered by the parents.</p>	IV and whose parents answered 'Yes' for all the questions on the OSAS questionnaire were diagnosed with sleep apnea.	canines and incisors where the worn borders of the teeth fit the wear facet of the antagonist tooth during excursive movements.	<p>was found between the presence of SB and OSA, where 11.03% of subjects with SB also exhibit OSA, and 97% of subjects without SB did not present OSA. SB was associated with OSA.</p> <p>Descriptive statistics, and possible associations were tested using a Chi-square test. ANOVA was used to compare the average age among children who were and were not diagnosed with SB and OSA.</p>	<p>research agency) and the University of Taubate' for the grant received by the first author (NMRF) to develop this study (PIBIC #072/12).</p> <p>The authors deny any conflict of interest.</p>
Goyal et al., 2018 [51] India Cross-Sectional	To estimate the prevalence of OSA in school children aged 5–10 years and its association with academic performance.	1346 (M=836, F= 510) NR 5–10 years	Children aged between 5 and 10 years at three purposively selected schools of Bhopal, India, from July	OSA: SRBD > 33%.	NR	Students with positive SRBD had higher chances of having bruxism (29% vs. 15.4%; P <0.0001, adjusted	No There are no conflicts of interest.

			2015 to November 2015. OSA: Validated 22-item pediatrics sleep-related breathing disorder (SRBD). SB: Questionnaire.			OR: 1.7; 95% CI: 1.1–2.6). Logistic regression analysis		
Gregório et al., 2008 [50] Brazil Cross-Sectional	To investigate the symptoms most frequently found in children with a PSG diagnosis of OSA.	38 (M=19, F=19) ** 3 children who were non-apneic. 8.4 ± 3.99 years	Children consecutively referred to the sleep laboratory with suspicion of OSAHS between June of 2003 and December of 2004. OSA: PSG SB: Pre-sleep questionnaire	OSA: was defined as a decrease of at least 50% of the baseline flow associated with the desaturation of 4% or more and/or micro-arousals. AHI: was calculated based on the number of obstructive apneas and hypopnea events occurring during one hour of sleep. The adopted classification were: normal	NR	In children with OSA, bruxism was seen in 31.3%. All the children diagnosed with severe OSA also presented bruxism. Student's t-test or Mann-Whitney test	31.3 % (11/35)	NR NR

				with an AHI < 1; mild with an AHI between 1 and 5; moderate when the AHI is between 5 and 10 and serious when the AHI is >10.				
Segu et al., 2020 [52] Italy Cross-Sectional	To assess such correlations in a large sample of school children between SB and sleep disorders.	741 (M=409, F=332) 11.26 ± 4.05 years	A group of 741 consecutive children of a private orthodontic practice between January 2016 and May 2019. OSA and SB: Clinical evaluation and questionnaire Sleep Disturbance Scale for Children.	NR	NR	The Spearman test reported a significant correlation between parental-reported tooth grinding and sleep apnea (r=0.092). There is a significant correlation between parental-reported tooth grinding and OSA. Spearman test		NR The authors declare that there are no conflicts of interest.
Sheldon SH, 2010 [54] United States Cross-Sectional	To evaluate the presence of SB noted on comprehensive polysomnography in 119 consecutive patients with possible OSA.	119 (NR) 7.0 ± 4.0 years	Patients between ages 3 and 16 years, referred to the Pediatric Sleep Medicine Center with symptoms of snoring.	OSA: was scored when a greater than 90% decrease was present in the signal amplitude for 90% or greater of the entire	Bruxism was defined as three or more rhythmic contractions of the temporalis muscles, as measured with temporalis muscle	Sleep-related rhythmic temporalis muscle activity associated with arousal is significantly associated with indices of respiratory disturbance,		NR NR

			<p>OSA and SB: PSG</p>	<p>respiratory event compared with pre-event baseline amplitude.</p> <p>Hypopnea: was scored if the event was associated with a 50% or greater decline in the amplitude of the nasal pressure for at least two respiratory efforts, the fall in nasal pressure lasted 90% or more of the entire respiratory event compared with the amplitude preceding the event, and the event was associated with an arousal, awakening, or 3% or greater oxygen desaturation.</p>	<p>electromyogram (EMG), occurring during NREM or REM sleep lasting more than 3 seconds, but less than 15 seconds.</p>	<p>particular the Arousal Index, and AHI, as measured using standard pediatric polysomnographic techniques.</p> <p>Mann-Whitney.</p>	
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<p>Singh N, 2011 [55]</p> <p>United States</p> <p>Cross-Sectional</p>	<p>To investigate whether sleep bruxism-related tooth wear could be a clinical marker for pediatric OSA.</p>	<p>50 (M=25, F=25)</p> <p>7.6±1.5 years</p>	<p>Pediatric subjects were recruited for this study from a pediatric sleep disorder center and a private dental practice.</p> <p>OSA: PSG SB: Dental wear score.</p>	<p>OSA was measured utilizing the AHI. The AHI scored the OSA events, and was used to classify the subjects into those with OSA and those with no OSA (controls; AHI<1).</p>	<p>Score 1 = no dental wear, or obvious wear of enamel or wear through the enamel to the dentin in single spots.</p> <p>Score 2 = wear of the dentin up to one-third of the crown height</p> <p>Score 3 = wear of the dentin more than one-third of the crown height.</p>	<p>The results revealed no statistically significant association between both the presence and severity of OSA and the presence and severity of sleep bruxism related dental wear. SB related dental wear is not a clinical indicator of pediatric OSA.</p> <p>Descriptive</p>		<p>Dr. Nischal Singh is NOT receiving any financial sponsorship or remuneration to conduct this study.</p> <p>There is no conflict of interest that would compromise his position or this research study.</p>
<p>Tachibana et al., 2016 [53]</p> <p>Japan</p> <p>Cross-Sectional</p>	<p>To investigate the prevalence of sleep bruxism in children in Japan, and its relationships with sleep-related factors and daytime problematic behavior.</p>	<p>6023 (M=2975, F=3048)</p> <p>NR</p>	<p>2191 preschoolers and 3832 elementary school students from Japan were subject to analysis.</p> <p>OSA and SB: Japanese Sleep Questionnaire</p>	<p>Higher scores indicated greater signs of sleep disorders or deleterious sleep habits.</p>	<p>SB Does he/she grind his/her teeth during sleep by rating on a 6-point intensity Likert scale.</p>	<p>Sleep bruxism significantly correlated with OSA.</p> <p>Logistic regression</p>	<p>21% (1263/6023)</p>	<p>Fundings from the Challenge to Intractable Oral Diseases and the Grant-in-Aid for Scientific Research and from the Center of Innovation Science and Technology based Radical Innovation and Entrepreneurship Program.</p>

								None to declare.
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Legend: AHI: Apnea-Hypopnea Index; F: female; M: male; NR: Not Reported; OSA: Obstructive Sleep Apnea; PSG: Polysomnography; SB: Sleep Bruxism; SDB: Sleep-disordered breathing.

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

	Study	SB Mean ± SD	Non-SB Mean ± SD	p value
Oxygen Desaturation Index (ODI)	Hosoya et al., 2014	33.7 ± 25.0	22.4 ± 19.3	<0.05*
	Smardz et al., 2020	5.09 ± 8.24	3.28 ± 3.22	0.90
	Tan et al., 2018	35.05 ± 24.75	26.1 ± 28.65	0.005*
Sleep Efficiency (SE)	Kim et al., 2020	86.2 ± 14.1	81.5 ± 14.7	0.14
	Hosoya et al., 2014	16.7 ± 8.6	17.6 ± 8.6	>0.05
	Tan et al., 2018	85.20 ± 12.40	85.49 ± 13.01	0.83
Arousal Index (AI)	Kim et al., 2020	6.9 ± 9.3	21.0 ± 26.3	0.13
	Hosoya et al., 2014	40.0 ± 18.8	31.3 ± 15.9	>0.05
	Tan et al., 2018	49.92 ± 18.05	43.44 ± 21.07	0.03*
Minimum Oxygen Saturation (minOSAT)	Kim et al., 2020	83.8 ± 8.5	80.4 ± 9.6	0.26
	Smardz et al., 2020	88.90 ± 6.34	88.16 ± 9.39	0.93
	Tan et al., 2018	78.47 ± 10.21	81.47 ± 10.76	0.04*
Apnea-Hypopnea Index (AHI)	Kim et al., 2020	25.5 ± 16.6	45.1 ± 28.2	0.03*
	Hosoya et al., 2014	37.2 ± 22.9	27.0 ± 19.7	<0.05*
	Smardz et al., 2020	5.52 ± 9.40	3.49 ± 3.55	0.82

Table 5. Summary of suggestions for future studies.

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

5 ARTIGO 2

Artigo a ser submetido após defesa, nas normas do *Journal SLEEP*.

Sleep bruxism and obstructive sleep apnea: is there a pattern of occurrence of events between them?

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Abstract

Study Objectives: To investigate the prevalence of sleep bruxism (SB) in obstructive sleep apnea patients and the patterns of occurrence between SB episodes and apnea-hypopnea events in a polysomnographic (PSG) analysis.

Methods: A descriptive study in which participants were submitted to PSG exam in a sleep laboratory was done. From 95 individuals, sixty-two had their PSGs eligible for evaluation. Both SB and OSA were detected by the one-night PSG. The occurrence patterns were analyzed within a 2-min time window: T1 - the interval between apnea-hypopnea events (AHE) termination and SB episode onset (SB episode after AHE), and T2 - the interval between SB episode termination and AHE onset (SB episode before AHE). An overall average of T1 and T2 was obtained and was statistically compared using paired t-test. Five occurrence patterns between SB episode and AHE was tested (ANOVA repeated measure). Statistical significance was determined at $p < 0.05$.

Results: Out of 62 participants, 61 presented OSA. The prevalence of SB in the population studied was 74.2%. There was no statistical difference between the occurrence of SB episode after $15.36 (\pm 12.52)$ seconds or before apneic events $13.92 (\pm 11.04)$, $p = 0.51$. Most SB episodes (47.7%) occurred without relationship with AHE.

Conclusions: The prevalence of SB in OSA patients was very high. We did not find a pattern of occurrence between SB episodes and AHE. The most SB episodes occurred isolated, that is means without relationship with AHE.

Keywords: sleep bruxism, obstructive sleep apnea, sleep disorders, polysomnography

Statement of Significance

The identification of a pattern of occurrence between sleep bruxism and obstructive sleep apnea could be helpful for dentists and sleep physicians to better understand if there is a synergistic mechanism between these two conditions and, perhaps, establish a better management treatment plan. This study did not find a pattern of occurrence between sleep bruxism and obstructive sleep apnea.

INTRODUCTION

Sleep bruxism (SB) has been described as a “masticatory muscle activity during sleep that is characterized as rhythmic or non-rhythmic”. Moreover, this activity is not considered a movement or sleep disorder in otherwise healthy individuals¹. The prevalence of SB in adults based on clinical investigation is around 12.8% ($\pm 3.1\%$)², while when it is detected by polysomnography (PSG) reached 7.4%³. Sleep bruxism may be associated with undesirable consequences such as facial muscle pain⁴, decreased bite force⁵, implant complications^{6,7}, poor sleep quality and lower oral-related quality of life^{8,9}, therefore the importance of its early and adequate detection.

The SB detection can be performed by non-instrumental approaches (questionnaires, oral history, clinical inspection) associated or not to instrumental approaches (electromyographic recordings). SB is categorized as “possible” when is recognized based on a positive self-report only; “probable” based on a positive clinical inspection, with or without a positive self-report; and “definitive” based on a positive instrumental assessment, with or without a positive self-report and/or a positive clinical inspection. Therefore, the standard reference for SB detection is based on positive instrumental approaches as electromyography (EMG) or PSG¹.

Obstructive sleep apnea (OSA) is a sleep-related disorder characterized by a collapse of the upper airways in the setting of continued respiratory effort, leading to airflow cessation and arterial oxygen desaturation, often terminated in an arousal¹⁰. In adults, the OSA is classified by its severity: severe OSA means that the apnea-hypopnea index (AHI) is greater than 30 episodes per hour; it is considered moderate OSA when the AHI is between 15 and 30, and mild OSA when the AHI is between 5 and 15 episodes per hour.

An association between SB and OSA has been previously hypothesized¹¹⁻¹³. The hypothesis was based on the understanding that both SB and OSA are related to sleep arousal episodes, i.e. micro-awakenings featuring physiological activations¹³. Four systematic reviews¹⁴⁻¹⁷ on the

subject have been published with inconclusive scientific evidence to establish a clear relation between them. Other hypotheses are also reported, regarding the temporality of the occurrence of SB and OSA events^{11,18,19}. One study reports the possible hypotheses related to the temporality of the two conditions, which would be: (1) the two phenomena are unrelated; (2) the onset of the OSA event precedes the onset of the SB event within a limited time span, with SB having a potential OSA-protective role; (3) the onset of the SB event precedes the onset of the OSA event within a limited time span, with SB having an OSA-inducing effect; and (4) the onset of the OSA and SB event occurs at the same moment¹².

The evidence on the temporal relationship between SB and OSA is scarce and controversial^{11,18,19}. As recommended by a recent scoping review, the new studies should focus their analysis not only on identifying a relationship between SB and OSA, but to investigate the temporal association of the episodes²⁰. The identification of a pattern of occurrence between SB and OSA could be helpful for dentists and sleep physicians to better understand if there is a synergistic mechanism between these two conditions and, perhaps, establish a better management plan²⁰.

Thus, the aims for this article are: first, to describe the prevalence of SB in a population with OSA. Second, to analyze the patterns of occurrence between SB episodes and apnea-hypopnea events.

METHODS

Ethical Considerations

The development of this study was approved by the Human Research Ethics Committee of the Federal University of Santa Catarina, under number 84783518.6.0000.0121. The study was conducted following the Helsinki Declaration of 1964¹³.

Study Design

This is an observational descriptive study reported following the “Strengthening the Reporting of Observational Studies in Epidemiology” (STROBE)¹² (Appendix 1).

Setting and Participants

Previously, a pilot study was carried out with 10 patients, to test the research procedures. With no change in the evaluated outcomes, these patients were subsequently incorporated into the final sample. A convenience sample was composed of patients suspected of sleep-disordered breathing consecutively referred to Baía Sul Hospital - a private hospital located in Florianópolis, Brazil, for an overnight PSG. Participants were invited to participate in the research before the conduction of the PSG exam.

Inclusion Criteria

Adult patients (>18 years old) scheduled for PSG between January 2019 and October 2019.

Exclusion Criteria

- Patients with neurological or movement impairment in general health conditions and PSG exams that did not have good signal quality for interpretation.

- **Variables / Data sources / Measurement**

Demographic and anthropometric variables

Demographic and anthropometric data including age, sex, and body mass index (BMI) were recorded. The data collection related to the characteristics of the patients was carried out, on the same night of the PSG exam, using an electronic questionnaire (Google Docs) accessed via internet on a tablet (Ipad Air 2, Apple). The patient self filled the information on the tablet. A calibrated digital scale (Digital Glass G-Tech, ACCUMED, China) was used to measure the patient's weight in kilograms. The measure was performed and noted by a dentist responsible for data collection on the day of PSG exam. The same scale was used for all patients. A tape measure was used to measure the patient's height. For measurement the patient was standing

with arms extended along the body and spine straight; eyes at a fixed point and head at 90° from the floor; heels and knees with a point of contact between them and the buttocks against the wall. In the same way, the responsible for this measure was a dentist. The BMI (ratio in which weight is divided by height squared) was calculated by BMI formula in Microsoft® Excel 16.29.1 (Microsoft Office 2019, Microsoft, Redmond, United States).

Polysomnography exam

A single overnight sleep was conducted in a dark and quiet hospital room using conventional PSG testing equipment (Alice III; Respironics, Andover, MA, USA), following the American Academy of Sleep Medicine (AASM)²¹. An experienced sleep medicine technician led the calibration of the instruments and performed an adequate recognition of the signs. The participants were instructed, before the beginning of the records, to perform voluntary leg movement, opening, closing and moving the eyes, teeth clenching, mandibular laterality, mandibular protrusion, as well as swallowing and coughing movements to record baseline amplitudes. Sleep recordings were registered with surface electrodes.

The electrodes were inserted by the same trained technician blinded to the research objectives in all participants. Three electroencephalograms (EEG) channels: (F4-M1, C4-M1, O2-M1) and other three backup leads (F3-M2, C3-M2, O1-M2; 2). Two electrooculography (EOG) channels: E1-M2 leads (E1 placed 1 cm below and 1 cm lateral to the distal end of the left eye and E2-M2 placed 1 cm below and 1 cm lateral to the distal end of the right eye). Three electrodes for EMG: central position 1 cm above the lower edge of the mandible, another 2 cm below the lower edge of the mandible 2 cm to the right of the midline and another in the same position, but to the left of medium line; Leg EMG with one electrode on each leg in the anterior tibial region to be recorded in just one channel. To detect bruxism, two electrodes were placed in the masseter region bilaterally. The variables collected during the polysomnographic

examination included airflow (through a nasal/oral thermistor), nasal pressure transducer, thoracic and abdominal movement for monitoring respiratory effort.

Obstructive Sleep Apnea diagnosis

A single trained sleep medicine specialist performed the PSG analysis. The OSA diagnosis was done exclusively based on AHI values. An apnea event was determined when there was an interruption in airflow $\geq 90\%$ for a minimum of 10 seconds. Mild OSA was defined as when the AHI is between 5 and 15 episodes per hour; moderate OSA, between more than 15 episodes and less than 30 events per hour; and severe OSA, as more than 30 events per hour²¹.

Sleep Bruxism Assessment

The SB events were estimated through the activity of the masseter muscle, assessed through EMG. Events separated by 3-second intervals were considered episodes of SB when following one of the patterns: 1) tonic (at least one masseteric EMG shot greater than 2 seconds); 2) phasic (three or more shots of masseteric EMG lasting between 0.25 and 2 seconds); or 3) mixed (both types of triggers). SB scoring rubrics were adapted from AASM 2016 manual to score sleep and associated events^{15,16}. Patients were diagnosed with SB when they demonstrated more than two bruxism episodes per hour of sleep². Sleep bruxism analyses were performed by another blinded evaluator who underwent specific training to analyze SB and who did not participate in the data collection.

Prevalence of Sleep Bruxism in patients with Obstructive Sleep Apnea

The prevalence of SB in patients with OSA was calculated by dividing the number of patients who had SB in the PSG exam, by the number of patients with OSA.

Patterns of Occurrence between SB episodes and Apnea-Hypopnea events

The patterns of occurrence between both conditions were obtained in two analyses. In the first analysis, the difference in mean duration of occurrence between apnea-hypopnea events (AHE) and SB episode was verified. A trained dentist performed the manual analysis of the recordings

of PSG and registered the interval periods. Two time periods were evaluated: (A) the interval between the end of the AHE and the beginning of the SB episode, called T1 (SB episode after AHE) (Figure 1A); and (B) the interval between the end of the SB episode and the beginning of the AHE, termed T2 (SB episode before AHE) (Figure 1B). The SB episodes were used as a reference, and from them, the closest AHE was marked before and after each SB episode. The analysis was scored within 2 minutes (min), 1-min after the SB episode and 1-min before SB episode. The mean distributions of the two intervals were calculated for all participants, and later an overall average of T1 and T2 was obtained.

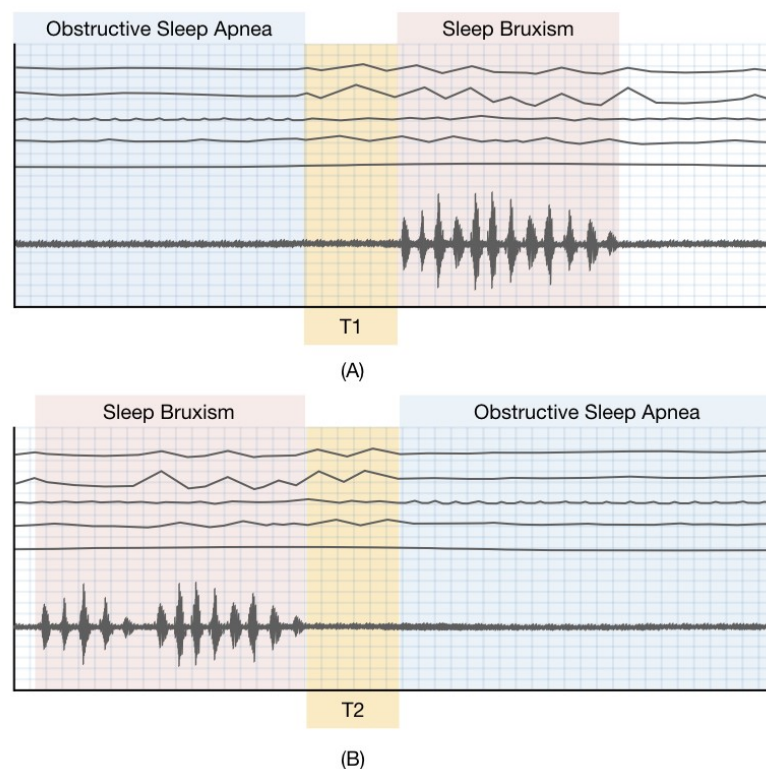


Figure 1. Mean time between apnea-hypopnea event (AHE) and sleep bruxism (SB) episode within a 2-minute scoring window. Where, T1 = time interval between the end of the AHE and the beginning of the SB (A), and T2 = time interval between end of SB episode and start AHE (B).

In the second analysis five temporal patterns occurrences were evaluated (Figure 2), where SB episodes were used as a reference:

- 1) Isolated SB episode: only SB episode in the 2-min window;
- 2) Antecedent (AHE before SB episode): AHE that occurred before the SB episode in the 2-min window;
- 3) Subsequent (AHE after SB episode): AHE that occurred after the SB episode in the 2-min window;
- 4) Antecedent + Subsequent (AHE before and after SB episode): AHE that occurred before and after the SB episode in the 2-min window;
- 5) Concomitant (AHE and SB concomitant): episodes of both conditions that occur concurrently.

Each temporal relationship was counted for each patient and at the end, an overall mean was obtained.

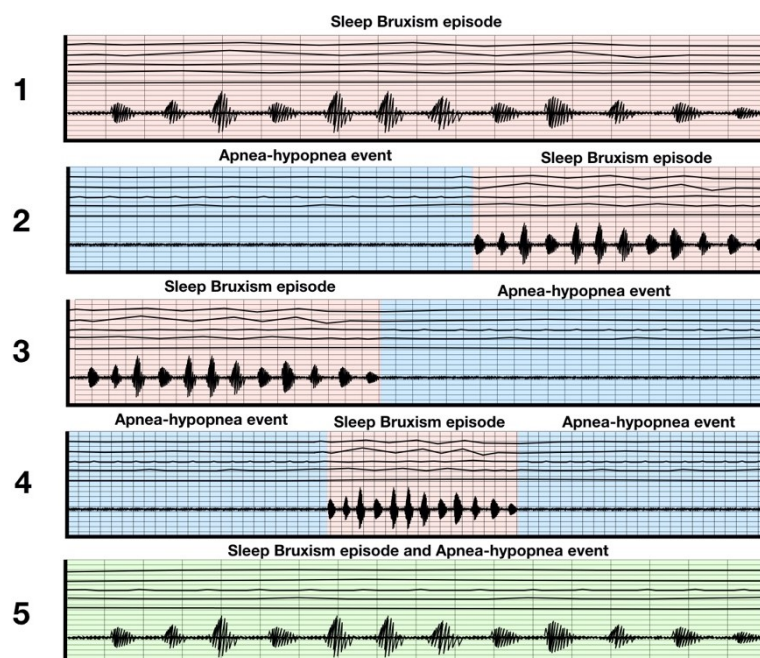


Figure 2. Occurrence patterns between apnea-hypopnea event (AHE) and sleep bruxism (SB) episode within a 2-minute scoring window. (1) Isolated SB episode; (2) Antecedent (AHE

before SB episode); (3) Subsequent (AHE after SB episode); (4) Antecedent + Subsequent (AHE before and after SB episode); (5) Concomitant (AHE and SB concomitant).

Bias

The main bias in the study that should be mentioned is the selection bias because it is a convenience sample.

Statistical methods

The statistical analysis was performed using the statistical software Jamovi (Version 1.8.4.0). T-test was used for independent variables (age, BMI, AHI). For the first analysis, it was used a paired t-test, and T1 and T2 within the 2-min windows were graphically expressed. Previously, the Kolmogorov-Smirnov test was performed to test the normality of the distribution of these variables. For analysis of occurrence patterns, the Kolmogorov Smirnov test was performed to test the normality and repeated measures ANOVA (non-parametric) was done. The Durbin-Conover was used as post-Hoc test. Statistical significance was determined at $p < 0.05$.

RESULTS

Participants

The study flow chart is shown in Figure 3. Ninety-five patients underwent PSG, and they all accepted to participate in the research. Data from 33 participants could not be evaluated all completely due to the bad signal quality of the PSG exam. Hence, the final sample was composed of 62 participants.

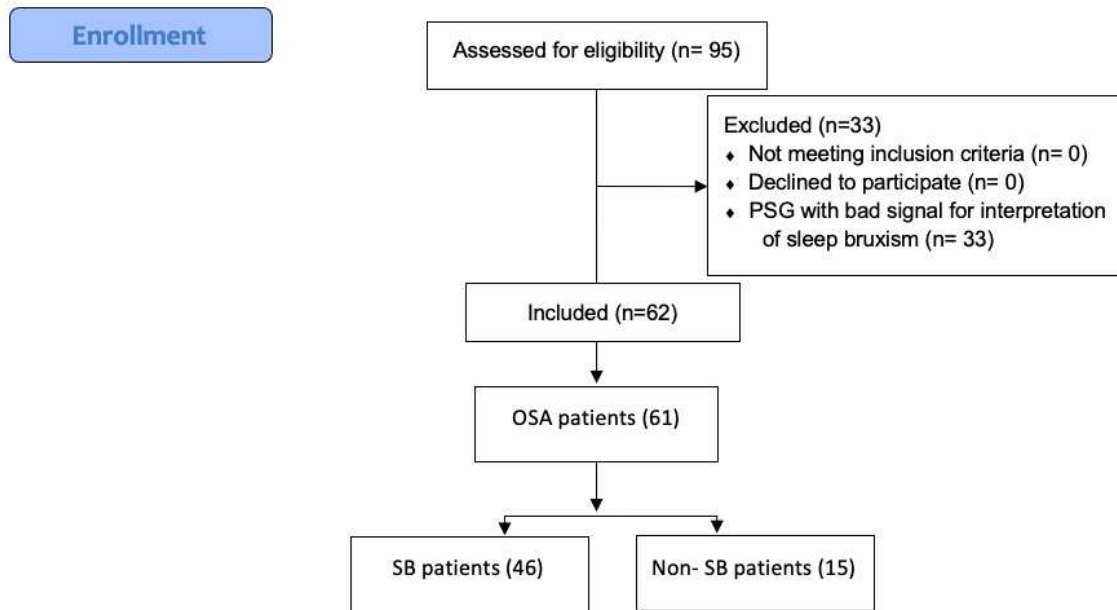


Figure 3. Flowchart of study participants adapted from CONSORT (2010).

Legend: SB: Sleep Bruxism; OSA: Obstructive Sleep Apnea

Descriptive and data outcome

The demographic and anthropometric characteristics of the participants with and without SB can be seen in Table 1. Of 62 participants, 30 are females, 32 males, with age mean (46.7 ± 14.3), BMI mean (29.7 ± 5.32) and AHI mean (106.0 ± 87.1).

Obstructive sleep apnea was detected in 61 patients (98.4%). Definitive SB was detected in 46 patients with OSA (75.4%). There was no difference in the mean age of patients with SB (46.8 ± 15.1) and without SB (46.6 ± 11.9), ($p=0.95$; t-test for independent variables). Moreover, there was no difference in AHI events ($p=0.41$) and BMI ($p= 0.33$) in the patients with and without SB (Table 1). 75.4% of the sample presented both conditions (SB and OSA).

A total of 1,361 episodes of SB were detected. During non-rapid eye movement (NREM) sleep 1,209 occurred, 27.2% ($n=370$) in stage N1, 55.6% ($n=757$) in stage N2 and 6.0% ($n=82$) in stage N3. During the rapid eye movement (REM) occurred 11.2% ($n=152$) of SB episodes (Figure 4).

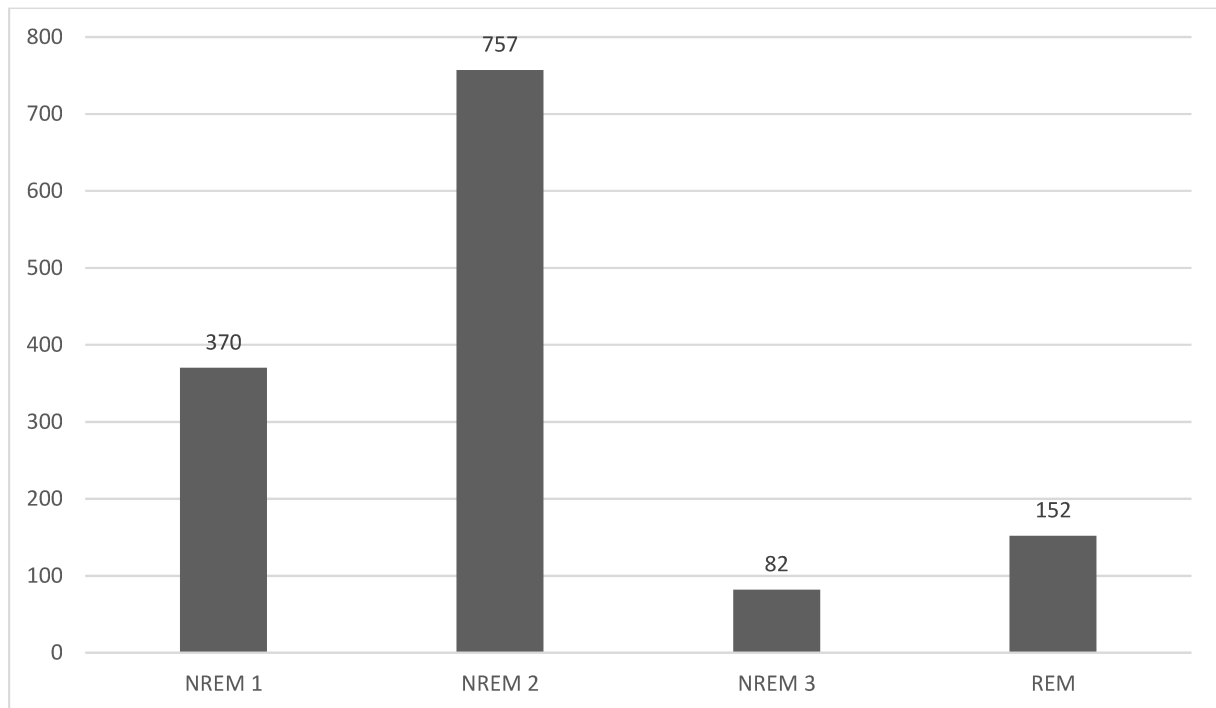


Figure 4. Barr graphic representation of the distribution of number of sleep bruxism episodes in relation to sleep phase. Legend: REM: rapid eye movement; NREM: non-rapid eye movement.

Main results

Prevalence of SB in OSA patients

The prevalence of SB in OSA patients was 75.4%, of 61 patients diagnosed with OSA, 46 had definitive SB.

Patterns of Occurrence between SB episodes and Apnea-Hypopnea events

In the 2-minutes window observed, the mean interval for T1 (SB episode after AHE) was 15.36 (± 12.52) seconds, and for T2 was 13.92 (± 11.04) (SB episode before AHE) seconds. There was no statistical difference ($p=0.51$).

Most of SB episodes ($n=650$ - 47.7%) occurred without a temporal relationship with AHE. Considering temporally related events, with SB episode as reference, the AHE events occurred

as antecedent, subsequent, antecedent + subsequent and concomitant and in (n=142 - 10.5%), (n= 273 - 20%), (n=231 - 17%) and (n= 65 - 4.8%) of the cases, respectively (Figure 5).

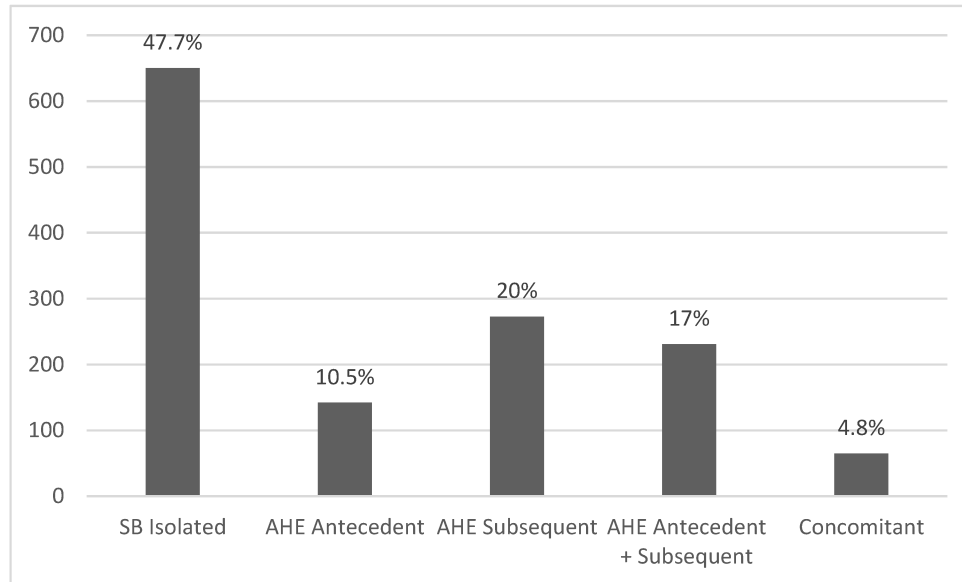


Figure 5. Distribution of sleep bruxism (SB) episodes according to 5 patterns of occurrence. (1) SB episode isolated; (2) AHE antecedent (AHE before SB episode); (3) Subsequent (AHE after SB episode); (4) Antecedent + Subsequent (AHE before and after SB episode); (5) Concomitant (AHE and SB concomitant).

Most groups showed statistical difference between themselves, $p < 0.05$. The only group that showed no statistical difference was between the groups of AHE antecedent to SB episode and AHE antecedent + subsequent (Table 3).

DISCUSSION

The prevalence of SB in OSA patients was high. This study did not identify a pattern of occurrence between SB episodes and apnea-hypopnea events. Also, most episodes of SB occur without a previous or later AHE. The relationship between the two conditions has been studied since 1986²², due to the belief that SB would be an activity induced by the central nervous system and that the same system partially originates OSA. Over time, primary studies were

conducted in view of the great interest in this subject²³⁻²⁶. In addition, four systematic reviews were conducted, seeking to verify a possible association between SB and OSA¹⁴⁻¹⁷. However, the results showed insufficient scientific evidence to show beyond doubt such an association. SB appears to be highly prevalent in the OSA population. In the present study, 75.4% of the participants with OSA also had SB. A recent systematic review¹⁷ reported that the prevalence of SB in adult patients with OSA ranges from 26% to 53.7%. These values are much higher than that in the general population from the State of São Paulo, Brazil which reported a prevalence of 7.4% of SB assessed by PSG³. Despite the high prevalence of the two concomitant conditions, an association between them is still controversial. In this study, an assessment of the association between the two conditions could not be performed, as this is a population mostly with OSA. Regarding the association, there are studies that failed to detect an association between SB and OSA^{19,23-27}. However, it differs from other studies, in which also used PSG as the definitive method for the detection of SB and found association between the two conditions.^{22,29} A possible explanation for these inconsistent results is the variability in the methodology of these studies²⁰. Or that, the association could be related to specific SB phenotypes, for example, being the association more specifically found in the tonic type of SB²⁵.

Although most studies focused their efforts on analyzing the association between OSA and SB^{19,23-27}, an analysis of the occurrence patterns between both conditions was still lacking. Some authors have considered SB a protective factor for OSA^{1,12,28}. Their hypothesis is that when SB episodes immediately follow the OSA episodes, this occurrence may be viewed to quickly re-establish the air passage when the patient is undergoing obstructive apnea. However, the literature presents contradictory results^{11,19}. Sjöholm et al¹⁹ did not find statistical difference in the occurrence of SB episodes being before or after apneic events. The percentage of masseter contraction episodes which ceased at the end of the apnea was only 3.5% in the mild

OSA group and 14.4% in the moderate group¹⁹. Whereas, in the study of Saito et al¹¹, a significantly higher percentage of SB episodes (54.9%) showed the T1 pattern (AHE before SB episode) than the T2 pattern (SB episode to AHE). A recent study that used the cardiorespiratory polygraphy to detect OSA and SB, investigated the correlation between SB-related masseter muscle activity and found no correlation between AHI and SB¹⁸. The same study showed that most of SB episodes (66.8%) occurred without a temporal relationship with AHEs, in agreement with the present study, that presents a larger sample¹⁸.

It was possible to notice differences in the time window observed in these studies between episodes. The Sjöholm et al¹⁹ did not mention the observed time window, Saito et al.¹¹, observed a 5-min window, while Colonna et al., observed a 5 seconds time window.

In this study, a previous analysis was performed, adopting a 5-min window before and after SB episodes. The average occurrence between episodes of OSA and SB was around 1-min, therefore, for the analysis of the temporal relationship and the occurrence of episodes, the 1-min window was used as a reference.

Regarding demographic and anthropometric variables, age and BMI were not associated with SB, demonstrating that SB does not necessarily cover a population with specific characteristics. The interest in studying the patterns of occurrence is related to the importance of multidisciplinary management of patients with respiratory disorders. A significant portion of patients with OSA are not adequately diagnosed and therefore do not receive appropriate treatment³³. Various clinical signs of respiratory disorders such as complaints of breath sounds reported by the sleeping partner, snoring, frequent daytime sleepiness, presence of hypertension, wide neck, retrognathia, deep palate, and large tonsils could be identified by the dentist³⁴. In addition, the dentist often has more contact with patients due to the duration of treatment and regular visits. Thus, by identifying potential patients, the dentist could

intermediate and refer the patient to an appointment with a sleep physician for a more appropriate diagnosis and management³⁵.

One of the study's strengths is the use of a SB detection method considered a reference standard^{1,36}. There is a dearth of studies using PSG to detect SB, since the high cost, time investment, and limited technologic access.³⁷ Another important point to be mentioned is that no other temporal evaluation study took into account that AHEs can happen before and after the SB episode, in the evaluated time window. For the main outcome of this research (temporal relationship), our study presents the biggest sample of current literature (n=62)^{11,18,19}. The previous studies published on the same topic evaluated 21 PSGs¹⁹; 10 PSGs¹¹, and the most recent study included 30 cardiorespiratory polygraphy analyses¹⁸.

Despite the limitations, it can be suggested that the study has internal validity due to the relative absence of systematic errors. As for the external validity, these results can be applied to a similar population, that is, adults around 47 years old, suspected sleep disorders.

CONCLUSION

Three quarters of patients with OSA have SB. No occurrence patterns were found between SB and OSA. The most SB episodes in the PSG exam occurred without relationship between beginning or ending OSA events.

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Disclosure Statement

The authors declare that they have no conflict of interest.

Authors' Contributions

PP, JD, HP, JCR, CM, HMH, IM, CFM, GDLC actively took part in the conceptualization and preparation of this manuscript. PP, CM and HMH performed the analysis of data. PP drafted a first version the manuscript. All co-authors revised and approval the final version of the manuscript.

Figure Legends

Figure 1. Temporal relationship between apnea-hypopnea event (AHE) and sleep bruxism (SB) episode within a 2-minute scoring window. Where, T1 = time interval between the end of the AHE and the beginning of the SB (A), and T2 = time interval between end of SB episode and start AHE (B).

Figure 2. Occurrence patterns between apnea-hypopnea event (AHE) and sleep bruxism (SB) episode within a 2-minute scoring window. (1) Isolated SB episode; (2) Antecedent (AHE before SB episode); (3) Subsequent (AHE after SB episode); (4) Antecedent + Subsequent (AHE before and after SB episode); (5) Concomitant (AHE and SB concomitant).

Figure 3. Flowchart of study participants adapted from CONSORT (2010).

Figure 4. Barr graphic representation of the distribution of number of sleep bruxism episodes in relation to sleep phase.

REFERENCES

1. 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(11):837-844.
2. Manfredini D, Winocur E, Guarda-Nardini L, Paesani D, Lobbezoo F. Epidemiology of Bruxism in Adults: A Systematic Review of the Literature. *Journal of Orofacial Pain.* 2013;27(2):99-110.
3. 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(7 Suppl):97S-103S.
4. Stuginski-Barbosa J, Porporatti AL, Costa YM, Svensson P, Conti PC. Agreement of the International Classification of Sleep Disorders Criteria with polysomnography for sleep bruxism diagnosis: A preliminary study. *J Prosthet Dent.* 2017;117(1):61-66.
5. Palinkas M, Bataglioni C, de Luca Canto G, et al. Impact of sleep bruxism on masseter and temporalis muscles and bite force. *Cranio.* 2016;34(5):309-315.
6. Chrcanovic BR, Kisch J, Albrektsson T, Wennerberg A. Factors influencing the fracture of dental implants. *Clin Implant Dent Relat Res.* 2018;20(1):58-67.
7. Chrcanovic BR, Kisch J, Larsson C. Analysis of technical complications and risk factors for failure of combined tooth-implant-supported fixed dental prostheses. *Clin Implant Dent Relat Res.* 2020;22(4):523-532.
8. Duarte J, Pauletto P, Massignan C, et al. Association Between Sleep Bruxism and Quality of Life: A Systematic Review. *J Oral Facial Pain Headache.* 2020;34(4):341-352.
9. Camara-Souza MB, de Figueredo OMC, Rodrigues Garcia RCM. Association of sleep bruxism with oral health-related quality of life and sleep quality. *Clin Oral Investig.* 2019;23(1):245-251.
10. Guilleminault C, Tilkian A, Dement WC. The sleep apnea syndromes. *Annual review of medicine.* 1976;27(1):465-484.
11. Saito M, Yamaguchi T, Mikami S, et al. Temporal association between sleep apnea-hypopnea and sleep bruxism events. *J Sleep Res.* 2013.
12. Manfredini D, Guarda-Nardini L, Marchese-Ragona R, Lobbezoo F. Theories on possible temporal relationships between sleep bruxism and obstructive sleep apnea events. An expert opinion. *Sleep Breath.* 2015;19(4):1459-1465.
13. Lavigne GJ KT, Kolta A, Sessle BJ. Neurobiological Mechanisms Involved in Sleep bruxism. *Critical Reviews in Oral Biology & Medicine.* 2013;14(1):30-46.
14. De Luca Canto G, Singh V, Gozal D, Major PW, Flores-Mir C. Sleep bruxism and sleep-disordered breathing: a systematic review. *J Oral Facial Pain Headache.* 2014;28(4):299-305.
15. da Costa Lopes AJ, Cunha TCA, Monteiro MCM, Serra-Negra JM, Cabral LC, Junior PCS. Is there an association between sleep bruxism and obstructive sleep apnea syndrome? A systematic review. *Sleep Breath.* 2020;24(3):913-921.
16. Jokubauskas L, Baltrusaityte A. Relationship between obstructive sleep apnoea syndrome and sleep bruxism: a systematic review. *J Oral Rehabil.* 2017;44(2):144-153.
17. Kuang B, Li D, Lobbezoo F, et al. Associations between sleep bruxism and other sleep-related disorders in adults: A systematic review. *Sleep Medicine.* 2022;89:31-47.
18. Colonna A, Cerritelli L, Lombardo L, et al. Temporal relationship between sleep-time masseter muscle activity and apnea-hypopnea events: A pilot study. *J Oral Rehabil.* 2022;49(1):47-53.

19. Sjöholm TT, Lowe AA, Miyamoto K, Fleetham JA, Ryan CF. Sleep bruxism in patients with sleep-disordered breathing. *Archives of Oral Biology*. 2000;45:889-896.
20. Pauletto P, Polmann H, Conti Reus J, et al. Sleep bruxism and obstructive sleep apnea: association, causality or spurious finding? A scoping review. *Sleep*. 2022;45(7).
21. Richard B. Berry RB, Charlene E. Gamaldo, Susan M. Harding, Robin M. Lloyd, Carole L. Marcus, and Bradley V. Vaughn. The AASM Manual for the Scoring of Sleep and Associated Events - Rules, Terminology and Technical Specifications. Scoring Manual Version 2.2. In: American Academy of Sleep Medicine; 2015.
22. Phillips BA, Okeson J, Paesani D, Gilmore R. Effect of sleep position on sleep apnea and parafunctional activity. *Chest*. 1986;90(3):424-429.
23. Jeffrey P. Okeson BAP, David T.R. Berry, Yvette R. Cook, John F. Cabelka,. Nocturnal Bruxing Events in Subjects With Sleep-Disordered Breathing and Control Subjects. *Journal of Craniomandibular Disorders Facial & Oral Pain*. 1991;5:258-264.
24. Maluly M, Dal Fabbro C, Andersen ML, Herrero Babiloni A, Lavigne GJ, Tufik S. Sleep bruxism and its associations with insomnia and OSA in the general population of Sao Paulo. *Sleep Med*. 2020;75:141-148.
25. 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;68:131-137.
26. 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(2):703-709.
27. de Holanda TA, Castagno CD, Barbon FJ, Costa YM, Goettems ML, Boscato N. Sleep architecture and factors associated with sleep bruxism diagnosis scored by polysomnography recordings: A case-control study. *Arch Oral Biol*. 2020;112:104685.
28. Manfredini D, Ahlberg J, Lobbezoo F. Bruxism definition: Past, present, and future - What should a prosthodontist know? *J Prosthet Dent*. 2021.
29. 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(4):837-844.
30. Jordan AS, McSharry DG, Malhotra A. Adult obstructive sleep apnoea. *The Lancet*. 2014;383(9918):736-747.
31. Rezaie L, Maazinezhad S, Fogelberg DJ, Khazaie H, Sadeghi-Bahmani D, Brand S. Compared to Individuals with Mild to Moderate Obstructive Sleep Apnea (OSA), Individuals with Severe OSA Had Higher BMI and Respiratory-Disturbance Scores. *Life (Basel)*. 2021;11(5).
32. 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(10).
33. Park JG, Ramar K, Olson EJ. Updates on definition, consequences, and management of obstructive sleep apnea. *Mayo Clin Proc*. 2011;86(6):549-554; quiz 554-545.
34. Lavigne G, Khoury S, Abe S, Yamaguchi T, Raphael K. Bruxism physiology and pathology: an overview for clinicians. *Journal of oral rehabilitation*. 2008;35(7):476-494.
35. Herrero Babiloni A, Beetz G, Dal Fabbro C, et al. Dental sleep medicine: Time to incorporate sleep apnoea education in the dental curriculum. *Eur J Dent Educ*. 2020;24(3):605-610.
36. Casett E, Reus JC, Stuginski-Barbosa J, et al. Validity of different tools to assess sleep bruxism: a meta-analysis. *J Oral Rehabil*. 2017;44(9):722-734.

37. Raphael KG, Sirois DA, Janal MN, et al. Sleep bruxism and myofascial temporomandibular disorders: a laboratory-based polysomnographic investigation. *The Journal of the American Dental Association*. 2012;143(11):1223-1231.
38. Saczuk K, Lapinska B, Wilmont P, Pawlak L, Lukomska-Szymanska M. The Bruxoff Device as a Screening Method for Sleep Bruxism in Dental Practice. *J Clin Med*. 2019;8(7).
39. Lavigne G, Kato T, Herrero Babiloni A, et al. Research routes on improved sleep bruxism metrics: Toward a standardised approach. *J Sleep Res*. 2021:e13320.
40. 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(4):e13066.

Table 1. Descriptive characteristics of the sample with and without sleep bruxism (SB) (n=62).

	SB	No SB	P-value
Female	21	9	
Male	25	7	
Total	46	16	0.47
Age (mean)	46.8 ± 15.1	46.6 ± 11.9	0.95
BMI (mean)	29.3 ± 5.35	30.8 ± 5.26	0.33
AHI (mean)	111.0 ± 91.0	90.3 ± 78.0	0.41

Legend: Sleep Bruxism (SB); Body Mass Index (BMI); Apnea-Hypopnea Index (AHI).

Table 2. Mean, Standard Deviation (SD), Median and Interquartile Range (IQR) of sleep bruxism (SB) episodes according to occurrence patterns.

Occurrence Patterns	SB episodes	SB episodes
	Mean (SD)	Median (IQR)
Isolated	16.3 (15.8) a	10.5 (12.5) a
AHE antecedent	3.55 (4.28) b	2.0 (4.25) b
AHE subsequent	6.83 (9.54) c	4.0 (6.0) c
AHE antecedent + subsequent	5.78 (9.11) b	3.0 (6.5) b
Concomitant	1.63 (3.87) d	0.5 (1.25) d

Legend: Sleep Bruxism (SB); Apnea-hypopnea event (AHE). Equals letters in columns mean no statistical difference.

6 CONSIDERAÇÕES FINAIS

Esta tese foi composta de dois estudos com delineamento diferentes, buscando responder a mesma questão de pesquisa: existe associação entre bruxismo do sono e apneia obstrutiva do sono?

Com o primeiro estudo, a revisão de escopo, foi possível conhecer os mais de 30 anos de pesquisas que buscaram entender a relação entre BS e AOS. Pode-se observar uma grande variabilidade nos métodos de pesquisa, gerando variabilidade também nos resultados. O objetivo da revisão de escopo foi mapear essa literatura, identificar *gaps*, e sobretudo entender como a pesquisa sobre este tema foi desenvolvida até agora e sugerir melhorias nas pesquisas futuras. Com um mapeamento exaustivo da literatura, e uma equipe de destaque internacional na área, novamente, não foi possível confirmar a relação entre BS e AOS em adultos. Em pacientes pediátricos, embora essa associação pareça plausível, atualmente ainda não há evidências suficientes para afirmar ou refutar a associação, levando em consideração a variabilidade dos métodos de detecção tanto de BS como da AOS. Na revisão de escopo não foi possível obter uma evidência sólida em relação a questão da temporalidade das duas condições devido poucos estudos disponíveis sem concordância nos resultados. Porém foi possível deixar algumas recomendações para pesquisas futuras: (1) investigação da relação temporal entre BS e AOS e não apenas a relação entre as duas condições; (2) que o objetivo e as hipóteses sejam específicas para verificar a associação de BS e AOS; (3) que o estudo apresente uma amostra suficiente para apoiar a comparação estatística, controle de variáveis que podem atuar como confundidoras, uso de metodologia e interpretação do diagnóstico de acordo com padrões reconhecidos; (4) coleta de informações baseadas em questionários sobre o histórico médico dos pacientes e, quando possível, usando questionários validados; (5) sempre que possível avaliar o papel do fenótipo não anatômico para BS e AOS.

Na sequência, realizamos um estudo observacional descritivo. Com base no estudo observacional descritivo, não foi possível constatar um padrão de ocorrência entre os episódios de BS e eventos de AOS. A maioria dos episódios de BS aconteceram de forma isolada, sem um padrão de ocorrência de episódios de apneia antes ou depois.

Os estudos mais recentes têm apresentado bases mais sólidas e metodologias mais consistentes, em relação ao padrão de detecção de ambas as condições. Novos estudos na área analisando o padrão temporal de ocorrência entre as duas condições, com amostras mais amplas complementarão a análise da hipótese e beneficiarão pacientes, bem como profissionais da área médica e odontológica no diagnóstico e tratamento de ambas as condições.

REFERÊNCIAS

- ACADEMIA AMERICANA DE MEDICINA DO SONO. **Obstructive Sleep Apnea**. 2008. Disponível em: <https://aasm.org/resources/factsheets/sleepapnea.pdf>. Acesso em: 10 set. 2022.
- BERTAZZO-SILVEIRA, Eduardo et al. Association between sleep bruxism and alcohol, caffeine, tobacco, and drug abuse: A systematic review. **The Journal of the American Dental Association**, v. 147, n. 11, p. 859-866. e4, 2016.
- CASETT, E. et al. Validity of different tools to assess sleep bruxism: a meta-analysis. **Journal of oral rehabilitation**, v. 44, n. 9, p. 722-734, 2017.
- CASTROFLORIO, Tommaso et al. Sleep bruxism and related risk factors in adults: A systematic literature review. **Archives of oral biology**, v. 83, p. 25-32, 2017.
- COLONNA, Anna et al. Temporal relationship between sleep-time masseter muscle activity and apnea-hypopnea events: A pilot study. **Journal of Oral Rehabilitation**, v. 49, n. 1, p. 47-53, 2022.
- DA COSTA LOPES, Ana Júlia et al. Is there an association between sleep bruxism and obstructive sleep apnea syndrome? A systematic review. **Sleep and Breathing**, v. 24, n. 3, p. 913-921, 2020.
- DE HOLANDA, Thiago Azario et al. Sleep architecture and factors associated with sleep bruxism diagnosis scored by polysomnography recordings: A case-control study. **Archives of Oral Biology**, v. 112, p. 104685, 2020.
- DE LUCA CANTO, Graziela et al. Sleep bruxism and sleep-disordered breathing: a systematic review. **Journal of Oral & Facial Pain & Headache**, v. 28, n. 4, 2014.
- DE SOUZA, Francinete Silva et al. Síndrome da apneia e hipopneia obstrutiva do sono e principais comorbidades associadas. **Revista de Ciências Médicas**, v. 29, p. 1-9, 2020.
- HOSOYA, Hisashi et al. Relationship between sleep bruxism and sleep respiratory events in patients with obstructive sleep apnea syndrome. **Sleep and Breathing**, v. 18, n. 4, p. 837-844, 2014.
- HOZ AIZPURUA, José Luis de la et al. Sleep bruxism. Conceptual review and update. **Medicina Oral, Patología Oral Y Cirugía Bucal**. v.16, n.2, p.231-238, 2011.
- JOHANSSON, Anders; OMAR, Ridwaan; CARLSSON, Gunnar E. Bruxism and prosthetic treatment: a critical review. **Journal of prosthodontic research**, v. 55, n. 3, p. 127-136, 2011.
- JOKUBAUSKAS, Laurynas; BALTRUŠAITYTĖ, Aušra. Relationship between obstructive sleep apnoea syndrome and sleep bruxism: a systematic review. **Journal of oral rehabilitation**, v. 44, n. 2, p. 144-153, 2017.

KHOURY, Samar et al. Sleep bruxism-tooth grinding prevalence, characteristics and familial aggregation: a large cross-sectional survey and polysomnographic validation. **Sleep**, v. 39, n. 11, p. 2049-2056, 2016.

KIM, Dong Hyun; LEE, Sang Hwa; LEE, Sang Haak. Sleep bruxism episodes in patients with obstructive sleep apnea syndrome determined by in-laboratory polysomnography. **Applied Sciences**, v. 10, n. 23, p. 8587, 2020.

KUANG, Boyuan et al. Associations between sleep bruxism and other sleep-related disorders in adults: A systematic review. **Sleep Medicine**, v. 89, p. 31-47, 2022.

LAVIGNE, G. J. et al. Critical issues in dental and medical management of obstructive sleep apnea. **Journal of dental research**, v. 99, n. 1, p. 26-35, 2020.

LOBBEZOO, Frank et al. International consensus on the assessment of bruxism: Report of a work in progress. **Journal of oral rehabilitation**, v. 45, n. 11, p. 837-844, 2018.

MALULY, M. et al. Sleep bruxism and its associations with insomnia and OSA in the general population of Sao Paulo. **Sleep Medicine**, v. 75, p. 141-148, 2020.

MALULY, M. et al. Polysomnographic study of the prevalence of sleep bruxism in a population sample. **Journal of dental research**, v. 92, n. 7_suppl, p. S97-S103, 2013.

MANFREDINI, Daniele et al. Theories on possible temporal relationships between sleep bruxism and obstructive sleep apnea events. An expert opinion. **Sleep and Breathing**, v. 19, n. 4, p. 1459-1465, 2015.

MANFREDINI, Daniele et al. Epidemiology of bruxism in adults: a systematic review of the literature. **J Orofac Pain**, v. 27, n. 2, p. 99-110, 2013.

MARTYNOWICZ, Helena et al. The relationship between sleep bruxism and obstructive sleep apnea based on polysomnographic findings. **Journal of clinical medicine**, v. 8, n. 10, p. 1653, 2019.

MASSAHUD, Maria Letícia de Barros et al. Association between sleep bruxism, use of antidepressants, and obstructive sleep apnea syndrome: A cross-sectional study. **Journal of Oral Rehabilitation**, v. 49, n. 5, p. 505-513, 2022.

MELO, Gilberto et al. Bruxism: an umbrella review of systematic reviews. **Journal of oral rehabilitation**, v. 46, n. 7, p. 666-690, 2019.

OKESON, Jeffrey P. et al. Nocturnal bruxing events in subjects with sleep-disordered breathing and control subjects. **Journal of Craniomandibular Disorders**, v. 5, n. 4, 1991.

PALINKAS, Marcelo et al. Impact of sleep bruxism on masseter and temporalis muscles and bite force. **CRANIO®**, v. 34, n. 5, p. 309-315, 2016.

PAULETTO, Patrícia et al. Sleep bruxism and obstructive sleep apnea: association, causality or spurious finding? A scoping review. **Sleep**, 2022.

PHILLIPS, Barbara A. et al. Effect of sleep position on sleep apnea and parafunctional activity. **Chest**, v. 90, n. 3, p. 424-429, 1986.

SAITO, Miku et al. Weak association between sleep bruxism and obstructive sleep apnea. A sleep laboratory study. **Sleep and Breathing**, v. 20, n. 2, p. 703-709, 2016.

SAITO, Miku et al. Temporal association between sleep apnea–hypopnea and sleep bruxism events. **Journal of sleep research**, v. 23, n. 2, p. 196-203, 2014.

SENARATNA, Chamara V. et al. Prevalence of obstructive sleep apnea in the general population: a systematic review. **Sleep medicine reviews**, v. 34, p. 70-81, 2017.

SJÖHOLM, T. T. et al. Sleep bruxism in patients with sleep-disordered breathing. **Archives of oral biology**, v. 45, n. 10, p. 889-896, 2000.

SMARDZ, Joanna 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 Medicine**, v. 68, p. 131-137, 2020.

TAN WAN YONG, Madeleine et al. Prevalence of Sleep Bruxism and Its Association with Obstructive Sleep Apnea in Adult Patients: A Retrospective Polysomnographic Investigation. **Journal of Oral & Facial Pain & Headache**, v. 33, n. 3, 2019.

WETSELAAR, Peter et al. Associations between tooth wear and dental sleep disorders: a narrative overview. **Journal of oral rehabilitation**, v. 46, n. 8, p. 765-775, 2019.

APÊNDICE A

MEMORIAL DE ATIVIDADES ACADÊMICAS

Nasci aos 19 dias do mês de julho de 1990, em uma pequena cidade do interior, localizada na serra gaúcha, chamada Nova Bassano. Uma cidade de pouco mais de oito mil habitantes, de colonização italiana, pequena e aconchegante. Meus pais e meus padrinhos sempre me contam que estava nevando no dia em que saí do hospital, talvez venha daí todo meu encanto pela neve.

Sou filha de Donatila Trevisan Pauletto (*in memoriam*) e Alcírio Pauletto, os dois seres humanos mais incríveis que já conheci. Simples, honestos, trabalhadores, sempre me ensinaram valores dos quais eu me orgulho e desejo transmitir aos meus filhos, o dia que vier a tê-los. Tenho dois irmãos mais velhos, Gláucia (*in memoriam*) e Diego. Minha mãe com muito esforço concluiu o Ensino Médio e se formou técnica em contabilidade, meu pai estudou até o quinto ano primário. Eles trabalhavam duro, inicialmente em um bar-restaurant e depois com uma padaria. Minha mãe era muito famosa na cidade pelos seus doces, salgados e bolos. Apesar de não terem muita instrução na educação, eles sempre incentivaram seus filhos a estudar. Minha irmã formou-se em Direito e meu irmão em Odontologia. Eu desde pequena acompanhei a jornada intensa de trabalho dos meus pais. De dia, frequentava a Creche Criança Feliz no meu município e à noite ficava com eles no estabelecimento, por vezes dormia ali mesmo, em uma caminha improvisada de sacos de farinha. Depois de um tempo, iniciei meus estudos no Colégio

Estadual Padre Colbachini, onde sempre fui uma boa aluna, muito dedicada e minha matéria favorita era português. Durante o período do ensino fundamental eu também participava da Escolinha Municipal de vôlei, um esporte que amo até hoje e dançava no Centro de Tradições Gaúchas (CTG). No CTG, além da dança eu participava de concursos de declamação e também tive a alegria de ser Primeira Prenda Dente de Leite, Primeira Prenda Mirim e Segunda Prenda Mirim da Região. Meus pais sempre me incentivavam a participar de atividades extras, e isso fez com que eu pudesse me desenvolver, perder a timidez e viver sempre ao redor de muitos amigos. Eu dividia meu tempo de estudos, brincando de algumas profissões: florista, bancária, cantora e professora. Eu e minha amiga vizinha, vivíamos para cima e para baixo, com livros, cadernos, dando aula para nossos alunos imaginários, fazendo chamada, escrevendo com giz na mesa de futebol de botão do meu irmão. Sem dúvida era minha brincadeira favorita.

Já com 15 anos de idade, na época do Ensino Médio, passei a estudar em um colégio particular em uma cidade vizinha, com uma bolsa de estudos. Era um colégio católico, chamado Sagrado Coração e eu estudava no período da noite. Ao findar o ensino médio, a preocupação com que carreira que seguir tomou conta. Eu não tinha certeza do que eu queria ser na minha vida, a única certeza que eu tinha era de que eu queria estudar em uma universidade pública. Inicialmente a aprovação na pública não aconteceu. O primeiro vestibular que fui aprovada foi para psicologia na Universidade de Caxias do Sul. Animada para iniciar minha vida acadêmica, iniciei meus estudos, no entanto, depois de seis meses, a dúvida voltou a rondar e eu decidi voltar para minha cidade. Fiquei um tempo trabalhando em um banco da minha cidade, mas com meu pai sempre dizendo para eu escolher um curso, porque eu tinha que estudar.

Fiz um ano de curso pré-vestibular, com intuito de amadurecer melhor qual carreira seguir e também me preparar para uma universidade pública. Foi um ano de muita dedicação e esforço que renderam frutos. Eu ainda estava em dúvida de qual caminho seguir: Psicologia ou Odontologia (naquele período eu também fiquei ajudando meu irmão na clínica, e confesso que o ato dele devolver sorriso às pessoas, chamou muito a minha atenção). No final daquele ano prestei vestibular para várias universidades públicas e tive a alegria de ser chamada para compor o quadro de alunos de Odontologia da Universidade Federal de Santa Catarina (UFSC), isso foi em 2010. No segundo semestre de 2010, iniciei meus estudos na UFSC, onde obtive o grau de Cirurgiã-Dentista, cinco anos depois. Um fato curioso aconteceu justamente no meu primeiro dia de aula de Anatomia com o Professor Wilson Pacheco entrando na sala com um macromodelo de dente. Meus olhos brilharam e vi no professor Pacheco o sentimento de alegria e paixão por ensinar, que eu também sentia em dar aula para meus alunos imaginários.

Durante a graduação, sempre estive envolvida em projetos de pesquisa e extensão, já visando uma possível carreira acadêmica. E não deu outra, fiz minha inscrição para o Mestrado em Implantodontia da UFSC. Fui aprovada, e dei início à Pós-Graduação, dois dias após a minha formatura. O Mestrado (2015- 2017) foi um período de muito aprendizado e desafios. Além da vida acadêmica que estava construindo, sem estar nos meus planos, recebi o presente mais lindo que o Mestrado poderia me dar, meu colega de mestrado, meu companheiro de vida, meu esposo, meu amor, Edwin Ruales Carrera. Apesar dos obstáculos, eu estava certa do meu propósito, ser professora universitária. Em conjunto com o mestrado eu também fazia especialização em Implantodontia. Após o fim do Mestrado, eu fiquei um ano trabalhando em uma clínica privada, quando senti falta do meio acadêmico. Resolvi voltar para UFSC para fazer meu Doutorado (2018/02), agora na área de Clínicas Odontológicas. Tive o prazer de ser orientada pela Professora Graziela De Luca Canto, que muito aprendi não somente sobre

metodologia, disfunção temporomandibular e bruxismo, mas sobre trabalho em equipe, pontualidade, prazos, dedicação. Certamente vou levar para sempre os ensinamentos obtidos neste período acadêmico.

O período do Doutorado foi muito enriquecedor, além de cursar as disciplinas, trabalhava com pesquisas de revisões sistemáticas, desenvolvia um estudo observacional, participava das reuniões semanais do grupo de pesquisa, cursos de revisão sistemática. Ainda, tive a oportunidade de atuar como professora nos cursos de revisões sistemáticas e escrita científica, compartilhar conhecimento com pesquisadores de muitas universidades brasileiras de forma remota. Ainda, participei de vários cursos de capacitação, publiquei 19 artigos científicos e escrevi quatro capítulos de livros. Também tive a linda oportunidade de ser aluna visitante na Universidade de Zurique, na Suíça, uma experiência que me fez crescer muito como profissional e como pessoa.

Entre 2018 e 2022 fui aprovada em alguns processos seletivos para professora substituta da UFSC. Em agosto de 2022, fui chamada para assumir o cargo, no entanto, meus planos haviam mudado um pouco e eu já estava morando no Equador. Atualmente sou Professora da disciplina de Oclusão e Clínica III da Universidad de Las Américas em Quito, no Equador. A seguir, uma pequena descrição das atividades realizadas durante o Doutorado (2018-2022).

Capacitações e Atividades Complementares

- ITI *Academy* campus UFSC, Universidade Federal de Santa Catarina, Brasil. 2022.
- Aluna visitante Universidade de Zurique, Suíça, Clínica de Odontologia Reconstructiva (2021);
- Bioestatística Essencial (40 h). Universidade de São Paulo, USP, Brasil. 2021;
- Curso de Meta-Análise de Estudos Observacionais (12 h). Universidade Federal de Santa Catarina, UFSC, Brasil. 2021;
- Curso de Meta-Análise de Estudos de Intervenção (8 h). Universidade Federal de Santa Catarina, UFSC, Brasil. 2021;
- Curso Intensivo de Revisão Sistemática (40 h). Universidade Federal de Santa Catarina, UFSC, Brasil. 2021;
- Curso de Redação e Publicação de Artigos Científicos (30 h). Universidade Federal de Santa Catarina, UFSC, Brasil. 2021;

- Curso Intensivo de Revisão Sistemática (45 h). Universidade Federal de Santa Catarina, UFSC, Brasil. 2021;
- Semana de Imersão em Disfunção Temporomandibular e Bruxismo (15h). Universidade Federal de Santa Catarina, UFSC, Brasil. 2021;
- I Curso Avançado de Avaliação de Risco de Viés e Qualidade da Evidência (16h). HT Educacional, Brasil. 2020;
- Materiais e Soluções Estéticas em Odontologia Digital (2h). Congresso Internacional de Odontologia de São Paulo, CIOSP, Brasil. 2019 – 2019;
- Research Connect (21h). Conselho Britânico, BC, Inglaterra. 2019;
- Curso de Revisão Sistemática Avançado (30h). Universidade Federal de Santa Catarina, UFSC, Brasil. 2019;
- Curso de Meta-Análise de Estudos de Intervenção (19 h). Universidade Federal de Santa Catarina, UFSC, Brasil. 2019;
- VI Curso de Revisão Sistemática e Meta-análise: Estudos Observacionais (8 horas). HTANALYZE, Brasil; 2019
- Capacitação em Disfunção Temporomandibular e Bruxismo CEMDOR UFSC. (Carga Horária: 36h). Universidade Federal de Santa Catarina, UFSC, Brasil. 2021-2022;
- Mini Residência em Harmonização Orofacial. (Carga Horária: 96h). Instituto Lucila Largura, ILL, Brasil. 2021-2022;
- Aperfeiçoamento em Prótese Dentária. (Carga Horária: 160h). Centro Universitário Avantis, UNIAVAN. 2018;
- Integrante do Centro de Dor Orofacial da UFSC (2020);
- Integrante do Grupo de Pesquisa em Prótese Digital (2019–2020).

Artigos Científicos publicados em Revistas Internacionais

1. Da Paz RLP, De Oliveira JM, Pauletto P, de Andrade EM, Guerra ENS, Massigan C, De Luca Canto G. Worldwide prevalence of geographic tongue in adults: a systematic review and meta-analysis. *Oral Diseases*. 2022 Oct 8.
2. Denardin AC, do Nascimento LP, Valesan LF, Da Cas CD, Pauletto P, Garanhani RR, Januzzi E, Hilgert LA, De Souza BD. Disocclusion guides in occlusal splints on

temporomandibular disorders and sleep bruxism: A systematic review. *Oral Surgery, Oral Medicine, Oral Pathology and Oral Radiology*. 2022 Jul 23.

3. Gaio DC, Sebastiani AM, Meger MN, Duarte J, Polmann H, Pauletto P, Réus JC, Juliana Feltrin de Souza, de Souza AV, Machado-Souza C, Maia IS, De Luca Canto G, João Armando Brancher JA. Association between genetic polymorphisms in the Melatonin Receptor Type 1 A gene and sleep bruxism. *Archives of Oral Biology*. 2022.

4. Pauletto P, Polmann H, Réus JC, de Oliveira JM, Chaves D, Lehmkuhl K, Massignan C, Stefani CM, Martins CC, Flores-Mir C, De Luca Canto G. Critical appraisal of systematic reviews of intervention in dentistry published between 2019-2020 using the AMSTAR 2 tool. *Evidence-Based Dentistry*. 2022 Sep 14:1-8.

5. Pauletto P, Polmann H, Conti Réus J, Massignan C, de Souza BD, Gozal D, Lavigne G, Flores-Mir C, De Luca Canto G. Sleep bruxism and obstructive sleep apnea: association, causality or spurious finding? A scoping review. *Sleep*. p. 1-7, 2022.

6. Oliveira JM, Butini L, Pauletto P, Lehmkuhl KM, Stefani CM, Bolan M, Guerra E, Dick B; De Luca Canto G, Massignan C. Mental health effects prevalence in children and adolescents during the COVID-19 pandemic: A systematic review. *Worldviews on Evidence-Based Nursing*, v. 19, p. 130-137, 2022.

7. Honnelf LR, Pauletto P, Réus JC, Massignan C, Michelotti A, Flores-Mir C, De Luca Canto G. Effects of Stabilization Splints on the Signs and Symptoms of Temporomandibular Disorders of Muscular Origin: A Systematic Review. *Cranio-The Journal of Craniomandibular Practice*, v. 1, p. 1-12, 2022.

8. Badaró MM, Marin DOM, Pauletto P, Gonçalves TMSV, Porporatti AL, De Luca Canto G. Failures in Single Extra-Short Implants (≤ 6 mm): A Systematic Review and Meta-analysis. *International Journal of Oral & Maxillofacial Implants* v. 36, p. 669-689, 2021.

9. Meller J, Pauletto P, Werlich MO, Massignan C, Lehmkuhl KM, Porfirio GJM, Hallal ALC, De Luca Canto G. Prevalence of orofacial injuries in wheeled non-motor sports athletes: A systematic review and meta-analysis. *Dental Traumatology*, p. 546-556, 2021.
10. Pauletto P, Réus JC, Bolan M, Massignan C, Flores-Mir C, Maia I, Gozal D, Hallal ALC, Porporatti AL, De Luca Canto G. Association between obstructive sleep apnea and health-related quality of life in untreated adults: a systematic review. *Sleep and Breathing* v. 1, p. 1, 2021.
11. Pauletto P, Ruales-Carrera E, Mezzomo LA, Stefani CM, Taba M, Gonçalves RB, Flores-Mir C, De Luca Canto G. Clinical performance of short versus standard dental implants in vertically augmented bone: an overview of systematic reviews. *Clinical Oral Investigations*, v. 00, p. 1-24, 2021.
12. Hallal ALC, Vidor AC, Machado CA, Garcia LP, Pauletto P, Honnef LR, Espirito-Santo TB, De Luca Canto G, Garcia-Larsen V. Características clínicas e epidemiológicos dos casos de covid-19 em Florianópolis, Brasil. *Saúde Coletiva (Barueri)*, v. 11, p. 8825-8829, 2021.
13. Lehmkuhl KM, De Luca Canto G, Pauletto P, Hallal ALC, Bastos RC, Cisneros O, Massignan C, ; De Luca Canto G. Covid-19 and the challenges for higher education: a scoping review. *Saúde Coletiva (Barueri)*, v. 11, p. 8747-8761, 2021.
14. Massignan C, Butini LO, Honnef LR, Meller JO, Lehmkuhl KM, Pauletto P, Stefani CM, De Luca Canto G. Methodological quality assessment in systematic reviews in health sciences that included observational studies: a cross-sectional study/ Avaliação da qualidade metodológica em revisões sistemáticas na área das ciências da saúde que incluíram estudos observacionais: um estudo transversal. *Brazilian Journal of Health Review*, v. 4, p. 23002-23018, 2021.
15. Werlich MO, Honnef LR, Bett JVS, Domingos FL, Pauletto P, Mendes de Souza BD, Mageste DT, Hallal ALC, De Luca Canto G. Prevalence of dentofacial injuries in contact sports players: A systematic review and meta-analysis. *Dental Traumatology*, v. 00, p. 1-12, 2020.

16. Escobar M, Pauletto P, Benfatti CA, Cruz AC, Flores-Mir C, Henriques BA. Effect of cyanoacrylate tissue adhesive in postoperative palatal pain management: a systematic review. *Clinical oral investigations*. 2020 Nov 17:1-4.
17. Duarte J, Pauletto P, Massignan C, Bolan M, Domingos FL, Curi Hallal AL, De Luca Canto G. Association Between Sleep Bruxism and Quality of Life: A Systematic Review. *Journal of Oral & Facial Pain & Headache*. 2020 Oct 1(4).
18. Oliveira Werlich M, Honnef LR, Silva Bett JV, Domingos FL, Pauletto P, Dulcinea Mendes de Souza B, Mageste Duque T, Curi Hallal AL, De Luca Canto G. Prevalence of dentofacial injuries in contact sports players: A systematic review and meta-analysis. *Dental traumatology*. 2020.
19. Melo G, Duarte J, Pauletto P, Porporatti AL, Stuginski-Barbosa J, Winocur E, Flores-Mir C, De Luca Canto G. Bruxism: An umbrella review of systematic reviews. *Journal of oral rehabilitation*. 2019 Jul;46(7):666-90.
20. Pauletto P, Ruales-Carrera E, Simek Vega Gonçalves TM, Gebler Philippi A, Donos N, Mezzomo LA. Fixed and Removable Full-Arch Restorations Supported by Short (≤ 8 mm) Dental Implants in the Mandible: A Systematic Review and Meta-Analysis. *International Journal of Oral & Maxillofacial Implants*. 2019 Jul 1;34(4).
21. Ruales-Carrera E, Pauletto P, Apaza-Bedoya K, Volpato CA, Özcan M, Benfatti CA. Peri-implant tissue management after immediate implant placement using a customized healing abutment. *Journal of Esthetic and Restorative Dentistry*. 2019 Nov;31(6):533-41.
22. Caetano GM, Pauletto P, Mezzomo LA, Rivaldo EG. Crestal bone changes in different implants designs: a prospective clinical trial. *European journal of dentistry*. 2019 Oct;13(4):497.

Capítulos de Livros Publicados

1. De Luca Canto G, Massignan C, Pauletto P, Polmann H, Meller J, Réus CJ, Guerra E, Perspectivas Futuras para as Revisões Sistemáticas. Fundamentos das Revisões Sistemáticas em Saúde. 1ed.São Paulo: Santos Publishing, 2021.
2. Pauletto P, Massignan C, Stefani CM, Martins CC, De Luca Canto G, Análise do Risco de Viés de Revisões Sistemáticas com a Ferramenta ROBIS. Risco de Viés em Revisões Sistemáticas: guia prático. 1ed.São Paulo: Câmara Brasileira do Livro, 2021, v. 1, p. 1.
3. Pauletto P, Stefani CM, Massignan C, De Luca Canto G. Análise do Risco de Viés de Revisões Sistemáticas com a Ferramenta AMSTAR 2. Risco de Viés em Revisões Sistemáticas: guia prático. 0ed.São Paulo: Câmara Brasileira do Livro, 2021, v. 1, p. 1-.
4. De Luca Canto G, Pauletto P, Síntese do Conhecimento. Revisões sistemáticas da literatura: guia prático. 1ed.: Brazil Publishing, 2020, v.1, p. 1.

Aprovações em Processos Seletivos

- Aprovada no processo seletivo simplificado para professor substituto na área/subárea do conhecimento Odontologia/ Prótese Dental 2018, quinta colocada.
- Aprovada no processo seletivo para professor substituto na área/sub-área do conhecimento Odontologia/ Oclusão (2019), segunda colocada.
- Aprovada no processo seletivo para professor substituto na área/sub-área do conhecimento Odontologia/ Oclusão (2022), quarta colocada.
- Professora de Oclusão Universidad de Las Américas, Quito, Equador (2022).

APÊNDICE B

PREFERRED REPORTING ITEMS FOR SYSTEMATIC REVIEWS AND META-ANALYSES EXTENSION FOR SCOPING REVIEWS (PRISMA – ScR) CHECKLIST

SECTION	ITEM	PRISMA-ScR CHECKLIST ITEM	REPORTED ON PAGE #
TITLE			
Title	1	Identify the report as a scoping review.	1
ABSTRACT			
Structured summary	2	Provide a structured summary that includes (as applicable): background, objectives, eligibility criteria, sources of evidence, charting methods, results, and conclusions that relate to the review questions and objectives.	3
INTRODUCTION			
Rationale	3	Describe the rationale for the review in the context of what is already known. Explain why the review questions/objectives lend themselves to a scoping review approach.	5-7
Objectives	4	Provide an explicit statement of the questions and objectives being addressed with reference to their key elements (e.g., population or participants, concepts, and context) or other relevant key elements used to conceptualize the review questions and/or objectives.	7
METHODS			
Protocol and registration	5	Indicate whether a review protocol exists; state if and where it can be accessed (e.g., a Web address); and if available, provide registration information, including the registration number.	7
Eligibility criteria	6	Specify characteristics of the sources of evidence used as eligibility criteria (e.g., years considered, language, and publication status), and provide a rationale.	7-8

SECTION	ITEM	PRISMA-ScR CHECKLIST ITEM	REPORTED ON PAGE #
Information sources*	7	Describe all information sources in the search (e.g., databases with dates of coverage and contact with authors to identify additional sources), as well as the date the most recent search was executed.	68
Search	8	Present the full electronic search strategy for at least 1 database, including any limits used, such that it could be repeated.	Table 1 / Appendix S2
Selection of sources of evidence†	9	State the process for selecting sources of evidence (i.e., screening and eligibility) included in the scoping review.	8-9
Data charting process‡	10	Describe the methods of charting data from the included sources of evidence (e.g., calibrated forms or forms that have been tested by the team before their use, and whether data charting was done independently or in duplicate) and any processes for obtaining and confirming data from investigators.	9
Data items	11	List and define all variables for which data were sought and any assumptions and simplifications made.	9
Critical appraisal of individual sources of evidence§	12	If done, provide a rationale for conducting a critical appraisal of included sources of evidence; describe the methods used and how this information was used in any data synthesis (if appropriate).	No
Synthesis of results	13	Describe the methods of handling and summarizing the data that were charted.	10
RESULTS			
Selection of sources of evidence	14	Give numbers of sources of evidence screened, assessed for eligibility, and included in the review, with reasons for exclusions at each stage, ideally using a flow diagram.	10
Characteristics of sources of evidence	15	For each source of evidence, present characteristics for which data were charted and provide the citations.	10
Critical appraisal within sources of evidence	16	If done, present data on critical appraisal of included sources of evidence (see item 12).	No
Results of individual sources of evidence	17	For each included source of evidence, present the relevant data that were charted that relate to the review questions and objectives.	10-11
Synthesis of results	18	Summarize and/or present the charting results as they relate to the review questions and objectives.	12-13

SECTION	ITEM	PRISMA-ScR CHECKLIST ITEM	REPORTED ON PAGE #
DISCUSSION			
Summary of evidence	19	Summarize the main results (including an overview of concepts, themes, and types of evidence available), link to the review questions and objectives, and consider the relevance to key groups.	13-17
Limitations	20	Discuss the limitations of the scoping review process.	17
Conclusions	21	Provide a general interpretation of the results with respect to the review questions and objectives, as well as potential implications and/or next steps.	19
FUNDING			
Funding	22	Describe sources of funding for the included sources of evidence, as well as sources of funding for the scoping review. Describe the role of the funders of the scoping review.	19

JBIG = Joanna Briggs Institute; PRISMA-ScR = Preferred Reporting Items for Systematic reviews and Meta-Analyses extension for Scoping Reviews.

* Where *sources of evidence* (see second footnote) are compiled from, such as bibliographic databases, social media platforms, and Web sites.

† A more inclusive/heterogeneous term used to account for the different types of evidence or data sources (e.g., quantitative and/or qualitative research, expert opinion, and policy documents) that may be eligible in a scoping review as opposed to only studies. This is not to be confused with *information sources* (see first footnote).

‡ The frameworks by Arksey and O'Malley (6) and Levac and colleagues (7) and the JBI guidance (4, 5) refer to the process of data extraction in a scoping review as data charting.

§ The process of systematically examining research evidence to assess its validity, results, and relevance before using it to inform a decision. This term is used for items 12 and 19 instead of "risk of bias" (which is more applicable to systematic reviews of interventions) to include and acknowledge the various sources of evidence that may be used in a scoping review (e.g., quantitative and/or qualitative research, expert opinion, and policy document).

From: Tricco AC, Lillie E, Zarin W, O'Brien KK, Colquhoun H, Levac D, et al. PRISMA Extension for Scoping Reviews (PRISMA-ScR): Checklist and Explanation. *Ann Intern Med.* 2018;169:467–473. doi: [10.7326/M18-0850](https://doi.org/10.7326/M18-0850).

APÊNDICE C
SEARCH STRATEGY USED FOR EACH OF THE DATABASES

Database	Search March 17 th , 2021
Embase	(bruxism) AND ("obstructive sleep apnea" OR "OSA" OR "SDB" OR "sleep disordered breathing")
LILACS	((bruxism OR bruxismo)) AND (("obstructive sleep apnea" OR "apneia obstrutiva do sono" OR "AOS" OR "OSA" OR "distúrbios respiratórios do sono" OR "SDB" OR "sleep disordered breathing"))
Livivo	(bruxism) AND ("obstructive sleep apnea" OR "OSA" OR "SDB" OR "sleep disordered breathing")
PubMed	("bruxism"[MeSH Terms] OR "bruxism"[All Fields]) AND ("sleep apnea, obstructive"[MeSH Terms] OR "obstructive sleep apnea"[All Fields] OR ("OSA"[All Fields] OR "SDB"[All Fields] OR "sleep disordered breathing"[All Fields]))
Scopus	TITLE-ABS-KEY (bruxism) AND TITLE-ABS KEY ("obstructive sleep apnea" OR "OSA" OR "SDB" OR "sleep disordered breathing")
Web of Science	TS=(bruxism) AND TS=("obstructive sleep apnea" OR "OSA" OR "SDB" OR "sleep disordered breathing")
Google Scholar	allintitle: "bruxism" AND "obstructive sleep apnea"

OpenGrey	("bruxism" AND "obstructive sleep apnea")
Proquest - Dissertation and Theses	noft (("Bruxism" OR "bruxismo")) AND noft ("Obstructive Sleep Apnea" OR "apneia obstrutiva do sono")

APÊNDICE D

EXCLUDED ARTICLES AND REASONS FOR EXCLUSION (N = 20)

Study	Reason for exclusion
Cahlin et al., 2017	1
Chae et al., 2011	3
Duran-Cantolla et al., 2015	2
Hesselbacher et al., 2014	4
Inoko et al., 2004	3
Kang et al., 2006	1
Korostovtseva et al., 2015	3
Maluly-Filho et al., 2019	3
Martínez et al., 2013	3
Michalek-Zrabkowska et al., 2020	1
Ohayon et al., 2001	3
Oksenberg & Arons, 2002	1
Palinkas et al., 2019	1
Puvanendran, Thomas & Chien, 2020	1
Simmons, Mohamed & Meskill, 2019	3
Solbach et al., 2018	1
Trindade & Rodriguez, 2014	3
Udwadia et al., 2004	1
Wetselaar et al., 2019	3
Wieczorek et al., 2020	1

Reasons for exclusion:

1. Studies that did not evaluate the relationship between SB and OSA
2. Studies in adults that did not detect the SB and OSA by PSG;
3. Reviews, letters, books, conference abstracts, case report, case series, opinion article, technique articles, posters, guidelines, short paper, pilot study, mechanism evaluation studies;

Full-text or data not available, even after three attempts to contact the corresponding authors over three weeks.

APÊNDICE E

UNIVERSIDADE FEDERAL DE SANTA CATARINA

CENTRO DE CIÊNCIAS DA SAÚDE

PROGRAMA DE PÓS GRADUAÇÃO EM ODONTOLOGIA

TERMO DE CONSENTIMENTO LIVRE E ESCLARECIDO

Eu, Joyce Duarte, doutoranda do Programa de Pós-Graduação em Odontologia, do Centro Ciências da Saúde da Universidade Federal de Santa Catarina (UFSC), sob orientação da Prof.^a Dr.^a Graziela De Luca Canto, convido você a participar da pesquisa intitulada “Associação entre bruxismo do sono e síndrome da apneia e hipopneia obstrutiva do sono em adultos”. O objetivo deste documento é dar a você informações suficientes sobre a pesquisa a qual você está sendo convidado a participar.

OBJETIVO DO ESTUDO

Este estudo tem por objetivo avaliar se existe associação entre o bruxismo do sono, caracterizado por rangimento ou apertamento dos dentes durante o sono e a síndrome da apneia e hipopneia obstrutiva, um problema respiratório onde há bloqueio total ou parcial da respiração durante o sono, através de dados obtidos por exame físico, questionários e exames de polissonografia.

PROCEDIMENTOS

- Em um primeiro momento, serão aplicados questionários específicos para identificar sinais e sintomas de bruxismo do sono e síndrome da apneia e hipopneia obstrutiva do sono.
- Será realizado um exame físico da sua boca e músculos da mastigação e posteriormente uma raspagem da mucosa oral, para avaliar as possíveis causas genéticas do Bruxismo do Sono. O exame físico e raspagem da mucosa oral terão a duração entre 5 e 10 minutos. O exame físico é como qualquer exame odontológico de rotina, e não

terá nenhuma consequência. Se não se sentir à vontade para a realização do exame, este será interrompido imediatamente. Os usos das células da mucosa bucal serão unicamente para essa pesquisa e saliento que logo após essa análise, elas serão descartadas de forma apropriada.

- Serão coletados também dados demográficos básicos como sexo, idade, estado civil e ocupação. Poderão também ser requisitadas outras informações, tais como peso, altura ou informações relevantes para o tema de pesquisa.
- Se constatadas necessidades de tratamento odontológico durante a avaliação clínica, você será orientado a buscar tratamento nas clínicas odontológicas da Universidade Federal de Santa Catarina.
- Toda a pesquisa envolve riscos, por isto se você não se sentir confortável para realizá-la, poderá desistir a qualquer momento. Nesta pesquisa os riscos são: desconforto ao realizar o exame clínico bucal, cansaço e aborrecimento em responder os questionários, quebra de sigilo, ainda que involuntário e não intencional. Para minimizar o risco de quebra de sigilo, seus dados terão um número para identificação, e não o seu nome. No entanto, como benefício direto, você terá uma avaliação clínica e levantamento de necessidades odontológicas, assim como os devidos encaminhamentos para o serviço. Como benefício indireto, você poderá contribuir para a elucidação e compreensão da fisiopatologia do bruxismo do sono e da apneia e hipopneia obstrutiva do sono.
- Os procedimentos da pesquisa (exame físico e aplicação de questionários), bem como os materiais utilizados, serão custeados pela Instituição proponente da pesquisa (UFSC).

PARTICIPAÇÃO VOLUNTÁRIA

Sua participação nesse estudo não é obrigatória e não haverá custos nem pagamentos pela mesma. Uma vez que você decidiu participar do estudo, você pode retirar seu consentimento de participação a qualquer momento, sem prejuízos de qualquer natureza. Não haverá reembolso, uma vez que com a participação na pesquisa você não terá custo.

JUSTIFICATIVA DO ESTUDO

O estudo servirá para a compreensão dos fatores associados ao bruxismo do sono, ou seja, se problemas respiratórios do sono causam, ou não, o ranger/apertar de dentes durante o sono.

PERMISSÃO PARA REVISÃO DE REGISTROS, CONFIDENCIALIDADE E ACESSO AOS REGISTROS

Será solicitada permissão para acesso aos registros dos exames de polissonografia realizados pelo hospital. Sua identidade não será revelada e os dados serão analisados e mantidos em sigilo. Os dados obtidos por meio dos questionários, exames físico e exames de polissonografia serão utilizados em publicações futuras e você terá acesso a eles a qualquer momento da pesquisa.

CONTATO COM OS PESQUISADORES

Se você tiver alguma dúvida em relação ao estudo, você deverá entrar em contato com a pesquisadora do estudo Joyce Duarte, pelo telefone (48) 3721-4952 ou via e-mail: joyceduarteortodtm@gmail.com ou da pesquisadora responsável, Graziela De Luca Canto, professora do Departamento de Odontologia, Centro de Ciências da Saúde – UFSC (Campus Trindade), telefone (48) 3721-4952 e e-mail delucacanto@gmail.com. Esta pesquisa atende a Resolução do CNS 466/2012 e conta com a aprovação do CEPESH/UFSC (Rua Desembargador Vitor Lima, nº 222, Trindade, Florianópolis, Prédio Reitoria II, 4º andar, sala 401). Caso você apresente alguma dúvida em relação a questões éticas, o contato com o Comitê de Ética dessa Instituição pode ser realizado por meio do telefone (48) 3721-9206 ou e-mail: cep@reitoria.ufsc.br.

DECLARAÇÃO DO PESQUISADOR

A pesquisadora responsável por esta pesquisa, Prof.^a Dr.^a Graziela De Luca Canto, se compromete a seguir a Resolução CNS n. 466/12 em todos os seus itens, entre os quais destacam-se: ressarcimento ou indenização de custos gerados em função da pesquisa, desde que estes sejam comprovados. O suporte, custeio, ressarcimento ou indenização serão de responsabilidade dos pesquisadores deste projeto, seguindo o que rege a resolução CNS n. 466/12.

DECLARAÇÃO DE CONSENTIMENTO DO PACIENTE

Eu _____, CPF _____, RG _____, residente à _____, estou ciente que me é garantido o livre acesso a todas as informações e esclarecimentos adicionais sobre o estudo durante e depois da minha participação. Declaro ter sido informado e estar devidamente esclarecido sobre os objetivos deste estudo. Recebi garantias de total sigilo e de obter novos esclarecimentos sempre que desejar, assim como afirmo também ter recebido uma via do presente Termo de Consentimento Livre e Esclarecido. Assim, concordo em participar voluntariamente deste estudo e sei que posso retirar meu consentimento a qualquer momento, sem qualquer prejuízo.

Florianópolis ___ / ___ / _____

Assinatura do participante

Assinatura da pesquisadora: Joyce Duarte

Assinatura da pesquisadora responsável: Graziela De Luca Canto

APÊNDICE F
STROBE STATEMENT – CHECKLIST OF ITEMS THAT SHOULD BE INCLUDED
IN REPORTS OF OBSERVATIONAL STUDIES

	Item No	Recommendation	
Title and abstract	1	(a) Indicate the study's design with a commonly used term in the title or the abstract	1
		(b) Provide in the abstract an informative and balanced summary of what was done and what was found	3
Introduction			
Background/rationale	2	Explain the scientific background and rationale for the investigation being reported	5,6
Objectives	3	State specific objectives, including any prespecified hypotheses	6
Methods			
Study design	4	Present key elements of study design early in the paper	7
Setting	5	Describe the setting, locations, and relevant dates, including periods of recruitment, exposure, follow-up, and data collection	7
Participants	6	(a) <i>Cohort study</i> —Give the eligibility criteria, and the sources and methods of selection of participants. Describe methods of follow-up <i>Case-control study</i> —Give the eligibility criteria, and the sources and methods of case ascertainment and control selection. Give the rationale for the choice of cases and controls <i>Cross-sectional study</i> —Give the eligibility criteria, and the sources and methods of selection of participants (b) <i>Cohort study</i> —For matched studies, give matching criteria and number of exposed and unexposed <i>Case-control study</i> —For matched studies, give matching criteria and the number of controls per case	7
Variables	7	Clearly define all outcomes, exposures, predictors, potential confounders, and effect modifiers. Give diagnostic criteria, if applicable	7,8,9,10
Data sources/ measurement	8*	For each variable of interest, give sources of data and details of methods of assessment (measurement). Describe comparability of assessment methods if there is more than one group	7,8,9,10
Bias	9	Describe any efforts to address potential sources of bias	11
Study size	10	Explain how the study size was arrived at	NA
Quantitative variables	11	Explain how quantitative variables were handled in the analyses. If applicable, describe which groupings were chosen and why	NA
Statistical methods	12	(a) Describe all statistical methods, including those used to control for confounding	12

(b) Describe any methods used to examine subgroups and interactions

(c) Explain how missing data were addressed

(d) *Cohort study*—If applicable, explain how loss to follow-up was addressed

Case-control study—If applicable, explain how matching of cases and controls was addressed

Cross-sectional study—If applicable, describe analytical methods taking account of sampling strategy

(e) Describe any sensitivity analyses

Results

Participants	13*	(a) Report numbers of individuals at each stage of study—eg numbers potentially eligible, examined for eligibility, confirmed eligible, included in the study, completing follow-up, and analysed (b) Give reasons for non-participation at each stage (c) Consider use of a flow diagram	12, 13
Descriptive data	14*	(a) Give characteristics of study participants (eg demographic, clinical, social) and information on exposures and potential confounders (b) Indicate number of participants with missing data for each variable of interest (c) <i>Cohort study</i> —Summarise follow-up time (eg, average and total amount)	13,14
Outcome data	15*	<i>Cohort study</i> —Report numbers of outcome events or summary measures over time <i>Case-control study</i> —Report numbers in each exposure category, or summary measures of exposure <i>Cross-sectional study</i> —Report numbers of outcome events or summary measures	13,14
Main results	16	(a) Give unadjusted estimates and, if applicable, confounder-adjusted estimates and their precision (eg, 95% confidence interval). Make clear which confounders were adjusted for and why they were included (b) Report category boundaries when continuous variables were categorized (c) If relevant, consider translating estimates of relative risk into absolute risk for a meaningful time period	14,15
Other analyses	17	Report other analyses done—eg analyses of subgroups and interactions, and sensitivity analyses	

Discussion

Key results	18	Summarise key results with reference to study objectives	16,17,18
Limitations	19	Discuss limitations of the study, taking into account sources of potential bias or imprecision. Discuss both direction and magnitude of any potential bias	19
Interpretation	20	Give a cautious overall interpretation of results considering objectives, limitations, multiplicity of analyses, results from similar studies, and other relevant evidence	19
Generalisability	21	Discuss the generalisability (external validity) of the study results	19
Other information			
Funding	22	Give the source of funding and the role of the funders for the present study and, if applicable, for the original study on which the present article is based	19, 20

*Give information separately for cases and controls in case-control studies and, if applicable, for exposed and unexposed groups in cohort and cross-sectional studies.

Note: An Explanation and Elaboration article discusses each checklist item and gives methodological background and published examples of transparent reporting. The STROBE checklist is best used in conjunction with this article (freely available on the Web sites of PLoS Medicine at <http://www.plosmedicine.org/>, Annals of Internal Medicine at <http://www.annals.org/>, and Epidemiology at <http://www.epidem.com/>). Information on the STROBE Initiative is available at www.strobe-statement.org.

APÊNDICE G

LISTA DE FIGURAS

ARTIGO 1.....		23
FIGURE 1 -	PRISMA 2020 flow diagram for new systematic reviews which included searches of databases, registers and other sources.....	30
FIGURE 2 -	Association between SB and OSA in adults with SB and OSA detection by PSG. Green cells indicate a positive association between the two conditions; yellow indicates the absence of association, and amber color represents the negative association. *In the Martynowicz et al., 2019 study, this association was found in mild and moderate OSA.....	33
FIGURE 3 -	Association between SB and OSA in children. Green indicates a positive association between the two conditions, and yellow indicates the absence of association.....	34
ARTIGO 2.....		67
FIGURE 1 -	Temporal relationship between apnea-hypopnea event (AHE) and sleep bruxism (SB) episode within a 2-minute scoring window. Where, T1 = time interval between the end of the AHE and the beginning of the SB (A), and T2 = time interval between end of SB episode and start AHE (B).....	73
FIGURE 2 -	Occurrence patterns between apnea-hypopnea event (AHE) and sleep bruxism (SB) episode within a 2-minute scoring window. (1) Isolated SB episode; (2) Antecedent (AHE before SB episode); (3) Subsequent (AHE after SB episode); (4) Antecedent + Subsequent (AHE before and after SB episode); (5) Concomitant (AHE and SB concomitant).....	74
FIGURE 3 -	Flowchart of study participants.....	75

FIGURE 4 -	Barr graphic representation of the distribution of number of sleep bruxism episodes in relation to sleep phase.....	77
FIGURE 5 -	Distribution of sleep bruxism (SB) episodes according to 5 patterns of occurrence. (1) SB episode isolated; (2) AHE antecedent (AHE before SB episode); (3) Subsequent (AHE after SB episode); (4) Antecedent + Subsequent (AHE before and after SB episode); (5) Concomitant (AHE and SB concomitant).....	78

APÊNDICE H

LISTA DE TABELAS

ARTIGO 1.....		23
TABLE 1 -	Search strategy in PubMed Database.....	48
TABLE 2 -	Characteristics of the included studies in adults (n=13).....	49
TABLE 3 -	Characteristic of the included studies in children (n=8).....	58
TABLE 4 -	Results related to the respiratory variables analyzed in adults (ODI, SE, AI, AHI and minOSAT).....	65
TABLE 5 -	Summary of suggestions for future studies.....	66
ARTIGO 2.....		67
TABLE 1 -	Descriptive characteristics of the sample with and without obstructive sleep apnea (OSA) (n=62).....	85
TABLE 2 -	Descriptive characteristics of the sample with and without sleep bruxism (SB) (n=62).....	85
TABLE 3 -	Distribution of frequency and percentage of participants with and without definitive sleep bruxism (SB) and obstructive sleep apnea (OSA).....	86
TABLE 4 -	Mean, Standard Deviation (SD), Median and Interquartile Range (IQR) of sleep bruxism (SB) episodes according to occurrence patterns.....	87
TABLE 5 -	Distribution of frequency and percentage of participants with and without definitive sleep bruxism (SB) and obstructive sleep apnea (OSA) severity.....	87

ANEXO A

SCOPING REVIEW PROTOCOL

REVIEW TITLE AND TIMESCALE

REVIEW TITLE

Give the working title of the review.

Sleep bruxism and obstructive sleep apnea: a scoping review

ORIGINAL LANGUAGE TITLE (SOMENTE PREENCHER SE FOR FAZER A REVISÃO EM OUTRO IDIOMA QUE NÃO O INGLÊS)

For reviews in languages other than English, this field should be used to enter the title in the language of the review.

NA

ANTICIPATED OR ACTUAL START DATE	ANTICIPATED DATE	COMPLETION DATE	SEARCH DATE
February 2021	July 2021		February 2021

STAGE OF REVIEW AT TIME OF THIS SUBMISSION

REVIEW STAGE	STARTED	COMPLETED
Stage 1: identifying the research question	<input checked="" type="checkbox"/>	<input type="checkbox"/>
Stage 2: identifying relevant studies	<input type="checkbox"/>	<input type="checkbox"/>
Stage 3: study selection	<input type="checkbox"/>	<input type="checkbox"/>
Stage 4: charting the data	<input type="checkbox"/>	<input type="checkbox"/>
Stage 5: collating, summarizing and reporting the results	<input type="checkbox"/>	<input type="checkbox"/>

REVIEW TEAM DETAILS

NAMED CONTACT / EMAIL SALUTATION FOR CORRESPONDENCE

Patrícia Pauletto

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Federal University of Santa Catarina; Health Sciences Center; Department of Dentistry

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NAMED CONTACT AND PHONE NUMBER

Patrícia Pauletto / +55 (48) 999277768

ORGANIZATIONAL AFFILIATION OF THE REVIEW / WEBSITE ADDRESS

Brazilian Centre for Evidence Based Research – cobe.paginas.ufsc.br

REVIEW TEAM MEMBERS AND THEIR ORGANIZATIONAL AFFILIATIONS

TITLE	NAME	AFFILIATION	CONTACT (E-MAIL)	CONTRIBUTIONS*
Professor, Dr, Mr, Ms, Miss, Mrs	Completed name	Organisational affiliations of each member of the review team.	Contact email of the authors	Contributions of the authors*
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*1R=First reviewer (Study conceptualization and design/ Search and selection/ Data collection/ Data analysis/ Manuscript preparation). 2R=Second Reviewer (Search and selection/ Data collection/ Data analysis/ Manuscript preparation). 3R=Third Reviewer (Data analysis). E=Expert (Study conceptualization and design/ Data analysis). C=Coordinator (Study conceptualization and design/ Data analysis).

All authors: Review of the manuscript.

FUNDING SOURCES/SPONSORS (GRANT NUMBER)

MSc Patrícia Pauletto and MSc Helena Polmann are supported by Coordination of Superior Level Staff Improvement (CAPES).

CONFLICTS OF INTEREST

None

COLLABORATORS

Give the name and affiliation of any individuals or organizations who are working on the review but who are not listed as review team members.

NA

REVIEW METHODS

RESEARCH QUESTION

Co – Condition: Sleep bruxism and obstructive sleep apnea.

Co – Context: Actual knowledge about the subject (prevalence, association, risk factors)

Pop – Population: Human individuals

S – Study design: Observational studies (cohort, cross-sectional)

QUESTION: What is known about the possible association between sleep bruxism and obstructive sleep apnea?

STRATEGY TO IDENTIFY RELEVANT STUDIES

State the sources that will be searched. Give the search dates, and any restrictions (e.g. Language or publication period). Electronic databases; reference lists; hand-searching of key journals; existing networks, relevant organizations and conferences.

The search strategy will be developed with the help of an experienced health science librarian. Six databases will be searched (PubMed, Embase, LILACS, Web of Science, Livivo and Scopus). Additional literature will be searched by using the grey literature (on Proquest Dissertation and Theses, OpenGrey and Google Scholar). Additionally, experts will be contacted to further indication of studies to include and hand searches of bibliographies from included studies and on the key journals will be done.

The search will be carried out on February 1st, 2021 and import into a reference software manager (EndNote X9 @; Thomson Reuters, Philadelphia, PA, USA).

SEARCH STRATEGY

Search	Query (February 09 th , 2021)	Results
#8	<p>("bruxism"[MeSH Terms] OR "bruxism"[All Fields]) AND ("sleep apnea, obstructive"[MeSH Terms] OR "obstructive sleep apnea"[All Fields] OR ("OSA"[All Fields] OR "SDB"[All Fields] OR "sleep disordered breathing"[All Fields])</p> <p>("bruxism") AND ("obstructive sleep apnea" OR "OSA" OR "SDB" OR "sleep disordered breathing")</p>	150

CONDITION OR DOMAIN BEING STUDIED

Give a short description of the disease, condition or healthcare domain being studied.

Bruxism is a condition characterized by jaw-muscle activity disorder, which can occur based on state .e.g., sleep aka Sleep Bruxism (SB) (when there are rhythmic or non-rhythmic activities), and/or wakefulness, being Awake Bruxism (AB) (when there are repetitive or sustained tooth contact and/or with bracing or thrusting of the jaw) (LOBBEZOO et al., 2013). Obstructive sleep apnea (OSA) is defined as a collapse of the upper airway that leads to a reduction in airflow during sleep, despite the patient's attempt to resume normal respiration (SLEEP AND NEUROLOGIC DISEASE, 2017). Although several studies have assessed a potential association or comorbidity between SB and OSA, three systematic reviews have concluded that there is not enough conclusive scientific evidence to establish a clear link between them (CANTO et al. 2014; JOKUBAUSKAS & BALTRUŠAITYTĖ 2017; DA COSTA LOPES et al. 2019). The search for the most current systematic review was carried out in May 2019. In addition to updating the search for new studies, it will serve as an in-depth study for writing a doctoral thesis. Thus, with this scoping review we intend to re-evaluated the extant literature on this possible relationship between obstructive sleep apnea and sleep bruxism.

STUDY SELECTION

Give summary criteria for the inclusion and exclusion criteria that will be studied by the review. The preferred format includes details of both inclusion and exclusion criteria.

Example: Inclusion: adults with schizophrenia (as diagnosed using any recognized diagnostic criteria).

Exclusion: adolescents (under 18 years of age) and elderly people (over 70).

We will include observational studies that analyzed adults and/or children, with no year or language restrictions; the objective is to assess the relationship between SB and OSA. To be included, the study must use full-night ambulatory polysomnography to diagnose OSA and detect bruxism by clinic questionnaire/ self-report, physical exam, and/or PSG.

Exclusion criteria:

1. Studies that did not mention the purpose to assess the relationship between SB and OSA;
2. Studies that did not use the stated selection criteria for OSA and/or bruxism;
3. Studies that did not use polysomnography or polygraphy for OSA diagnosis;
4. Studies that included other sleep breathing disorder than OSA;
5. Reviews, letters, books, conference abstracts, case report, case series, opinion article, technique articles, posters and guidelines;
7. Full-text or data not available, even after trying to contact the corresponding authors (three attempts in a 3-week period).

DATA ITEMS:

List and define all variables for which data were sought and any assumptions and simplifications made.

- Sleep Bruxism
- Obstructive Sleep Apnea

CHARTING THE DATA:

State the process that will be used for selecting studies (e.g., two independent reviewers) through each phase of the review (i.e., screening, eligibility, and inclusion in meta-analysis). Describe planned method of extracting data from reports (e.g., piloting forms, done independently, in duplicate), any processes for obtaining and confirming data from investigators.

Three independent reviewers (P.P, H.P and J.C.R) will select the included articles in two phases. Firstly (phase-1), the 3 reviewers will evaluate the titles and abstracts according the eligibility criteria; secondly (phase-2), they will view full-texts and select articles by the same criteria as phase-1; then, they will crosscheck all the information found. If disagreements arise, a fourth reviewer (CM) will participate before a final decision is made of both phases. If important data for the review are missing or unclear, an attempt will be made to contact the study corresponding author to resolve or clarify the problem.

STRATEGY FOR COLLATING, SUMMARIZING AND REPORTING THE RESULTS

Provide details of the planned synthesis including a rationale for the methods selected.

The results will be synthesized through both a descriptive analysis akin to a 'narrative review' strategy (Pawson, 2002).

Data will be charted following the method presented by Hilary Arksey & Lisa O'Malley (2005), as follows:

1. Author(s), year of publication, country;
2. Study aim;
3. Study population (sample; groups; range and mean age);

4. Outcome measures; and
5. Main results.

Sub-group analysis:

Severity of apnea (mild, moderate or severe).

Three different reviewers (P.P., HP and JCR) will collect data and cross-check for accuracy. In case of any discrepancies, a consensus discussion will be performed, and, if necessary, a fourth reviewer (C.M.) will be involved to make a final decision.

CRITICAL APPRAISAL OF INDIVIDUAL SOURCES OF EVIDENCE *Provide a rationale for conducting a critical appraisal of included sources of evidence; describe the methods used and how this information was used in any data synthesis (if appropriate).*

We will conduct a critical appraisal based on each type of observational study.

For cross-sectional studies, we will use JBI Checklist for Analytical Cross Sectional Studies.

For case-control studies, we will use JBI Checklist for Analytical Case-Control Studies.

For cohort, we will use JBI Checklist for Analytical Cohort Studies.

DATABASES

- PubMed
- EMBASE
- Cochrane
- LILACS
- Web of Science
- Science Direct
- CINAHL
- Livivo
- Scopus
- Other:

ADDITIONAL LITERATURE

- Google Scholar web search (specify if limitations applied)
- Hand searches of bibliographies from included studies
- Experts
- OpenGrey
- Proquest (Dissertation and Theses)

DATA MANAGEMENT

- | | |
|---|---|
| <input checked="" type="checkbox"/> Endnote | <input type="checkbox"/> Zotero |
| <input type="checkbox"/> Refworks | <input type="checkbox"/> Covidence |
| <input type="checkbox"/> Mendeley | <input checked="" type="checkbox"/> Other: Rayyan |

REVIEW GENERAL INFORMATION

HEALTH AREA OF THE SCOPING REVIEW

Dentistry

KEYWORDS

Give words or phrases that best describe the review. Example: systematic review; meta-analysis; recurrence

Bruxism, obstructive sleep apnea, evidence-based dentistry, systematic review

OTHERS**PREVIOUS REVIEWS ABOUT THE SAME SUBJECT**

No

If yes, why perform another one? NA

SUGGEST 3 JOURNALS IN WHICH THIS RESEARCH COULD BE PUBLISHED AND WHY

JOURNAL	IF	QUALIS
Journal of Oral Rehabilitation	2.304	A1
Clinical Oral Investigations	2.812	A1
Journal of Oral & Facial Pain and Headache	1.260	A2

CITE 5 STUDIES THAT YOU READ (ABOUT THIS TOPIC) BEFORE PREPARE THIS PROTOCOL

Jokubauskas L, Baltrušaitytė A. Relationship between obstructive sleep apnoea syndrome and sleep bruxism: a systematic review. *J Oral Rehabil.* 2017;44(2):144-153.

Da Costa Lopes AJ, Cunha TCA, Monteiro MCM, Serra-Negra JM, Cabral LC, Júnior PCS. Is there an association between sleep bruxism and obstructive sleep apnea syndrome? A systematic review. *Sleep Breath.* Published online 2019.

De Luca Canto G, Singh V, Gozal D, Major PW, Flores-Mir C. Sleep bruxism and sleep-disordered breathing: a systematic review. *Journal of Oral & Facial Pain & Headache.* 2014 Oct 1;28(4).

Maluly M, Dal Fabbro C, Andersen ML, Babiloni AH, Lavigne GJ, Tufik S. Sleep bruxism and its associations with insomnia and OSA in the general population of Sao Paulo. *Sleep Medicine.* 2020 Nov 1;75:141-8.

Kim DH, Lee SH, Lee SH. Sleep Bruxism Episodes in Patients with Obstructive Sleep Apnea Syndrome Determined by In-Laboratory Polysomnography. *Applied Sciences.* 2020 Jan;10(23):8587.

Maluly M, Andersen ML, Dal-Fabbro C, Garbuio S, Bittencourt L, de Siqueira JTT, et al. Polysomnographic study of the prevalence of sleep bruxism in a population sample. *J Dent Res.* 2013;92(7):97-103.

Martynowicz H, Gac P, Brzecka A, Poreba R, Wojakowska A, Mazur G, et al. The relationship between sleep bruxism and obstructive sleep apnea based on polysomnographic findings. *J Clin Med.* 2019;8(10):1653-1663.

Phillips BA, Okeson J, Paesani D, Gilmore R. Effect of sleep position on sleep apnea and parafunctional activity. *Chest.* 1986;90(3):424-429

Hosoya H, Kitaura H, Hashimoto T, Ito M, Kinbara M, Deguchi T, et al. Relationship between sleep bruxism and sleep respiratory events in patients with obstructive sleep apnea syndrome. *Sleep Breath*. 2014;18(4):837–844.

Okeson JP, Phillips BA, Berry DTR, Cook YR, Cabelka JF. Nocturnal bruxing events in subjects with sleep-disordered breathing and control subjects. *J Craniomandib Disord*. 1991;5(4):258-264.

Saito M, Yamaguchi T, Mikami S, Watanabe K, Gotouda A, Okada K, et al. Weak association between sleep bruxism and obstructive sleep apnea. A sleep laboratory study. *Sleep Breath*. 2016;20(2):703–709.

Tan MWT, Yap AU, Chua AP, Wong JCM, Parot MVJ, Tan KBC. Prevalence of sleep bruxism and its association with obstructive sleep apnea in adult patients: A retrospective polysomnographic investigation. *J Oral Facial Pain Headache*. 2019;33(3):269-277.

ANEXO B
PARECER CONSUBSTANCIADO DO CEP SH

UNIVERSIDADE FEDERAL DE
SANTA CATARINA - UFSC



PARECER CONSUBSTANCIADO DO CEP

DADOS DO PROJETO DE PESQUISA

Título da Pesquisa: ASSOCIAÇÃO ENTRE BRUXISMO DO SONO E APNEIA OBSTRUTIVA DO SONO EM ADULTOS

Pesquisador: Graziela De Luca Canto

Área Temática: Genética Humana:

(Trata-se de pesquisa envolvendo Genética Humana que não necessita de análise ética por parte da CONEP.);

Versão: 2

CAAE: 84783518.6.0000.0121

Instituição Proponente: CENTRO DE CIÊNCIAS DA SAÚDE

Patrocinador Principal: Financiamento Próprio

DADOS DO PARECER

Número do Parecer: 2.620.213

Apresentação do Projeto:

Trata o presente projeto, intitulado "Associação entre bruxismo do sono e apneia obstrutiva do sono em adultos" de uma pesquisa submetida pelo Profa. Graziela De Luca Canto, que assina a folha de rosto como pesquisador responsável juntamente com a Profa. Elena Riet Correa Rivero, coordenadora do PPG Odontologia/CCS/UFSC. O projeto é um estudo do tipo transversal analítico com o objetivo de investigar a associação entre bruxismo do sono e síndrome da apneia obstrutiva do sono em adultos e, secundariamente, as associações destas com a qualidade de vida e características do sono. Os participantes da pesquisa serão pacientes encaminhados para o Hospital Bala Sul para realização de exames de polissonografia (PSG). Os participantes (n=110) serão convidados a participar do estudo em uma situação prévia ao exame de PSG pois já terão seu encaminhamento para a realização do exame por motivo de suspeita de apneia ou outros problemas relacionados ao sono. Os dados dos participantes serão coletados por meio de anamnese, exame físico da boca e músculos da mastigação, coleta de células epiteliais da mucosa oral e aplicação de diversos questionários específicos.

Objetivo da Pesquisa:

Objetivo geral

Investigar a associação entre bruxismo do sono e apneia obstrutiva do sono em adultos.

Endereço: Universidade Federal de Santa Catarina, Prédio Reitoria II, R: Desembargador Vitor Lima, nº 222, sala 401
Bairro: Trindade **CEP:** 88.040-400
UF: SC **Município:** FLORIANOPOLIS
Telefone: (48)3721-8094 **E-mail:** cep.propesq@contato.ufsc.br

Continuação do Parecer: 2.620.213

Objetivos específicos

- Avaliar se existe associação entre a presença de BS e de apneia obstrutiva do sono e qualidade de vida.
- Avaliar a acurácia de questionários e exame físico comparados ao padrão de referência PSG no diagnóstico de BS e SAHOS.
- Verificar a associação entre uso de medicamentos antidepressivos e ocorrência de bruxismo do sono.
- Verificar a associação entre presença de tórus mandibular e presença de bruxismo do sono.
- Realizar uma análise quantitativa da presença de atividade rítmica da musculatura mastigatória e apneias por hora;
- Realizar uma avaliação quantitativa e qualitativa da distribuição dos episódios de bruxismo nas diferentes fases do sono;
- Verificar a presença de outras características do sono associadas aos episódios de bruxismo do sono, exemplos: movimentos de membros, despertares, posições, presença de ronco.
- Analisar os polimorfismos genéticos relacionados ao bruxismo do sono.

Avaliação dos Riscos e Benefícios:

De acordo com o que foi citado no TCLE apresentado:

Riscos: Toda a pesquisa envolve riscos, por isto se você não se sentir confortável para realizá-la, poderá desistir a qualquer momento. Nesta pesquisa os riscos são: desconforto ao realizar o exame clínico bucal, cansaço e aborrecimento em responder os questionários, quebra de sigilo, ainda que involuntário e não intencional. Para minimizar o risco de quebra de sigilo, seus dados terão um número para identificação, e não o seu nome.

Benefícios: Como benefício direto, você terá uma avaliação clínica e levantamento de necessidades odontológicas, assim como os devidos encaminhamentos para o serviço. Como benefício indireto, você poderá contribuir para a elucidação e compreensão da fisiopatologia do bruxismo do sono e da apneia e hipopneia obstrutiva do sono.

Comentários e Considerações sobre a Pesquisa:

Pode contribuir para o conhecimento generalizável sobre o tema.

Endereço: Universidade Federal de Santa Catarina, Prédio Reitoria II, R: Desembargador Vitor Lima, nº 222, sala 401
Bairro: Trindade CEP: 88.040-400
UF: SC Município: FLORIANOPOLIS
Telefone: (48)3721-8094 E-mail: cep.propesq@contato.ufsc.br

Continuação do Parecer: 2.620.213

Considerações sobre os Termos de apresentação obrigatória:

Adequados.

Recomendações:

Sem recomendações.

Conclusões ou Pendências e Lista de Inadequações:

Considerando que todas as pendências indicadas foram devidamente atendidas, não há nenhuma inadequação no presente processo.

Considerações Finais a critério do CEP:

Este parecer foi elaborado baseado nos documentos abaixo relacionados:

Tipo Documento	Arquivo	Postagem	Autor	Situação
Informações Básicas do Projeto	PB_INFORMAÇÕES_BÁSICAS_DO_PROJETO_1081179.pdf	13/04/2018 10:41:53		Aceito
Outros	RESPOSTA_AS_PENDENCIAS.docx	13/04/2018 10:41:20	Joyce Duarte	Aceito
Declaração de Manuseio Material Biológico / Biorepositório / Biobanco	Declaracao_UFPR.pdf	12/04/2018 20:40:28	Joyce Duarte	Aceito
Outros	Instrumentos_da_Pesquisa.docx	12/04/2018 20:38:49	Joyce Duarte	Aceito
Projeto Detalhado / Brochura Investigador	Plataforma_Brasil_Projeto_Bruxismo.docx	12/04/2018 20:38:03	Joyce Duarte	Aceito
Declaração de Instituição e Infraestrutura	Declaracao_Baia_Sul.pdf	12/04/2018 20:35:55	Joyce Duarte	Aceito
TCLE / Termos de Assentimento / Justificativa de Ausência	TCLE_projeto.docx	12/04/2018 20:31:14	Joyce Duarte	Aceito
Folha de Rosto	FolhadeRosto.pdf	08/03/2018 10:30:08	Joyce Duarte	Aceito

Situação do Parecer:

Aprovado

Necessita Apreciação da CONEP:

Endereço: Universidade Federal de Santa Catarina, Prédio Reitoria II, R: Desembargador Vitor Lima, nº 222, sala 401
 Bairro: Trindade CEP: 88.040-400
 UF: SC Município: FLORIANOPOLIS
 Telefone: (48)3721-6094 E-mail: cep.propesq@contato.ufsc.br

UNIVERSIDADE FEDERAL DE
SANTA CATARINA - UFSC



Continuação do Parecer: 2.620.213

Não

FLORIANOPOLIS, 25 de Abril de 2018

Assinado por:
Luiz Eduardo Toledo
(Coordenador)

Endereço: Universidade Federal de Santa Catarina, Prédio Reitoria II, R: Desembargador Vitor Lima, nº 222, sala 401
Bairro: Trindade **CEP:** 88.040-400
UF: SC **Município:** FLORIANOPOLIS
Telefone: (48)3721-8094 **E-mail:** cep.propesq@contato.ufsc.br

ANEXO C

ARTIGO PUBLICADO NO JOURNAL SLEEP



SLEEP, 2022, 1–17

<https://doi.org/10.1093/sleep/zsac073>
 Advance Access Publication Date: 20 April 2022
 Review

REVIEW

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

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Abstract

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

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

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

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

Statement of Significance

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

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

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Introduction

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

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

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

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

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

and non-treated in a substantial portion of the population [28]. Furthermore, unrecognized OSA has a substantial economic impact on healthcare systems [29,30].

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

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

Methods

Protocol and registration

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

Eligibility criteria

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

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

Two types of studies were included:

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

Exclusion criteria

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

guidelines, short paper, pilot studies, mechanism evaluation studies;

4. Full-text or data not available, even after three attempts to contact the corresponding authors over three weeks.

Information sources

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

Search

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

Selection of sources of evidence

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

Data charting process

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

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

Data items

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

Synthesis of results

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

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

Results

Selection of sources of evidence

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

Characteristics of sources of evidence

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

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

Table 1. Search strategy in PubMed database

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

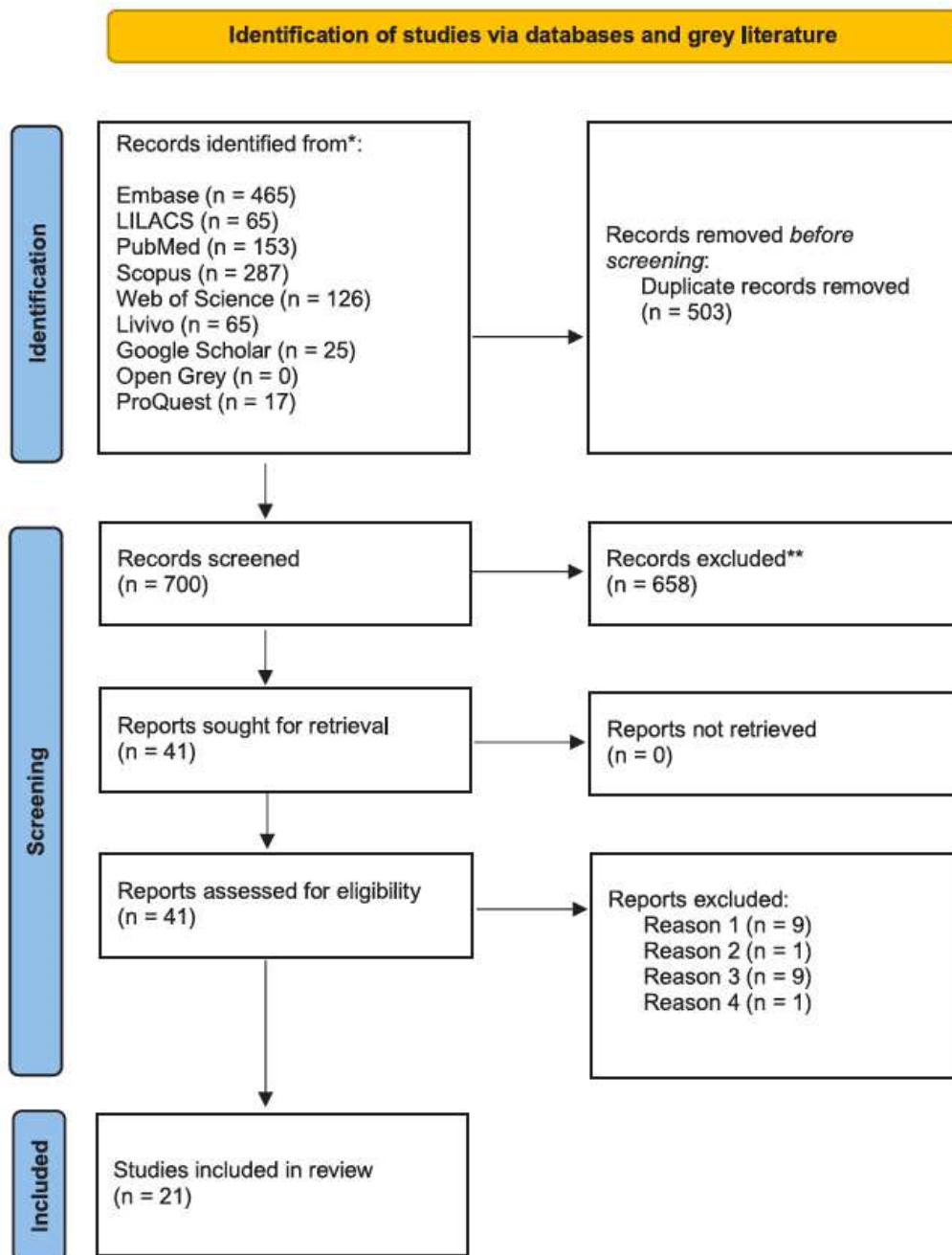


Figure 1. Flow diagram of literature search and selection criteria.

Results of individual sources of evidence

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

Sleep bruxism and obstructive sleep apnea assessment

Adults

SB and OSA conditions were assessed by using PSG.

Children

Sleep bruxism detection

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

Obstructive Sleep Apnea diagnosis

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

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

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

Table 2. Continued

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

Table 2. Continued

Study	Objective	Population	Methods			Results	Additional Information	
			Inclusion Criteria/Setting	OSA Diagnoses Criteria	SB Criteria			
Phillips et al., 1986 [42] United States Cross-Sectional	To determine the relationship between OSA and parafunctional activity.	Sample (N), Sex (M/F) Mean age (years) OSA:14 Non-OSA:10 OSA: 52 ± 15.9 years Non-OSA: 50.2 ± 16.4 years	Patients referred to the University of Kentucky Sleep Apnea Laboratory during the four-month period between July and October 1985 for evaluation of possible OSA.	Apnea: was defined as cessation of airflow at the nose and mouth for 10 s or longer during sleep. Hypopnea: was defined as a reduction in airflow associated with a 4% fall in oxygen saturation.	A clench was defined as activity of the masseter muscle exceeding 40% of the maximum clench of that muscle and lasting for two seconds or longer.	Findings/ Main Conclusion Statistical Analysis	Prevalence SB/OSA concomitant	Report on the sources of funding Conflict of Interest
Saito et al., 2013 [36] Japan Descriptive	To investigate the temporal association between SB events and OSA events.	N = 10 (male) 46.7 ± 11.5 years	Subjects with confirmed OSA and SB.	Apnea-hypopnea event: American Academy of Sleep Medicine	SB: Events were identified as RMMA. The amplitude was set twice the baseline activity.	Nocturnal clenching was slightly higher in patients with OSA than those without (12.2 vs 7.6 p = .18), and there was a correlation between the clench index and AHI by linear regression (r = 0.49, p < .05). There were significant falls in both the AHI (4.4 ± 28.8 vs 36.5 ± 36.7, p = .02) and clench index (12.5 ± 12.1 vs 7.0 ± 8.6, p = .04) in the lateral decubitus vs supine sleeping positions.	NR	NR
Saito et al., 2016 [32] Japan Cross-Sectional	To investigate, in a population reporting awareness of both OSA and SB, the associations between each specific breathing and jaw muscle event.	N = 59 (M = 47, F = 12) 44.8 ± 10.8 years	Japanese patients reporting awareness of breathing cessation and tooth grinding as well as signs and symptoms of OSA and SB.	Apnea and hypopnea events were scored according to standard criteria, with an AHI threshold of 5 events/h or more.	RMMA/SB Episode/h > 2 and/ or RMMA/SB bursts/h > 25.	AHI did not show a significant correlation with RMMA/SB episodes nor with RMMA/SB bursts. Sleep arousals in patients with concomitant SB and OSA are not strongly associated with onset of RMMA/SB. Spearman correlation test.	50.84% (30/59)	No No
Sjoholm et al., 2000 [35] Canada Cross-Sectional	To test the hypothesis of a direct association between SDB and SB.	N = 21 (M = 19, F = 2) 40.0 ± 29.2 years	Patients with OSA.	Apnea: was defined as the cessation of airflow for at least 10 s. Hypopneas: were defined as a decrease of more than 50% in thoracoabdominal amplitude for at least 10 s.	(1) Subjective estimation: teeth grinding, or clenching was reported by the patient one to two nights or more per week. (2) Clinical: the number and extent of visible wear facets on tooth enamel (attrition), the presence of masticatory muscle fatigue, and/or discomfort of the temporomandibular joint. (3) Masseter EMG: If the arbitrary cut-off point of 2.5 rhythmic jaw-movement episodes per hour was exceeded, the participant was as a bruxer during that night.	Masseter contraction episodes were associated with the termination of apnea or hypopnea episodes in 3.5% of the mild group and 14.4% of the moderate group (p < 0.05). It appears that SB is rarely directly associated with apneic events. Student's t-test	47.61% (10/21) Mild OSA 54% (6/11) Moderate OSA 40% (4/10)	NR NR

Table 2. Continued

Study	Objective	Population	Methods	Results	Additional Information			
Author, Year Country Study Design	Objective	Sample (N), Sex (M/F) Mean age (years) Inclusion Criteria/Setting	OSA Diagnoses Criteria SB Criteria	Findings/ Main Conclusion Statistical Analysis	Prevalence SB/OSA concomitant	Report on the sources of funding Conflict of Interest		
Smardz et al., 2020 [43] Poland Cross-Sectional	To evaluate the relationship between SDB and SB.	N = 77 (M = 21, F = 56) SB:58 Non-SB:19 34.8 ± 10.8 years	Patients above 18 years age, with a positive diagnosis of probable SB of the Clinic of Prosthetic Dentistry operating in the Department of Prosthetic Dentistry at Wrocław Medical University.	Apnea: defined as the absence of airflow through the airway for more than 10 s. Hypopnea: has been defined as a decrease in respiratory amplitude by more than 30% for more than 10 s, followed by subsequent blood desaturation of more than 3% or subsequent arousal.	Episodes qualified as bruxism if there was a RMMA, often accompanied by grinding sounds and characteristic movements in the mandible occurring after a minimum of 3s break from the last muscle activity. Episodes were classified as phasic (lasting 0.25 to 2s), tonic (lasting more than 2s), or mixed.	Both groups (SB and Non-SB) not differ statistically significantly in terms of oxygen desaturation index (U = 540.0, p = .90) and AHI (U = 531.5, p = .82). Quantitative analysis showed a lack of a statistically significant relationship between AHI and ODI in the patients with and without SB. Mann-Whitney U test	NR	Authors have no competing interests to declare. This study was co-financed by financial resources for Young Researchers of Wrocław Medical University, Poland (STM. 8022.17.011). The funding source was not involved in the study design, in the collection, analysis and interpretation of data, in the writing of the report, and in the decision to submit the article for publication. NR No Conflict of interest.
Tan et al., 2019 [16] Singapore Cross-Sectional	To determine the prevalence of SB in adult patients with OSA, to assess the association between SB and OSA in terms of sleep macrostructure and respiratory parameters and to determine possible OSA risk factors for SB.	N = 147 (M = 100, F = 47) SB: 49 (M = 37, F = 12) Non-SB: 98 (M = 63, F = 35)	Patients diagnosed with mild, moderate, or severe OSA and aged 25 years and above who underwent a PSG at the Ng Teng Fong General Hospital sleep clinic from July 2015 to February 2016.	Apnea: was determined as a cessation in airflow of 90% for a minimum period of 10 s. Hypopnea: was identified when the airflow dropped by 30% for a period of 10 s, accompanied by an oxygen desaturation of 3%. AHI: indicated the severity of OSA through the representation of number of apnea/hypopnea events per hour of sleep. Mild OSA was defined as having an AHI of 5 or more and less than 14 respiratory events per hour, moderate OSA between 15 or more and less than 30 events per hour, and severe OSA as 30 or more events per hour.	SB episodes were established when masseter RMMA exhibited twice the background EMG amplitude and were preceded by a period of > 3 second of stable background EMG.	Of the 147 patients with OSA, 49 (33.3%; 95% CI: 25.7 to 40.9%) were diagnosed with SB, while 98 (66.7; 95% CI: 59.1% to 74.3%) had no SB (descriptive analysis). An association was found between AHI and SB group. Patients with SB had significantly higher Respiratory Arousal Index and Oxygen Desaturation Index than patients without SB. This study demonstrated that SB occurs in about one-third of patients with OSA. Mann-Whitney U test.	33.33 % (49/147)	NR No Conflict of interest.

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

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

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

Table 3. Continued

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

Table 3. Continued

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

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

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

Obstructive sleep apnea diagnostic criteria

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

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

Sleep bruxism detection criteria

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

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

Synthesis of Results

Prevalence of concurrent sleep bruxism and obstructive sleep apnea

Adults

The prevalence of co-occurrence of the two conditions among studies that used PSG to detect both conditions ranged from

5.28% [44] to 50.84% [32] (median 39.3%; interquartile range 34.9%).

Children

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

Association between sleep bruxism and obstructive sleep apnea

Adults

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

Children

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

Temporal relationship

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

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

Respiratory variables in adult studies

Findings related to the respiratory variables analyzed (Oxygen Desaturation Index, Sleep Efficiency, Arousal Index,

Study	Association SB and OSA
Holanda et al., 2020 [34]	(-)
Hosoya et al., 2014 [17]	(+)
Kim et al., 2020 [46]	(-)
Maluly et al., 2020 [33]	No
Martynowicz et al., 2019*[45]	(+)
Okeson et al., 1991 [47]	No
Phillips et al., 1986 [42]	(+)
Saito et al., 2016 [32]	No
Smardz et al., 2020 [43]	No
Tan et al., 2019 [16]	(+)

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

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

Discussion

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

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

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

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

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

	Study	SB Mean±SD	No n-SB Mean±SD	p value
Oxygen Desaturation Index (ODI)	Hosoya et al., 2014 [17]	33.7 ± 25.0	22.4 ± 19.3	<.05*
	Smarcz et al., 2020 [43]	5.09 ± 8.24	3.28 ± 3.22	.90
	Tan et al., 2019 [16]	35.05 ± 24.75	26.1 ± 28.65	.005*
Sleep Efficiency (SE)	Kim et al., 2020 [46]	86.2 ± 14.1	81.5 ± 14.7	.14
	Hosoya et al., 2014 [17]	16.7 ± 8.6	17.6 ± 8.6	>.05
	Tan et al., 2019 [16]	85.20 ± 12.40	85.49 ± 13.01	.83
Arousal Index (AI)	Kim et al., 2020 [46]	6.9 ± 9.3	21.0 ± 26.3	.13
	Hosoya et al., 2014 [17]	40.0 ± 18.8	31.3 ± 15.9	>.05
	Tan et al., 2019 [16]	49.92 ± 18.05	43.44 ± 21.07	.03*
Minimum Oxygen Saturation (minOSAT)	Kim et al., 2020 [46]	83.8 ± 8.5	80.4 ± 9.6	.26
	Smarcz et al., 2020 [43]	88.90 ± 6.34	88.16 ± 9.39	.93
	Tan et al., 2019 [16]	78.47 ± 10.21	81.47 ± 10.76	.04*
Apnea-Hypopnea Index (AHI)	Kim et al., 2020 [46]	25.5 ± 16.6	45.1 ± 28.2	.03*
	Hosoya et al., 2014 [17]	37.2 ± 22.9	27.0 ± 19.7	<.05*
	Smarcz et al., 2020 [43]	5.52 ± 9.40	3.49 ± 3.55	.82

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

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

evaluated the temporal relationship between OSA and SB events, which could be a better strategy for potentially explaining the putative causal relationship between the two conditions.

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

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

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

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

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

Strengths, limitations

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

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

Scoping reviews do not require a quality assessment of the reviewed studies, as is the case for SRs [76]. Therefore, such limitations in the interpretation of results must be acknowledged. Another limitation that should be highlighted was the attempt to investigate confounding factors. However, studies

underreport this information, presenting simple statistics without adjustment for possible confounding factors.

Suggestions for future research

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

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

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

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

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

Table 5. Summary of suggestions for future studies

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

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

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

Supplementary material

Supplementary material is available at SLEEP online.

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References

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

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

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