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Gilberto de Souza Melo

BRUXISMO:
UMA *OVERVIEW* DE REVISÕES SISTEMÁTICAS

Florianópolis
2018

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UMA *OVERVIEW* DE REVISÕES SISTEMÁTICAS

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Orientadora: Profª. Drª. Graziela De Luca Canto

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Gilberto de Souza Melo

BRUXISMO: UMA *OVERVIEW* DE REVISÕES SISTEMÁTICAS

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Este trabalho é dedicado aos meus pais, amigos e professores.

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Esta dissertação foi originalmente escrita como um artigo na língua inglesa, com o objetivo de ser submetida ao periódico *Journal of Oral Rehabilitation*. Essa pesquisa foi realizada em parceria com os pesquisadores Ma. Joyce Duarte, Ma. Patrícia Pauletto, Dr. André Luís Porporatti e Drª. Graziela De Luca Canto, da Universidade Federal de Santa Catarina; a pesquisadora Drª. Juliana Stuginski-Barbosa, da Universidade de São Paulo; o pesquisador Carlos Flores-Mir, da *University of Alberta*; e o pesquisador Ephraim Winocur, da *Tel-Aviv University.*

RESUMO

Objetivos: Sumarizar e avaliar criticamente a evidência disponível em revisões sistemáticas (RS) publicadas sobre bruxismo do sono (BS) e/ou bruxismo em vigília (BV). **Métodos:** RS que investigaram qualquer desfecho relacionado ao bruxismo foram consideradas elegíveis e agrupadas em: 1) taxas de prevalência; 2) fatores associados; 3) acurácia diagnóstica; 4) efeitos sobre estruturas estomatognáticas; 5) efetividade de terapias. As buscas na literatura foram realizadas em sete bases de dados eletrônicas principais e em três bases da literatura cinzenta. Primeiramente, três revisores avaliaram os títulos e resumos dos artigos identificados, aqueles considerados elegíveis pelos critérios de elegibilidade foram lidos na íntegra. Em casos de discordâncias não resolvidas através de uma reunião de consenso, um quarto revisor seria consultado. A coleta de dados dos artigos e a avaliação do risco de viés foram realizadas independentemente por três revisores; foi utilizada a ferramenta "*University of Bristol's tool for assessing risk of bias in systematic reviews*" para a avaliação do risco de viés das RS incluídas. **Resultados:** De um total de 1038 estudos identificados, 112 foram lidos na íntegra e 49 RS foram finalmente incluídas. Em geral, três RS foram relacionadas às taxas de prevalência, duas à acurácia diagnóstica, dezoito aos fatores associados, dez aos efeitos sobre estruturas estomatognáticas e 16 sobre a efetividade de intervenções. Os achados das RS foram: 1) Em adultos, a prevalência do bruxismo em vigília foi 22-30%; do sono (1-15%) e o BS em crianças e adolescentes (3-49%); 2) os principais fatores consistentemente associados ao bruxismo foram o uso de álcool, cafeína, tabaco, alguns medicamentos psicotrópicos, acidificação esofágica, fumo passivo e alguns sinais e sintomas de disfunção temporomandibular; 3) comparados à polissonografia, os dispositivos portáteis (e.g. *BiteStrip*, *Bruxoff* e *GrindCare*) mostraram boa acurácia diagnóstica; 4) o bruxismo pode resultar em complicações relacionadas aos implantes dentários, no entanto, as evidências foram inconclusivas quanto às restaurações dentárias e danos ao periodonto; 5) dispositivos oclusais foram considerado efetivos para o manejo do bruxismo, no entanto, a evidência atual foi considerada insuficiente em relação à efetividade de outras terapias investigadas. **Conclusão:** Há uma grande quantidade de RS investigando desfechos relacionados ao BS, no entanto, apenas uma RS investigou o BV separadamente do BS. Além disso, a baixa acurácia dos métodos utilizados para o diagnóstico do bruxismo foi considerado uma limitação pela maioria das RS.

Palavras-chave: Odontologia baseada em evidências. Bruxismo. Bruxismo do sono. Bruxismo de vigília. Revisão sistemática.

ABSTRACT

Objectives: To summarize and critically appraise available evidence from published systematic reviews (SR) regarding sleep bruxism (SB) and/or awake bruxism (AB). **Methods.** SR investigating any bruxism related outcome were considered and grouped according to: 1) prevalence rates; 2) diagnostic accuracy; 3) associated factors; 4) effects on stomatognathic structures; and 5) therapies effectiveness. Searches were performed on seven electronic databases; additionally, a grey literature search was conducted on three databases.. Firstly, titles and abstracts of identified studies were independently screened by three reviewers. Studies considered eligible were read in full-text. In case of disagreements not solved by a consensus discussion, a fourth reviewer was consulted. Data collection and risk of bias assessment were performed independently by three reviewers; the tool "University of Bristol's tool for assessing risk of bias in SR" was used to assess bias in included SR. **Results:** From 1038 identified studies, 112 were read in full-text and 49 SR were finally included. Overall, three SR were related to prevalence rates, two to diagnostic accuracy of assessment tools, eighteen to associated factors, ten to effects on stomatognathic, and 16 to interventions' effectiveness. Findings from SR suggested that 1) among adults, prevalence of AB was 22-30%, SB (1-15%), and SB among children and adolescents (3-49%); 2) major factors consistently associated with bruxism were use of alcohol, caffeine, tobacco, some psychotropic medications, esophageal acidification, second-hand smoke, and several temporomandibular disorder signs and symptoms; 3) compared to polysomnography, portable diagnostic devices (e.g. BiteStrip, Bruxoff, and GrindCare) showed good diagnostic accuracy; 4) bruxism may result in complications regarding dental implants, however evidence was inconclusive regarding other dental restorations and damage to the periodontium; 5) occlusal appliances were considered effective on the management of bruxism, although evidence was considered weak regarding other investigated therapies. **Conclusion:** There are plenty of SR investigating particularly SB related outcomes, however, only one SR investigating AB separately from SB was found. Moreover, poor accuracy related to bruxism diagnosis was considered a limitation across the majority of SR.

Keywords: Evidence-based dentistry. Bruxism. Sleep bruxism. Awake bruxism. Systematic Review. Overview.

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LISTA DE ABREVIATURAS E SIGLAS

BS - Bruxismo do sono
BV - Bruxismo de vigília
e.g. - *Exempli grata* (do latim)
EMG - Eletromiografia
PSG - Polissonografia
RS - Revisão sistemática

Do artigo em inglês:

AB - Awake bruxism
CES - Contingent electrical stimulation
CI - Confidence interval
CNS - Central nervous system
DOR - Diagnostic odds ratio
EMG - Electromyography
HR - Hazard ratio
LR+ - Positive likelihood ratio
LR- - Negative likelihood ratio
MA - Meta analysis
N - No
NA - Not applicable
NI - Not informed
NPV - Negative predictive value
OR - Odds ratio
OSA - Obstructive sleep apnea
PN - Probably no
PPV - Positive predictive value
PR - Prevalante ratio
PRISMA - Preferred reporting items for systematic reviews and meta-analysis
PROSPERO - Prospective Register of Systematic Reviews
PSG - Polysomnography
PY - Probably yes
RCT - Randomized controlled trial
ROBIS - Risk of bias in systematic reviews
RR - Relative risk
SB - Sleep bruxism
SR - Systematic review
TMD - Temporomandibular disorder
Y - Yes

LISTA DE SÍMBOLOS

% - Percentual
± - Mais ou menos

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1 INTRODUÇÃO

1.1 BRUXISMO

O bruxismo é uma condição de grande interesse para pesquisadores e clínicos na área da Odontologia, Neurologia e Medicina do Sono. Apesar de existirem uma série de relatos na literatura em relação a complicações clínicas relacionadas ao bruxismo como, por exemplo, dor orofacial, desgastes dentários e falhas em procedimentos restauradores, o bruxismo continua sendo considerado difícil de ser manejado de modo efetivo e seguro (LAVIGNE, MANZINI e KATO, 2005).

A depender de sua manifestação circadiana, o bruxismo pode ser subdividido em bruxismo do sono (BS) ou bruxismo em vigília (BV) (LOBBEZOO et al., 2013). De acordo com um consenso internacional, o bruxismo pode ser definido como uma atividade repetitiva da musculatura mastigatória, caracterizado por apertamento ou rangimento dos dentes ou pelo forçamento da mandíbula (LOBBEZOO et al., 2013). Ademais, outros sinais e sintomas comumente associados ao bruxismo incluem dores de cabeça, endentações em língua e bochecha, hipersensibilidade dentária, hipertrofia dos músculos mastigatórios, limitação de abertura bucal e má-qualidade do sono (DE LA HOZ-AIZPURUA et al., 2011). Apesar de algumas preocupações terem sido levantadas nos últimos anos em relação a sua definição e manejo, parece existir alguma concordância em relação a definição de bruxismo como um comportamento, o qual não necessariamente exige um tratamento, e não efetivamente como uma desordem ou um fator de risco para uma desordem, nos quais um tratamento é usualmente recomendado (MANFREDINI et al., 2016; RAPHAEL, SANTIAGO e LOBBEZOO, 2016b).

1.2.1 Taxas de prevalência

Estudos epidemiológicos têm mostrado que, embora exista uma alta heterogeneidade em decorrência da falta de métodos diagnósticos padronizados, as taxas de prevalência entre adultos podem variar de 10-13% para BS e 22-31% para BV (MANFREDINI *et al.*, 2013b); nas populações mais jovens, no entanto, o bruxismo pode ser mais frequente, afetando até 40-50% dos participantes dos estudos (MANFREDINI *et al.*, 2013a; MACHADO *et al.*, 2014).

1.2.2 Etiologia e fatores associados

Tem sido proposto na literatura que a etiologia do bruxismo seja multifatorial e que vários mecanismos subjacentes possam desempenhar um papel em sua gênese, tais como fatores psicossociais (*e.g.* estresse e ansiedade), fisiológicos (*e.g.* fatores genéticos) e exógenos (*e.g.* consumo de álcool, uso de medicamentos ou tabagismo) (LAVIGNE et al., 2008; FALISI et al., 2014). Ainda, embora o conhecimento existente seja limitado, acredita-se que os fatores associados sejam distintos em relação às manifestações circadianas do bruxismo; enquanto aspectos psicossociais parecem ter alguma influência sobre o BV (MANFREDINI e LOBBEZOO, 2009), ativações relacionadas ao sistema nervoso autonômo/central podem ser os principais fatores envolvidos na gênese do BS (KATO et al., 2003).

1.2.3 Ferramentas para o diagnóstico

Embora o BV seja considerado mais prevalente, o BS é aquele que tem sido mais estudado, no entanto, há uma escassez de métodos diagnósticos válidos para a avaliação de ambas as condições (CASTRILLON et al., 2016). De acordo com uma revisão sistemática (RS) recente, o exame de polissonografia (PSG), apesar de algumas limitações em relação a sua validade interna, ainda é considerado o padrão de referência para o diagnóstico BS, enquanto que questionários, exames clínicos e dispositivos portáteis de diagnóstico são recomendados como ferramentas de triagem (CASETT et al., 2017). Até o momento, nenhuma RS investigou a validade de ferramentas de diagnóstico em relação ao BV, embora exista a recomendação da realização de um exame de eletromiografia (EMG) para um diagnóstico definitivo (CASTRILLON et al., 2016).

1.2.4 Efeitos sobre estruturas estomatognáticas

Ambas as formas de bruxismo podem ser prejudiciais às estruturas estomatognáticas (MANFREDINI et al., 2016) e algumas complicações clínicas mais relatadas incluem desgaste dentário anormal, mobilidade dental e falhas em restaurações, implantes ou próteses fixas/removíveis (JOHANSSON, OMAR e CARLSSON, 2011). Vale ressaltar que apesar dos inúmeros relatos sobre os efeitos negativos do bruxismo nos desfechos relacionados à saúde bucal, a literatura ainda é controversa, especialmente

devido às limitações diagnósticas observadas em grande parte dos estudos científicos (RAPHAEL, SANTIAGO e LOBBEZOO, 2016b).

1.2.5 Efetividade de intervenções

Na prática diária, os clínicos devem tomar decisões sobre a abordagem mais adequada para o manejo do bruxismo, o que inclui reconhecer se um tratamento é ou não recomendado (HUYNH et al., 2006; RAPHAEL, SANTIAGO e LOBBEZOO, 2016a). Portanto, apesar de não existir um tratamento único e definitivo, algumas terapias podem ser efetivas no manejo dessa condição, incluindo abordagens como 1) dispositivos oclusais; 2) tratamentos farmacológicos; 3) terapias comportamentais; e 4) outras abordagens (*e.g.* fisioterapia) (LOBBEZOO et al., 2008). Deve ser salientado que, apesar de muitas terapias estarem à disposição dos clínicos, a evidência disponível em relação a algumas modalidades terapêuticas foi em geral considerada fraca, portanto, recomenda-se cautela na interpretação desses estudos (MANFREDINI et al., 2015).

2 JUSTIFICATIVA

Numerosas RSs que investigaram tópicos relacionados ao bruxismo foram realizadas, especialmente na última década, no entanto, uma síntese e avaliação crítica desses estudos ainda não foram realizadas. Portanto, o objetivo deste trabalho foi sumarizar e avaliar criticamente as evidências disponíveis e responder à seguinte pergunta focada: "O que sabemos atualmente sobre as taxas de prevalência entre diferentes populações, acurácia diagnóstica dos instrumentos de avaliação, fatores associados, efeitos sobre estruturas estomatognáticas e efetividade de intervenções terapêuticas em relação ao bruxismo?"

3 OBJETIVOS

3.1 OBJETIVO GERAL

- Sumarizar e realizar uma avaliação crítica da evidência disponível em relação ao bruxismo do sono e em vigília.

3.2 OBJETIVOS ESPECÍFICOS

- Avaliar as taxas de prevalência relacionadas ao bruxismo em populações adultas e/ou pediátricas;
- Determinar os fatores endógenos e/ou exógenos associados ao bruxismo;
- Verificar a acurácia de instrumentos diagnósticos para bruxismo, tais como auto-relato, questionários, exame físico, dispositivos portáteis e exames de eletromiografia ou polissonografia;
- Investigar os efeitos do bruxismo sobre estruturas estomatognáticas;
- Avaliar a efetividade de diferentes terapias para o manejo do bruxismo;

4 ARTIGO

Artigo formatado conforme as normas da revista *Journal of Oral Rehabilitation* (acessadas em: 01/06/2018), exceto em relação ao idioma.

Bruxism: an overview of systematic reviews

Running title: Bruxism overview of systematic reviews

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CONFLICT OF INTEREST
Authors have no conflicts of interest to declare.

ABSTRACT

Objectives. To summarize and critically appraise available evidence from systematic reviews (SR) regarding sleep bruxism (SB) and/or awake bruxism (AB). **Methods.** SRs that investigated any bruxism-related outcome were considered eligible and selected in a two-phase process. Searches were performed on seven main electronic databases and on three grey literature databases. Risk of bias was assessed using the "University of Bristol's tool for assessing risk of bias in SR". **Results.** From 1038 identified studies, 49 SRs were included. Overall, three SRs were related to prevalence rates, eighteen to associated factors, two to diagnostic accuracy of assessment tools, ten to effects on stomatognathic structures, and 16 to interventions' effectiveness. Findings from SRs suggested that 1) among adults, prevalence of AB was 22-30%, SB (1-15%), and SB among children and adolescents (3-49%); 2) factors strongly associated with bruxism were use of alcohol, caffeine, tobacco, some psychotropic medications, esophageal acidification, and second-hand smoke; temporomandibular disorder signs and symptoms presented plausible association; 3) portable diagnostic devices showed the overall highest values of specificity (0.83-1.00) and sensitivity (0.40-1.00); 4) bruxism might result in biomechanical complications regarding dental implants, however, evidence was inconclusive regarding other dental restorations and periodontal damage; 5) occlusal appliances were considered effective for bruxism management, although current evidence was considered weak regarding other therapies. **Conclusions.** There are plenty of SRs assessing SB related outcomes, however, only one SR investigating AB separately from SB was found. Moreover, poor reliability related to bruxism diagnostic methods was considered a limitation across the majority of included SRs.

Keywords: Evidence-based dentistry; bruxism; sleep bruxism; awake bruxism; systematic review; overview.

INTRODUCTION

Depending on its circadian manifestation, bruxism may be sub-divided into sleep bruxism (SB) or awake bruxism (AB) and may be defined as a repetitive jaw-muscle activity characterized by clenching or grinding of the teeth and/or by bracing or thrusting of the mandible.[1] Although some concerns have been raised in the last years about bruxism definition and management, it appears there is some agreement regarding bruxism as a behavior or phenomenon rather than a disorder.[2, 3]

Even though high variability exists due to a lack of standardized diagnostic methods, epidemiological studies have shown that prevalence rates among adults may range from 10-13% for SB and 22-31% for AB[4]; in younger populations, however, bruxism could be more frequent, affecting up to 40-50% of studies' participants.[5, 6]

It has been proposed that bruxism etiology may be multifactorial and that several underlying mechanisms might play a role in its genesis, such as psychosocial (e.g stress and anxiety), physiological (e.g. genetics), and exogenous factors (e.g. alcohol consumption, medication use, smoking).[7, 8] More importantly, although existing knowledge is still limited, associated factors are thought to be distinct regarding both circadian manifestations of bruxism; whilst psychosocial aspects appears to have some influence on AB,[9] autonomic/central nervous system activation might be the primary factors involved in SB genesis.[10]

Although AB is considered more prevalent, SB is the one that has been most studied, nonetheless, there is a scarcity of reliable and valid diagnostic methods for detecting both conditions.[11] According to a recent systematic review (SR), despite some internal validity concerns, polysomnography (PSG) exam is still considered the reference-standard for SB diagnosis, whilst questionnaires, clinical exams, and portable diagnostic devices may be used as screening tools.[12] So far, no SR have investigated the validity of diagnostic tools regarding AB, although, for a definite diagnosis, an electromyography (EMG) exam is recommended.[11]

Moreover, both forms of bruxism might be harmful to the stomatognathic structures,[3] and some of the most reported harmful effects includes abnormal tooth wear, mobile teeth, and problems with dental restorations, implants, or fixed/removable prostheses.[13] It is worth mentioning that despite the numerous reports regarding bruxism negative

effects on oral health outcomes, the literature is still controversial, especially due to diagnostic limitations of the majority of studies.[2]

Nonetheless, in daily practice, clinicians are required to make decisions on the most suitable approach to manage bruxism, which includes recognizing whether or not a treatment is needed.[14, 15] Therefore, although there is no definitive treatment, some therapies might be useful in the management of this condition, including approaches like 1) occlusal appliances; 2) pharmacological treatments; 3) behavioral therapies; and 4) miscellaneous approaches (e.g. physical therapy).[16] It must be pointed out that despite many therapies are at clinicians disposal, evidence regarding some therapeutic methods is often weak, and therefore caution should be exercised.[17]

Numerous SR investigating the bruxism have been performed, especially in the last decade, however, a synthesis and appraisal of these reviews have not yet been performed. Therefore, the purpose of this overview was to summarize available evidence and answer the following focused question: "What do we currently know so far regarding bruxism about prevalence rates among different populations, associated factors, diagnostic accuracy of assessment tools, effects on stomatognathic structures, and interventions' effectiveness?"

MATERIALS AND METHODS

Protocol and registration

A SR protocol based on the Preferred Reporting Items for Systematic Reviews and Meta-Analyses Protocols (PRISMA-P)[18] was elaborated and registered at Prospective Register of Systematic Reviews (PROSPERO),[19] being made publicly available under the registration number CRD42018088560. In addition, the reporting of this study was based on the PRISMA checklist.[20]

Eligibility criteria

SRs and meta-analyses (MA) that investigated any bruxism-related outcome were considered eligible. Furthermore, studies were considered SRs if they matched the following description, as proposed by the Cochrane Collaboration's Handbook (chapter 1.2.2)[21]: "It uses explicit, systematic methods that are selected with a view to minimizing bias, thus providing more reliable findings from which conclusions can be drawn and decisions made". No time and language restriction were applied.

The exclusion criteria were based on the following: 1) SRs in which outcomes were not directly related to sleep and/or awake bruxism; 2) Studies that did not meet the minimum criteria regarding SRs; 3) Interventional studies, observational studies, laboratory research, abstracts, case-reports, protocols, personal opinions, letters, and posters, and 4) Full-text not available.

Information sources and search

Appropriate search strategies were elaborated and adapted for each of the following electronic databases: EMBASE, Latin American and Caribbean Health Sciences (LILACS), LIVIVO, PubMed, SCOPUS, The Cochrane Library, and Web of Science. In addition, a grey literature search was conducted on Google Scholar, OpenGrey, and ProQuest. All electronic database searches were performed from the starting coverage date through May 21, 2018. More information in regards to search strategies was provided in Appendix 1.

Reference lists of included SR were hand-searched to identify additional relevant papers, as proposed by Greenhalgh and Peacock.[22] A computer software was used to manage references (EndNote X7, Thomson Reuters).

Study selection

A two-phase selection process was performed; in phase-one, three reviewers (G.M.; J.D.; and P.P.) independently screened titles and abstracts to identify eligible studies using an online software (Rayyan, Qatar Computing Research Institute). Afterward, in phase-two, a full-text reading of eligible studies was performed by the same three reviewers. Any discrepancies were resolved by a consensus discussion and a fourth reviewer (A.L.P.) was involved to make a final decision, if necessary. Studies were included for qualitative analysis if minimum eligibility criteria were met.

Data collection process and data items

Three reviewers (G.M.; J.D.; and P.P.) independently collected pertinent data; information was then cross-checked to warrant integrity of contents. The following key features were collected regarding included SR: authors, year of publication, objectives or research questions, databases searched, number of included primary studies, risk of bias assessment tools, main results, and main conclusions. In addition, one reviewer (G.M.) collected data regarding included primary studies within SR and information was summarized in five supplementary tables (Supplementary Tables 1-5).

Risk of bias in individual studies

Risk of bias was independently assessed by three reviewers (G.M.; J.D.; and P.P.) using the University of Bristol's tool for assessing risk of bias in SR (ROBIS).[23] This tool targets four domains through which bias may be introduced into a SR: 1) study eligibility criteria; 2) identification and selection of studies; 3) data collection and study appraisal, and 4) synthesis and findings. In addition, each domain presents 5-6 signaling questions, of which possible answers were: "Yes (Y)", "Probably Yes (PY)", "Probably No (PN)", "No (N)", "Not Informed (NI)", or "Not Applicable (NA)".

Decisions about the scoring system and cut-off points were agreed upon by all reviewers prior to bias assessment. The grading system regarding bias within each domain were determined by the authors, according to the following: "low risk" if all signaling questions were scored as Y/PY, "unclear risk" if a single question was judged as PN/N/NI, and "high risk" if more than one question was judged as PN/N/NI. Furthermore, overall risk of bias regarding each SR was judged according to the following: 1) low, if all four domains were judged as "low risk" or only one as "unclear risk"; 2) moderate, if two or more domains were judged as "unclear risk"; and 3) high, if one or more domains were judged as "high risk".

In addition, the software RevMan 5.3 (Review Manager 5.3, The Cochrane Collaboration) was used to generate figures, which were edited by Adobe Photoshop CS6 (Adobe Systems Incorporated).

Summary measures and synthesis of results

A qualitative analysis of results was performed based on:

1) Prevalence rates, considering quantitative data reported in relative or absolute frequencies as main summary measures;
2) Associated factors, considering summary measures such as hazard ratio (HR), odds ratio (OR), relative risk (RR), and qualitative data;
3) Diagnostic accuracy of bruxism assessment tools, of which measures of sensitivity and specificity were considered.
4) Effects on stomatognathic structures, through relative or absolute frequencies, HR, RR, OR, and qualitative data;
5) Interventions' effectiveness, through relative or absolute frequencies, standardized or weighted mean differences, RR, and qualitative data.

In addition, evidence was considered "insufficient", "plausible", or "consistent" based on the conclusions of included SRs and overall risk of bias assessed by ROBIS.

Risk of bias across studies

Bias across studies was assessed by comparing variability among primary studies' methods (such as bruxism diagnostic methods and strength of evidence) and also by comparing risk of bias in individual SRs.

RESULTS

Study selection

From a total of 2140 references identified on electronic databases searches, 1038 remained after duplicates had been removed. Papers from grey literature were already within other databases, so no additional references were included. In phase-one, the title and abstract of identified studies were assessed, and 112 articles were considered eligible for full-text reading. Thereafter, 49 SRs were finally included for qualitative synthesis; further information regarding reasons for studies' exclusion is available in Appendix 2. Moreover, the complete process of studies' identification and selection is provided in Figure 1.

Study characteristics

Overall, three SRs investigated prevalence rates among different populations,[4-6] eighteen investigated associated factors,[24-41] ten evaluated effects on stomatognathic structures,[42-51] two evaluated diagnostic accuracy of bruxism assessment tools,[12, 52] and 16 assessed interventions' effectiveness.[17, 53-67] Statistical pooling of data using meta-analysis was available in 8 studies.[12, 35, 36, 42, 43, 51, 67] Regarding language of publication, most reviews were published in English, one in German,[39] and one in Portuguese.[29] Moreover, all SRs were published between 2007 and 2018. Overall characteristics of included SR are available in Table 1.

In addition, a total of 279 primary studies were identified within all SRs, from which 70 were cited twice across reviews, fourteen were cited three times, and one was cited four times (Figure 2). More information regarding primary studies is available in Supplementary Tables 1-5.

Risk of bias within studies

Overall, eleven SRs were judged with low risk,[12, 24, 25, 27, 41, 43, 56, 58, 60, 61, 67] eighteen with moderate risk,[4, 6, 17, 26, 28, 30-33, 35, 36, 46, 51, 52, 54, 55, 65, 66] and 20 with high risk of bias.[5, 29, 34, 37-40, 42, 44, 46-50, 53, 57, 59, 62-64] Major concerns

regarding risk of bias were observed, which included: 1) lack of *a priori* registration of the study protocol; 2) inappropriate range of database/electronic sources searched; 3) no risk of bias assessment; 4) study selection, data collection, or bias assessment performed by only one reviewer; 5) no publication bias assessment or sensitivity analysis; 6) high risk of bias in included primary studies. It is worth mentioning that a considerable number of primary studies were considered biased mainly due to inappropriate or poor bruxism diagnostic criteria. More details regarding risk of bias assessment is available in Figure 3 and Appendix 3.

Results of individual studies

Prevalence rates

From 3 SRs that had prevalence rates as primary outcomes, two investigated SB regarding young populations (children and adolescents) and the prevalence of SB in these studies ranged from 3.5%[6] to 49.6%.[5, 6] Moreover, a single SR investigated the prevalence of bruxism in adult populations and, overall, prevalence of generically identified bruxism ranged from 8% to 31.4%, AB from 22.1% to 31%, and SB 1.1% to 15.3%.[4] It is worth mentioning that two studies reported that SB prevalence decreased with age.[4, 6]

Associated factors

Five SRs investigated children and adolescents exclusively, [26, 30, 32, 35, 36] of which one concluded that available evidence was considered insufficient to credit or discredit any association between tension-type headache and migraine with SB.[30] Other SRs, based on consistent evidence, proposed that bruxism was associated with second-hand smoke,[26, 36] sleep disturbances,[26] and psychosocial factors.[32, 36] It is worth mentioning that two SRs investigated a wide range of sleep behaviors and risk factors and, based on consistent evidence, proposed that some were associated with bruxism in children, including snoring, mouth breathing, restless sleep, and others.[35, 36]

Association between bruxism and temporomandibular disorders (TMD) was assessed in 3 SRs[29, 37, 40]; evidence was considered insufficient or plausible in all 3 SRs. Manfredini *et al.* (2010)[40] suggested that investigations based on self-report or clinical bruxism showed a plausible association with TMD pain, however, potential bias and cofounders at diagnostic level were major concerns in included studies. Later, Cunali *et al.* (2012)[29] concluded that evidence was insufficient to support an association between SB in particular and TMD, whilst Jiménez-Silva *et al.* (2017)[37]

suggested that bruxism (SB or AB) could be plausibly associated with myofascial pain, arthralgia, and joint pathology (disc displacement and joint noises).

Regarding sleep breathing disorders in adult populations, De Luca Canto *et al.* (2014)[31] suggested that available evidence was insufficient to credit or discredit an association with SB. Similarly, a more recent SR (2018)[38] concluded that there are not enough scientific data to define a clear causative link between obstructive sleep apnea (OSA) and SB, although some clinical features appear to be common in both conditions.

Considering miscellaneous risk factors, history of SB during childhood, gastro-esophageal reflux disease, and genetic polymorphisms were considered consistent risk factors for SB in adults, as suggested by Castroflorio *et al.* (2017).[27] The association between signs and symptoms of bruxism and presence of tori was evaluated by Bertazzo-Silveira *et al.* (2017)[25] and, based on consistent evidence, it was suggested that abnormal tooth wear (not necessarily bruxism) was associated with torus, specially torus mandibularis.

Considering exogenous factors, Feu *et al.* (2013) suggested that smoking is consistently associated with SB in a dose-dependent manner and that esophageal acidification could also induce SB.[33] Similarly, a more recent SR (2017) [24] proposed that use of alcohol, caffeine, and tobacco were also consistently associated with SB. With regard to stress related outcomes, one SR[39] proposed that increased distress in everyday life, as generically described by the authors, could be a plausible risk factor for SB. Moreover, salivary cortisol levels (i.e. hormones related to stress) were investigated by Cruz *et al.* (2016),[28] however, no conclusive evidence regarding a possible association with bruxism was found.

Furthermore, two SR evaluated the possible association between bruxism and use of several psychotropic medications. Garret *et al.* (2018),[34] based on insufficient evidence from case-reports, suggested that antidepressant-associated bruxism may plausibly occur in pediatric and adult patients and that fluoxetine, sertraline, and venlafaxine were the most commonly reported agents. Moreover, Melo *et al.* (2018)[41] suggested that SB might be consistently associated with use of duloxetine, paroxetine, and venlafaxine among adults, whilst barbiturates and methylphenidate may exhibit a consistent association with the presence of SB among younger populations.

Diagnostic accuracy

Two SRs were identified regarding diagnostic accuracy of bruxism assessment tools. Manfredini *et al.* (2010)[52] evaluated portable diagnostic devices in particular (e.g. BiteStrip, and Bruxoff), reporting that evidence was still scarce to support any non-PSG technique and that further investigations on the topic are necessary. Moreover, Casett *et al.* (2017)[12] updated existing literature about portable devices and further evaluated diagnostic accuracy of questionnaires and clinical examinations compared to the reference standard PSG. Findings from this SR suggested that portable devices had the highest values of specificity (0.83-1.00) and sensitivity (0.40-1.00) of all methods, whilst questionnaires and clinical examinations presented somewhat similar specificity (0.68-0.99) but overall poorer sensitivity (0.13-0.94).[12]

Effects on stomatognathic structures

Five SRs investigated the effects of bruxism or generically identified "parafunctional habits" regarding dental implants, [42, 44, 45, 47, 51] from which bruxism was the main outcome in three of these studies.[42, 45, 51] Manfredini *et al.* (2014)[45] suggested that bruxism is unlikely to be a risk factor for biological complications regarding dental implants, whilst it may be a plausible risk factor for mechanical complications. Chrcanovic *et al.* (2015),[42] on the other hand, concluded that the effects of bruxing habits on the osseointegration and survival of endosteal dental implants are still not well established. Moreover, Zhou *et al.* (2016)[51] suggested that bruxism is a plausible contributing factor to dental implant technical/biological complications and plays a role in dental implant failure. In addition, although bruxism was not the primary outcome in the studies of Salvi *et al.* (2009)[47] and Hsu *et al.* (2012),[44] these SRs suggested that generically identified bruxism[47] or "bruxism/parafunctional habits"[44] were plausibly related to increased biomechanical complications related to dental implants.

Three SRs assessed the effects of bruxism on dental restorations. Schmitter *et al.* (2014)[48] concluded there is a lack of information about the effect of bruxism on the incidence of technical failure of veneered zirconia restorations. Melo *et al.* (2017)[43] concluded that available evidence did not favor any association between SB and increased odds of failure for ceramic restorations. Although bruxism was not the primary outcome in the study of Van de Sande *et al.* (2016),[49] the role of "bruxism or parafunctional habits" (as generically described by the authors) on restorations survival was

assessed, however, since conflicting results were reported, evidence was considered inconclusive. With regard to dental structures in particular, although bruxism was not the primary outcome in the study of Van't Spijker *et al.* (2007),[50] it was suggested that dental attrition seems plausibly coexistent with self-reported bruxism.

A single SR investigated possible harmful effects of bruxism on the periodontium, however, the authors (based on scarce quantity and quality of available literature) concluded that current evidence points out that bruxism cannot cause periodontal damage *per se*, although more and better studies were recommended to further clarify this issue.[46]

Interventions' effectiveness

The following therapeutic methods were assessed in included SR: a) occlusal appliances, b) pharmacological therapies (including botulinum toxin injections); c) biofeedback therapies; and d) miscellaneous therapies (e.g. prosthetic rehabilitation, adenoidectomy).

Regarding occlusal appliances, Macedo *et al.* (2007),[60] in a Cochrane review, concluded that available evidence was insufficient to state that occlusal splint is effective for SB management. Moreover, Stapelmann *et al.* (2008)[66] concluded nociceptive trigeminal inhibition tension suppression system (NTI-TSS) device might present plausible effectiveness on the management of bruxism. Furthermore, the most recent SRs on occlusal appliances reported that, although many studies support the efficiency of these devices for SB management, evidence was insufficient to support its role in the long-term reduction of SB activity, and further long-term studies are necessary.[55]

With regard to pharmacological therapies, Martin *et al.* (2012)[64] evaluated the effects of antidepressants on several facial pain disorders, concluding that the limited evidence makes the administration of antidepressants questionable. Similarly, Macedo *et al.* (2014),[61] in a Cochrane review, suggested that evidence was still insufficient on the effectiveness of pharmacotherapy for the treatment of SB. In addition, the most recent review on antidepressants (2017)[58] suggested that its efficacy has not yet been validated for cases of bruxism. Moreover, regarding botulinum toxin injections in particular, both studies of Long *et al.* (2012)[59] and De La Torres Canales *et al.* (2017)[53] suggested that this therapeutic method may reduce bruxism intensity, which might present plausible effectiveness in the management of this condition.

Concerning biofeedback therapies, Wang *et al.* (2014)[67] concluded that there was no powerful evidence to support the use of biofeedback technology on SB treatment. On the other hand, Jokubauskas *et al.* (2018)[56] updated the literature on the topic and suggested the contingent electrical stimulation (CES), one of the biofeedback modalities, was plausibly effective in reducing SB-related motor activities after a short-term treatment period. It is worth mentioning that the authors concluded that evidence of long-term effects was lacking, and therefore further studies are necessary.

Two SRs evaluated multiple treatment methods; Machado *et al.* (2011)[62] concluded there are lot of treatment options for SB, however many lacks scientific support. Similarly, Manfredini *et al.* (2015)[17] reported outcomes related oral appliances, pharmacological approaches, biofeedback and cognitive-behavioral approaches, and electrical stimulus to the masseter muscles. The authors concluded there was not enough evidence to define a standard of reference approach for SB treatment, except for the use of occlusal appliances.

Moreover, four SRs assessed miscellaneous therapies with regard to bruxism. Restrepo *et al.* (2009)[65] evaluated treatment of bruxism in children (including adenoidectomy and psychologic techniques), however, few studies met the quality criteria for evidence-based practice and the authors concluded that further investigations are required. Lang *et al.* (2009)[57] evaluated therapies for the management of bruxism in children with developmental disabilities, suggesting that evidence was extremely limited and no definitive statements regarding treatment efficacy can be made. Moreover, regarding prosthetic rehabilitation as treatment option for bruxism, the study of Manfredini *et al.* (2017)[63] revealed an absence of RCTs on the topic and, based on available evidence, prosthetic changes in dental occlusion were considered not yet acceptable strategies for bruxism management. It is worth mentioning that Hillier *et al.* (2015)[54] evaluated the effectiveness of the Feldenkrais method (a type of alternative exercise therapy which aims to improve self-awareness) and, based on a single bruxism-related primary study, suggested that it could present plausible effectiveness for children after a 10-week course of lessons.

Synthesis of results

AB was investigated separately from SB in a single SR assessing prevalence rates and, overall, was considered more prevalent than SB among adults. The other two prevalence SRs investigated SB in children and

adolescents, reporting highly heterogeneous prevalence-rates (3-49%). Associated factors were greatly heterogeneous among included SRs and bruxism was consistently associated gastro-esophageal reflux disease, esophageal acidification, and genetic polymorphisms. A plausible association was proposed regarding several TMD signs and symptoms. Exogenous factors consistently associated with bruxism were use of tobacco, alcohol, caffeine, and some psychotropic medications. The presence of tori was also consistently associated with abnormal tooth wear (not necessarily bruxism). In younger populations (children and adolescents), psychosocial factors and sleep disturbances were consistently associated with SB

With regard to diagnostic accuracy of assessment tools, considering the reference standard PSG, portable diagnostic devices showed the highest values of specitivity and sensitivity compared to questionnaires and clinical examination. Regarding effects to stomatognathic structures, whilst some SRs reported that bruxism may be a plausible risk factor for dental implants and implant-supported prostheses, available evidence did not credit or discredit any harmful effects of bruxism to other dental restoration or to the periodontium.

Although studies on the long-term were lacking, occlusal appliances were considered consistently effective for SB management. Pharmacological treatments, such as use of antidepressants, were overall not supported, however, some studies suggested that botulin toxin injections might reduce bruxism intensity and present plausible effectiveness. It is worth mentioning that SRs and primary studies investigating botulinum toxin were considered with high risk of bias and thus caution should be exercised. Overall, no recommendations on biofeedback therapy could be provided, with the exception of CES, which showed plausible effectiveness in the short-term management of SB.

Risk of bias across studies

A great variability was observed across included SRs. Regarding bruxism classification, most SRs investigated SB alone, several SRs used the generic term "bruxism" or "parafunctional habits", and a single investigated AB separately from SB.[4] In addition, bruxism diagnostic criteria were greatly heterogeneous; the majority of primary studies included in SRs have evaluated bruxism through questionnaires or clinical examinations, whilst few have adopted the use of PSG or EMG exams to confirm the diagnosis.

Considering associated factors, variables evaluated were often of different nature (e.g. exogenous and endogenous factors) across SRs and, therefore, not directly comparable. Short follow-up times were also observed in SRs evaluating bruxism effects on stomatognathic structures, which might hinder the assessment of possible harmful effects due to insufficient observation time. In addition, some SRs have pointed out that evidence of therapy effectiveness was limited to the short-term, thus long-term studies on the topic were recommended.

DISCUSSION

Summary of Evidence

This overview aimed to summarize and critically appraise current literature regarding bruxism-related SRs. Although evidence from SRs is usually considered of high quality, uncritically accepting the results of a single SR has risks, and some methodological flaws related to its methods might even generate inaccurate conclusions.[68] Therefore, caution should be exercised by healthcare practitioners and policy makers with regard to biomedical publishing and the need to improve standards in conducting and reporting SRs is highlighted.

Findings from SRs reporting bruxism prevalence rates were considered imprecise due to wide prevalence ranges observed. This may be due to inaccurate diagnostic methods, since several primary studies used single-question questionnaires to diagnose bruxism, especially in pediatric populations. Moreover, sample sizes were usually large, which might explain the lack of PSG and/or EMG exams.[1] Therefore, overall conclusions from epidemiological SR should be interpreted with caution.[4, 6]

With regard to factors associated with bruxism, primary studies included in SRs were considerable heterogeneous. However, it appears that current evidence from SRs is in accordance with previously proposed hypotheses regarding bruxism etiology, in which mechanisms involved in the genesis of this condition are distinct for both AB and SB.[8, 69] Whilst there is a lack of SRs investigating AB in particular, SB was associated with several variables that are proposed to affect central nervous system (CNS) neurotransmission pathways,[7] such as use of tobacco, alcohol, caffeine,[24] and some psychotropic medications.[41] Moreover, since bruxism diagnosis was based mostly on self-report and questionnaires, diagnostic limitations were a major concern across SRs investigating associated factors, and thus further

investigations with more accurate diagnostic methods are recommended to further explore this topic.

It is worth mentioning that Lobbezzo *et al.* (2018)[70] recently published a commentary regarding directions to which an updated consensus about bruxism definition and grading might be moving towards to. Overall, the authors pointed out that in otherwise healthy individuals, bruxism should not be considered as a disorder, but rather as a behavior that can be harmful or protective considering several clinical consequences. Moreover, regarding bruxism diagnostic criteria, it was proposed that both non-instrumental and instrumental approaches can be used to assess bruxism, however, further research is necessary to evaluate its clinical usefulness. In addition, the authors recommended that cut-off points for establishing presence or absence of bruxism should not be used in otherwise healthy individuals, rather, bruxism should be assessed considering a behavior's continuum classification. It is worth mentioning that these recommendations are proposals and not yet well established.

Current literature was considered absent regarding accuracy of diagnostic methods to assess AB, as no SRs on the topic were found. Moreover, although some SRs investigated methods to assess SB, it must be pointed out that a clear definition regarding bruxism as a behavior or a disorder is not yet well stablished.[2, 3, 15] Depending on future consensus updates, there may be a reappraisal of PSG criteria, which are currently used as reference for SB diagnosis.[12]

Several SRs proposed that poor homogeneity of primary study, as well as bruxism diagnostic methods, may hinder the evaluation of complications related to the stomatognathic structures, such as dental implants,[45] restorations,[43] and the periodontium.[46] In addition, retrospective studies were observed in several SRs, which could introduce bias related to gaps in information and incomplete records.[42] Further prospective studies with appropriate follow-up times and diagnostic methods are recommended in order to investigate possible harmful effects of bruxism to stomatognathic structures.

Current evidence regarding interventions for the management of bruxism is still inconclusive, as previously described by Lobbezzo *et al.* (2008).[16] Effectiveness of occlusal appliances in managing SB signs and symptoms was consistent across included SRs, however, it should be mentioned that primary studies with longer follow-up time spans are

necessary to assess its effects on the long-term.[17] There was not enough evidence to propose any recommendation regarding pharmacological treatment of bruxism, although some SRs proposed that botulinum toxin injections might present plausible effects on SB intensity reduction.[53, 59] However, it should be mentioned that real improvements in muscle pain levels might not be superior to placebo,[17] thus further studies are necessary to evaluate possible beneficial effects of botulinum toxin in bruxism management.

Moreover, evidence regarding biofeedback therapies was not strong enough to suggest real benefits on bruxism management,[67] with the exception of CES.[17, 56] Although stand-alone effectiveness of these therapies is somewhat doubtful, given its non-harmful nature, some authors recommended its inclusion in SB treatment protocols as a multimodal approach.[17] In addition, overall recommendations regarding future studies investigating bruxism therapies could be proposed, which include *a priori* calculation of an adequate sample size, accurate and valid methods to assess bruxism, and preferably randomized and double-blinded study designs.

Although SRs are considered to provide the most reliable form of evidence, systematic flaws or limitations in the design or conduct of a SR may result in misleading or inaccurate conclusions. In addition, since SRs are vital in clinical decision making and resource allocation, consistent and unbiased standards are expected across SR investigating different topics and, therefore, efforts should be made to minimize or prevent potential sources of bias.[23]

Limitations

The authors of this overview acknowledge that inclusion criteria regarding SR definition was considerably broad. Since older SRs often did not present strictly rigorous methods, especially regarding bias assessment in primary studies, a more restrictive inclusion criteria would have excluded a considerable number of SRs. It must be pointed out that poor designed SR were dealt with by using the ROBIS tool, therefore, conclusions based on those should be interpreted with caution.

CONCLUSIONS

Based on current evidence, some conclusions may be drawn:

1) Among adults, prevalence of AB was 22-30%, SB (1-15%), and SB among children and adolescents (3-49%);

2) Major factors consistently associated with SB were use of alcohol, caffeine, tobacco, several psychotropic medications, esophageal acidification, and second-hand smoke. Several TMD signs and symptoms presented a plausible association with SB. In pediatric populations, sleep disturbances and psychosocial factors were consistently associated with SB.

3) Portable diagnostic devices showed the highest values of both sensitivity and specificity, whilst questionnaires and clinical examinations presented similar specificity, but considerably poorer sensitivity;

4) Bruxism might result in biomechanical complications related to dental implants and implant-supported prostheses, however, available evidence did not supported harmful effects regarding other dental restorations or periodontal damage.

5) Occlusal appliances were consistently considered effective for bruxism management. Evidence regarding botulinum toxin was considered biased, although plausible effectiveness was reported. No treatment recommendations regarding other pharmacological treatments and biofeedback therapy could be provided, with the exception of CES.

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Table 1 - Summary of overall descriptive characteristics of included systematic reviews (n=49).

Author (Year); Journal	Objectives or research question	Subgroup	Databases searched (Search date)	Included primary studies	Risk of bias assessment tools	Main results	Main conclusion
Machado et al. (2014); Dental Press Journal of Orthodontics	Prevalence of sleep bruxism in children	Prevalence rates	MEDLINE, Cochrane, EMBASE, PubMed, LILACS, and BBO (from Janyary, 2000 to February, 2013)	4 cross-sectional	Authors' judgment (no specific tool)	The prevalence rates of SB ranged from 5.9% to 49.6%, and these variations showed possible associations with the diagnostic criteria used for SB.	There is a small number of studies with the primary objective of assessing SB in children. Additionally, there was a wide variation in the prevalence of SB in children. Thus, further,

							evidence-based studies with standardized and validated diagnostic criteria are necessary to assess the prevalence of SB in children more accurately.
Manfredini et al. (2013); Journal of Oral Rehabilitation	Prevalence of sleep bruxism in children	Prevalence rates	PubMed, SCOPUS, Google Scholar, and four journal Publishers' website, including Elsevier, Wiley-Blackwell,	8 cross-sectional	MORE checklist	The reported prevalence was highly variable between the studies (3.5–40.6%), with a commonly described decrease with age and no gender differences.	A very high variability in sleep bruxism prevalence in children was found, due to the different age groups under

			Quintessence Publishing, and Springer (August, 2012)				investigation and the different frequencies of self-reported sleep bruxism. This prevented from supporting any reliable estimates of the prevalence of sleep bruxism in children.
Manfredini et al. (2013); Journal of Orofaccial Pain	Prevalence of bruxism in adult populations	Prevalence rates	PubMed, SCOPUS, and Google Scholar (February, 2011)	7 cross-sectional	MORE checklist	Generically identified "bruxism" was assessed in two studies reporting an 8% to 31.4% prevalence, awake bruxism was	Findings must be interpreted with caution due to the poor methodologi

						investigated in two studies describing a 22.1% to 31% prevalence, and prevalence of sleep bruxism was found to be more consistent across the three studies investigating the report of "frequent" bruxism (12.8% ± 3.1%).	cal quality of the reviewed literature and to potential diagnostic bias related with having to rely on an individual's self-report of bruxism
Casett et al. (2017); Journal of Oral Rehabilitation	Which is the validity of questionnaires, clinical assessment, and portable diagnostic devices in diagnosing SB, when compared to the reference	Diagnostic accuracy	EMBASE, LILACS, PubMed, Science Direct, and Web of Science (August, 2016)	8 diagnostic accuracy studies	QUADAS-2	The MA indicated that portable diagnostic devices showed the best validity of all evaluated methods, especially as far as a four-channel EMG/ECG recording is concerned.	Questionnaires and the clinical assessment can be used as screening methods to identify non-SB individuals, although it is not that good in

	standard PSG?						identifying subjects with SB.
Manfredini et al. (2014); Journal of Oral Rehabilitation	What is the validity of the different portable instrumental devices that have been proposed to measure SB if compared with PSG recordings assumed as the gold standard?	Diagnostic accuracy	MEDLINE, SCOPUS, and Google Scholar (April, 2014)	4 diagnostic accuracy studies	QUADAS-2	The positive predictive value (PPV) of the Bitestrip device was 59–100%, with a sensitivity of 71–84.2%, whilst EMG-telemetry recordings had an unacceptable rate of false-positive findings (76.9%), counterbalanced by an almost perfect sensitivity (98.8%). The Bruxoff device had the highest accuracy values, showing an excellent agreement with PSG for both manual (area under ROC = 0.98)	Available information on the validity of portable instrumental diagnostic approaches with respect to PSG recordings is still scarce and not solid enough to support any non-PSG technique's employ as a stand-alone diagnostic

						and automatic scoring (0.96) options as well as for the simultaneous recording of events with respect to PSG (0.89–0.91).	method in the research setting, with the possible exception of the Bruxoff device that needs to be further confirmed with future investigations.
Bertazzo-Silveira et al. (2016); Journal of the American Dental Association	In adults, is there any association between SB and alcohol, caffeine, tobacco, or drug abuse?	Associated factors	LILACS, PsycINFO, PubMed, Science Direct, and Web of Science (April, 2016)	2 cross-sectional studies 3 cohort studies 2 descriptive studies	MAStARI (different questionnaires according to study design)	In one study, the investigators noted a positive and weak association for heavy coffee drinkers. The odds for SB seem to increase almost 2 times for those who drank alcohol, almost 1.5 times for those who drank more than 8 cups of coffee per day, and more than 2	SB was associated positively with alcohol, caffeine, and tobacco. The association between the studied drugs could not be

						times for those who were current smokers. The abuse of methylenedioxymethamphetamine associated with SB remained without sufficient evidence.	discredited; however, there is still a need for stronger evidence based on studies with greater methodological rigor.
Bertazzo-Silveira et al. (2017); Clinical Oral Investigations	Is there an association between any specific signs and symptoms of bruxism and the presence of tori?	Associated factors	EMBASE, LILACS, PubMed, Science Direct, and Web of Science (May, 2016)	2 case-control studies 3 cross-sectional studies	MAStARI for observational studies	Self-report of teeth grinding and/or clenching presented contradictory results. Presence of abnormal tooth wear increased the odds of having tori, mainly for torus mandibularis. The overall quality of evidence ranged from low to very low.	The presence of abnormal tooth wear might be associated with tori, mainly torus mandibularis. There is no sufficient evidence to credit or discredit the association

							of tori and other signs and/or symptoms of bruxism.
Castroflorio et al. (2015); Archives of Oral Biology	1. Which are the identified risk factors for bruxism in children? 2. Which is the weight of each risk factor?	Associated factors	PubMed, EMBASE, Scopus, Cochrane Oral Health Group's Trial Register and Cochrane Register of Controlled Trials, Web of Science, LILACs, SciELO (1950 to March, 2015)	3 case-control studies 2 cross-sectional studies 1 RCT	Simplified GRADE checklist	One randomized clinical trial suggested the increase of SB in heavily exposed patients to second hand smoke (OR = 4.5, CI = 2.2–9.4), two cross-sectional studies suggested neuroticism as determinant factor for the development of sleep bruxism (OR = 1.9, CI = 1.3–2.6), among children and three case-control studies suggested that children with sleep disturbances were more likely to have	Second hand smoke and sleep disturbances presented the strongest association with SB. The most recurrent source of bias was the lack of blinding procedures. Furthermore, the use of reliable SB diagnostic procedures should be

						SB (OR = 3.3, CI = 1.6–6.6). Parafunctional behaviours (OR = 2.3, CI = 1.2–4.3) had a moderate association.	recommend ed to increase the quality of future studies.
Castroflori o et al. (2017); Archives of Oral Biology	1. Which are the identified risk factors for SB in adults? 2. Which is the weight of each risk factor?	Associated factors	PubMed, EMBASE, Scopus, Cochrane Oral Health Group's Trial Register and Cochrane Register of Controlled Trials, Web of Science, LILACs and SciELO (March, 2017)	3 case-control studies 5 cross-sectional studies 1 RCT	Simplified GRADE checklist	Among the nine analyzed articles, associations between SB and gastro-esophageal reflux disease (GERD) (OR =6.6, CI =1.4–30.9) was found in one randomized clinical trial (RCT). Four cross-sectional studies suggested history of SB during childhood (OR =8.1 CI= 5.4–12–2), age (OR =3.1, CI =2.3–4.1) and chronic migraine (OR =3.8, C.I =1.8–7.8) as	History of SB during childhood, gastro-esophageal reflux disease and genetic polymorphis ms seem to be important risk factors associated to SB in adults. Dry mouth on awakening seems to be a protective

		determinant factors for the development of SB. In one case-control study, patients with genetic polymorphisms were more likely to present SB (OR =4.3, CI =1.6–11.3). Smoking (OR =2.8, CI=2.2–3.5) and alcohol intake (OR =1.9, CI =1.2–2.8) showed moderate association in two case-control studies.	factor. Association does not infer with causality. Even if the evidence emerged from the considered studies was clinically relevant, further studies are requested to better understand the biological mechanisms behind the described associations.

Cruz et al. (2016); International Journal of Odontostomatology	Verify the existence of scientific evidence of association between the daytime and/or nighttime bruxism and levels of salivary cortisol.	Associated factors	PubMed; OVID and VHL (Virtual Health Library, LILACS, IBECS; MEDLINE and Scielo (January, 2016)	2 cross-sectional studies	New Castle-Ottawa SCALE for cross-sectional studies modified by Herzog et al. (2013) (reference in original article)	Two articles were included in this review. One of them showed moderate positive correlation between the BiteStrip scores and the levels of salivary cortisol in patients with bruxism. On the other hand, the other research demonstrated that children with sleep bruxism are more likely to have low levels of salivary cortisol	There is no conclusive evidence of association between bruxism and salivary cortisol.
Cunali et al. (2012); Revista Dor *	Verify the possible association between sleep bruxism and temporomandibular joint disorders	Associated factors	MEDLINE, Cochrane, EMBASE, PubMed, LILACS, and BBO (January, 2000 to	3 cross-sectional studyes 1 longitudinal study	No risk of bias assessment	Evaluated studies were unable to establish a positive relationship between SB and TMD when keywords sleep bruxism, temporomandibular	Not enough evidence to support an association between SB and TMD.

			August, 2012)			disorders and polysomnography were crossed; however they reinforce the need for referring TMD patients with sleep disorders to polysomnographic evaluation.	
De Luca Canto et al. (2014a); Headache	Evaluate and synthesize the possible association between the most common primary headaches disorders (TTH and migraine) with SB.	Associated factors	The Cochrane Library, MEDLINE, EMBASE, PubMed, LILACS, and Google Scholar (January, 2014)	2 cross-sectional studies	QUIPS	The presence of SB significantly increased the odds (study 1: odds ratio [OR] 3.12 [1.25-7.7] and study 2:OR 3.8; 1.83-7.84) for headaches, although studies reported different headache type.	There is not enough scientific evidence to either support or refute the association between tension-type headache and migraine with SB in children. Adults with

							SB appear to be more likely to have headache.
De Luca Canto et al. (2014b); Journal of Orofacial Pain	Evaluate the association between SB and sleep-disordered breathing	Associated factors	MEDLINE, PubMed, Embase, the Cochrane Library, and LILACS (October, 2013)	1 experimental bruxism study	Qu-ATEBS	Only one study was finally selected for the qualitative/quantitative synthesis. This study did not support the putative association between SB and sleep-disordered breathing, since SB was not observed during or in temporal conjunction with snoring or apneic events in any of the evaluated patients. In addition, masseter activity was not observed during apneic episodes.	There is not sufficient scientific evidence either to confirm or discredit the association between SB and sleep-disordered breathing.

De Luca Canto et al. (2015); Clinical Pediatrics	Evaluate whether SB is associated with psychosocial factors in children and adolescents	Associated factors	Cochrane, EMBASE, MEDLINE, PubMed, Virtual Health Library (BVS -Database that include articles in Spanish and Portuguese from MEDLINE, LILACS, Wholis, BBO and AdoLec), and Google Scholar (Search date not reported)	4 case-control studies 3 other studies	QUIPS	No evidence supportive of an association between sleep bruxism and psychosocial factors in children younger than 5 years emerged. A significant association was present in children between 6 and 11 years old and in adolescents 12 to 17 years old. Risk of bias was low–to-moderate in most of the included studies	The current available evidence suggests an association between sleep bruxism and psychologic al factors in children older than 6 years.
Feu et al. (2013); Journal of Orthodont ics	To examine whether risk factors for bruxism can be identified	Associated factors	Cochrane Library, Medline, and Embase	1 double-blind clinical trial	Cochrane Collaboratio n risk of bias tool	There is some evidence that: 1. Disturbances in the central dopaminergic system are implicated	There is convincing evidence that (sleep-related)

	in children and adults.	(1980 to 2011)	1 cross-over, randomized, single-blinded trial 3 longitudinal studies	in the etiology of bruxism; 2. SB can be induced by esophageal acidification. 3. An important dose-dependent relationship exists between smoking and bruxism, and this is a behavior that may persist for long periods in some individuals. 4. the proposed role of stress and other psychological factors, such as affective disturbance and anxiety seems to be small in all probability, if present at all.	bruxism can be induced by esophageal acidification and also that it has an important relationship with smoking in a dose-dependent manner. Disturbances in the central dopaminergic system are also implicated in the etiology of bruxism.

Garret et al. (2018); Neurology Clinical Practice	The objective of this article was to review the existing literature for the clinical features of antidepressant associated bruxism, to identify common offending agents, and to explore successful treatment strategies.	Associated Factors	PubMed (Search date not reported)	37 case-reports	No risk of bias assessment	Antidepressant-associated bruxism may occur in pediatric and adult patients, most commonly among female patients. Patients may develop symptoms with short-term and long-term antidepressant use. Fluoxetine, sertraline, and venlafaxine were the most commonly reported offending agents. Symptoms may begin within 3–4 weeks of medication initiation and may resolve within 3–4 weeks of drug discontinuation, addition of buspirone, or substitution with another	Antidepressant-associated bruxism may be an underreported condition, particularly in the neurology clinic. Further prospective trials may help to elucidate optimal therapies for this condition.

						pharmacologic agent. The incidence of this phenomenon is unknown.	
Guo et al. (2017); Sleep & Breathing	What sleep behaviors are associated with bruxism in children?	Associated factors	Pubmed, Excerpta Medica Database (Embase), Cochrane Library database, Web of Science, Chinese National Knowledge Infrastructure (CNKI), Chinese Biomedical Literature Database (CBM), and Wanfang	11 case-control studies 3 cross-sectional studies	1. Newcastle-Ottawa Scale on case-control studies 2. Criteria of the cross-sectional/prevalence study quality (reference in original article)	Of 5637 initially identified articles, 14 met inclusion criteria. Study qualities of all case-control studies were high. Quality of cross-sectional studies was more variable. The pooled ORs, 95% CIs, and P values were as follows: snoring (2.86, 1.85–4.42, <0.0001), mouth breathing (1.51, 1.04–2.18, 0.029), restless sleep (2.31, 1.89–2.83, <0.0001), drooling (1.79, 1.07–2.97, 0.026), stomach position during sleep	Snoring, mouth breathing, restless sleep, drooling, stomach position during sleep, and lack of sleep were the risk factors related to bruxism in children.

			Data (WF) (September, 2016)			(1.70, 1.0–2.39, 0.003), and inadequate sleep time (2.56, 1.48–4.43, 0.001).	
Guo et al. (2018); Archives of Oral Biology	The risk factors related to bruxism in children	Associated factors	Pubmed, Embase, Cochrane Library database, Web of Science, CNKI, CBM, and WF (October, 2016)	18 case-control studies 2 cohort studies 1 RCT	1. Newcastle-Ottawa Scale on cohort and case-control studies 2. Cochrane risk of bias table	Gender, age, gene, mixed position, anxiety, the nervous, secondhand smoke, high psychological reactions, responsibility, move a lot during sleep, sleeps with mouth open, snores loudly, restless sleep, sleep hours, sleep with light on, noise in room, headache, biting, cheeks tonus, perioral musculature participation, conduct problems, peer problems, emotional symptoms, mental health problems,	The risk factors related to bruxism were as follows: Male, gene, mixed position, moves a lot, anxiety, the nervous, psychological reactions, responsibility, secondhand smoke, snore loudly, restless

						birth weight, occupation of family head, maternal marital status, hyperactivity, family income seemed to have statistical significance from the present systematic review and meta-analysis	sleep, sleep with light on, noise in room, "sleep hours, ≤8 h", headache, objects biting, conduct problems, peer problems, emotional symptoms and mental health problems.
Jiménez-Silva et al. (2017); Acta Odontologica	Sleep and awake bruxism in adults and its relationship with temporomand	Associated factors	PubMed, Cochrane Library, Medline, Embase, BIREME, Lilacs and	34 case-control studies 5 cohort studies	Newcastle-Ottawa Scale for case-control and cohort studies	Thirty-nine studies (n=39) were analyzed in this review. According to bruxism diagnosis, articles were grouped as follows:	The evidence based on PSG was not as conclusive as the

Scandinavica	ibular disorders	Scielo (From 2003 and 2014)	polysomnographic diagnosis (PSG) (n=7), clinical diagnosis (n=11) and survey/self-report (n=21). Thirty three articles (n=33) established a positive relation between bruxism and TMD and six (n=6) did not. Quality of evidence was low to moderate. In general, the most part of the studies showed shortcomings on their design with bias risk, and also had a low sensitivity on bruxism diagnosis.	studies that used surveys and clinical exam to diagnosis bruxism, when bruxism was related to TMD. Sleep bruxism could be associated with myofascial pain, arthralgia and joint pathology as disc displacement and joint noises. Although the evidence

							at present is inconclusive and does not provide information according to the type of bruxism (bruxism sleep and wakefulness), it is possible to suggest that bruxism would be associated with TMD.
Jokubauskas et al. (2017); Journal of Oral Rehabilitation	What is the relationship between OSA and SB, which can be determined using full-night	Associated factors	PubMed, ScienceDirect, Wiley Online Library, SAGE Journals, and EBSCOhost	3 experimental bruxism studies	Qu-ATEBS	Two studies gave evidence that OSA is associated with the occurrence of SB events: (i) SB events frequently occur during micro-arousal events consequent on	There are not enough scientific data to define a clear causative link

	polysomnography (PSG), in adult patients diagnosed with OSA and/or SB?		(January, 2006 to September, 2016)			apnoea–hypopnoea (AH) events and (ii) most SB events occur in temporal conjunction with AH events termination. However, one study did not report a strong association between AH and SB events.	between OSA and SB. However, they appear to share common clinical features. Further studies should focus on the intermediate mechanisms between respiratory and SB events.
Kulis et al. (2008); Schweizer Monatsschrift für Zahnmed*	What variables have been identified as risk factors for sleep and / or awake	Associated factors	PubMed, MEDPILOT. DE (URL: www.medpilot.de), publisher database the	6 cross-sectional studies 1 longitudinal study	No risk of bias assessment	1. Three variables – severe stress experience; age between 25 and 44 years; age between 45 and 64 years – were grouped into	Considering the risk factors in categories A and B, it is apparent that the only

	bruxism in adults?	German doctors Publishing (URL: www.dzz.de), publishing database of Quintessence Publishing (URL: www.quintessenz.de) and Google Scholar (June, 2007)		category A (very strong indication for clinically relevant risk factor: OR > 2; CILL > 2). 2. Five variables fell into category B (strong indication for clinically relevant risk factor: OR > 2; 1 < CILL ≤ 2). 3. Category C (indication for risk factor: 1 < OR ≤ 2; CILL > 1) was composed of 16 variables. 4. Category D (possible indication for risk factor: 1 < OR ≤ 2; CILL ≤ 1) embraced 11 variables.	modifiable risk factor is a very stress full life. It follows the recommend ation to try to reduce the daily distress and its effects on the organism. Given the clinical significance of bruxism and the small number of published findings on risk factors further epidemiolog ical and

							clinical studies should be planned and carried out with the help of our knowledge deepens on this subject
Manfredini et al. (2010); Oral Surgery, Oral Medicine, Oral Pathology	Is there a relationship between bruxism and temporomandibular joint disorders?	Associated factors	PubMed (May, 2006)	46 studies	Authors' judgment (no specific tool)	A total of 46 articles were included for discussion in the review and grouped into questionnaire/self-report (n=21), clinical assessment (n=7), experimental (n=7), tooth wear (n=5), polysomnographic (n=4), or electromyographic (n=2) studies. In several studies, the	Investigations based on self-report or clinical bruxism diagnosis showed a positive association with TMD pain, but they are characterized by some potential bias and

		level of evidence was negatively influenced by a low level of specificity for the assessment of the bruxism-TMD relationship, because of the low prevalence of severe TMD patients in the studied samples and because of the use of self-report diagnosis of bruxism with some potential diagnostic bias.	confounders at the diagnostic level (eg, pain as a criterion for bruxism diagnosis). Studies based on more quantitative and specific methods to diagnose bruxism showed much lower association with TMD symptoms. Anterior tooth wear was not found to be a major risk

	factor for TMD. Experimental sustained jaw clenching may provoke acute muscle tenderness, but it is not analogous to myogenous TMD pain, so such studies may not help clarify the clinical relationship between bruxism and TMD.

Melo et al. (2018); Journal of Oral Rehabilitation	Is there an association between psychotropic medications and presence of sleep bruxism?	Associated factors	Embase, LILACS, LIVIVO, PubMed, PsycINFO, SCOPUS, Web of Science, Google Scholar, OpenGrey, and ProQuest (November, 2017)	5 cross-sectional studies	Joanna Briggs Institute Critical Appraisal Checklist for Analytical Cross-Sectional Studies	Overall, one study was categorized as low risk of bias, three as moderate risk, and one as high risk. Antidepressants were evaluated only in adult populations, and duloxetine (Odds Ratio [OR]=2.16; 95% Confidence Interval [95%CI]=1.12-4.17), paroxetine (OR=3.63; 95%CI=2.15-6.13), and venlafaxine (OR=2.28; 95%CI=1.34-3.86) were positively associated with SB. No increased odds were observed considering the use of citalopram, escitalopram,	Medications such as duloxetine, paroxetine, venlafaxine, barbiturates, and methylphenidate may exhibit a positive association with the presence of SB.

						fluoxetine, mirtazapine, and sertraline. With regard to anticonvulsants, only barbiturates were associated with SB in children (OR=14.70; 95%CI =1.85-116.90), while no increased odds were observed for benzodiazepine, carbamazepine, and valproate. The only psychostimulant evaluated was methylphenidate, and an association with SB was observed in adolescents (OR=1.67; 95%CI=1.03-2.68)	
Chrcanovic et al. (2015);	In patients being	Effects on stomatognat	PubMed, Web of Science, and	2 controlled	Newcastle-Ottawa Scale	Ten publications were included with a total of 760 implants	These results cannot

Implant Dentistry	rehabilitated with dental implants, what is the effect of bruxism on the implant failure rates, postoperative infection, and marginal bone loss?	hic structures	the Cochrane Oral Health Group Trials Register (Jnue, 2014)	clinical trials 3 prospectiv e noncontrol led trials 5 retrospecti ve studies	inserted in bruxers (49 failures; 6.45%) and 2989 in non-bruxers (109 failures; 3.65%). Due to lack of information, meta-analyses for the outcomes “postoperative infection” and “marginal bone loss” were not possible. A risk ratio of 2.93 was found (95% confidence interval, 1.48–5.81; P=0.002).	suggest that the insertion of dental implants in bruxers affects the implant failure rates due to a limited number of published studies, all characterize d by a low level of specificity, and most of them deal with a limited number of cases without a control group.

							Therefore, the real effect of bruxing habits on the osseointegration and survival of endosteal dental implants is still not well established.
De Souza Melo et al. (2017); Journal of Prosthetic Dentistry	Is sleep bruxism associated with an increased frequency of ceramic restoration failures?	Effects on stomatognathic structures	Embase, Latin American and Caribbean Health Sciences (LILACS), LIVIVO, PubMed (including Medline), Science	8 retrospective cohort studies	MAStARI	Eight studies were included for qualitative synthesis, but only 5 for the meta-analysis. Three studies were categorized as moderate risk and 5 as high risk of bias. Clinical and methodological heterogeneity across	Within the limitations of this systematic review, the overall result from the meta-analysis did not favor any association

	Direct, the Cochrane Library, and Web of Science	studies were considered high. Increased hazard ratio (HR=7.74; 95% confidence interval [CI]=2.50 to 23.95) and odds ratio (OR=2.52; 95% CI=1.24 to 5.12) were observed considering only anterior ceramic veneers. Nevertheless, limited data from the meta-analysis and from the restricted number of included studies suggested that differences in the overall odds of failure concerning SB and other types of ceramic restorations did not favor or disfavor any	between SB and increased odds of failure for ceramic restorations.

						association (OR=1.10; 95% CI=0.43 to 2.8). The overall quality of evidence was considered very low according to the GRADE criteria	
Hsu et al. (2012); International Journal of Oral & Maxillofacial Implants**	How can biomechanical implant complications be identified and managed?	Effects on stomatognathic structures	PubMed for English-language articles (May, 2011)	5 bruxism-related studies (from 15 included)	No risk of bias assessment	Examination of the included studies revealed that bruxism or parafunctional habits were related to increased susceptibility to biomechanical implant treatment complications and peri-implant bone loss.	Occlusal overloading was thought to be the primary etiologic factor in biomechanical implant treatment complications, which commonly included marginal bone loss, fracture of resin/cerami

				c veneers and porcelain, retention device or denture base fracture of implant-supported overdentures, loosening or fracture of abutment screws, and even implant failure. Occlusal overloading was positively associated with parafunctional habits

							such as bruxism.
Manfredini et al. (2014); Clinical Implant Dentistry and Related Research	Role of bruxism as a risk factor for the different complications on dental implant-supported rehabilitations	Effects on stomatognathic structures	Medline for English-language articles (May, 2012)	21 studies	Authors' judgment (no specific tool)	A total of 21 papers were included in the review and split into those assessing biological complications (n=14) and those reporting mechanical complications (n=7). In general, the specificity of the literature for bruxism diagnosis and for the study of the bruxism's effects on dental implants was low. From a biological viewpoint, bruxism was not related with implant failures in six papers,	Bruxism is unlikely to be a risk factor for biological complications around dental implants, while there are some suggestions that it may be a risk factor for mechanical complications.

						while results from the remaining eight studies did not allow drawing conclusions. As for mechanical complications, four of the seven studies yielded a positive relationship with bruxism.	
Manfredini et al. (2015); Journal of Periodontology	Is there any evidence that bruxism may cause periodontal damage *per se*?	Effects on stomatognathic structures	Medline and Scopus for English-language articles (January, 2014)	1 case-control study 5 cohort studies	CASP cohort study checklist	The six included articles covered a high variability of topics, without multiple papers on the same argument. Findings showed that the only effect of bruxism on the periodontal structures was an increase in periodontal sensation, whilst a relationship with periodontal lesions was absent. Based on the analysis	It seems reasonable to suggest that bruxism cannot cause periodontal damage per se, but it is also important to emphasize that due to methodological problems

						of Hill's criteria, the validity of causation conclusions was limited, mainly due to the absence of a longitudinal evaluation of the temporal relationship and dose-response effects between bruxism and periodontal lesions.	regarding particularly SB assessment, more and better studies should be performed in order to further clarify this issue.
Salvi et al. (2009); International Journal of Oral & Maxillofacial Implants**	Which mechanical/ technical risk factors have an impact on implant-supported reconstructions?	Effects on stomatognathic structures	MEDLINE (PubMed) database (1966 to April, 2008)	5 bruxism-related studies (from 35 included)	No risk of bias assessment	The present literature search indicated five studies in which bruxers were compared to nonbruxers. In two of the clinical reports, statistically significantly higher rates of mechanical/technical complications (ie, 17.3% and 23%) and	Increased mechanical/ technical risks for FDPs were observed in bruxers in four of five studies (two retrospective and two consecutive case studies)

						failures (ie, 60% and 39%) were found in bruxers compared with nonbruxers. In two additional publications, trends toward more frequent mechanical/technical complications and implant losses were observed in bruxers. However, one study found no increased rate of complications in FDPs and verdentures in bruxers compared to nonbruxers.	comparing bruxers and nonbruxers.
Schmitter et al. (2014); International Journal of Prosthodontics	Investigate the influence of patient-related factors on restoration survival as well as to report the	Effects on stomatognathic structures	Medline (via PubMed), Cochrane library, and OpenSIGLE (July, 2012)	No bruxism-related included study	Not applicable	Not applicable	There is a lack of information about the effect of bruxism on the incidence of

	methods used to collect these factors.						technical failure of veneered zirconia restorations because all available studies failed to use suitable instruments for diagnosis of bruxism.
Van de Sande et al. (2016); Operative Dentistry **	Investigate the influence of patient-related factors on restoration survival as well as to report the methods used to collect these factors.	Effects on stomatognathic structures	PubMed/Medline, Scopus, and Cochrane library (April, 2015)	8 bruxism-related studies (from 51 included)	No risk of bias assessment	Most of the studies included in the present review, assessing bruxism, have not objectively stated the cutoff points applied to determine the condition. Thus, a direct comparison of methods is not feasible. Among the	Few studies were found investigating the role of bruxism/parafunctional habits on restoration survival, and different

		studies evaluating ceramics, no significant effect on the failure rates for inlay/onlay restorations was found. However, for extensive partial crowns, a significant effect for bruxism was shown in restoration survival. Regarding other materials, only two studies have investigated the effect of bruxism, and in both cases, this variable significantly influenced the survival of amalgam and composite restorations. Other reports were found presenting	results were reported.

						information regarding bruxism behavior only in the discussion of the results, where more failures were seen in bruxing patients	
Van't Spijker et al. (2007); Clinical Oral Implants Research**	To systematically assess relationships, if any, between attrition and occlusal factors and oral (dys)function in terms of management of attrition	Effects on stomatognathic structures	PubMed and Cochrane Library (February, 2006)	10 bruxism-related studies (from 37 included)	No risk of bias assessment	All other reports in this category dealt with TMD or bruxism and as such they were considered addressing dysfunction. A few trends could be distinguished. Seven studies reported positive correlations between attrition and self-reported bruxism. Two studies including self-reported bruxism reported no such correlation. Another study reported no	Attrition seems coexistent with self-reported bruxism.

						significant correlation between attrition and clinically diagnosed bruxism.	
Zhou et al. (2016); Clinical Implant Dentistry and Related Research	Does bruxism contribute to dental implant failure?	Effects on stomatognathic structures	MEDLINE (PubMed) and Embase (November, 2013)	7 cohort studies	Newcastle-Ottawa Scale for cohort studies	In this meta-analysis review, extracted data were classified into two groups based on different units. Units were based on the number of prostheses (group A) and the number of patients (group B). In group A, the total pooled OR of bruxers versus nonbruxers for all subgroups was 4.72 (95% CI: 2.66–8.36, p = .07). In group B, the total pooled OR of bruxers versus nonbruxers for all subgroups was	In contrast to nonbruxers, prostheses in bruxers had a higher failure rate. It suggests that bruxism is a contributing factor of causing the occurrence of dental implant technical/biological complications and plays a role in

						3.83 (95% CI: 2.12–6.94, p = .22).	dental implant failure.
Canales et al. (2017); Clinical Oral Investigations	Is there enough evidence to use botulinum toxin injections for bruxism management?	Therapy effectiveness	PubMed, Scopus, Web of Science, Embase, Cochrane, Scielo, and Lilacs on English-language articles (1980 to March, 2016)	2 RCT 3 before-after studies	1. CASP checklist 2. Cochrane Collaboration's risk of bias tool	Three RCTs and two uncontrolled before–after studies out of 904 identified citations were included in this review. All five articles dealt with sleep bruxism and featured a small sample size. None of them was about awake bruxism. Two randomized clinical trials were double-blinded, with a control group using saline solution. Two studies used polysomnography/electromyography for	BoNT-A seems to be a possible management option for sleep bruxism, minimizing symptoms and reducing the intensity of muscle contractions, although further studies are necessary especially as far as the treatment

						sleep bruxism diagnosis, whilst others were based on history taking and clinical examination. All studies using subjective evaluations for pain and jaw stiffness showed positive results for the BoNT-A treatment. In contrast, the two studies using objective evaluations did not demonstrate any reduction in bruxism episodes, but a decrease in the intensity of muscles contractions.	indications for bruxism itself is concerned.
Hillier et al. (2015); Evidence-Based Complem	1. Systematically identifying and appraising the	Therapy effectiveness	AMED, Embase, MEDLINE (Ovid), Cochrane,	1 bruxism-related study (from 20 included)	Cochrane tables	After intervention 77% parents in feldenkrais method reported no nocturnal bruxism compared	Reduction in nocturnal bruxism in young children

entary and Alternative **	evidence for the effectiveness of the Feldenkrais Method across domains 2. Determining what is the nature and order of magnitude of any beneficial effects and for which population		PsycINFO, PubMed, and Google Scholar (July, 2014)			with 15.38% for controls.	after 10-week course of feldenkrais method lessons
Jokubauskas et al. (2017); Journal of Oral Rehabilitation	What is the effect of oral appliances on various treatment outcomes in adult patients with SB	Therapy effectiveness	Cochrane Library and MEDLINE (via PubMed) (January, 2017)	7 before-after studies 7 RCTs 2 RCTs (cross-over)	1. Cochrane risk of bias tool (RCT) 2. CASP checklist for cohort studies 3. Cochrane	Analysis of the included articles revealed a high variability of study designs and findings. Generally, the risk of bias was lowto-unclear for RCTs and	Although many positive studies support the efficiency of OA treatment

		risk of bias tool (cross-over studies)	high for crossover studies, whilst the before–after studies exhibited several structural limitations. Nine studies used polysomnography/polygraphy/electromyography for SB diagnosis, whilst others were based on history taking and clinical examination. Most of them featured small samples and were short term. Of the studies using objective SB evaluations, eight showed positive results for almost every type of OA in reducing SB activity, with a higher decrease for devices	for SB, accepted evidence is insufficient to support its role in the long-term reduction of SB activity. Further studies with larger samples and sufficient treatment periods are needed to obtain more acknowledgements for clinical application.

						that are designed to provide a certain extent of mandibular advancement. Among the studies using a subjective SB evaluation, one demonstrated a significant reduction in SB activity, and additional two showed a myorelaxant effect of OA in SB patients.	
Jokubauskas et al. (2018); Journal of Oral Rehabilitation	Assessing the most recent literature and providing a comprehensive summary of the efficacy of any biofeedback treatment approach for the reduction	Therapy effectiveness	MEDLINE (searched via PubMed), EMBASE (searched via ScienceDirect), System for Information on Grey Literature in Europe, The Cochrane	4 RCTs 2 uncontrolled before-after studies	GRADE criteria	The meta-analysis indicated a non-significant difference in electromyographic-measured SB episodes per hour after one night of contingent electrical stimulation (CES) compared with placebo control, yet a	One of the biofeedback modalities, CES, is effective in reducing SB-related motor activities after a short-term treatment

	or control of SB.		Library (Cochrane Central Register of Controlled Trials) and LILACS (January, 2018)			significant difference was shown after five nights of CES. The quality of evidence identified through GRADEpro, was from low-to-moderate, due to imprecision and inconsistency between studies. Qualitative synthesis did not present a reliable reduction in clinical pain levels, however, no substantial sleep disturbances were indicated following the intervention	period. However, evidence of long-term effects is lacking. Further longitudinal studies with larger samples are necessary to acknowledge the clinical application of biofeedback.
Lang et al. (2009); Research in Developmental	This review involved a systematic analysis of studies that focused on	Therapy effectiveness	Education Resources Information Center (ERIC), MEDLINE,	11 studies	No risk of bias assessment	Across the11 studies, intervention was provided to a total of 19 participants aged 4–43 years. Assessment	Overall, the evidence base is extremely limited and no definitive

Disabilities	the treatment of bruxism in individuals with developmental disabilities.		Psychology and Behavioral Sciences Collection, and PsycINFO (December, 2008)			procedures included dental screening under sedation and interviews with caregivers. Intervention approaches included prosthodontics, dental surgery, injection of botulinum toxin-a, behavior modification, music therapy, and contingent massage. Positive outcomes were reported in 82% of the reviewed studies.	statements regarding treatment efficacy can be made.
Lino et al. (2017); Oral Diseases **	The aim of this systematic review was to search for scientific evidence of	Therapy effectiveness	PubMed, Web of Science, Cochrane Library, Scopus, Bireme, US	4 bruxism-related studies (from 15 included)	PEDro scale	Clinical trials involving patients with bruxism indicated a lack of efficacy for pain and muscle activity, suggesting that this	Efficacy has not been validated for cases of bruxism

	the efficacy of antidepressant drugs for the treatment of oral problems.		National Institute for Heal th and Clinical Trials (June, 2017)			class of therapy has little utility in such cases.	
Long et al. (2012); International Dental Journal	The objective of this study was to assess the efficacy of botulinum toxins on bruxism.	Therapy effectiveness	PubMed, Embase and Science Citation Index, websites of the Cochrane Central Register of Controlled Trials (CENTRAL) and ClinicalTrials.gov, and the literature database of SIGLE (System for Information	2 RCT 2 controlled before-after studies	Cochrane risk of bias tool	These studies showed that botulinum toxin injections can reduce the frequency of bruxism events, decrease bruxism-induced pain levels and satisfy patients' self-assessment with regard to the effectiveness of botulinum toxins on bruxism. In comparison with oral splint, botulinum toxins are equally effective on bruxism. Furthermore, botulinum toxin injections at a dosage	Botulinum toxin injections are effective on bruxism and are safe to use. Therefore, they can be used clinically for otherwise healthy patients with bruxism.

			on Grey Literature in Europe)			of <100 U are safe for otherwise healthy patients.	
Macedo et al. (2007); Cochrane Database of Systematic Reviews	To evaluate the effectiveness of occlusal splints for the treatment of sleep bruxism with alternative interventions, placebo or no treatment.	Therapy effectiveness	Cochrane Oral Health Group's Trials Register (to May 2007), The Cochrane Central Register of Controlled Trials (CENTRAL) (The Cochrane Library 2007, Issue 1), MEDLINE (1966 to May 2007). EMBASE (1980 to May	3 RCT 1 RCT (cross-over) 1 quasi-randomized controlled trial	Cochrane Collaboration's risk of bias tool for randomized controlled trials	Thirty-two potentially relevant RCTs were identified. Twenty-four trials were excluded. Five RCTs were included. Occlusal splint was compared to: palatal splint, mandibular advancement device, transcutaneous electric nerve stimulation, and no treatment. There was just one common outcome (arousal index) which was combined in a meta-analysis. No statistically	There is not sufficient evidence to state that the occlusal splint is effective for treating sleep bruxism. Indication of its use is questionable with regard to sleep outcomes, but it may be that there is some benefit with

			2007). LILACS (1982 to May 2007). Dissertation, Theses and Abstracts (1981 to May 2007). Biblioteca Brasileira de Odontologia (1982 to May 2007).			significant differences between the occlusal splint and control groups were found in the meta-analyses.	regard to tooth wear.
Macedo et al. (2014); Cochrane Database of Systematic Reviews	To evaluate the effectiveness and safety of pharmacological therapy for the treatment of sleep bruxism compared with other drugs, no	Therapy effectiveness	The Cochrane Central Register of Controlled Trials (CENTRAL) (Issue 8, 2014); MEDLINE (1966 to August 2014);	7 RCTs (cross-over)	Cochrane Collaboration's risk of bias tool for randomized controlled trials	Resultswere imprecise and consistentwith benefit, no difference or harm.Thesewere the specific findings for each of the drugs according to specific outcomes: 1. Amitriptyline versus placebo for masseteric	There was insufficient evidence on the effectiveness of pharmacotherapy for the treatment of sleep bruxism.

	treatment or placebo.	EMBASE (1980 to August 2013); LILACS (1982 to August 2014).	electromyography (EMG) activity per minute: standardized mean difference (SMD) -0.28 (95% confidence interval (CI) -0.91 to 0.34; P value = 0.37), 2. bromocriptine versus placebo for bruxism episodes per hour: mean difference (MD) 0.60 (95%CI -2.93 to 4.13), bruxism bursts per hour: MD -2.00 (95%CI -53.47 to 49.47), bruxism bursts per episode: MD 0.50 (95% CI 1.85 to 2.85) or number of episodes with grinding noise: MD 2.40 (95% CI -24.00 to 28.80), 3. clonidine versus

		placebo for number of bruxism episodes per hour: MD -2.41 (95% CI -4.84 to 0.02), 4. Propranolol versus placebo for the number of bruxism episodes per hour: MD 1.16 (95% CI -1.89 to 4.21), 5. L tryptophan versus placebo for masseteric EMG activity per second: SMD 0.08 (95% CI -0.90 to 1.06) and 6. levodopa versus placebo for bruxism episodes per hour of sleep: MD -1.47 (95% CI -3.64 to 0.70), for bruxism bursts per episode: MD 0.06 (95% CI -2.47 to 2.59).

Machado et al. (2011); Dental Press Journal of Orthodontics	The objective of this systematic literature review is to discuss, based on scientific evidence, treatment alternatives for the control and management of SB	Therapy effectiveness	MEDLINE, Cochrane, EMBASE, PubMed, Lilacs and BBO for articles in English, Spanish, or Portuguese (January 1990 until July 2008)	11 RCTs	No risk of bias assessment	1. Occlusal splint seems to be an acceptable and safe treatment alternative in the short and medium terms, while the clonazepam, among pharmacological treatments, stood out as a therapeutic option in the short term, because in the long term it can cause dependence. 2. Mandibular advancement device and clonidine are the most promising experimental treatments for the SB, however both are associated with secondary adverse effects.	There is a lot of treatment options for the SB, but many of the therapies have no scientific support. Thus, the choice therapy should be based on scientific evidences and in clinical common sense, for an improvement in quality of life of the bruxist patient.

						3. Cognitive-behavioral therapies such as psychotherapy, biofeedback, physical exercise and lifestyle changes, which are aimed at stress reduction, may be auxiliary in the treatment of SB.	
Manfredini et al. (2015); Journal of Oral Rehabilitation	The review focuses on the most recent literature on management of sleep bruxism (SB) in adults	Therapy effectiveness	PubMed for articles in English (March, 2015)	12 RCTs 2 before-after studies	1. Cochrane Collaboration's risk of bias tool for randomized controlled trials 2. CASP checklist for cohort studies	The studies' results suggest that (i) almost every type of oral appliance (OA) (seven papers) is somehow effective to reduce SB activity, with a potentially higher decrease for devices providing large extent of mandibular advancement; (ii) all tested pharmacological	There is not enough evidence to define a standard of reference approach for SB treatment, except for the use of OA. Future studies on the indications

	approaches [i.e. botulinum toxin (two papers), clonazepam (one paper) and clonidine (one paper)] may reduce SB with respect to placebo; (iii) the potential benefit of biofeedback (BF) and cognitive–behavioural (CB) approaches to SB management is not fully supported (two papers); and (iv) the only investigation providing an electrical stimulus to the masseter muscle supports its effectiveness to reduce SB.	for SB treatment are recommend ed.

Manfredini et al. (2017); Journal of Prosthetic Dentistry	The purpose of this systematic review was to evaluate the relationship between prosthetic rehabilitation and TMDs and bruxism	Therapy effectiveness	PubMed (July, 2016)	No included study	Not applicable	No clinical trials of the reviewed topics were found, and a comprehensive review relying on the best available evidence was provided. Bruxism is not linearly related to TMDs, and both of these conditions are multifaceted. Based on the diminished causal role of dental occlusion, prosthetic rehabilitation cannot be recommended as a treatment for the 2 conditions. In theory, they may increase the demand for adaptation beyond the stomatognathic system's tolerability. No evidence based guidelines were	There is an absence of RCTs on the various topics concerning the relationship between TMD and bruxism and prosthodontics.

						available for the best strategy for managing prosthetic needs in patients with TMDs and/or bruxism.	
Martin et al. (2012); International Journal of Oral and Maxillofacial Surgery **	The present review was designed to investigate the evidence of the use of antidepressants in orofacial pain disorders. Which treatment modalities are effective for specific orofacial pain disorders or for orofacial pain in general.	Therapy effectiveness	PubMed for articles in Dutch or English (April, 2012)	1 bruxism-related cross-over study	15-item criteria score	After 4 weeks of treatment there was no significant improvement in pain reduction. The level of perceived stress was reduced significantly in the treatment group. The authors advised small doses of amitriptyline for the management of perceived stress in patients with bruxism.	There was a lack of randomized trials concerning the use of these treatment modalities in facial pain disorders. The limited evidence of their effectiveness in pain management and their side effects, make the

							administration of antidepressant in the treatment of pain in patients with orofacial pain questionable.
Restrepo et al. (2009); Quintessense International	To conduct a systematic review to assess and analyze the scientific evidence about the available therapies for bruxism in children.	Therapy effectiveness	Medline, PubMed, Ovid, Biomed Central, EBSCOhost, ISI, Cochrane Library, Embase, LILACS, Scielo, Scirus (March 1985 to September 2007)	1 quasi-experimental study 1 RCT	Chalmers scoring system	From 52 records found, 2 fulfilled the inclusion criteria. In 1 study, bruxism was treated by widening the upper airway through adenoidectomy, and the other study proposed to treat bruxism in children with psychologic techniques. When analyzed, the 2	The available literature does not provide adequate support to treat bruxism in children, as the diagnosis methods in the studies

		considered studies did not fully accomplish the requirements to treat the etiology of bruxism in children.	are insufficient and are not comparable to confirm the presence of bruxism. Very few studies about therapies for bruxism in children meet the quality criteria required for the evidence based practice. Treatment for bruxism in children requires further study.

Stapelmann et al. (2008); BMC Oral Health **	The aim of this systematic review was to appraise the currently available evidence regarding the efficacy and safety of the NTI-tss splint.	Therapy effectiveness	The Cochrane Library, PubMed, TRIP database, MEDPILOT. DE, BIREME, Deutscher Arzte-Verlag database, Quintessenz Database, Google Scholar, Web of Science (December, 2007).	2 bruxism-related RCTs	Jadad Quality Score	Two RCTs concentrated on electromyographic (EMG) investigations in patients with TMDs and concomitant bruxism or with bruxism alone; in both studies, compared to an occlusal stabilization splint the NTI-TSS device showed significant reduction of EMG activity.	Evidence from RCTs suggests that the NTI-TSS device may be successfully used for the management of bruxism and TMDs.
Wang et al. (2014); Sleep & Breathing	The aim of this systematic review was to evaluate the efficacy of any biofeedback	Therapy effectiveness	Cochrane Central Register of Controlled Trials, MEDLINE, Embase, ISI Web of	7 RCTs	Cochrane Collaboration's risk of bias tool for randomized controlled trials	Seven eligible studies involving 240 participants were finally included. Three of them had moderate risk of bias, and four had high risk of bias. In an	There is no powerful evidence to support the use of biofeedback technology on sleep

	treatment on sleep bruxism.	Science, System for Information on Grey Literature in Europe, Chinese Biomedical Literature Database, and PsycINFO (October 2012)	electromyographicmeasured sleep bruxism episode, meta-analysis showed no significant difference between contingent electrical stimulation and blank control (95 % confidence interval=−12.33, 3.38, P=0.26). Moreover, five studies reported electromyographic activity index. Due to the diversity of biofeedback modalities (auditory, electrical, and visual stimulus) and controls (splint, occlusal adjustment, etc.), these data were unable to be pooled, so only qualitative	bruxism treatment.

	description was provided.

Legend: AH: Apnea-Hypopnea; BF: Biofeedback; BoNT-A: Type-A Botulinum Toxin; CB: Cognitive-Behavioural; CI: Confidence Interval; CASP: Critical Appraisal Skills Programme; CES: Contingent Electrical Stimulation; EGC: Electrocardiography; EMG: Electromyography; FDP: Fixed Dental Prosthesis; GERD: Gastroesophageal Reflux Disease; GRADE: Grading of Recommendations, Assessment, Development and Evaluation; HR: Hazard Ratio; MA: Meta-Analysis; MAStARI: Meta-Analysis of Statistics Assessment and Review Instrument; MD: Mean Difference; MORE: Methodological Evaluation of Observational Research; NA: Not Available; NTI: Nociceptive Trigeminal Inhibition; NTI-TSS: Nociceptive Trigeminal Inhibition Tension Suppression System; OA: Oral Appliance; OR: Odds Ratio; OSA: Obstructive Sleep Apnea; PEDro: Physiotherapy Evidence Database; PPV: Positive Predictive Value; PSG: Polysomnography; Qu-ATEBS: Quality-Assessment Tool for Experimental Bruxism Studies; QUADAS: Quality Assessment of Diagnostic Accuracy Studies; QUIPS: Quality in Prognosis Studies; RCT: Randomized Controlled Trial; ROC: Receiver Operating Characteristic SB: Sleep Bruxism; SMD: Standardized Mean Difference; TMD: Temporomandibular disorder; (*) Translated by overview authors; (**) Bruxism was not the primary outcome, only data regarding bruxism were considered.

Figure 1 - Flow diagram of literature search and selection criteria (adapted from Preferred Reporting Items for Systematic Reviews and Meta-Analysis and generated using the software Review Manager 5.3, The Cochrane Collaboration).

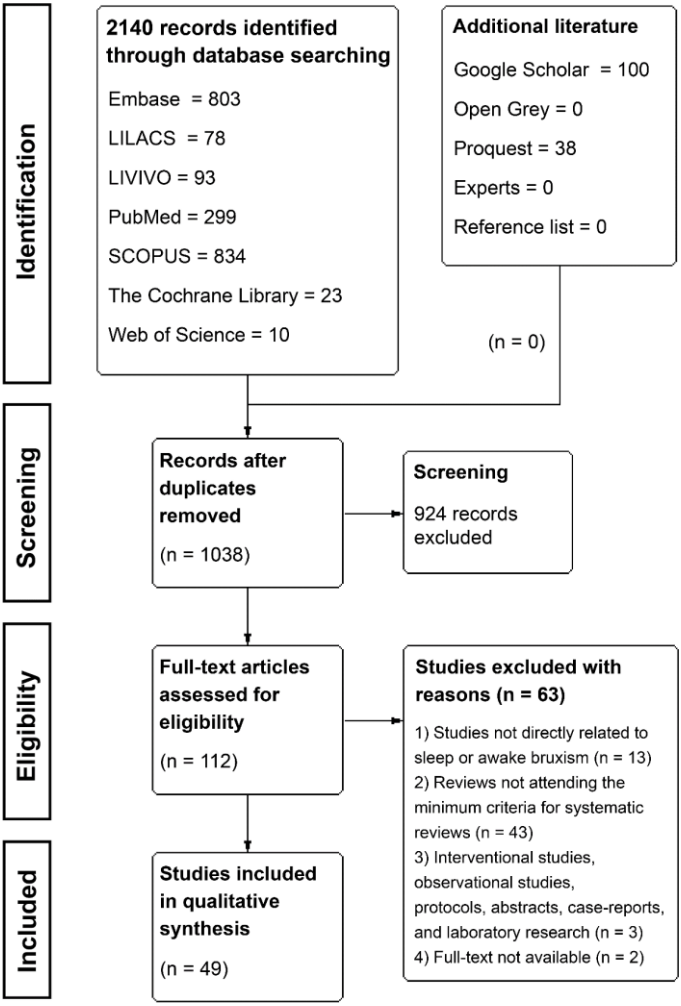


Figure 2 - Percentage of same primary study cited in one or more of the different systematic reviews, for each subgroup.

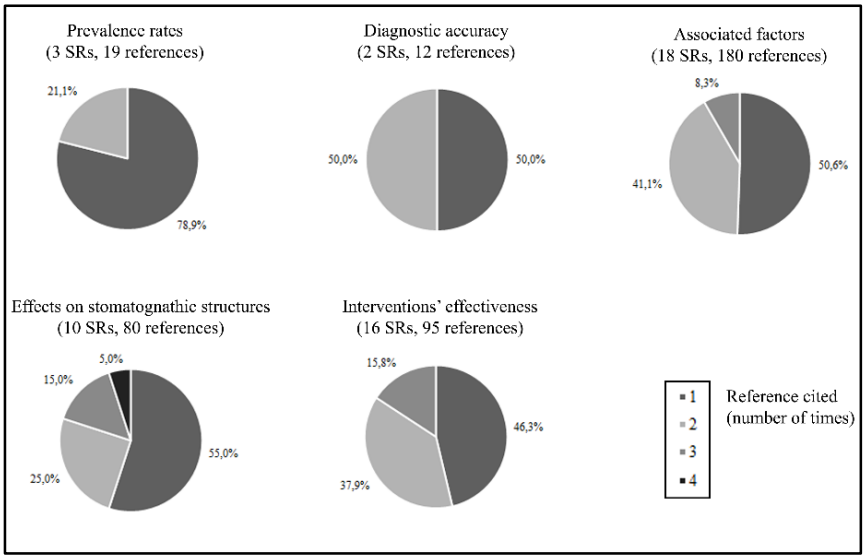
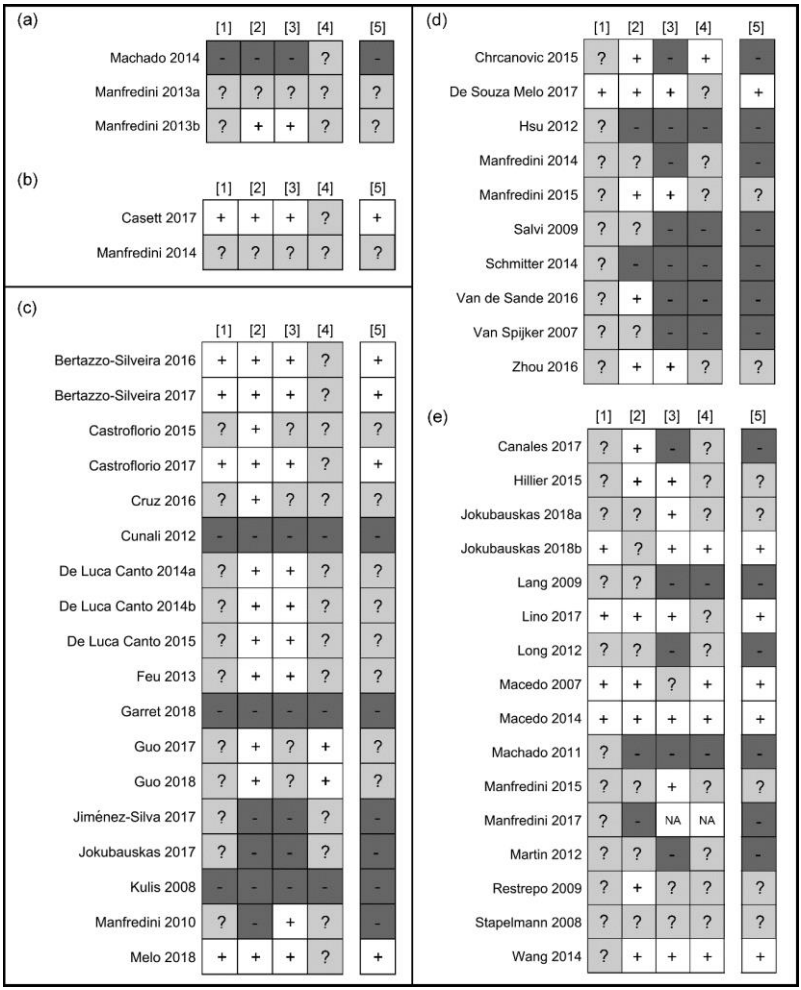


Figure 3 - Risk of bias summary, assessed by the University of Bristol's tool for assessing risk of bias in Systematic Reviews (generated using the software Review Manager 5.3, The Cochrane Collaboration).



Legend: (a) Prevalence-rates; (b) Diagnostic accuracy; (c) Associated factors; (d) Effects on stomatognathic structures; (e) Interventions' effectiveness; [1] Study eligibility criteria; [2] Identification and selection of studies; [3] Data collection and study appraisal; [4] Synthesis and findings; [5] Overall risk of bias; (+) Low risk; (?) Unclear risk; (-) High risk.

5 CONCLUSÃO

Com base nas evidências disponíveis, pode-se concluir que:

1) Em adultos, a prevalência do bruxismo em vigília foi 22-30%; do sono (1-15%) e o BS em crianças e adolescentes (3-49%);

2) Os principais fatores concistentemente associados ao bruxismo foram: uso de álcool, cafeína, tabaco, alguns medicamentos psicotrópicos, acidificação esofágica, fumo passivo e alguns sinais e sintomas de DTM. Não houve forte evidência de uma associação entre bruxismo e distúrbios do sono ou cefaléia do tipo tensional/enxaqueca.

3) Dispositivos portáteis mostraram os maiores valures de sensibilidade e especificidade, enquanto questionários e exame clínico apresentaram especificidade similar, porém sensibilidade consideravelente mais baixa.

4) O bruxismo pode resultar em complicações biomecânicas relacionadas aos implantes dentários e próteses implantossuportadas, apesar disso, as evidências disponíveis não corroboram ou refutam qualquer efeito negativo considerando falhas de outras restaurações dentárias ou danos ao periodonto.

5) Dispositivos oclusais foram consistementemente considerados efetivos para o manejo do bruxismo. A evidência em relação a toxina botulínica foi considerada com alto risco de viés, porém uma efetividade plausível para o manejo do bruxismo foi reportada. Outras terapias farmacológicas e terapias de *biofeedback* não foram recomendadas, com exceção da estimulação elétrica contingente.

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APÊNDICES

Apêndice A - Registro do protocolo

PROSPERO
International prospective register of systematic reviews

NHS National Institute for Health Research

Bruxism: an overview of systematic reviews

Gilberto Melo, Joyce Duarte, Patricia Pauletto, Juliana Stuginski-Barbosa, André Porporatti, Ephraim Winocur, Carlos Flores-Mir, Graziela De Luca Canto

Citation

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Review question

What do we currently know so far about associated factors, diagnosis, effects on oral health, prevalence rates, and interventions in regard to sleep and/or awake bruxism?

Searches

Appropriate truncation and word combinations will be elaborated and adapted for each of the following electronic databases: Embase, Latin American and Caribbean Health Sciences (LILACS), LIVIVO, PubMed, Scopus, The Cochrane Library, and Web of Science. In addition, a grey literature search will be conducted on Google Scholar, OpenGrey, and ProQuest.

Types of study to be included

Inclusion: Systematic reviews and meta-analyses
Exclusion: 1) Studies in which outcomes were not directly related to sleep and/or awake bruxism;

2) Studies that did not use explicit, systematic methods that are selected with a view to minimizing bias, thus not providing reliable findings from which conclusions can be drawn and decisions made;
3) Interventional studies, observational studies, laboratory research, abstracts, case-reports, protocols, personal opinions, letters, and posters.

Condition or domain being studied

According to a recent international consensus (Lobbezoo, 2013), bruxism is defined as a repetitive jaw-muscle activity characterized by clenching or grinding of the teeth and/or by bracing or thrusting of the mandible. Bruxism has two distinct circadian manifestations: it can occur during sleep (indicated as sleep bruxism) or during wakefulness (indicated as awake bruxism). It is considered a topic of interest since previous studies have reported a prevalence of approximately 8% to 20% in the general population. Bruxism etiopathogenesis is not fully understood; however, it is proposed that it may present multifactorial etiology, and recent studies have proposed that sleep bruxism and awake bruxism may even present distinct etiologies. Several systematic reviews on Bruxism have been performed in the last two decades, evaluating topics such as associated factors, diagnostic accuracy of bruxism assessment tools, effects of bruxism on oral health, prevalence rates among different populations, and intervention effectiveness.

Participants/population

Inclusion: Humans
Exclusion: None

Intervention(s), exposure(s)

Inclusion: Presence of sleep bruxism, awake bruxism, or sleep-awake bruxism.
Exclusion: None

Comparator(s)/control

Inclusion: Non-bruxism samples or no comparison
Exclusion: None

Context

Systematic reviews and meta-analyses that investigated any bruxism-related outcome will be considered.

Only systematic reviews that met the minimum criteria proposed by the Cochrane Handbook for Systematic Reviews of Interventions will be considered: "It uses explicit, systematic methods that are selected with a view to minimizing bias, thus providing more reliable findings from which conclusions can be drawn and decisions made (Antman 1992, Oxman 1993)."
No time and language restriction will be applied.

Primary outcome(s)

1. Associated factors

2. Diagnostic accuracy
3. Effects on oral health
4. Prevalence rates
5. Therapy effectiveness

Secondary outcome(s)

Not applicable

Data extraction (selection and coding)

Three independent reviewers (1R, 2R, and 3R) will collect data from the selected articles. Subsequently, the retrieved information will be crosschecked. Any disagreement will be discussed between them and the fourth reviewer (4R). The following data will be extracted and recorded in duplicate by two reviewers for each included study: author; year of publication; country; research question; characteristics of the participants (n, age); outcome measure(s); pertinent result(s) and conclusion(s).

Risk of bias (quality) assessment

The methodology of selected systematic reviews will be evaluated by using the “A Measurement Tool to Assess systematic Reviews 2” (AMSTAR-2) checklist.

Strategy for data synthesis

We will perform a narrative synthesis. If a quantitative synthesis is possible, we will do it for each subgroup.

Analysis of subgroups or subsets

If applicable, we will classify into subgroups based on bruxism related outcomes, such as associated factors, diagnostic accuracy, effects on oral health, prevalence rates, and therapy effectivity.

Contact details for further information

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Organisational affiliation of the review

Federal University of Santa Catarina

Review team members and their organisational affiliations

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Anticipated or actual start date

01 February 2018

Anticipated completion date

31 July 2018

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Conflicts of interest

Language

English

Country

Canada, Israel, Brazil

Stage of review

Review_Ongoing

Subject index terms status

Subject indexing assigned by CRD

Subject index terms

Bruxism; Humans

Date of registration in PROSPERO

29 March 2018

Date of publication of this version

29 March 2018

Details of any existing review of the same topic by the same authors

Stage of review at time of this submission

Stage	Started	Completed
Preliminary searches	Yes	Yes

Stage	Started	Completed
Piloting of the study selection process	Yes	No
Formal screening of search results against eligibility criteria	No	No
Data extraction	No	No
Risk of bias (quality) assessment	No	No
Data analysis	No	No

Versions

29 March 2018

PROSPERO
This information has been provided by the named contact for this review. CRD has accepted this information in good faith and registered the review in PROSPERO. CRD bears no responsibility or liability for the content of this registration record, any associated files or external websites.

Apêndice B - Estratégias de busca das bases de dados

Do artigo em inglês:

Appendix 1 - Data search strategy.

Database	Search query 2018, May 21th
EMBASE	#1 = ('bruxism'/exp OR bruxism OR 'sleep bruxism'/exp OR 'sleep bruxism' OR 'awake bruxism') #2 = ('systematic review' OR 'integrative review' OR 'meta-analysis' OR 'meta analysis' OR overview OR review OR 'systematic literature review' OR 'rapid review') #3 = (#1 AND #2)
LILACS	tw:(bruxismo OR "bruxismo do sono" OR "bruxismo noturno" OR "bruxismo de vigília" OR "bruxismo diurno" OR "bruxismo del sueño" OR "bruxismo de la vigilia") AND tw:("revisão sistemática" OR "revisão integrativa" OR "meta-análise" OR "meta análise" OR revisão OR "revisão sistemática da literatura" OR "revisión sistemática" OR "revisión integradora" OR "meta análisis" OR "meta-análisis" OR "metaanálisis" OR revisión OR "revisión sistemática de la literatura")
LIVIVO (Articles)	TI=(bruxism OR "sleep bruxism" OR "awake bruxism") AND TI=("systematic review" OR "integrative review" OR "meta-analysis" OR "meta analysis" OR overview OR review OR "systematic literature review" OR "rapid review")
PubMed	("bruxism"[MeSH Terms] OR "sleep bruxism"[MeSH Terms] OR bruxism OR "sleep bruxism" OR "awake bruxism") AND ("systematic review"[Title/Abstract] OR "integrative review"[Title/Abstract] OR "meta-analysis"[Title/Abstract] OR "meta analysis"[Title/Abstract] OR overview[Title/Abstract] OR review[Title/Abstract] OR "systematic literature review"[Title/Abstract] OR "rapid review"[Title/Abstract])
SCOPUS	TITLE-ABS-KEY(bruxism OR "sleep bruxism" OR "awake bruxism") AND TITLE-ABS-KEY("systematic review" OR "integrative review" OR "meta-analysis" OR

	"meta analysis" OR overview OR review OR "systematic literature review" OR "rapid review")
The Cochrane Library (Reviews)	#1 = (bruxism or 'sleep bruxism' or 'awake bruxism') #2 = ('systematic review' or 'integrative review' or 'meta-analysis' or 'meta analysis' or overview or review or 'systematic literature review' OR 'rapid review') #3 = (#1 AND #2)
Web of Science (Articles)	TI=(bruxism OR "sleep bruxism" OR "awake bruxism") AND TI=("systematic review" OR "integrative review" OR "meta-analysis" OR "meta analysis" OR overview OR review OR "systematic literature review" OR "rapid review")

Grey Literature

Google Scholar	(bruxism OR "sleep bruxism" OR "awake bruxism") AND ("systematic review" OR "integrative review" OR "meta-analysis" OR "meta analysis" OR "systematic literature review" OR "rapid review")
Open Grey	(bruxism OR "sleep bruxism" OR "awake bruxism") AND ("systematic review" OR "integrative review" OR "meta-analysis" OR "meta analysis" OR overview OR review OR "systematic literature review" OR "rapid review")
Proquest	all(bruxism OR "sleep bruxism" OR "awake bruxism") AND all("systematic review" OR "integrative review" OR "meta-analysis" OR "meta analysis" OR overview OR review OR "systematic literature review" OR "rapid review")

Apêndice C – Artigos excluídos e justificativas

Do artigo em inglês:
Appendix 2 - Articles excluded and the reasons for exclusion (n=63).

Reference	Author	Reasons for Exclusion*
1.	Abreu et al. (2016)	1
2.	Ahmed et al. (2016)	1
3.	Amaral et al. (2012)	2
4.	Amaral et al. (2011)	2
5.	Aurora et al. (2012)	2
6.	Awan et al. (2017)	1
7.	Barbosa et al. (2008)	2
8.	Barclay et al. (2013)	2
9.	Biondi et al. (2014)	2
10.	Bou Khalil et al. (2012)	2
11.	Bueno Torcato et al. (2014)	2
12.	Cockburn et al. (2017)	2
13.	Dao et al. (1998)	2
14.	Demarco et al. (2012)	2
15.	Dimova-Gabrovska et al. (2017)	2
16.	Ella et al. (2017)	2
17.	Falisi et al. (2014)	2
18.	Fuertes-Gonzáles et al. (2011)	1
19.	Goldstein et al. (2017)	2
20.	Hernández Reyes et al (2017)	2
21.	Hollway et al. (2011)	2
22.	Hoque et al. (2009)	2
23.	Ihde et al. (2007)	2
24.	Ilovar et al. (2014)	3
25.	Jagger, R (2008)	3
26.	Johansson et al. (2011)	2
27.	Kalamir, A (2007)	2
28.	Kalamir et al. (2007)	4

29.	Karila et al. (2016)	2
30.	Klein et al. (2014)	1
31.	Kotagal et al. (2012)	1
32.	Kulis et al. (2007)	2
33.	List et al. (2010)	1
34.	Lobbezoo et al. (2012)	2
35.	Lobbezoo et al. (2006)	2
36.	Lobbezoo et al. (2008)	2
37.	Lobbezoo et al. (2014)	2
38.	Lupoli et al. (2007)	2
39.	Luther et al. (2007)	2
40.	Madhusoodanan et al. (2010)	2
41.	Manfredini et al. (2009)	2
42.	Molina-Garcia et al. (2016)	1
43.	Nishi et al. (2016)	1
44.	Ohkubo et al. (2013)	2
45.	Osiewicz et al. (2013)	4
46.	Pecie et al. (2011)	2
47.	Perinetti et al. (2011)	1
48.	Persaud et al. (2013)	2
49.	Reichow et al. (2015)	1
50.	Rodríguez Lozano et al. (2011)	3
51.	Santos et al. (2017)	2
52.	Shetty et al. (2010)	2
53.	Sommer et al. (2015)	2
54.	Squires et al. (2014)	1
55.	Stanciu et al. (2017)	2
56.	Thanish et al. (2016)	2
57.	Tinastepe et al. (2015)	2
58.	Torcato et al. (2014)	2
59.	Torres et al. (2008)	2
60.	Veiga et al. (2013)	1
61.	Wadia, R. (2018)	3
62.	Walters et al. (2007)	2
63.	Winocur et al. (2001)	2

Legend: 1) Studies in which results were not directly related to sleep and/or awake bruxism; 2) Studies that did not use explicit, systematic methods that are selected with a view to minimizing bias, thus not providing reliable findings from which conclusions can be drawn and decisions made; 3) Interventional studies, observational studies, laboratory research, abstracts, case-reports, protocols, personal opinions, letters, and posters; and 4) Full-text not available.

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Apêndice D – Tabela suplementar 1

Do artigo em inglês:
Supplementary table 1 - Summary of descriptive characteristics of included articles in prevalence systematic reviews (n=3).

SYSTEMATIC REVIEW CHARACTERISTICS		INCLUDED STUDIES CHARACTERISTICS (n=17)					MAIN FINDINGS
Author (Year); Journal	**Objectives or research question**	**Included studies**	**Sample (n/female)**	**Geographical area**	**Mean age±SD or age range, in years**	**Bruxism diagnostic criteria**	**Prevalence rates**
Machado et al. (2014);[1] Dental Press Journal of Orthodontics	Prevalence of sleep bruxism in children	Fonseca et al. (2010)[2]	170	Brazil	Children (0 to 12)	Clinical examination according to the AASM associated with a questionnaire filled in by parents	15.3%
		Insana et al. (2013)[3]	1953	USA	Preschool (2.5 to 6.9)	Parents's report based on a	36.8% (preschool)

					First graders (3 to 8.6)	questionnaire according to the criteria of the American Academy of Sleep Disorders	49.6% (first graders)
		Lam et al. (2011)[4]	6389	China	Children (MA 9.2±1.8)	Parents' validated questionnaire	5.9%
		Serra-Negra et al. (2010)[5]	652	Brazil	Children (7 to 10)	Parents's report based on a questionnaire according to the criteria of the American Academy of Sleep Disorders	35.3%
Manfredini et al. (2013);[6] Journal of Oral Rehabilitation	Prevalence of sleep bruxism in children	Agargun et al. (2004)[7]	971	Turkey	Children (7 to 11)	Single-item questionnaire (unspecified question)	7 years: 5.2% 8 years: 7.1% 9 years: 9.3% 10 years: 8.4% 11 years: 1.9%
		Lam et al. (2011)[4]	6389	Hong Kong	Children (8 to 11)	Single-item questionnaire	5.9%

				(parents' report of teeth grinding during sleep more than thrice weekly over the past year)	
Liu et al. (2005)[8]	5979	China	Children (2 to 12)	Single-item questionnaire (unspecified question)	Overall: 6.5% 2 years: 3.5% 3–5 years: 8.5% 6–10 years: 6.7% 11–12 years: 3.7%
Reding et al. (1966)[9]	568	USA	Children (3 to 12)	Single item questionnaire (did your child ever grind the teeth during his/her sleep?)	3-7 years: 12.1% 8-12 years: 5.6%
Renner et al. (2012)[10]	1674	Brazil	Children (7 to 11)	Single item questionnaire (does your child grind the teeth at night?)	7-9 years: 39.1% 9-11 years: 35.7%

		Serra-Negra et al. (2010)[5]	652	Brazil	Children (7 to 10)	Single item questionnaire (parents' report of audible night teeth grinding – AASM)	Overall: 35.3% 7-8 years: 34.7% 9-10 years: 40.0%
		Shur-Fen Gau et al. (2006)[11]	2463	Taiwan	Children (6 to 16)	Single item questionnaire (unspecified question)	Class I: 25.1% Class II: 23.8% Class III: 16.0% Class IV: 17.7% Class V: 17.1% Class VI: 14.7%
		Simola et al. (2010)[12]	904	Finland	Children (3 to 6)	Single item questionnaire (does your child grind teeth during sleep?)	40.5%
Manfredini et al. (2013);[13] J Orofac Pain	Prevalence of bruxism in adult populations	Agerberg et al. (1972)[14]	1106 (51.6%F)	Sweden	Adults (15 to 74)	Unspecified self-reporting	AB (25-34 years): 34.6% AB (35-44 years): 34.6%

					AB (45-54 years): 34.6% AB (55-64 years): 34.6% AB (65-74 years): 34.6% SB (25-34 years): 34.6% SB (35-44 years): 34.6% SB (45-54 years): 34.6% SB (55-64 years): 34.6% SB (65-74 years): 34.6%
Bernhardt et al. (2004)[15]	2529 (52%F)	Germany	Adults (20 to 79)	1 self-reported item for "frequent" bruxism	Bruxism: 8%
Ciancaglini et al. (2001)[16]	483 (62.1%F)	Italy	Adults (18 to 75)	1 self-reported item: "Would you say that you have any clenching and/or	Bruxism: 31.4% Bruxism (<30 years): 34.6% Bruxism (31-40 years): 33.8%

				grinding of the teeth?"	Bruxism (41-50 years): 29.5% Bruxism (51-60 years): 29.4% Bruxism (>60 years): 26.9%
	Jensen et al. (1993)[17]	735 (NA)	Denmark	Adults (25 to 64)	1 self-reported item: "Do you often press (or grind) your teeth (during sleep)"	AB: 22.1% SB: 15.3%
	Ohayon et al. (2001)[18]	13057 (52%F)	UK Germany Italy	Adults (15 to 100)	2 self-reported items: Teeh grinding plus at least one of tooth wear, muscle stiffness, or loud grinding.	SB (19-24 years): 5.8% SB (25-44 years): 5.8% SB (45-64 years): 4.7% SB (>64 years): 1.1%
	Santos-Silva et al. (2010)[19]	1101 (53.6%F)	Brazil	Adults (MA 28)	1 unspecified self-reported item using "three times a week" as cutoff	SB: 9.3%

	Winocur et al. (2011)[20]	402 (62.4%F)	Israel	Adults (18 to 70)	3 self-reported items: Grinding and/or worn dentition plus one of six "symptoms" ("frequently" for sleep bruxism; no specification for awake bruxism)	AB: 31% SB: 14%

Legend: AASM: American Academy of Sleep Medicine; AB: Awake Bruxism; F: Femela; MA: mean age; NA: Not Available; OR: Odds Ratio; SB: Sleep Bruxism; SD: Standard Deviation; UK: United Kingdom; USA: United States of America.

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Apêndice E - Tabela suplementar 2

Do artigo em inglês:
Supplementary table 2 - Summary of descriptive characteristics of included articles in diagnostic accuracy systematic reviews (n=2).

SYSTEMATIC REVIEW CHARACTERISTICS		INCLUDED STUDIES CHARACTERISTICS (n=9)					DIAGNOSTIC TESTS		MAIN FINDINGS		
Author (Year); Journal	**Objectives or research question**	**Included studies**	**Sample (n/female)**	**Age group**	**Presence of bruxism (n)**	**Absence of bruxism (n)**	**Index Test**	**Reference standard**	**Test**	**Sensitivity [95% CI]**	**Specificity [95% CI]**
Casett et al. (2017);[1] Journal of Oral Rehabilitation	Which is the validity of questionnaires, clinical assessment, and portable	Abe et al. (2009) [2]	130 (58.5% F)	Adults	107	23	Clinical assessment (tooth wear), ccording to the extension of the wear facet	Laboratory-based PSG	Clinical assessment (tooth wear)	0.94 [0.88, 0.98]	0.87 [0.66, 0.97]

	diagnostic devices in diagnosing SB, when compared to the reference standard PSG?	Castroflorio et al. (2014) [3]	25 (12F)	Adults	13	12	Bruxoff device (EMG/ECG recorder) Manual: 10% MVC +25% increase in heart rate Automatic: 10% MVC + 20% increase in heart rate	Polygraphic studies in the home environment (type II device)	Diagnostic devices (AS)	0.92 [0.64, 1.00]	0.92 [0.62, 1.00]
									Diagnostic devices - Contemporaneity (AS)	0.92 [0.64, 1.00]	0.83 [0.52, 0.98]
									Diagnostic devices - Contemporaneity (MS)	0.85 [0.55, 0.98]	0.83 [0.52, 0.98]
									Diagnostic devices (MS)	0.92 [0.64, 1.00]	1.00 [0.74, 1.00]
		Mainieri et al. (2012) [4]	49 (32F)	Adults	32	17	BiteStrip EMG device - 30% MVC	Laboratory-based PSG	BiteStrip is a moderate screening method for SB diagnosis. It is more accurate in detecting presence or absence of SB but is less accurate in detecting its intensity.		

	Maluly et al. (2013)[5]	1019 (NR)	Adults	75	934	Questionnaire	Laboratory-based PSG	Questionnaires (grinding and sounds)	0.85 [0.75, 0.92]	0.68 [0.65, 0.71]
	Palinkas et al. (2015)[6]	90 (58F)	Adults	45	45	Self-report and signs and symptoms through questionnaire; and clinical assessment (tooth wear)	Laboratory-based PSG	Questionnaire (grinding and sounds)	0.49 [0.34, 0.64]	0.80 [0.65, 0.90]
								Questionnaire (jaw locking)	0.16 [0.06, 0.29]	0.80 [0.65, 0.90]
								Questionnaire (muscle fatigue)	0.78 [0.63, 0.89]	0.73 [0.58, 0.85]
								Questionnaire (muscle pain)	0.18 [0.08, 0.32]	0.93 [0.82, 0.99]
								Questionnaire (sounds)	0.49 [0.34, 0.64]	0.80 [0.65, 0.90]
								Questionnaire (temporal headache)	0.67 [0.51, 0.80]	0.82 [0.68, 0.92]

							Clinical assessment (tooth wear)	0.33 [0.20, 0.49]	0.80 [0.65, 0.90]
Raphael et al. (2015) [7]	170 (170F)	NR	124	46	Questionnaire, self-report through an interview	Laboratory-based PSG	Questionnaires (grinding and sounds, moderate SB)	0.13 [0.00, 0.53]	0.84 [0.69, 0.94]
							Questionnaires (grinding and sounds, severe SB)	0.40 [0.05, 0.85]	0.88 [0.74, 0.96]
							Questionnaires (grinding and sounds)	0.14 [0.05, 0.29]	0.80 [0.44, 0.97]
Shochat et al. (2007) [8]	18 (NR)	Adults	6 SB 4 OSA	8	BiteStrip EMG device 30%	Laboratory-based PSG	Diagnostic devices	0.67 [0.22, 0.96]	0.88 [0.47, 1.00]

							MVC for more than 0.25 seconds				
		Stuginski-Barbosa et al. (2015)[9]	20 (15F)	Adults	10	10	GrindCare (portable single-channel EMG device) 20% MVC + amplitude of the EMG signals exceeds the threshold for more than 100 ms for up to 1 s.	Laboratory-based PSG	Diagnostic devices (1st night)	0.40 [0.12, 0.74]	0.90 [0.55, 1.00]
									Diagnostic devices (3 nights)	0.50 [0.19, 0.81]	0.90 [0.55, 1.00]
									Diagnostic devices (5 nights)	0.50 [0.19, 0.81]	0.90 [0.55, 1.00]
Manfredini et al.	What is the	Castrofiorio et	25 (12F)	Adults	14 proba	11	Bruxoff device;	SB present	Diagnostic devices	83.3%	84.6%

(2014);[10] Journal of Oral Rehabilitation	validity of the different portable instrumental devices that have been proposed to measure SB if compared with PSG recordings assumed as the gold standard?	al. (2014)[10]			ble SB		10% MVC + 20% increase in heart rate	or absent - 10% MVC	(manual scoring)		
									Diagnostic devices (automatic scoring)	91.6%	84.6%
		Mainieri et al. (2012)[4]	49 (32F)	Adults	49 with clinical history of SB		Bitestrip device; 30% MVC	SB present or absent - 20% MVC	Diagnostic devices	Agreement = 87.8% (75.8–94.3%) Kappa = 0.71 (0.44–0.97) Sensitivity = 84.2% (68.7–93.9%) PPV = 100% (89.1–100%)	
		Yamaguchi et al. (2012)[11]	8 (4F)	Adults	8 tooth grinders		EMG-telemetry; two times higher than baseline)	SB present or absent - 10% MVC	Diagnostic devices	Sensitivity = 98% PPV = 23.1%	
		Shochat et al. (2007)[8]	18 (13F)	Adults	6 SB 4 OSA	8 non-patients	Bitestrip device; 30% MVC	SB present or absent -	Diagnostic devices	Sensitivity = 71-72% PPV = 59-81%	

								20% MVC		

Legend: AS: Automatic Scoring; CI: Confidence Interval; EGC: Electrocardiography; EMG: Electromyography; F: Female; MS: Manual Scoring; MVC: Maximum Voluntary Clenching; OR: Odds Ratio; OSA: Obstructive Sleep Apnea; PPV: Positive Predictive Value; PSG: Polysomnography; SB: Sleep Bruxism.

Supplementary table 2 - references

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Apêndice F - Tabela suplementar 3

Do artigo em inglês:
Supplementary table 3 - Summary of descriptive characteristics of included articles in association systematic reviews (n=18).

SYSTEMATIC REVIEW CHARACTERISTICS		INCLUDED STUDIES CHARACTERISTICS (n=131)					EXPOSITION CHARACTERISTICS	MAIN RESULTS
Author (Year); Journal	**Objectives or research question**	**Included studies**	**Study design**	**Participants enrolled**	**Mean age (SD) or age range, in years**	**Bruxism diagnostic criteria**		
Bertazzo-Silveira et al. (2016);[1] Journal of the American Dental Association	In adults, is there any association between SB and alcohol, caffeine, tobacco, or drug abuse?	Ahlberg et al. (2004)[2]	Cohort	205	46.0 (6.0)	Questionnaire and clinical examination	Tobacco consumption	OR=2.9 (95%CI, 2.26-3.61)
		Cohen (1995)[3]	Descriptive	500	18-25	Questionnaire	Drug abuse (MDMA)	Analysis by means of percentage: no significant association

						between MDMA consumption and prolonged occurrence of bruxism
Hojo et al. (2007)[4]	Cross-sectional	51	23 (1.9)	Questionnaire and EMG	Alcohol consumption	Mean (standard deviation) muscle activity duration calculated at EMG with alcohol consumption (35.2 [14.6]) and without alcohol consumption (30.3 [22.9])
Lavigne et al. (1997)[5]	Cross-sectional	1874	Smokers with SB 24.5 (4.7)	Questionnaire and polysomnography	Tobacco consumption	OR=1.9 (95%CI, 1.37-2.63)

			Nonsmokers with SB 28.6 (4.7)			
Peroutka et al. (1988)[6]	Descriptive	100	18-25	Questionnaire	Drug abuse (MDMA)	Analysis by means of percentage: no significant association between MDMA consumption and prolonged occurrence of bruxism
Rintakoski et al. (2010)[7]	Cohort	3124	24.0 (NR) 23-27	Questionnaire	Tobacco consumption	Heavy tobacco smoker: OR=2.45 (95% CI, 1.75-3.44)
	Cohort	10229	44.0 (7.8)	Questionnaire	Alcohol consumption	Binge drinking

	Rintakoski et al. (2013)[8]			OR=1.8 (95% CI, 1.36-2.39) Heavy drinking OR=1.7 (95% CI, 1.11-2.67)
			Caffeine consumption	Model I (adjusted for age and sex) OR=1.9 (95% CI, 1.38-2.66) Model II (adjusted for age, sex, and smoking status) OR=1.4 (95%CI, 1.01-1.98)
			Tobacco consumption	Current tobacco smoker:

								OR=2.9 (95%CI, 2.26-3.61)
Bertazzo-Silveira et al. (2017);[9] Clinical Oral Investigations	Is there an association between any specific signs and symptoms of bruxism and the presence of tori?	De Luca Canto et al. (2012)[10]	Case-control	200	41 (10.5) 20-26	Clinical interview and signs of abnormal tooth wear	Association between SB and TM	SB with abnormal tooth wear OR=20.89 (95%CI, 8.36–52.02) SB without abnormal tooth wear OR=4.122 (95%CI, 1.35–12.51)
		Kerdpon et al. (1999)[11]	Cross-sectional	609	32.1 (14.2) 10-80	Generic bruxism and signs of abnormal tooth wear	Association between SB and TM/TP	TM OR=25.30 (95%CI, 15.65–40.92) TP OR=0.96 (95%CI, 0.66–1.40)

	Morrison et al. (2013)[12]	Case-control	166	TM 47.3 (4.7) TP 44.5 (7.8) 14-83	Abnormal tooth wear	Association between SB and TM/TP	The subjects with abnormal tooth wear showed an increased risk for the presence of TP and/or TM.
	Sawair et al. (2009)[13]	Cross-sectional	618	33.6 (13.1) 10-82	Self-report of teeth grinding and/or clenching	Association between SB and TM/TP	Patients who had abnormal tooth wear had significantly more prevalent TM and/or TP. Abnormal tooth wear and self-report of parafunctionalhabits (clenching, grinding or

								bruxism) could be important factors.
		Yoshinaka et al. (2010)[14]	Cross-sectional	664	66.5 (4.2) 60-82	Self-report of teeth grinding and/or clenching	Association between SB and TM	Self-report of AB: N = 24 21.4% (P = 0.198) OR = 1.31 (95%CI, 0.55–3.09) P=0.539 Self-report of SB: N = 21 18.6% (P = 0.933) OR=1.13 (95%CI, 0.60–2.12) P=0.705
Castroflorio et al. (2015);[15] Archives of Oral Biology	1. Which are the identified risk factors	Montal do et al. (2012)[16]	RCT	498	7-11	Self-reported questionnaire, interview, clinical	Second-hand smoke	High exposure to SHS is associated to SB

for bruxism in children? 2. Which is the weight of each risk factor?					examination		
	Serra-Negra et al. (2009)[17]	Cross-sectional	652	7-10	Questionnaire	Psycho-social factors	Neuroticism and high degree of responsibility are determinant factors for the development of SB among children
	Castelo et al. (2010)[18]	Cross-sectional	94	6-7	Parents' report, clinical examination	Quality of life	Children from the youngest mothers were more likely to present SB
	Serra-Negra et al. (2012a)[19]	Case-control	360	7-11	Parents' report, clinical examination	Clinical signs and symptoms, parafunctions	Children that presenting parafunctions (object biting and wake-time bruxism) were more

								susceptible to SB
		Serra-Negra et al. (2012b)[20]	Case-control	360	7-11	Parents' report	Stress levels, personality traits	High levels of stress are associated to SB
		Serra-Negra et al. (2014)[21]	Case-control	360	7-11	Questionnaire	Environmental factors, sleep duration	Children sleeping for less than 8 h a night are more likely to have SB. Light and noise in the room were associated to SB
Castroflorio et al. (2017);[22] Archives of Oral Biology	1. Which are the identified risk factors for SB in adults? 2. Which is the weight of each risk factor?	Abe et al. (2012)[23]	Case-control	114	22-69	Questionnaire, clinical examination	Genetic, psychological, behavioral factors	The study revealed that the C allele carrier of HTR2A single nucleotide polymorphism rs6313 (102C>T)

						was associated significantly with an increased risk of sleep bruxism.
Blanco Aguilera et al. (2014)[24]	Cross-sectional	1220	> 18	Questionnaire	Gender, age, clinical subtypes of temporomandibular disorders (TMD)	The results of the regression showed high statistical significance for gender and age.
Fernandes et al. (2013)[25]	Cross-sectional	301	18-76	Clinical diagnostic criteria proposed by AASM, Research Diagnostic Criteria for Temporomandibular Disorders	Primary headaches	Prevalence of sleep bruxism was higher among individuals with headaches. Among individuals with chronic migraine 74.6%

						presented with sleep bruxism and the association was significant.
	Fernandes et al. (2014)[26]	Cross-sectional	261	37.0 (NR)	Clinical diagnostic criteria proposed by AASM, Research Diagnostic Criteria for Temporomandibular Disorders	Tinnitus	Association was observed between SB and the presence of self-reported tinnitus.
	Kato et al. (2012)[27]	Cross-sectional	1930	18-69	Self-reported questionnaire, clinical examination	Age, parafunctions	The study confirmed a significant relationship between self-reported SB and the groups of 30–39 and 40–49 years

						of age, snoring and childhood teeth grinding.
Mengatto et al. (2013)[28]	RCT	45	30-58	Self-reported questionnaire, clinical examination	Gastroesophageal reflux disease (GERD), stress levels, morphological parameters	GERD is highly associated with SB.
Ohayon et al. (2001)[29]	Cross-sectional	12454	19-64	Self-reported questionnaire, interview	Lifestyle (smoking, alcohol intake), age, problems during sleep	Subjects with obstructive sleep apnea syndrome, loud snorers, subjects with moderate daytime sleepiness, heavy alcohol drinkers, caffeine drinkers, smokers, subjects with

						a highly stressful life, and those with anxiety are at higher risk of reporting sleep bruxism. Among the associated risk factors, patients with anxiety and sleep-disordered breathing have a higher number of risk factors for sleep bruxism.
Rintakoski et al. (2010)[30]	Case-control	824	44.0 (NR)	Questionnaire	Nicotine dependence	Nicotine dependence may be a significant predisposing

								factor for bruxism.
		Rintakoski et al. (2013)[8]	Case-control	7774	44.0 (NR)	Questionnaire	Legal psychoactive substances intake	The results support our hypothesis of an independent association of both alcohol use, and coffee consumption with bruxism.
Cruz et al. (2016);[31] International Journal of Odontostomatology	Verify the existence of scientific evidence of association between the daytime and/or nighttime bruxism and levels	Castelo et al. (2012)[32]	Cross-sectional	127	6-8	Questionnaires to caregivers/siblings	Salivar cortisol levels	Cildren with sleep bruxism are more likely thave lower concentrations of salivary cortisol OR=0.882 (95%CI, 0.74-0.98).

	of salivary cortisol.	Karakoulaki et al. (2015)[33]	Cross-sectional	45	25-52	Questionnaires and EMG	Salivary cortisol levels	Higher salivary cortisol levels in patients with bruxism than in those without bruxism ($p<0.001$). There was still a positive correlation between the BiteStrip scores in patients with bruxism and their salivary cortisol level ($r=0.401$, $P=0.047$).
Cunali et al. (2012);[34] Revista Dor	Verify the possible association between sleep	Camparis et al. (2006a)[35]	Cross-sectional (one night)	40	NR	Questionnaires and PSG	Association with TMD, evaluated by using the RDC/TMD	No statistically significant differences between SB

	bruxism and temporomandibular joint disorders						and TMD regarding sleep variables evaluated.
		Rossetti et al. (2008a)[36]	Cross-sectional (two nights)	60	NR	PSG	Association with TMD, evaluated by using the RDC/TMD	Sleep RMMA is associated with myofascial pain and is a low risk factor for TMD, while diurnal clenching may be a risk factor for TMD.
		Smith et al. (2009)[37]	Cross-sectional (two nights)	54	NR	Interview and PSG	Association with TMD, evaluated by using the RDC/TMD	Insomnia might play a role in the physiophatology of TMD.
		Saueressig et al. (2010)[38]	Longitudinal (30 days)	28	NR	EMG	Association with TMD, evaluated by using the RDC/TMD	Mandibular advancement device showed

								positive effects regarding SB and sleep variables and did not increase TMD prevalence.
De Luca Canto et al. (2014);[39] Headache	Evaluate and synthesize the possible association between the most common primary headaches disorders (TTH and migraine) with SB.	Fernandes et al. (2013)[25]	Cross-sectional	286	37.3 (NR) 18-76	AASM criteria	Association with primary headache (TTH and migraine) evaluated by using the ICDH criteria	SB is associated with primary Headache (TTH and migraine).
		Troeltzsch et al. (2011)[40]	Cross-sectional	1031	49.6 (NR)	AASM criteria	Association with chronic migraine evaluated by using the ICDH-II criteria	The presence of SB significantly increased the risk for chronic migraine.
De Luca Canto et al. (2014);[41] Journal of	Evaluate the association between SB and sleep-	Sjohom l et al. (2000)[42]	Experimental bruxism study	21	40.0 (9.2)	AASM Criteria	Association with sleep-disordered breathing (diagnosed with PSG)	SB was diagosed in 54% of patients with mild OSA

Orofacacial Pain	disordered breathing							and 40% of patients with moderate OSA. SB was not observed during snoring or apnea in any of these patients. Masseter actvity was not observed during apneic episodes.
De Luca Canto et al. (2015);[43] Clinical Pediatrics	Evaluate whether SB is associated with psychosocial factors in children and adolescents	Kuch et al. (1979)[44]	Case-control	100	5-6	AASM Criteria	To determine if correlation between bruxism and personality characteristics	None of the test group means scores differed significantly from the control group mean scores (P>0.05)

	Vander as et al. (1999)[45]	Not case-control	167	6-8	AASM Criteria	To investigate the association between urinary catecholamines as biomarkers of stress and the presence of bruxism	Bruxer children had a higher mean epinephrine, norepinephrine, and dopamine. The relative risk between 1 and 9.69, and 1 to 15.38, respectively, depending on the catecholamine levels, in a 95%CI. Epinephrine (P=0.03) and dopamine (P=0.01) had a significant association with bruxism

	Herrera et al. (2006)[46]	Case-control	20	5-15	AASM Criteria	To assess the daytime behavior and cognitive impact of bruxism	The K-BIT score correlated strongly with the internalizing problems (r=0.76, P=0.047, analysis of variance), and externalizing problems scale (r=0.74, P=0.006, analysis of variance). The most significant of the individual subscales were the somatic problems scale

						(r=0.85, P=0.010, analysis of variance) and conduct problems (r=0.760, P=0.04, analysis of variance)
Katayoun et al. (2008)[47]	Not case-control	50	12-14	AASM Criteria	To determine the correlation between psychosocial disorders and bruxism	Reported higher prevalence of thought disorders (P<0.005), conduct disorders(P< 0.050) and antisocial disorders (P< 0.060) in bruxers. The odds ratio revealed that a bruxer adolescent has 16 times

						greater probability for psychosocial disorders than a nonbruxer one
Restrepo et al. (2008)[48]	Case-control	52	8-11	AASM Criteria	To describe the personality traits and the anxiety level of bruxer children	Statistically significant difference between the control and bruxism group regarding tense personality (P=0.024) and anxiety (P=0.0007)
Ferreira-Bacci et al. (2012)[49]	Not case-control	29	7-11	AASM Criteria	To evaluate the behavioral profile of a group of bruxer children	82.76% of the sample needed psychological or psychiatric intervention

						and 18.75% presented significant physical and psychological manifestations of stress
Türkoglu et al. (2013)[50]	Case-control	70	8-17	AASM Criteria	To examine statetrait anxiety, anxiety sensitivity, depressive symptoms levels, and psychiatric disorders in children and adolescents with SB	At least 1 psychiatric disorder was present in 42.9% of the patient group and 17.1% of the control group (P<0.05). Trait and state anxiety, anxiety sensitivity, and the severity of depression symptoms were also

								higher in the SB group (P<0.05). After the multivariate analysis, the associations between state and trait anxiety, depression, and SB became statistically insignificant, while the association with anxiety sensitivity persisted
Feu et al. (2013);[51] Journal of Orthodontics	To examine whether risk factors for bruxism can be identified in children and adults.	Ahlberg et al. (2004)[2]	Longitudinal study	211	46.0 (NR)	Questionnaires	Questionnaires regarding tobacco use, levels of perceived bruxism, affective disturbance,	Affective disturbance [tiredness (P=0.03); anxiety (P=0.03); worry about health

		sleep disturbance, somatic symptoms, pain symptoms and TMD symptoms	(P=0.01); sex dysfunction (P=0.01)] and early insomnia (P=0.03) were significantly more prevalent in frequent bruxers, as well as pain symptoms, smoking and TMD-related symptoms. According to the logistic regression, smokers were 1.2–4.9 times more likely to report frequent bruxism than

						non-smokers (P=0.01).
Carlsson et al. (2003)[52]	Longitudinal study	402	7-15	Questionnaires and clinical examination	Presence of symptoms associated with the masticatory system, headaches or previous trauma, and whether the subject often felt stressed, worried or depressed. There were also questions on whether the subjects experienced TMD or requested TMD treatment.	Subjective reports in childhood of bruxism [tooth clenching during daytime (P=0.02) and/or tooth grinding at night (P=0.05)] were predictors of the same oral parafunctions 20 years later. A patient that reported bruxism at the first evaluation was

						approximately 3 times more likely to have bruxism at the end of the follow-up [OR=53.1]. The association between SB and psychological factors had a very weak correlation (r<0.2), and this factor may explain less than 5% of the variance in bruxism.
Lobbezoo et al. (1997)[53]	Double-blind clinical trial	10	27.5 (5.4)	PSG and EMG exams	Association between disturbances in the central	L-dopa resulted in a significant decrease in

				neurotransmitter system and SB	the average number of bruxism episodes per hour of sleep, as well as in a significant reduction in the average value of the root-mean-square (RMS) EMG level per bruxism burst. This indicates that L-dopa exerts an attenuating effect on SB and caused a reduction in the variance in RMS values, which

						suggests that L-dopa normalizes EMG activity patterns associated with SB.
Ohmure et al. (2011)[54]	Cross-over, randomized, single-blinded trial	12	24.2 (2.8)	PSG and EMG exams	Test the hypothesis that experimental intra-esophageal acid infusion induces SB	The frequencies of EMG bursts, rhythmic masticatory muscle activity (RMMA) episodes, grinding noise, and the RMMA/microarousal ratio were significantly higher in the 20-minute period after

						acidic infusion than after saline infusion, whereas no significant difference was observed between saline infusion and no intervention. RMMA episodes including SB were induced by esophageal acidification
Rintakoski et al. (2010)[30]	Longitudinal study	445 twin pairs (concordant for heavy	44.0 (NR)	Questionnaires	Association between smoking and bruxism	Bruxism was more frequent among cigarette smokers in both

	smoking) 142 twin pairs (discordant for smoking status)			genders. Alcohol dependence and depression were not related. In an age and gender-controlled multinomial logistic regression, both monthly and rarely reported bruxism were associated with current cigarette smoking (OR=51.74 and 1.64) and with former cigarette

								smoking (OR=51.64 and 1.47). Weekly bruxism wasassociated with current smoking (OR=52.85). Current smokers smoking 20 or more cigarettes a day reported weekly bruxism more often (OR=51.61–1.97) than those who smoked less.
Garret et al. (2018);[55] Neurology Clinical Practice	The objective of this article was to review the	37 case-reports (please see	Case-reports only	Total of 46 patients	39.8 (NR) 7-81	NR	Association between bruxism and antidepressants	Bruxism may develop as an adverse reaction to antidepressa

	existing literature for the clinical features of antidepressant associated bruxism, to identify common offending agents, and to explore successful treatment strategies.	original article)			nt therapy, and is most likely to develop within 2–3 weeks of medication introduction or dose titration. This phenomenon may be seen in a variety of serotonergic antidepressants, and may be most associated with fluoxetine, sertraline, or venlafaxine. Patients who experience this condition

								may benefit from the addition of buspirone 5 and 10 mg in daily, twice daily, or 3 times daily dosing; dose reduction and antidepressant cessation may also be considered.
Guo et al. (2017);[56] Sleep and Breathing	What sleep behaviors are associated with bruxism in children?	Junqueira et al. (2013)[57]	Cross-sectional	937	2-6	Questionnaire for parents	Restless sleep	OR=2.4 (95%CI, 1.8-3.3)
		Tachibana et al. (2016)[58]	Case-control	6023	2-12	Questionnaire	1. Sleeps alone 2. Moves a lot during sleep 3. Sleeps with mouth open 4. Sleeps with head arched back 5. Snores loudly 6. Stops breathing	1. OR=2.4 (95%CI, 1.8-3.3) 2. OR=0.86 (95%CI, 0.64-1.13) 3. OR=1.47 (95%CI, 1.29-1.68)

		7. Snorts and gasps 8. Cries at night 9. Wakes screaming and hard to be calmed down 10. Woken by scary dreams 11. Wakes up at any little sound 12. Awakes more than once during the night 13. Sleeps without being tucked in 14. Goes to bed by himself/herself 15. Stays up later than usual the day before a holiday 16. Wakes up later on holidays	4. OR=1.56 (95%CI, 1.35-1.81) 5. OR=1.25 (95%CI, 0.99-1.59) 6. OR=0.99 (95%CI, 0.65-1.51) 7. OR=1.1 (95%CI, 0.78-1.53) 8. OR=0.89 (95%CI, 0.59-1.32) 9. OR=0.9 (95%CI, 0.54-1.47) 10. OR=1.12 (95%CI, 0.91-1.37) 11. OR=1.22 (95%CI, 0.84-1.75) 12. OR=0.72 (95%CI, 0.47-1.07)

						13. OR=1.0 (95%CI, 0.84-1.19) 14. OR=0.95 (95%CI, 0.8-1.13) 15. OR=0.93 (95%CI, 0.81-1.08) 16. OR=1.1 (95%CI, 0.95-1.26)
Nahas-Scocate et al. (2014)[59]	Case-control	873	2-6	Questionnaire for parents	Restless sleep	OR=2.1 (95%CI, 1.6-2.9)
Serra-Negra et al. (2014)[21]	Case-control	360	7-10	Questionnaire for parents	1. Sleep hours, ≤8 h, >8 h (ref) 2. Does the child sleep well? no, yes (ref) 3. Sleep with light on, yes, no(ref) 4. Noise in room, yes, no (ref)	1. OR=2.56 (95%CI, 1.48-4.43) 2. OR=3.25 (95%CI, 1.6-6.61) 3. OR=2.37 (95%CI, 1.45-3.88) 4. OR=2.7 (95%CI, 1.65-4.43)

	Simoes-Zenari et al. (2010)[60]	Case-control	141	4-6	Questionnaire for parents	1. Sialorrhea during sleep, yes, no (ref) 2. Oral breathing during sleep, yes, no(ref) 3. Awakening at night 4. Hours of sleep (10 h–11 h), altered, adequate (ref)	1. OR=2.23 (95%CI, 0.99-4.98) 2. OR=1.13 (95%CI, 0.48-2.87) 3. OR=1.32 (95%CI, 0.59-2.91) 4. OR=5.1 (95%CI, 2.27-11.47)
	Alencar et al. (2016)[61]	Cross-sectional	66	3-7	Interview with parents	1. Nightmares 2. Drooling 3. Snoring 4. Sleep talking 5. Awakening at night 6. Sleepwalking	1. OR=18.09 (P=0.002) 2. OR=1.37 (P=0.739) 3. OR=0.14 (P=0.013) 4. OR=0.33 (P=0.722) 5. OR=0.46 (P=0.306) 6. OR=0.2 (P=0.207)
	Soares et al. (2016)[62]	Cross-sectional	151	3-5	Questionnaires	1. Position during sleep, on side, on back (ref)	1. OR=1.45 (95%CI, 0.66-3.16)

	2. Position during sleep, on stomach, on back (ref) 3. Sleeps with hand on face, yes, no (ref) 4. Nightmares, more than once a week, none (ref) 5. Nightmares, once a week, none (ref) 6. Nightmares, none, once a month(ref) 7. Nightmares, yes, none (ref) 8. Snoring, yes, no (ref) 9. Drooling during sleep, yes, no (ref) 10. Talking during sleep, yes, no (ref)	2. OR=1.41 (95%CI, 0.55-3.64) 3. OR=2.41 (95%CI, 1.22-4.79) 4. OR=5.62 (95%CI, 1.14-27.66) 5. OR=1.53 (95%CI, 0.62-3.77) 6. OR=2.85 (95%CI, 0.31-26.31) 7. OR=1.73 (95%CI, 0.83-3.64) 8. OR=2.63 (95%CI, 1.35-5.1) 9. OR=1.58 (95%CI, 0.82-3.01) 10. OR=1.98 (95%CI, 1.02-3.89)

					11. Awakes at night, yes, no (ref) 12. Mouth breathing, yes, no (ref)	11. OR=1.02 (95%CI, 0.78-2.88) 12. OR=1.49 (95%CI, 0.78-2.88)
Miamoto et al. (2011)[63]	Case-control	61	0-12	Questionnaires	Breathing, mouth, nasal (ref)	OR=3.31 (95%CI, 0.42-25.84)
Zhu et al. (2009)[64]	Case-control	117	4-10	Questionnaires	Sleep talking	P>0.05
Serra-Negra et al. (2012a)[19]	Case-control	360	8.0 (NR)	Questionnaires	Mouth breathing	OR=1.6 (95%CI, 0.9-2.6)
Suwa et al. (2009)[65]	Case-control	1956	6-12	Questionnaires for parents	1. Sleep starts, high frequency, low frequency(ref) 2. Snoring, high frequency, low frequency (ref) 3. Difficulty arising, difficulty, facility (ref)	1. OR=2.8 (95%CI, 2.05-3.84) 2. OR=3.39 (95%CI,2.55-4.50) 3. OR=1.35 (95%CI, 0.94-1.94)

	Zhang et al. (2000)[66]	Case-control	243	6-12	Questionnaires	1. Position during sleep, on stomach, on back (ref) 2. Position during sleep, mixed position, on back (ref)	1. OR=1.31 (95%CI, 0.44-3.92) 2. OR=4.99 (95%CI, 0.46-4.08)
	Wang et al. (2011)[67]	Case-control	64	4-6	Questionnaires	Sleep talking	OR=1.36 (95%CI, 0.46-4.08)
	Jiang et al. (2010)[68]	Case-control	2706	3-12	Questionnaires	1. Position during sleep, on side, on back (ref) 2. Position during sleep, on stomach, on back (ref) 3. Snore 4. Awakening or cry at night 5. Not having nap habit	1. OR=1.49 (95%CI, 1.04-2.14) 2. OR=1.81 (95%CI, 1.22-2.67) 3. OR=4.16 (95%CI, 2.93-5.91) 4. OR=1.50 (95%CI, 1.11-2.03) 5. OR=1.35 (95%CI, 1.03-1.77)

Guo et al. (2018);[69] Archives of Oral Biology	The risk factors related to bruxism in children	Renner et. al (2012)[70]	Cohort	689	9-11	Parents questionnaire	1. Male, female (ref) 2. Birth weight 1500–2499 g, ≥2500 g (ref) 3. Birth weight 500–1499g, ≥2500 g (ref) 4. Occupation of family head, skilled and semiskilled manual, nonmanual (ref) 5. Occupation of family head, unskilled manual and unemployed, nonmanual (ref) 6. Maternal marital status, cohabiting, married (ref) 7. Maternal marital status, No companion, Married (ref)	1. OR=1.84 (95%CI, 1.37–2.49) 2. OR=1.09 (95%CI, 0.77–1.55) 3. OR=1.92 (95%CI, 1.02–3.62) 4. OR=1.58 (95%CI, 1.03–2.42) 5. OR=2.18 (95%CI, 1.31–3.63) 6. OR=1.71 (95%CI, 1.19–2.46) 7. OR=1.52 (95%CI, 0.99–2.32) 8. OR=1.307 (95%CI, 0.864–1.977) 9. OR=2.3 (95%CI, 1.725–3.235)

					8. Maternal marital status, no companion, companion (ref) 9. Emotional symptoms 10. Conduct problems 11. Peer problems 12. Hyperactivity 13. Mental health problems	10. OR=1.87 (95%CI, 1.386–2.533) 11. OR=1.75 9 (95%CI, 1.284–2.408) 12. OR=1.61 2 (95%CI, 1.194–2.178) 13. OR=2.31 4 (95%CI, 1.715–3.123)
Renner et al. (2012)[70]	Cohort	805	7-9	Parents questionnaire	1. Family income, low, medium (ref) 2. Family income, high, medium (ref) 3. Family income, unknown, medium (ref) 4. Emotional symptoms 5. Peer problems	OR=1.389 (95%CI, 0.927–2.079) OR=1.668 (95%CI, 1.108–2.512) OR=1.627 (95%CI, 0.98–2.70) OR=1.583 (95%CI, 1.155–2.17)

					6. Mental health problems	OR=1.716 (95%CI, 1.237–2.381) OR=1.856 (95%CI, 1.368–2.519)
Serra-Negra et al. (2012b)[20]	Case-control	360	7-11	Parents questionnaire	1. Neuroticism, high, low (ref) 2. Responsibility, high, low (ref) 3. Total-stress levels, high, low (ref) 4. Physical reactions, high, low (ref) 5. Psychological reactions, high, low (ref) 6. Psychological reactions/ depression 7. Component, high, low (ref)	1. P>0.05 2. P<0.05; OR=1.6 (95%CI, 1.0-2.5) 3. P>0.05 4. P>0.05 5. P<0.05; OR=1.8 (95%CI, 1.1-2.9) 6. P>0.05 7. P>0.05
Montaldo et al. (2012)[16]	RCT	498	7-11	Questionnaire, interview, and clinical	1. Secondhand smoke (SHS), heavily exposed children, no	1. RR=4.5 (95%CI, 2.17-9.35)

				examination	exposed children (ref) 2. Secondhand smoke (SHS), moderately exposed, no exposed children (ref) 3. Secondhand smoke (SHS), lightly exposed, no exposed children (ref) 4. Secondhand smoke (SHS), occasionally exposed, no exposed children (ref) 5. Secondhand smoke (SHS), exposed, no exposed children (ref)	2. RR=2.2 (95%CI, 1.01-4.91) 3. RR=1.23 (95%CI, 0.72-2.1) 4. RR=0.97 (95%CI, 0.61-1.55) 5. RR=3.11 (95%CI, 2.24-4.32)
Tachibana et al. (2016)[58]	Case-control	6023	2-12	Questionnaire	1. Male, female (ref) 2. Age 2-4, 11-12 (ref)	1. OR=1.09 (95%CI, 0.96-1.24)

		3. Age 5-7, 11-12 (ref) 4. Age 8–10, 11–12 (ref) 5. Sleeps alone 6. Says legs hurt at night 7. Says legs feel hot at nigh 8. Says legs feel strange at night 9. Moves a lot during sleep 10. Sleeps with mouth open 11. Sleeps with head arched back 12. Snores loudly 13. Stops breathing 14. Snorts and gasps 15. Grumpy in the morning 16. Needs much time to wake up	2. OR=1.03 (95%CI, 0.81-1.31) 3. OR=1.72 (95%CI, 1.38-2.15) 4. OR=1.15 (95%CI, 0.93–1.43) 5. OR=0.86 (95%CI, 0.64–1.13) 6. OR=1.13 (95%CI, 0.66–1.93) 7. OR=1.08 (95%CI, 0.75–1.54) 8. OR=0.89 (95%CI, 0.54–1.45) 9. OR=1.47 (95%CI, 1.29–1.68) 10. OR=1.56 (95%CI, 1.35–1.81

		17. Takes a long time to get out of bed	11. OR=1.25 (95%CI, 0.99–1.59)
		18. Cries at night	12. OR=1.8
		19. Wakes screaming and hard to be calmed down	(95%CI, 1.47–2.20) 13. OR=0.99
		20. Woken by scary dreams	(95%CI, 0.65–1.51) 14. OR=1.1
		21. Wakes up at any little sound	(95%CI, 0.78–1.53)
		22. Awakes more than once during the night	15. OR=1.17 (95%CI, 0.92–1.30)
		23. Late for (nursery) school due to waking up late	16. OR=1.03 (95%CI, 0.84–1.26)
		24. Falls asleep during the daytime	17. OR=0.97 (95%CI, 0.79–1.19)
		25. Snoozes at (nursery) school or kindergarten	18. OR=0.89 (95%CI, 0.59–1.32)
		26. Goes to bed after 10 pm	19. OR=0.9 (95%CI, 0.54–1.47)

		27. Gets excited at night	20. OR=1.12 (95%CI,
		28. Gets grumpy at night	0.91–1.37)
		29. Has no fixed pattern in sleep–wake cycle	21. OR=1.22 (95%CI,
		30. Day–night reversal	0.84–1.75
		31. Seems sleepy in the daytime	22. OR=0.72 (95%CI,
		32. Looks run down in the daytime	0.47–1.07)
		33. Restless in the daytime	23. OR=0.93 (95%CI,
		34. Poor concentration in the daytime	0.67–1.28)
		35. Sleeps without being tucked in	24. OR=0.74 (95%CI,
		36. Goes to bed by himself/herself	0.53–1.02)
		37. Stays up later than usual	25. OR=0.61 (95%CI,
			0.32–1.09)
			26. OR=0.92 (95%CI,
			0.75–1.12)
			27. OR=1.33 (95%CI,
			0.95–1.84)
			28. OR=0.87 (95%CI,
			0.62–1.20)

		the day before a holiday 38. Wakes up later on holidays 39. Rubs feet at night 40. Touches feet at night	29. OR=1.05 (95%CI, 0.82–1.34) 30. OR=1.76 (95%CI, 0.85–3.54) 31. OR=0.86 (95%CI, 0.65–1.14) 32. OR=1.13 (95%CI, 0.83–1.52) 33. OR=1.15 (95%CI, 0.92–1.44) 34. OR=1.06 (95%CI, 0.85–1.30) 35. OR=1.00 (95%CI, 0.84–1.19) 36. OR=0.95 (95%CI, 0.80–1.13) 37. OR=0.93 (95%CI, 0.81–1.08)

						38. OR=1.1 (95%CI, 0.95–1.26) 39. OR=0.9 (95%CI, 0.52–1.52) 40. OR=1.42 (95%CI, 0.84–2.37)
Nahassc ocate et al. (2014)[59]	Case-control	873	2-6	Parents questionnaire	1. Male, female (ref) 2. Absence of posterior crossbite, Presence of posterior crossbite (ref) 3. Headache 4. Restless sleep 5. Race	1. OR=1.19 (95%CI, 0.887-1.597) 2. OR=2.2 (95%CI, 1.4-3.6) 3. OR=1.5 (95%CI, 1.1-2.2) 4. OR=2.1 (95%CI, 1.6-2.0 5. P>0.05
Tehrani (no referenc e)	Case-control	100	3-6	Parents questionnaire	Parasitic infections	OR=1.481 (95%CI, 0.54-4.064)

	Motta et al. (2011)[71]	Case-control	42	3-6	Parents questionnaire	Male, Female (ref)	OR=1.467 (95%CI, 0.434-4.951)
	Serra-Negra et al. (2014)[21]	Case-control	360	7-10	Parents questionnaire	1. Sleep hours ≦8h,>8h (ref) 2. Does the child sleep well? No, Yes (ref) 3. Times mother has checked on child in room, 0–1, ≥2 (ref) 4. Proximity of parent/children rooms, near rooms, fast rooms (ref) 5. Sleep with light on, Yes, No (ref) 6. Noise in room, Yes, No (ref) 7. Sialorrhea during sleep, Yes, No (ref)	1. OR=2.561 (95%CI, 1.480-4.433) 2. OR=3.253 (95%CI, 1.600-6.615) 3. OR=1.069 (95%CI, 0.689-1.660) 4. OR=1.172 (95%CI, 0.298–4.614) 5. OR=2.37 (95%CI, 1.446–3.884) 6. OR=2.699 (95%CI, 1.645–4.429) 7. OR=2.227 (95%CI, 0.995–4.985)

	Simoes-Zenari et al. (2010)[60]	Case-control	141	4-6	Parents fulfilled and returned an investigation protocol in a week, at the most.	1. Use of pacifier, Yes, No (ref) 2. Digital suction, Yes, No (ref) 3. Use of nursing bottle, Yes, No (ref) 4. Lips biting, Yes, No (ref) 5. Objects biting, Yes, No (ref) 6. Oral breathing during sleep, Yes, No (ref) 7. Nail biting, Yes, No (ref) 8. Lingual frenulum, altered, adequate (ref) 9. Cheeks tonus, altered, adequate (ref) 10. Inferior lip tonus, altered, adequate (ref)	1. OR=7.164 (95%CI, 0.870–58.965) 2. OR=0.568 (95%CI, 0.222–1.453) 3. OR=1.167 (95%CI, 0.477–2.853) 4. OR=4.932 (95%CI, 2.131–11.415) 5. OR=1.965 (95%CI, 0.993–3.889) 6. OR=1.132 (95%CI, 0.477–2.868) 7. OR=2.037 (95%CI, 1.029–4.033) 8. OR=1.632 (95%CI, 0.145–18.418)

		11. Superior lip tonus, altered, adequate (ref)	9. OR=2.509 (95%CI, 1.065–5.913)
		12. Tongue tonus, altered, adequate (ref)	10. OR=1.563 (95%CI, 0.767–3.184)
		13. Tongue posture, altered, adequate (ref)	11. OR=0.921 (95%CI, 0.439–1.932)
		14. Lips posture, altered, adequate (ref)	12. OR=1.737 (95%CI, 0.835–3.612)
		15. Bite, altered, adequate (ref)	13. OR=1.571 (95%CI, 0.668–3.695)
		16. Hard palate shape, adequate, altered (ref)	14. OR=1.104 (95%CI, 0.566–2.152)
		17. Tonsils,altered,adequate (ref)	15. OR=1.869 (95%CI, 0.946–3.689
		18. Frequent headache	16. OR=1.091 (95%CI, 0.558–2.132)
		19. Awakening at night	17. OR=1.733 (95%CI, 0.869–3.458)
		20. Hours of sleep (10h–11h), altered, adequate (ref)	

		21. Head moviment, present, absent (ref) 22. Tongue posture, altered, adequate (ref) 23. Perioral musculature participation, present, absent (ref) 24. Head moviment, present, absent (ref) 25. Tongue posture, adequate, altered (ref) 26. Perioral musculature participation, present, absent (ref) 27. Food waste, present, absent (ref)	18. OR=1.527 (95%CI, 0.684–3.409) 19. OR=1.319 (95%CI, 0.599–2.906 20. OR=5.1 (95%CI, 2.268–11.467) 21. OR=3.515 (95%CI, 0.713-17.223) 22. OR=1.111 (95%CI, 0.537-22.298) 23. OR=2.647 (95%CI,1.293-5.418) 24. OR=4.275 (95%CI, 0.486-37.632)

					28. Lateralization,absent, present (ref) 29. Lips posture, adequate, altered (ref) 30. Moving jaw, adequate, altered (ref) 31. Rhythm, adequate, altered (ref) 32. Pattern, adequate, altered (ref)	25. OR=1.589 (95%CI, 0.717-3.518) 26. OR=1.251 (95%CI, 0.592-2.643) 27. OR=1.585 (95%CI, 0.797-3.154) 28. OR=1.336 (95%CI, 0.672–2.657) 29. OR=1.481 (95%CI, 0.656-3.345) 30. OR=1.065 (95%CI, 0.513-2.211) 31. OR=1.686 (95%CI, 0.764-3.719) 32. OR=1.044 (95%CI, 0.532-2.046)
Diaz-Serrano (no	Case-control	57	6-11	Intraoral clinical examination,	Intestinal parasitic infestation	OR=0.623 (95%CI, 0.209-1.863)

reference)				questionnaires Questionnaires		
Restrepo et al. (2008)[48]	Case-control	52	8-11	Questionnaires	1. Reserved, Outgoing 2. Dull, Intelligent 3. Ego strength, weakness 4. Excitable, placid 5. Submissive, dominant 6. Happy-go-lucky, serious 7. Frivolous, conscientious 8. Shy, venturesome 9. Sensitive, tough 10. Restrained, Vigorous 11. Artless, Shrewd 12. Self-assured, Apprehensive	1. OR=1.5 (95%CI, 0.371-6.061) 2. OR=1.228 (95%CI, 0.349-4.322) 3. OR=1.0 (95%CI, 0.294-3.406) 4. OR=1.206 (95%CI, 0.363-4.013) 5. OR=2.204 (95%CI, 0.721-6.733) 6. OR=1.364 (95%CI, 0.457-4.071) 7. OR=2.577 (95%CI, 0.803-8.142) 8. OR=1.169 (95%CI, 0.391-3.495)

		13. Self-controlled, Lax 14. Tense, Relaxed 15. Low anxiety, High anxiety 16. Introversion, Extraversion 17. Mindedness, Tough 18. Anxiety	9. OR=1.0 (95%CI, 0.252-3.972) 10. OR=1.406 (95%CI, 0.446–4.432) 11. OR=1.364 (95%CI, 0.457–4.071) 12. OR=1.394 (95%CI, 0.279–6.953) 13. OR=1.765 (95%CI, 0.522–5.969) 14. OR=4.545 (95%CI, 1.370–15.077) 15. OR=1.31 (95%CI, 0.309–5.551) 16. OR=1.173 (95%CI, 0.387–3.556) 17. OR=1.619 (95%CI, 0.530–4.946)

						18. P<0.05
Miamoto et al. (2011)[63]	Case-control	NR	NR	Questionnaires	1. Cerebral palsy 2. Down syndrome 3. Gender, Male, Female (ref) 4. Age, ≦10years, > 10years (ref) 5. Premature Birth, Yes, No (ref) 6. Sucking habits, Yes, No (ref) 7. Worn facets, Yes, No (ref) 8. Facial type, Long face, Average (ref) 9. Facial type, Short face, Average (ref) 10. Breathing, Mouth, Nasal (ref) 11. Type of malocclusion,	1. OR=0.913 (95%CI, 0.396–2.107) 2. OR=0.913 (95%CI, 0.396–2.107) 3. OR=0.727 (95%CI, 0.198–2.672) 4. OR=2.5 (95%CI, 0.312–3.762) 5. OR=1.5 (95%CI, 0.126–17.831) 6. OR=2.313 (95%CI, 0.619–8.637) 7. P>0.05 8. OR=3.469 (95%CI, 0.940–12.799) 9. P>0.05 10. OR=3.308 (95%CI,

		Class I, Class II (ref) 12. Type of malocclusion, Class I, Class III (ref) 13. Posterior crossbite, Present, Absent (ref) 14. Severity of malocclusion, Moderate, Absent or mild (ref) 15. Severity of malocclusion, Severe, Absent or mild (ref) 16. Caries, 1 to 2 teeth with cavities, Absent (ref) 17. Caries, 3 to 7 teeth with cavities, Absent (ref)	0.423–25.843) 11. OR=0.924 (95%CI, 0.266–3.209) 12. OR=0.292 (95%CI, 0.035–2.462) 13. OR=7 (95%CI, 1.429–34.286) 14. OR=0.541 (95%CI, 0.086–3.388) 15. OR=0.36 (95%CI, 0.069–1.880) 16. OR=0.5 (95%CI, 0.097–2.584) 17. OR=2.8 (95%CI, 0.658–11.923) 18. OR=1.242 (95%CI, 0.336–4.588)

					18. Caries, present, Absent (ref)	
Zhu et al. (2009)[64]	Case-control	117	4-10	Questionnaires	1. Parents bruxism 2. Relatives bruxism 3. Posterior teeth relationship 4. Anterior deep jaw 5. Anterior deep overjet 6. Pediatric joint abnormality 7. Conduct problems 8. Age 9. Gender, male, female (ref) 10. Caries 11. Astriction 12. Oral ulcer 13. Dysfunction of gastralintestinal tract 14. Sleeptalking	1. OR=11.164 (P<0.05) 2. OR=8.575 (P<0.05) 3. OR=0.047 (95%CI, 0.006–0.369) 4. OR=0.945 (95%CI, 0.463–1.932) 5. OR=0.839 (95%CI, 0.405–1.738) 6. P>0.05 7. OR=1.704 (P<0.05) 8. P>0.05 9. P>0.05 10. P>0.05 11. P>0.05 12. P>0.05 13. P>0.05 14. P>0.05 15. P>0.05 16. P>0.05

					15. Moving mouth during sleep 16. Faulty nutrition	
Serra-Negra et al. (2012a)[19]	Case-control	360	8	Examination	1. Facial symmetry 2. Lip incompetence 3. Masseter muscle pain 4. Temporal muscle pain 5. Temporomandibular disorders 6. Headaches 7. Headaches temporal muscle 8. Headaches frontal muscle 9. Headaches occipital muscle 10. Headaches on top part of head 11. Buccal mucosa ridging	1. P>0.05 2. P>0.05 3. P>0.05 4. P>0.05 5. P>0.05 6. OR=1.177 (95%CI, 0.744-1.862) 7. P>0.05 8. P>0.05 9. P>0.05 10. P>0.05 11. P>0.05 12. P>0.05 13. P>0.05 14. OR=0.696 (95%CI, 0.394–1.228) 15. OR=2.3 (95%CI, 1.2-4.3) 16. P>0.05 17. P>0.05

					12. Tongue indentation 13. Anterior crossbite 14. Posterior crossbite 15. Primary canine wear 16. Primary first molar wear 17. Primary second molar wear 18. Nail biting 19. Biting of objects 20. Clenching teeth when awake 21. Mouth breathing	18. OR=1.352 (95%CI, 0.871-2.098) 19. OR=2.0 (95%CI, 1.2–3.3) 20. OR=2.3 (95%CI, 1.2–4.3) 21. OR=1.6 (95%CI, 0.9–2.6)
De Oliveira et al. (2015)[72]	Case-control	84	6-8	Exams	1. Anxious 2. Nervous 3. Fearful 4. Aggressive 5. Timid	1.OR=19.25 (95%CI, 7.453–49.722) 2.OR=2.818 (95%CI, 1.128–7.043)

						3.OR=1.673 (95%CI, 0.683–4.096) 4.OR=4 (95%CI, 0.779–20.531) 5.OR=0.4 (95%CI, 0.142–1.125)
Zhang et al. (2000)[66]	Case-control	243	6-12	Questionnaires	1. Position during sleep,on stomach, on back (ref) 2. Position during sleep, mixed position, on back (ref) 3. Gender, male, female(ref)	1.OR=1.312 (95%CI, 0.440–3.917) 2.OR=4.986 (95%CI, 2.254–11.027) 3.OR=1.743 (95%CI, 0.980–3.100)
Wang et al. (2011)[67]	Case-control	NR	4-6	Questionnaires	1. Parents bruxism 2. Sleeptalking 3. Caries 4. Oral ulcer 5. Premature contact	1. OR=1.364 (95%CI, 0.456–4.076) 2. OR=0.117 (95%CI, 0.03–0.464)

					6. Unilateral mastication 7. Astriction 8. Dysfunction of gastralintestinal tract 9. Conduct problems	3. OR=1.615 (95%CI, 0.409–6.377) 4. OR=0.644 (95%CI, 0.100–4.142) 5. OR=0.644 (95%CI, 0.100–4.142) 6. OR=0.716 (95%CI, 0.229–2.234) 7. OR=0.636 (95%CI, 0.216–1.879) 8. P>0.05 9. P<0.05
Chen et al. (2004)[73]	Case-control	779	0-12	Questionnaires	1. Gender, male, female (ref) 2. Father bruxism 3. Mother bruxism 4. Parents bruxism	1. OR=1.173 (95%CI, 0.878–1.567) 2. OR=4.525 (95%CI, 2.795–7.324) 3. OR=7.356 (95%CI, 3.751–14.426) 4. P<0.05

Jiménez-Silva et al. (2017);[74] Acta Odontologica Scandinavica	Sleep and awake bruxism in adults and its relationship with temporomandibular disorders	Raphael et al. (2013)[75]	Cases and controls study	170	39.2 (14.6)	PSG	Association between sleep and/or awake bruxism with temporomandibular disorders	These data are not able to determine whether the EMG activity during sleep is a risk factor for developing myofascial pain, but supports the hypothesis that a high EMG activity in the dream would be a risk factor for the course of myofascial pain.
		Raphael et al. (2012)[76]	Cases and controls study	170	39.2 (14.6)	PSG	Association between sleep and/or awake bruxism with	There would be no relationship between SB

					temporomandibular disorders	and course of myofascial pain in TMD. Treatment of SB should not be considered to maintain or exacerbate TMD myofascial pain.
Rossetti et al. (2008b) [77]	Cases and controls study	26	17-40	PSG	Association between sleep and/or awake bruxism with temporomandibular disorders	Sleep bruxism is not associated with TMD or with tenderness. Pain associated only in some individuals with SB.

	Rosseti et al. (2008a) [36]	Cases and controls study	60	19-42	PSG	Association between sleep and/or awake bruxism with temporomandibular disorders	SB and tooth clenching significantly associated with MP. Tooth clenching more power than SB as a risk factor.
	Rompré et al. (2007) [78]	Cases and controls study	100	25.6 (0.6)	PSG	Association between sleep and/or awake bruxism with temporomandibular disorders	SB-RDC has a high level of discrimination between subjects with sleep bruxism and controls. The pain is often reported among subjects with low frequency SB mandibular

						muscle contractions.
Camparis et al. (2006a)[35]	Cases and controls study	40	36.1 (11.3)	PSG	Association between sleep and/or awake bruxism with temporomandibular disorders	There is no conclusive evidence that relationship TMD and sleep bruxism.
Baba et al. (2005)[79]	Cases and controls study	103	F 23.7 (2.6) M 24.7 (NR)	PSG	Association between sleep and/or awake bruxism with temporomandibular disorders	Joint noises significantly related to duration of the EMG activity of the masseter muscle when sleeping.
Alves et al. (2013)[80]	Cases and controls study	80	NR	Clinical diagnosis, with or without self-report	Association between sleep and/or awake bruxism with temporomandibular disorders	Masticatory function was reduced in G1, it may be the result of hyperactivity of the masticatory muscles

						caused by increased muscle tension.
Fernandes et al. (2012)[81]	Cases and controls study	272	36.9	Clinical diagnosis, with or without self-report	Association between sleep and/or awake bruxism with temporomandibular disorders	SB patients showed increased myofascial pain and arthralgia.
Manfredini et al. (2010)[82]	Cases and controls study	276	32.2 (5.7) 25-44	Clinical diagnosis, with or without self-report	Association between sleep and/or awake bruxism with temporomandibular disorders	Overbite greater than or equal to 4mm combined with clinical diagnosis of bruxism (OR=4.62), greater than or equal 5mm overjet (OR=2.83) and asymmetric molar ratio combined with

						clinically diagnosed bruxism (OR=2.77) have higher chance of TMD IIIa and IIIb group
Li et al. (2009)[83]	Cases and controls study	40	NR	Clinical diagnosis, with or without self-report	Association between sleep and/or awake bruxism with temporomandibular disorders	In the TMJ vibration analysis, it was concluded that bruxism induces abnormal vibrations in the TMJ. Moreover, alterations in the TMJ produced by bruxism may be related to the pathogenesis of TMD

	Mehulic et al. (2009)[84]	Cases and controls study	42	18-65	Clinical diagnosis, with or without self-report	Association between sleep and/or awake bruxism with temporomandibular disorders	Bruxers with more common muscle disorders (neuromuscular incoordination). Patients without bruxism have disorders in diskcondyle complex. There are differences in TMD symptoms between the two study groups.
	Janal et al. (2007)[85]	Cases and controls study	51	34.5 (11.0)	Clinical diagnosis, with or without self-report	Association between sleep and/or awake bruxism with temporomandibular disorders	Study fails to support the model in which tooth wear keeps pain.

						Without demonstrating that tooth grinding or tightening start pain.
Schierz et al. (2007)[86]	Cases and controls study	646	35-44	Clinical diagnosis, with or without self-report	Association between sleep and/or awake bruxism with temporomandibular disorders	Anterior tooth wear does not define a relevant increase in risk for TMD in individuals aged 35–44 years.
Storm et al. (2007)[87]	Cases and controls study	68	13.1 (49.7) 21-70	Clinical diagnosis, with or without self-report	Association between sleep and/or awake bruxism with temporomandibular disorders	The engine of the jaw, especially 'tooth clenching' behaviour is significant in patients with TMD.

Güler et al. (2003)[88]	Cases and controls study	64	29.0 (NR) 13.63	Clinical diagnosis, with or without self-report	Association between sleep and/or awake bruxism with temporomandibular disorders	High prevalence condyle changes in patients with bruxism.
Manfredini et al. (2003)[89]	Cases and controls study	289	34.4 (13.8)	Clinical diagnosis, with or without self-report	Association between sleep and/or awake bruxism with temporomandibular disorders	Bruxism has a greater relationship with muscle disorders than with joint pathology.
Pergamalian et al. (2003)[90]	Cases and controls study	84	29.1 (8.1)	Clinical diagnosis, with or without self-report	Association between sleep and/or awake bruxism with temporomandibular disorders	No association between myofascial pain (MP) and TW. Bruxism is related to high levels of muscle pain. Report of minimum bruxism and nonbruxism

						was associated with high levels of TMJ pain.
Blanco Aguilera et al. (2014)[24]	Cases and controls study	1220	18-60	Questionnaires or self-report	Association between sleep and/or awake bruxism with temporomandibular disorders	Strong association between SB and the presence of painful symptoms of TMD, especially muscle pathology accompanied by arthralgia. No significant difference in reporting the presence of bruxism and disc displacement.

	Ferreira et al. (2014)[91]	Cases and controls study	201	20.5 (NR) 17-34	Questionnaires or self-report	Association between sleep and/or awake bruxism with temporomandibular disorders	Only tooth clenching and overjet were associated with myofascial pain.
	Bortolleto et al. (2013)[92]	Cases and controls study	172	34.8 (NR) 17-78	Questionnaires or self-report	Association between sleep and/or awake bruxism with temporomandibular disorders	Awake bruxism habit is the most common and is associated with joint pain, followed sleep bruxism associated with muscle pain, both are risk factor for TMD. The other habits studied did not have the

						same association.
Anastasaki Köhler et al. (2012)[93]	Cases and controls study	1704	20-70	Questionnaires or self-report	Association between sleep and/or awake bruxism with temporomandibular disorders	Report bruxism increased during the study period and deterioration of health perception were mostly associated with TMD symptoms and dysfunction index.
Manfredini et al. (2012)[94]	Cases and controls study	Padova University 219 Tel Aviv University 397	Padova University 42.9 (16.1) 18-81 Tel Aviv	Questionnaires or self-report	Association between sleep and/or awake bruxism with temporomandibular disorders	The characteristics of samples studied and the different interpretation of the same pattern of diagnosis may

			Univer sity 35.6 (14.7) 18-84			influence the epidemiologi cal reports of bruxism and TMD and relationship between them.
Yachida et al. (2012)[95]	Cases and controls study	115	M 36.8 (14.0) F 32.9 (10.2)	Questionnai res or self-report	Association between sleep and/or awake bruxism with temporomandibu lar disorders	There are no major differences between patients with different conditions of craniofacial pain and patients without pain in terms of EMG activity during sleep.
Marklu nd et al. (2010)[96]	Cohort	280	NR	Questionnai res or self-report	Association between sleep and/or awake bruxism with	The self-reported bruxism and crossbite increase the

					temporomandibular disorders	risk in the incidence and duration of signs and symptoms of TMD
Michelloti et al. (2010)[97]	Cases and controls study	668	11-79	Questionnaires or self-report	Association between sleep and/or awake bruxism with temporomandibular disorders	Parafunctional daytime activities can be a risk factor for TMD subgroup. More specifically, tooth clenching and grinding of the day was a risk factor for myofascial pain and disk displacement.
Osterberg et al. (2007)[98]	Cohort study	Born in 1922 422	Seventh	Questionnaires or self-report	Association between sleep and/or awake	TMD symptoms associated

		Born in 1930 484	decade of life		bruxism with temporomandibular disorders	with bruxism and psychosomatic factors and overall health.
Campar is et al. (2006b) [99]	Cases and controls study	100	36.1 (11.3) 13-66	Questionnaires or self-report	Association between sleep and/or awake bruxism with temporomandibular disorders	There are significant differences in long-standing bruxism with and without chronic facial pain.
Johansson et al. (2006) [100]	Cases and controls study	Sample 50 years 12468 Sample 60 years 6322	50 and 60 years	Questionnaires or self-report	Association between sleep and/or awake bruxism with temporomandibular disorders	There is a relationship between bruxism and TMD.
Sato et al. (2006) [101]	Cases and controls study	508	NR	Questionnaires or self-report	Association between sleep and/or awake bruxism with	Half TMD patients had TCH.

					temporomandibular disorders	TCH could be a prolongation factor TMD pain.
Van der Meulen et al. (2006)[102]	Cohort study	Cohort frequency 226 Cohort degree of stress 303	Corrert frequency 38.5 (13.3) 13-76 Cohort degree of stress 37.2 (14.2) 14-83	Questionnaires or self-report	Association between sleep and/or awake bruxism with temporomandibular disorders	No clinical relevance related to different types of oral parafunctions with selfreport and discomfort for TMD. Causal relationship between TMD and bruxism if exists, is small.
Ahlberg et al. (2005)[103]	Cases and controls study	1500	Group work shifts M 45.0 (10.6)	Questionnaires or self-report	Association between sleep and/or awake bruxism with	Association between perception of orofacial pain and

			F 42.6 (10.7) Group worker s day M 47.4 (9.7) F 45.5 (10.1)		temporomandibu lar disorders	bruxism report. Bruxism together with sleep disruption may participate simultaneous ly in developing orofacial pain.
Glaros et al. (2005)[10 4]	Cases and controls study	96	NR	Questionnai res or self-report	Association between sleep and/or awake bruxism with temporomandibu lar disorders	Parafunction s that increase muscle tension and emotional states are good predictors of levels of mandibular pain in TMD patients and healthy subjects.

	Kobs et al. (2005)[105]	Cases and controls study	307	35.4 (NR) 20-54	Questionnaires or self-report	Association between sleep and/or awake bruxism with temporomandibular disorders	There is a relationship between the incidence of dental clench and pathological phenomena in the muscles and joints.
	Magnusson et al. (2005)[106]	Cases and controls study	320	7-15	Questionnaires or self-report	Association between sleep and/or awake bruxism with temporomandibular disorders	A significant correlation between reported bruxism and TMD symptoms. Baseline report of toothgrinding at night was a predictor of TMD treatment during the

						observation period.
Mundt et al. (2005)[107]	Cases and controls study	2963	35-74	Questionnaires or self-report	Association between sleep and/or awake bruxism with temporomandibular disorders	In men and women, the presence of bruxism is associated with TMD.
Miyake et al. (2004)[108]	Cases and controls study	3557	20.4 (2.1) 18-26	Questionnaires or self-report	Association between sleep and/or awake bruxism with temporomandibular disorders	Association between parafunctional activities and symptoms of TMD.
Fujita et al. (2003)[109]	Cases and controls study	57	23.6 (NR)	Questionnaires or self-report	Association between sleep and/or awake bruxism with temporomandibular disorders	Comparing primary habits, patients with bruxism and unilateral chewers were more complex symptoms of TMD.
Velly et al.	Cases and	183	18-60	Questionnaires or self-	Association between sleep	Tooth clenching

		(2003)[110]	controls study			report	and/or awake bruxism with temporomandibular disorders	(with or without grinding) are associated with chronic MP.
Jokubauskas et al. (2017);[111] Journal of Oral Rehabilitation	What is the relationship between OSA and SB, which can be determined using full-night polysomnography (PSG), in adult patients diagnosed with OSA and/or SB?	Hosoya et al. (2014)[112]	Experimental bruxism study	67	54.3 (13.2)	PSG (AASM criteria)	Relationship between SB and sleep respiratory events in patients with OSA	1) Significantly higher risk of SB in the OSA group. 2) Frequency of the phasic type of SB correlated positively with that of obstructive apnoea, micro-arousal and oxygen desaturation. 3) Significantly higher frequency of SB events

						during micro-arousal events consequent on AH events in the OSAS group.
Saito et al. (2015)[113]	Experimental bruxism study	59	44.8 (10.8)	PSG (AASM criteria)	Associations between each specific breathing and jaw muscle event in a population reporting awareness of both OSA and SB.	1) OSA and SB were concomitant in only 50.8% of subjects. 2) Moderate correlations were found in the following combinations (P<0.05): RMMA/SB episode with AI, RMMA/SB burst with AI and age,

						sleep-OMA burst with AHI and wake-OMA burst with BMI.
Saito et al. (2013)[114]	Experimental bruxism study	10	46.7 (11.5)	PSG (AASM criteria)	Association between sleep apnoea–hypopnoea (AH) events and SB events	1) Of the intervals between SB and the nearest AH events, 80.5% were scored within 5 min. 2) Most intervals were distributed within a period of <30 s, with peak at 0–10 s. 3) Significantly more SB

								events were scored in the interval between AH events termination and SB events onset (P<0.05).
Kulis et al. (2008);[115] Schweizer Monatsschrift für Zahnmed	What variables have been identified as risk factors for sleep and / or awake bruxism in adults?	Ahlberg et al. (2002)[116]	Cross-sectional	133	Mid-40s	Questionnaires	1.Very stress full life (self-report) 2.Adult woman	1.OR=5.0 (95%CI, 2.8-8.8) 2.OR=2.3 (95%CI, 1.4-3.6)
		Ahlberg et al. (2005)[103]	Cross-sectional	874	Mid-40s	Questionnaires	1.Syndrome of restless legs 2.Dissatisfaction with work shifts 3.Irregular work shift	1.OR=2.0 (95%CI, 1.1-3.8) 2.OR=1.8 (95%CI, 1.8-3.1) 3.OR=1.2 (95%CI, 0.7-2.1)
		Carlsson et al. (2003)[52]	Longitudinal study	402	7 to 15	Questionnaires	1.Jaw clenching a day in childhood (self-report), for	1.OR=6.8 (95%CI, 1.6-28.3)

					bruxism as an adult 2.Bruxism in childhood (self-report) 3.Bruxism in childhood (self-report) for bruxism as an adult	2.OR=3.1 (95%CI, 1.6-6.3) 3.OR= 2.9, (95%CI, 1.3-6.3)
Johansson et al. (2004)[117]	Cross-sectional	6343	50.0 (NR)	Questionnaires	1.Dissatisfaction with the dental care 2.Education: High School / University 3.When not healthy rated health 4.Tobacco use 5.Marital status single 6.Office Worker 7.Occupation: Entrepreneur 8.Higher School education	1.OR=1.5 (95%CI, NR) 2.OR=1.4 (95%CI, NR) 3.OR=1.4 (95%CI, NR) 4.OR=1.35 (95%CI, NR) 5.OR=1.3 (95%CI, NR) 6.OR=1.2 (95%CI, NR)

					9.High importance of dental care	7.OR=1.1 (95%CI, NR) 8.OR=1.1 (95%CI, NR) 9.OR=1.1 (95%CI, NR)
Lavigne et al. (1997)[5]	Cross-sectional	2019	NR	Interview	Smoke cigarettes	OR=1.9 (95%CI, 1.4-2.6)
Manfredini et al. (2004)[118]	Cross-sectional	160	NR	NR	Possible link between occlusal factors and bruxism	Laterotrusive interference OR=2.5 (95%CI, NR); conditional, see results from original article
Ohayon et al. (2001)[29]	Cross-sectional	13057	14.0 (NR)	Telephone survey	1. People between 25 and 44 years 2. People between 45 and 64 years	1. OR=3.1 (95%CI, 2.3-4.1) 2. OR=2.7 (95%CI, 2.1-3.6)

		3.Persons aged 19 to 24 4.Three or more glasses of alcohol a day 5.People aged between 15 and 18 years 6.Obstructive sleep apnea syndrome 7.Somniloquy (speaking in his sleep) 8.Automatic (stereotypical) behavior during the day 9.One to two glasses of alcohol a day 10.Six or more cups of coffee a day 11.Loud snoring 12.Hypnagogic hallucinations	3.OR=2.8 (95%CI, 2.0-3.8) 4.OR=1.8 (95%CI, 1.4-2.4) 5.OR=1.8 (95%CI, 1.2-2.7) 6.OR = 1.8 (95%CI, 1.2-2.6) 7.OR = 1.7 (95%CI, 1.4-2.0) 8.OR = 1.5 (95%CI, 1.3-1.9) 9.OR = 1.5 (95%CI, 1.1-1.9) 10.OR = 1.4 (95%CI, 1.2-1.8) 11.OR = 1.4 (95%CI, 1.1-1.8)

		13.Much distress in everyday life 14.20 cigarettes / day 15.Nighttime awakenings 16.Moderate sleepiness 17.Anxiety disorder (according to DSM-IV classification) 18.Abnormal behavior during sleep 19.Morning headaches 20.One to two cups of coffee a day 21.No loud snoring 22.severe drowsiness 23.Hallucinogens once a month	12.OR = 1.3 (95%CI, 1.1-1.5) 13.OR = 1.3 (95%CI, 1.1-1.6) 14.OR = 1.3 (95%CI, 1.1-1.5) 15.OR = 1.3 (95%CI, 1.1-1.5) 16.OR = 1.3 (95%CI, 1.1-1.6) 17.OR = 1.3 (95%CI, 1.0-1.6) 18.OR = 1.3 (95%CI, 0.9-1.9) 19.OR = 1.3 (95%CI, 0.9-1.7) 20.OR = 1.2 (95%CI, 1.0-1.5)

							24.Three to five cups of coffee a day 25.Completely unrefreshed after the morning awakening 26.Depression (according to DSM-IV classification)	21.OR = 1.2 (95%CI, 1.0-1.4) 22.OR = 1.2 (95%CI, 0.9-1.7) 23.OR = 1.1 (95%CI, 0.9-1.4) 24.OR = 1.1 (95%CI, 0.9-1.4) 25.OR = 1.1 (95%CI, 0.9-1.3) 26.OR = 1.1 (95%CI, 0.8-1.5)
Manfredini et al. (2010);[119] Oral Surgery, Oral Medicine, Oral Pathology	Is there a relationship between bruxism and temporomandibular joint disorders?	Costa et al. (2008)[120]	NR	42	18-63	Questionnaire or self-report	Association between SB and TMD	Bruxing behavior risk factor for headaches in TMJ
		Osterberg et al. (2007)[98]	NR	904	70.0 (NR)	Questionnaire or self-report	Association between SB and TMD	TMD symptoms associated with bruxism

						Uncertainty in self-reported bruxism, caution in interpretation of results
Chen et al. (2007)[121]	NR	9	35.-(NR) 18-67	Questionnaire or self-report	Association between SB and TMD	MFP nearly 4 times more NTC during wake time and higher stress levels than controls. NTC frequency not correlated with stress levels
Sato et al. (2006)[101]	NR	508	NR	Questionnaire or self-report	Association between SB and TMD	TCH in about half of chronic TMD TCH potential risk factor for

						TMD pain prolongation
Johansson et al. (2006)[100]	NR	Cohort 1: 12468 Cohort 2: 6232	Cohort 1: 50 Cohort 2: 60	Questionnaire or self-report	Association between SB and TMD	Positive ssociation between bruxism and TMD signs and symptoms
Van der Meulen et al. (2006)[102]	NR	Cohort 1: 226 Cohort 2: 303	Cohort 1: 38.5 (13.3) 13-76 Cohort 2: 37.2 (14.2) 14-83	Questionnaire or self-report	Association between SB and TMD	Causal relation between bruxism and TMD, if existing, is small
Camparis et al. (2006b)[99]	NR	100	36.1 (11.3) 13-66	Questionnaire or self-report	Association between SB and TMD	Clear differences between longstanding bruxism, with and without chronic facial pain

						Bruxers with CFP: bilateral pain, uncomfortable bite, stiffness in the morning (statistically different from bruxers without pain)
Kobs et al. (2005)[105]	NR	307	35.4 (NR) 20-54	Questionnaire or self-report	Association between SB and TMD	"Solid relationship" between "incidence of clenching" and muscle palpation findings
Magnusson et al. (2005)[106]	NR	329	7-15	Questionnaire or self-report	Association between SB and TMD	Significant correlations between reported bruxism and TMD

						symptoms Baseline report of tooth-grinding at night predictor of TMD treatment during the observation period
Ahlberg et al. (2005)[103]	NR	750	>45 years	Questionnaire or self-report	Association between SB and TMD	Association between perceived orofacial pain and selfreported bruxism
Mundt et al. (2005)[107]	NR	2963	35-74	Questionnaire or self-report	Association between SB and TMD	Significant associations between bruxism and TMD signs in females and males

	Glaros et al. (2005)[104]	NR	96	Controls 35.4-44.9	Questionnaire or self-report	Association between SB and TMD	Parafunctional behaviors related with jaw pain levels in subjects with TMD and controls
	Gesch et al. (2005)[122]	NR	4290	20-79	Questionnaire or self-report	Association between SB and TMD	"Frequent clenching" significantly and clinically connected with subjective TMD symptoms
	Velly et al. (2003)[110]	NR	83	35.0 (NR) 19-59	Questionnaire or self-report	Association between SB and TMD	Clenching alone or combined with grinding, contributing factors to chronic MFP

	Velly et al. (2002)[123]	NR	152	NR	Questionnaire or self-report	Association between SB and TMD	Generalized TMD groups ("dysfunctionals") strongly related to clenching-grinding and depression
	Celic et al. (2002)[124]	NR	230	21.3 (2.1) 19-28	Questionnaire or self-report	Association between SB and TMD	Clinical TMD signs and symptoms weak association with awareness of parafunctional habits and with some occlusal parameters Caution to not overestimate findings in the clinical setting

	Huang et al. (2002)[125]	NR	261 (clinic cases) 1016 (matched controls)	NR	Questionnaire or self-report	Association between SB and TMD	Clenching identified as a risk factor for subjects with MP and MP+A
	MacFarlane et al. (2001)[126]	NR	131	36.0 (NR) 18-65	Questionnaire or self-report	Association between SB and TMD	PDS patients characterized by frequent headaches, history of facial trauma, teeth grinding, sleep problems, pain elsewhere in the body and high levels of psychological distress
	Ciancaglini et al.	NR	383	44.9 (14.8) 18-75	Questionnaire or self-report	Association between SB and TMD	Bruxism potentially harmful to

	(2001)[127]						the masticatory system Bruxism likely to have a direct relation with TMD and play an etiologic role
	Yamada et al. (2001)[128]	NR	94	NR	Questionnaire or self-report	Association between SB and TMD	SR bruxism associated with condylar bony change and DD in orthognathic surgery patients with TMJ disorders
	Israel et al. (1999)[129]	NR	83	35.0 (NR)	Questionnaire or self-report	Association between SB and TMD	Significant relationship between parafunctional masticatory activity and

						TMJ osteoarthritis, but not with synovitis
Marklund et al. (2008)[130]	Longitudinal design	308	23.0 (NR) 18-48	Clinically based diagnosis of bruxism	Association between SB and TMD	Hypothesis of a positive relationship between awareness of bruxism and MP not rejected TMD signs and symptoms only in a minor proportion of subjects with awareness of bruxism
Storm et al. (2007)[87]	Longidutinal design	22	NR	Clinically based diagnosis of bruxism	Association between SB and TMD	Muscle and TMJ pain elicited with loading test as a discriminant between

						cases and controls Association between parafunctions and TMD
Güller et al. (2003)[88]	NR	64	29.0 (NR) 13-63	Clinically based diagnosis of bruxism	Association between SB and TMD	High prevalence of condylar bony changes in patients with bruxing behavior
Manfredini et al. (2003)[89]	NR	212	34.7	Clinically based diagnosis of bruxism	Association between SB and TMD	Bruxism more strongly associated muscle disorders than with DD and joint pathologies Association independent from other concurrent RDC/TMD

	Molina et al. (2003)[131]	NR	394	NR	Clinically based diagnosis of bruxism	Association between SB and TMD	TMD/bruxing and DAP patients more impaired by their functional disorders when compared with a group of TMD/bruxing and non-DAP patients and to controls
	Chung et al. (2000)[132]	Longitudinal study	26	16-54	Clinically based diagnosis of bruxism	Association between SB and TMD	Nocturnal bruxism mainly in the form of grinding rather than clenching No conclusions on bruxism-

						TMD relation
Molina et al. (1999)[133]	NR	276	34.8 (NR) 12-73	Clinically based diagnosis of bruxism	Association between SB and TMD	Higher prevalence of specific muscle and joint disorders in severe bruxers when compared to mild and moderate bruxers, and to the CMD nonbruxing group
Torisu et al. (2007)[134]	Experimental study	23	F 25.5 (1.0) M 23.5 (0.9)	EMG	Association between clenching or grinding tasks with onset of TMD-like symptoms	Combination of muscle fatigue (clenching task) and pain (injection of saline or glutamate) different

						effect on exteroceptive suppression response and resting EMG activity Potential clinical interaction between muscle fatigue and nociceptive regulation
Torisu et al. (2006)[135]	Experimental study	23	F 25.5 (1.0) M 23.5 (0.9)	NR	Association between clenching or grinding tasks with onset of TMD-like symptoms	Gender differences in the neuromuscular system as a potential contributor to a greater female susceptibility to develop chronic musculoskel

						etal pain problems
Glaros et al. (2004)[136]	Experimental study	14	21-35	NR	Association between clenching or grinding tasks with onset of TMD-like symptoms	Parafunctional activities increase pain and can lead to a TMD diagnosis
Svensson et al. (2001)[137]	Experimental study	11	23-27	NR	Association between clenching or grinding tasks with onset of TMD-like symptoms	Sustained, low-intensity clenching likely involved causally in the development of fatigue Short-lasting pain sensation in some individuals (other factors needed for longlasting pain)

	Arima et al. (1999)[138]	Experimental study	12	25.0 (2.0) 28-42	NR	Association between clenching or grinding tasks with onset of TMD-like symptoms	45 min grinding activity: marginal and self-limiting TMJ and masticatory muscles symptoms in the day following the exercise
	Glaros et al. (1998)[139]	Experimental study	5	23-29	NR	Association between clenching or grinding tasks with onset of TMD-like symptoms	Low-level parafunctional activity-pain relationship in some subjects
	Plesh et al. (1998)[140]	Experimental study	14	25.0 (3.0)	NR	Association between clenching or grinding tasks with onset of TMD-like symptoms	Postexertional pain 24 h later only in female, interpreted as true gender difference

						Unclear mechanisms for such pain, and no apparent relation with chronic pain pathology
Janal et al. (2007)[85]	Longitudinal design	51	34.5 (11.0)	Diagnosis based on tooth wear	Association between SB and TMD	Failure to show more tooth grinding in MP than control subjects Failure to support a model of MP maintenance by tooth grinding (no information on clenching or on the role of grinding in pain initiation)

	Schierz et al. (2007)[86]	NR	646	35-44	Diagnosis based on tooth wear	Association between SB and TMD	Exclusion of a clinically relevant increased risk for TMD from anterior TW
	Seligman et al. (2006)[141]	NR	300	13-78	Diagnosis based on tooth wear	Association between SB and TMD	Suggestion for a peculiar attrition pattern in MP Anterior attrition as a differentiating factor in the intracapsular models vs with the asymptomatic controls Asymptomatics: low anterior attrition severity and some

						mediotrusive wear
Pergamalian et al. (2003)[90]	NR	84	29.1 (8.1)	Diagnosis based on tooth wear	Association between SB and TMD	TW modestly correlated with age No association between TMD and TW No indication for bruxism as a TW accelerator in TMD Bruxism not associated with higher levels of muscle pain severity. Inverse relationship between bruxism and TMJ pain

	John et al. (2002)[142]	NR	274	13-76	Diagnosis based on tooth wear	Association between SB and TMD	Incisal TW not associated with TMD Exclusion of a clinically relevant increased risk for TMD from incisal TW
Manfredini et al. (2010); continued	Rossetti et al. (2008a)[36]	NR	60	19-42	PSG	Association between SB and TMD	RMMA during sleep associated with MFP and risk factor (although small) for MFP Daytime clenching potential risk factor for MFP
	Rossetti et al.	NR	26	19-42	PSG	Association between SB and TMD	SB neither associated with general

	(2008b) [77]					TMD nor pain on palpation Pain only in some SB individuals
	Rompré et al. (2007) [78]	NR	143	SB 26.5 (0.6) Controls 24.5 (0.9)	PSG	Association between SB and TMD	SB-RDC: high level of discrimination between SB and controls Pain frequently reported among SB with low frequencies of jaw muscle contractions
	Campar is et al. (2006a) [35]	NR	40	36.1 (11.3)	PSG	Association between SB and TMD	Inconclusive evidence for the association between facial pain and SB

		Van Selms et al. (2008)[143]	NR	8	23-43	EMG	Association between SB and TMD	Changes in chronic MMP more related to changes in stress than to those in parafunctional activities
		Baba et al. (2005)[79]	NR	103	F 23.7 (2.6) M 24.7 (2.0)	EMG	Association between SB and TMD	Association between masseter muscle activity and click sounds
Melo et al. (2018);[144] Journal of Oral Rehabilitation	Is there an association between psychotropic medications and presence of sleep bruxism?	Dias et al. (2014)[145]	Cross-sectional	100	43.4 (NR) 10 to 70	Questionnaire and clinical examination	Association between fluoxetine, paroxetine, and sertraline with SB	No association between use of SSRI and presence of SB was observed (OR=1.024; 95%CI=0.331-3.171)
		Gau et al.	Cross-sectional	467	10 to 17	Sleep Disturbance Questionnai	Association between	Methylphenidate use significantly

	(2009)[146]				re	methylphenidate and SB	increased the odds for the presence of SB (OR=1.670; 95%CI=1.030-2.680)
	Hermesh et al. (2005)[147]	Cross-sectional	75	SP 32.8 (10.1) Controls 32.7 (11.5)	Questionnaire and clinical examination (AASM criteria)	Association between citalopram, escitalopram, fluoxetine, and other unspecified SSRI with SB	No association between use of medication and presence of SB was observed, neither between the presence of SB in SP participants vs. controls (P=0.070)
	Ortega et al. (2014)[148]	Cross-sectional	207	CP-drug 8.9 (3.8) CP-no drug	Caregivers were interviewed using questionnaires	Association between barbiturate, benzodiazepine, carbamazepine,	Only the use of barbiturate was associated with

			9.0 (4.0) Control 8.4 (3.3)		and valproate with SB	increased odds for SB (OR=14.70; 95%CI=1.850-116.90), while no increased odds were observed with the use of benzodiazepine, carbamazepine, and valproate
Uca et al. (2015)[149]	Cross-sectional	807	38.4 (11.49)	Questionnaire and clinical examination (AASM criteria)	Association between citalopram, duloxetine, escitalopram, fluoxetine, mirtazapine, paroxetine, setraline, and venlafaxine with SB	With regard to different medications, the use of duloxetine (OR=2.16; 95%CI=1.12-4.17), paroxetine (OR=3.63; 95%CI=2.15-6.13), and

							venlafaxine (OR=2.28; 95%CI=1.34 -3.86) was associated with increased odds for SB. No increased odds were observed with citalopram, escitalopram , fluoxetine, mirtazapine, and sertraline.

Legend: AASM: American Academy of Sleep Medicine; AB: Awake Bruxism; AH: Apnea-Hypopnea; AHI: Apnea-Hypopnea Index; BMI: Body Mass Index; CI: Confidence Interval; CFP: Chronic Facial Pain; CMD: Craniomandibular Disorders; CP: Cerebral Palsy; DD: Disk Displacement; DSM-IV: Diagnostic and Statistical Manual of Mental Disorders (fourth edition); EMG: Electromyography; F: Female; GERD: Gastroesophageal Reflux Disease; ICDH: International Classification of Headache Disorders; M: Male; MDMA: Methylenedioxymethamphetamine; MFP: Myofascial Face Pain; MP: Myofascial Pain; MP+A: Myofascial Pain+Arthralgia; NA: Not Available; NR: Not Reported; NTC: Nonfunctional Teeth Contact; OMA: Oromotor Acticity; OR: Odds Ratio; OSA: Obstructive Sleep Apnea; PDS: Pain-Dysfunction Syndrome; PSG: Polysomnography; RCT: Randomized Controlled Trial; RDC/TMD: Research Diagnostic Criteria for Temporomandibular Disorders; RMMA: Rhythmic Masticatory

Muscle Activity; RMS: Root-Mean-Square; RR: Relative Risk; SB: Sleep Bruxism; SB-RDC: Research Diagnostic Criteria for Sleep Bruxism; SD: Standard Deviation; SHS: Second-hand smoke; SP: Social Phobia; SSRI: Selective Serotonin Reuptake Inhibitor; TCH: Teeth Contact Habit; TM: Torus Mandibularis; TMD: Temporomandibular disorder; TMJ: Temporomandibular joint TP: Torus Palatinus; TTH: Tension-type headache; TW: Tooth wear.

Supplementary table 3 - references

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Apêndice G - Tabela suplementar 4

Do artigo em inglês:
Supplementary table 4 - Summary of descriptive characteristics of included articles in prognostic systematic reviews (n=10).

SYSTEMATIC REVIEW CHARACTERISTICS		INCLUDED STUDIES CHARACTERISTICS (n=60)						EFFECTS ON STOMATOGNATHIC STRUCTURES	MAIN FINDINGS
Author (Year); Journal	**Objectives or research question**	**Included studies**	**Study design**	**Sample**	**Mean age (SD) or age range, in years**	**Follow-up period**	**Bruxism diagnostic criteria**		
Chrcanovic et al. (2015);[1] Implant Dentistry	In patients being rehabilitated with dental implants, what is the effect	Naert et al. (1992)[2]	Retrospective analysis	91 patients	53.7 (NR) 15-88	6 y 10 mo	NR	Effect of bruxism on dental implant failure	RR=7.49 (95%CI, 3.59-15.64)
		Glauser et al. (2001)[3]	Prospective noncontrolled study	41 patients	52.0 (NR) 19-72	12 mo after loading	NR	Effect of bruxism on dental implant failure	RR=3.30 (95%CI, 1.62-6.75)

	of bruxism on the implant failure rates, postoperative infection, and marginal bone loss?	Engstrand et al. (2003)[4]	Prospective noncontrolled study	95 patients	68.5 (NR) 45-89	2.5 y (mean) 1-5 y	NR	Effect of bruxism on dental implant failure	RR=2.25 (95%CI, 0.57-8.90)
		Nedir et al. (2004)[5]	Controlled clinical trial	236 patients	18-89	7 y	NR	Effect of bruxism on dental implant failure	RR=12.67 (95%CI, 1.16-137.90)
		Ibañes et al. (2005)[6]	Prospective noncontrolled study	41 patients	62.1 (NR) 38-82	12-74 mo	Patients were considered bruxers when they presented with teeth grinding or/and clenching, in combination with other symptom	Effect of bruxism on dental implant failure	Not estimable No implant failures observed in both groups.

					s like a sore or painful jaw, headache, earache, anxiety, stress and tension, and eating disorders		
Bischof et al. (2006)[7]	Retrospective analysis	212 patients	49.9 (NR) 22-88	5 y	NR	Effect of bruxism on dental implant failure	RR=2.46 (95%CI, 0.42-14.39)
Siebers et al. (2010)[8]	Controlled clinical trial	76 patients	52.0 (13.0) 22-85	38 mo (mean)	NR	Effect of bruxism on dental implant failure	RR=0.78 (95%CI, 0.09-6.81)
Maló et al. (2011)[9]	Retrospective analysis	221 patients	56.8 (NR) 34-84	5 y	Classified in absent or present, and diagnose	Effect of bruxism on dental implant failure	RR=0.66 (95%CI 0.30-1.47)

						d by evaluating the degree of tooth wear vs the patient's age or the degree of prosthesis wear vs the prosthesis time in function and by asking the patient		
Ji et al. (2012)[10]	Retrospective analysis	45 patients	61.5 (NR) 22-88	42.1 mo (mean) 1-125.5	NR		Effect of bruxism on dental implant failure	RR=6.37 (95%CI, 3.16-12.85)

						mo			
		Schneider et al. (2012)[11]	Retrospective analysis	70 patients	50.7 (NR) 19.8-76.6	6.2 y (mean) 4.73-11.7 y	NR	Effect of bruxism on dental implant failure	RR=2.44 (95%CI, 0.49-12.28)
De Souza Melo et al. (2017);[12] Journal of Prosthetic Dentistry	Is sleep bruxism associated with an increased frequency of ceramic restoration failures?	Beier et al. (2012a)[13]	Retrospective cohort	84 participants 318 teeth	44.42 (NR) (13.14)	118 (63) mo	Self-report; Clinical inspection (signs of clenching or grinding)	Effect of sleep bruxism on ceramic restoration failure (laminate veneers)	HR=7.74 (95%CI, 2.5-24.14), P=0.0012
		Beier et al. (2012b)[14]	Retrospective cohort	302 participants 1335 teeth	46.51 (13.14)	102 (60) mo	Self-report; Clinical inspection (signs of occlusal wear)	Effect of sleep bruxism on ceramic restoration failure (inlays, onlays, laminate veneers, single-crowns)	HR=2.31 (95%CI, 1.28-4.06), P=0.0045
		Beier et al. (2012c)[15]	Retrospective cohort	120 participants	46.2 (12.5)	113 (63) mo	Means of direct questions and	Effect of sleep bruxism on ceramic restoration	No greater risk of failure

		587 teeth			visual observation of patient behavior and teeth (presence of facets by clenching, grinding, and gnashing)	failure (inlays and onlays)	P=0.408
Fabbri et al. (2014)[16]	Retrospective cohort	312 participants 808 teeth	Men 19-61 Woman 19-71	12-72 mo	Questionnaire (muscle or teeth tenderness in the morning or evening, morning headache; reported	Effect of sleep bruxism on ceramic restoration failure (onlays, single-crowns, veneers)	OR=0.72 (95%CI, 0.27-1.92)

					sounds of teeth grinding from partner; diurnal feeling of teeth clenching, and frequent fractures of teeth or direct restorations) Intraoral clinical evaluation		
Granell-Ruíz et al. (2014)[17]	Retrospective cohort	70 participants 323 teeth	46.0 (NR) 18-74	36-132 mo	Clinical inspection of teeth (consequences of clenching or	Effect of sleep bruxism on ceramic restoration failure (laminate veneers)	OR=2.52 (95%CI, 1.24-5.12)

					grinding activities, visible in the dentition and consistent with a bruxing habit)		
Monaco et al. (2013)[18]	Retrospective cohort	398 participants 1132 teeth	48.6 (NR) 18-84	12-60 mo	Presence of parafunctions (clenching or bruxism); Parafunctions in combination with the absence of wear facets	Effect of sleep bruxism on ceramic restoration failure (single crowns)	OR=2.60 (95%CI, 1.45-4.66)

		Simeone et al. (2015)[19]	Retrospective cohort	107 participants 275 teeth	52.0 (15.0)	12-132 mo	Occlusal signs and clinical symptoms of bruxism	Effect of sleep bruxism on ceramic restoration failure (single crowns)	OR=0.05 (95%CI, 0.01-0.39)
		Smales et al. (2004)[20]	Retrospective cohort	50 participants 97 teeth	12-72	12-72 mo	Matching facets on extensively worn opposing teeth and the enlargement of masseter muscles	Effect of sleep bruxism on ceramic restoration failure (onlays)	OR=1.43 (95%CI, 0.48-4.26)
Hsu et al. (2012);[21] The International Journal of Oral & Maxillofa	How can biomechanical implant complications be identified and managed?	Wahlstrom et al. (2010)[22]	Retrospective	43 patients 264 implants	NR	61.3 mo	NR	Bruxism effects on implants biomechanical failures	Veneer fractures were not significantly correlated with use of an occlusal

cial Implants **								appliance or bruxism
	Kinsel et al. (2009)[23]	Retrospective	105 patients 729 implants	NR	<5 y or >5 y	NR	Bruxism effects on implants biomechanical failures	1. Prosthetic fractures were significantly associated with bruxism 2. Risk of prosthetic porcelain fracture was seven times greater in patients with bruxism compared to patients without bruxism

	Bragger et al. (2001)[24]	Retrospective	85 patients 103 implants	NR	4 to 5 y	NR	Bruxism effects on implants biomechanical failures	Bruxism and cantilevers were associated with more mechanical failures
	Esposito et al. (2000)[25]	Retrospective	9 patients 10 implants	NR	11 mo to 6 y	NR	Bruxism effects on implants biomechanical failures	Four failed implants in patients with bruxism
	Rangert et al. (1995)[26]	Retrospective	39 patients 297 implants	NR	32 mo	NR	Bruxism effects on implants biomechanical failures	Bruxism or excessive occlusal force was associated with fracture of the prostheses

Manfredini et al. (2014);[27] Clinical Implant Dentistry	Role of bruxism as a risk factor for the different complications on dental implant-supported rehabilitations	Ji et al. (2012)[10]	NR	45 patients	61.6 (NR) 25-88	1-125.5 mo	NR	Bruxism and biological complications in implant-supported restorations	Higher failure rates in bruxers (29.3% implants [17/58] vs 4.6% [11/239])
		Zupnik er al. (2011)[28]	NR	341 implants	52.4 (13.0) 20-81	NR	Self-reported clenching history	Bruxism and biological complications in implant-supported restorations	Clenching: OR=0.22 (95%CI, 0.04–1.41) for implant failure
		Luongo et al. (2010)[29]	NR	218 patients 273 implants	51 (NR) 19-89	1 y	Bruxism history	Bruxism and biological complications in implant-supported restorations	No failures after loading
		Siebers et al. (2010)[8]	NR	76 222 implants	59.2 (13.1) 22-85	1.5-7.2 y	Bruxism history	Bruxism and biological complications in implant-	No association between bruxism history

						supported restorations	and implant failure (data not shown)
Fischer et al. (2008)[30]	NR	24 patients 142 implants	64.0 (NR)	5 y	Assessment of bruxism signs (unspecified criteria and number of patients)	Bruxism and biological complications in implant-supported restorations	Four implants (in two patients) failed after loading – one of the two patients had bruxism/ poor hygiene
Herzberg et al. (2006)[31]	NR	70 patients 212 implants	52.0 (NR) 32-75.6	6-56.5 mo	Bruxism habits Protective mouth guard to bruxers	Bruxism and biological complications in implant-supported restorations	No association between bruxism and marginal bone loss

Ibanez et al. (2005)[6]	NR	41 patients 343 implants	62.1 (NR) 38-82	12-74 mo	Clinical assessment of bruxism-related symptoms	Bruxism and biological complications in implant-supported restorations	The only two implants that failed were in a bruxer (plus other risk factors)
Nedir et al. (2004)[5]	NR	236 patients 528 implants	18-89	7 y	Bruxism habits	Bruxism and biological complications in implant-supported restorations	Two out of three implant failures were in a bruxers (plus other risk factors: age of 81, poor hygiene, and smoking)
Henry et al. (2003)[32]	NR	51 patients 153 implants	62.3 (9.2) 43-79	1 y	Bruxism signs before treatment	Bruxism and biological complications in implant-	Four out of seven subjects reporting implant

					Bruxism signs during treatment	supported restorations	failures were bruxers (plus other risk factors) One out of six before-treatment bruxers lost all implants
Eckert et al. (2001)[33]	NR	63 patients 85 implants	NR	280 days (median) 0-734 days	Bruxism history (single-item bruxism diagnosis)	Bruxism and biological complications in implant-supported restorations	Bruxism: HR=1.7, P=0.56
Ekfeldt et al. (2001)[34]	NR	53 patients 301 implants	41 to >70	NR	Diagnostic signs of bruxism	Bruxism and biological complications in implant-supported restorations	Bruxism attributed as cause of implant failure in 4/27 patients

							(clinicians' opinion)
Glauser et al. (2001)[3]	NR	41 patients 127 implants	52.0 (NR) 19-72	1 y	Assessment of bruxism (unspecified criteria)	Bruxism and biological complications in implant-supported restorations	41% failure rate out of 22 implants in bruxers versus 12% out of 105 implants in nonbruxers (at patients' level: P=0.086; at fixture level: P=0.002) – OR=0.20
Wannfors et al. (2000)[35]	NR	40 patients	31-78	1 y	Bruxism history (unspecified	Bruxism and biological complications in implant-	6 patients out of 17 with failures in

		150 implants			number of subjects)	supported restorations	one-stage surgery were bruxers versus 4/23 in two-stage surgery Correlation between bruxism and implant failure at fixture level ($p<0.05$), no correlation at the individual level ($P>0.05$) – OR=3.0
Lindquist et al. (1996)[36]	NR	47 patients	51.0 (NR)	12-15 y	Tooth clenching (unspecif	Bruxism and biological complications in implant-	No correlation between tooth

		273 implants			ied criteria for diagnosis)	supported restorations	clenching and marginal bone loss
Schneider et al. (2012)[11]	NR	70 patients 100 implants	50.7 (NR) 19.8-76.6	6.2 y (mean) 4.7-11.7 y	Self-report bruxism	Bruxism and mechanical complications in implant-supported restorations	Bruxism did not predict mechanical or biological complications
Malò et al. (2011)[9]	NR	221 patients 995 implants	56.8 (NR) 34-84	5 y	Bruxism (anamnesis plus tooth wear; unspecified number of bruxers)	Bruxism and mechanical complications in implant-supported restorations	Bruxism is a risk factor for mechanical complications OR=60.9 (95%CI, 21.4–173), P=0.000
Wahlstrom et al. (2010)[22]	NR	46 patients	59.0 (NR) 36-84	61.3 mo (mean	Self-reported bruxism	Bruxism and mechanical complications in	Four implants lost in

			116 implants		) 40-84 mo		implant-supported restorations	two patients were in two bruxers Frequency of veneer fractures not related with bruxism
	Kinsel et al. (2009)[23]	NR	152 patients 729 implants	NR	NR	NR	Bruxism and mechanical complications in implant-supported restorations	15/43 (34.9%) patients with signs of bruxism had metal ceramic fracture(s) versus 20/109 (18.3%) patients without

							bruxism (P=0.030) At dental unit level bruxers had fractures in 59/312 (18.9%) versus 35/686 (5.1%) in nonbruxers (P<0.001) Protective effect of oral appliances
Tawil et al. (2006)[37]	NR	109 patients 262 implants	53.6 (NR) 25-86	53 mo	Bruxism habits	Bruxism and mechanical complications in implant-supported restorations	No differences in complications between bruxism

								groups (P=0.51)
De Boever et al. (2006)[38]	NR	105 patients 283 implants	59.1 (13.5) 25-86	62.5 (25.3) mo	Bruxism habits		Bruxism and mechanical complications in implant-supported restorations	Mechanical complications: 17/43 (39%) reconstructions in bruxers versus 29/126 (23%) in nonbruxers - P<0.001
Bragger et al. (2001)[24]	NR	85 patients 105 implants	55.7 (NR) 23-83	4-5 y	NR		Bruxism and mechanical complications in implant-supported restorations	Mechanical complications: 6/10 (60%) in bruxers versus 13/75 (17.3%) in nonbruxe

									rs – P<0.001
Manfredini et al. (2015);[39] Journal of Periodontology	Is there any evidence that bruxism may cause periodontal damage *per se*?	Calderon et al. (2009)[40]	Case-control	115 individuals	14-37	NR	Clinical bruxism diagnosis (tooth wear, shiny spots, masseter hypertrophy) – three examiners	Influence of bruxism on periodontal perception by the assessment of interdental tactile threshold	Minimum interdental threshold of 0.013-0.016 mm for both bruxers and nonbruxers; P=0.74
		Tokiwa et al. (2008)[41]	Unspecified cohort	50 individuals	41.2 (NR) 23-74	NR	Assessment of grinding types (canine vs molar grinding)	Prevalence of periodontal problems in individuals with different grinding patterns	Individuals with grinding patterns involving the molars have higher values of attachment loss, tooth

							mobility, non-carious cervical lesions, and dental hypersensitivity
Ono et al. (2008)[42]	Case-control	28 students	26.3 (NR) 21-30	NR	Nocturnal masseter EMG	Influence of bruxism on periodontal perception by the assessment of interdental tactile threshold	1. Mean periodontal sensation by interocclusal tactile threshold in bruxers lower than controls (P<0.0001) 2. Same pattern of force voluntary clenching

							-induced tooth displacement, irrespective of bruxism status, but higher displacement in bruxers (P<0.05)
Bernhardt et al. (2006)[43]	Cohort study	2980 indviduals	20-79	NR	Self-reported bruxism	Association between self-reported bruxism and periodontal problems at the general population level	Bruxism not associated with plaque score or clinical attachment loss
Martinez-Canut et al. (1997)[44]	Cohort study	825 peridontal patients	42.5 (NR) 19-72	NR	Self-reported bruxism or clenching	Prevalence of pathological tooth migration in a cohort sample of periodontal	Pathological tooth migration: 15% of bruxers vs 12% of

							Attrition to confirm bruxism	patients in relation to self-reported bruxism	non-bruxers (P=0.159); 26% of clenchers vs 28% non-clenchers (p=0.551)
		Hanamura et al. (1987)[45]	Cohort study	51 patients with modera to-to-severe periodontitis 40 patients with bruxism-tooth wear	51 patients 48.2 (NR) 35-60 40 patients 48.9 (NR) 37-62	NR	Self-reported bruxism	Differences in periodontal parameters between two cohorts of periodontal or bruxism patients	Higher bone level in bruxers (88% of root length vs 72%; P<0.001)
Salvi et al. (2009);[46] The	Which mechanical/	Bragger et al. (2001)[24]	Retrospective cohort	85 patients	55.7 (NR) 23-83	56.8 mo (mean)	NR	Effects of bruxism on implant failure	13/75 nonbruxers (17.3%)

Inernational Journal of Oral & Maxillofacial Implant **	technical risk factors have an impact on implant-supported reconstructions?			103 implants 1042 teeth					had technical complications and 6/10 bruxers (60%) had complications P<0.01 No influence on implant loss
		Ekfeldt et al. (2001)[34]	Clustered failures as test group gersus matched control group	54 patients 301 implants	NR	NR	NR	Effects of bruxism on implant failure	In the test group with clustered losses, there were 7 patients with bruxism Heavy influence

								on implant loss
	De Boever et al. (2006)[38]	Consecutive	105 patients 283 implants	25-86	65.2 (25.3) mo	NR	Effects of bruxism on implant failure	17/43 (39%) had complications in the bruxing group 29/126 (23%) had complications in the non-bruxing group P<0.01 No influence on implant loss
	Tawil et al. (2006)[37]	Consecutive patients	109 patients	53.6 (NR) 22-80	53 mo (mean)	NR	Effects of bruxism on implant failure	22.6% of the patients

				12-108 mo			were defined as bruxers; they had 50% of the veneer fractures; however; however, not significant No significant influence on implant loss
Nedir et al. (2006)[47]	Consecutive patients	215 patients 72 implants	NR	8 y life table	NR	Effects of bruxism on implant failure	No statistically significant increase in complication rate

									for fixed dental prostheses and overdentures Not significant
Schmitter et al. (2014);[48] International Journal of Prosthodontics	Investigate the influence of patient-related factors on restoration survival as well as to report the methods used to collect these factors.	Eligible studies have excluded bruxers or did not present reliable bruxism diagnostic criteria	Not applicable	Not applicable	Not applicable	Not applicable	Not applicable	Influence of patient-related factors on restoration survival	Although several studies assess the survival of veneered zirconia restorations, there is a lack of information about the effect of bruxism on the incidence

									of technical failures because none of the available studies used a reliable and valid instrument to diagnose bruxism.
Van de Sande et al. (2016);[49] Operative Dentistry **	Investigate the influence of patient-related factors on restoration survival as well as to report the methods used to	Adolphi et al. (2007)[50]	NR	NR	NR	NR	Signs of bruxism	Bruxism	NR
		Beier et al. (2012c)[15]	Historical cohort	120 patients 547 restorations	46.0 (NR) 14-72	12 y	Self-reporting by direct questions and inspection of clinical signs consisten	Bruxism (ceramic)	Not statistically significant on restoration survival

collect these factors.					t with past bruxism behavior from the presence of clear wear facets caused by clenching, gnashing, and grinding activities of the teeth not interpreted to be a result of masticatory function		

	Palllesen et al. (2003)[51]	NR	NR	NR	NR	Presence of bruxism was self-reported in interviews.	Bruxism	NR
	Smales et al. (1993)[52]	Cohort	105 patients 582 restorations	<20->41	5 y	Extensive tooth wear (obvious evidence of bruxism)	Bruxism (amalgam)	Statistically significant on restoration survival
	Smales et al. (2004)[20]	Historical cohort	50 patients 97 restorations	15->51	6 y	Evidence of parafunction was collected from dental records. Authors stated that occlusal splints	Bruxism (ceramic)	Not statistically significant on restoration survival

						were generally made for patients when multiple onlays were placed or parafunctional habits were obvious, as shown by matching facets on extensively worn opposing teeth and the enlargement of masseter muscles.		

	Van de Sande et al. (2013)[53]	Historical cohort	44 patients 306 restorations	47.0 (NR) 25-71	15 y	Self-reporting by six direct questions and clinical signs of bruxism were visually inspected (wear facets, loss of contour, dentin exposure). Patients were classified as having high occlusal stress risk	Bruxism (composite resin)	HR/OR= 2.78 (95%CI, 1.39-5.59)

					when answered positively on two or more questions and presented at least one of the clinical parameters. In other cases, they were classified as low risk.		
Van Dijken et al. (2013)[54]	Cohort	121 patients 117 restorations	52.0 (NR) 26-81	15 y	Bruxism was estimated as low or high by the	Bruxism (ceramic)	HR/OR= 0.38 (95%CI, 0.19-0.77)

							treating clinician by means of clinical signs and history at the annual examinations.		
		Zimmer et al. (2008)[55]	Historical cohort	95 patients 308 restorations	44.0 (NR) 22-65	1 y	In addition to personal data, the presence of bruxism by wear facets was noted.	Bruxism (ceramic)	Not statistically significant on restoration survival
Vant't Spijker et al. (2007);[56]	To systematically assess	Baba et al. (2004)[57]	Cross-sectional	16	Young adult 19-30	NR	NR	Relationships between attrition and bruxism activity	No significant relationsh

Clinical Oral Implants Research **	relationships, if any, between attrition and occlusal factors and oral (dys)function in terms of management of attrition							ip between tooth wear and current bruxism
		Carlsson et al. (2003)[58]	Longitudinal	320	Young adult 19-30	NR	NR	Relationships between attrition and bruxism or oral parafunctions	Anterior tooth wear at 15 years of age predicts reported tooth grinding at night 20 years later
		Pergamalian et al. (2003)[59]	Cross-sectional	84	Young adult 19-30	NR	Self-report	Relationships between attrition and history of self-reported bruxism	No correlation between tooth wear and TMD pain. Tooth wear not correlated

							with reported bruxism
Pintado et al. (1997)[60]	Longitudinal	18	Yong adult 19-30	NR	NR	Relationships between attrition and bruxism	Bruxers show more volume loss per time period than non-bruxers
Ekfeldt et al. (1990)[61]	Cross-sectional	220	Young adult 19-30 Adult 31-64 Elderly >65	NR	NR	Relationships between attrition and bruxism	Higher prevalence of bruxism in subjects with tooth wear compared with subjects without
Seligman et al. (1988)[62]	Cross-sectional	222	Adolescents 12-18	NR	NR	Relationships between attrition and bruxism	Dental attrition not

			Adults 31-64				associated with TMJ clicking. In male: attrition of canines and premolars associated with reported bruxism
Szentpetery et al. (1987)[63]	Cross-sectional	600	All age groups	NR	NR	Relationships between attrition and bruxism	Correlation between excessive tooth wear and dysfunction signs and between excessive tooth wear and reported bruxism

	Egemark-Eriksson et al. (1987)[64]	Longitudinal	240	Children <11 Adolescents 12-18	NR	NR	Relationships between attrition and occlusal factors/temporomandibular joint disorders/bruxism	Tooth wear correlated with reported bruxism for ages 11 and 15 years. No correlation between tooth wear and TMD
	De Laat et al. (1986)[65]	Cross-sectional	121	Children <11 Adolescents 12-18	NR	Reported bruxism	Relationships between attrition and bruxism	More dental wear in subjects with reported bruxism. Dental wear correlated with muscle pain

		Droukas et al. (1984)[66]	Cross sectional	48	Yong adult 19-30 Adult 31-64	NR	Reported bruxism	Relationships between attrition and bruxism	Negative correlation between attrition of premolars and clinical dysfunction index. No correlation between attrition and reported bruxism
Zhou et al. (2016);[67] Clinical Implant Dentistry and Related Research	Does bruxism contribute to dental implant failure?	Papaspyridakos et al. (2014)[68] and Papaspyridakos et al. (2013)[69]	Cohort	14 patients	58.0 (NR) 35-71	3 y (mean) 2-4 y	NR	Ceramic chipping	Analysis based on number of prosthesis OR=77.00 (95%CI, 2.67-2222.91)

							Analysis based on number of patients OR=189.00 (95%CI, 3.22-11095.09)
Ji et al. (2012)[10]	Cohort	45 patients	61.5 (NR) 25-88	1-10 y	NR	Acrylic resin base fracture, broken denture teeth, screw loosening, screw fracture, and/or framework misfit	Analysis based on number of prosthesis OR=8.59 (95%CI, 3.75-19.67)
Kinsel et al. (2009)[23]	Cohort	152 patients	<60 y 102 individuals >60 y 50 individuals	5 y	NR	Porcelain fracture	Analysis based on number of prosthesis OR=4.34 (95%CI, 2.79-6.75)

							Analysis based on number of patients OR=2.38 (95%CI, 1.08-5.27)
De Boever et al. (2006)[38]	Cohort	105 patients	59.1 (NR) 25-86	62.5 (5.3) mo 4-144 mo	NR (may be available within full-text)	Mechanical complications	Analysis based on number of prosthesis OR=2.19 (95%CI, 1.04-4.58)
Glauser et al. (2001)[3]	Cohort	41 patients	52 (NR) 19-72	1 y	NR (may be available within full-text)	Implant loss	Analysis based on number of prosthesis OR=4.90 (95%CI, 1.75-13.71)

	Mangano et al. (2014)[70]	Cohort	194 patients	49.1 (11.5) 24-74	1-10 y	NR (may be available within full-text)	Implant loss, bone loss, porcelain fractures, abutment loosening	Analysis based on number of patients OR=5.24 (95%CI, 0.93-33.18)
	Bragger et al. (2001)[24]	Cohort	85 patients	55.7 (NR) 23-83	56.8 mo (mean) 40-78 mo	NR	Technical complications: loss of retention, porcelain fracture, screw loosening	Analysis based on number of patients OR=7.15 (95%CI, 1.77-28.99)

Legend: CI: Confidence Interval; EMG: Electromyography; HR: Hazard Ratio; MO: Months; NA: Not Available; NR: Not Reported; OR: Odds Ratio; RR: Relative Risk; SD: Standard Deviation; TMD: Temporomandibular Disorder; Y: Years; (**) Data were colleted only from bruxism-related primary studies.

Supplementary table 4 - references

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Apêndice H - Tabela suplementar 5

Do artigo em inglês:
Supplementary table 5 - Summary of descriptive characteristics of included articles in intervention systematic reviews (n=16).

SYSTEMATIC REVIEW CHARACTERISTICS		INCLUDED STUDIES CHARACTERISTICS (n=67)							
Author (Year); Journal (2016 Impact Factor)	**Objectives or Research Question**	**Included studies**	**Study design**	**Sample**	**Mean age (SD) or age range, in years**	**Follow-up period**	**Bruxism diagnostic criteria**	**INTERVENTIONS**	**MAIN FINDINGS**
Canales et al. (2017);[1] Clinical Oral Investigations	Is there enough evidence to use botulinum toxin injections for	Shim et al. (2014)[2]	RCT	24	20.2-38.7	4 weeks	Recent history of tooth grinding at least three nights per week, morning	Botulinum toxin injections	BoNT-A injection did not reduce the frequency, number of bursts or duration for

	bruxism management?					jaw stiffness and clinical presence of tooth wear.		RMMA episodes in the two groups. The injection decreased the peak amplitude of EMG burst of RMMA episodes in the injected muscles in both groups.
		Redaelli et al. (2011)[3]	Before-after study	120	NR	NR	Nocturnal bruxism (unspecified criteria)	Botulinum toxin injections	94.1% of the patients declared a fairly good to excellent result after BoNT-A injection.
		Lee et al. (2010)[4]	RCT	12	M 25 (2.3) F 24.8 (0.8)	12 weeks	Nocturnal bruxism (unspecified criteria)	Botulinum toxin injections	The injection of botulinum toxin in the

								masseter muscle reduces the number of bruxism events during sleep for up to 12 weeks.
	Guarda-Nardini et al. (2008)[5]	RCT	20	25-45	6 months	Nocturnal bruxism (screening-oriented clinical diagnostic criteria)	Botulinum toxin injections	Patients treated with BoNT-A had a higher subjective improvement in their perception of treatment efficacy and reduction in pain whilst chewing, after 6 months.
	Bolayir et al. (2005)[6]	Before-after study	12	18-35	3 months	Nocturnal bruxism individuals who had	Botulinum toxin injections	The injection of BoNT-A in the masseter

							not responded to splint and medication treatment		muscle reduces pain degree up to 3 months.
Hillier et al. (2015);[7] Evidence-Based Complementary and Alternative Medicine **	1. Systematically identifying and appraising the evidence for the effectiveness of the Feldenkrais Method across domains 2. Determining what is the nature and order of magnitud	Quintero et al. (2009)[8]	RCT (cross-over design)	NR	3-6	10 weeks	NR	Feldenkrais method	After intervention 77% parents in feldenkrais method reported no nocturnal bruxism compared with 15.38% for controls.

	e of any beneficial effects and for which populatio n								
Jokubaus kas et al. (2018); Journal of Oral Rehabilit ation	What is the effect of oral appliance s on various treatment outcomes in adult patients with SB	Matsum oto et al. (2015)[9]	RCT	20	28.9 (NR)	4 weeks	Portable EMG system	Occlusal splint	Intermittent use of occlusal splint may reduce SB activity for a longer period compared with that of continuous use.
		Dalews ki et al. (2015)[10]	RCT	30	26.6 (NR)	4 weeks	Four-channel EMG system (diurnal)	1. Occlusal splint 2. Nociceptive trigeminal inhibition splint	Neither device affected the asymmetry index or postural activity/max imum voluntary

							contraction ratio of the temporal and masseter muscles.
Singh et al. (2015)[1] [1]	RCT	28	34.7 (NR)	3 months	PSG (plus AV) in sleep laboratory	1. Mandibular advancement appliance (MAA) 2. Occlusal splint	Both devices significantly reduced the PSQI and SB episodes and bursts after 3 months. MAA provided greater reduction in SB episodes per hour, yet caused more discomfort than occlusal splint.

	Gu et al. (2015)[12]	RCT	24	25.6 (NR)	12 weeks	Mini device analysing bite force, and software for SB analysis	1. Maxillary occlusal splint + vibratory feedback 2. Maxillary occlusal splint without vibration	There were no significant differences in the episodes and duration of SB events in the occlusal splint group without vibration (in contrast to the other group).
	Gomes et al. (2014)[13]	RCT	15/15/15/15	28.0 (NR)	4 weeks	Eight-channel EMG system (diurnal)	1. Masseter and temproal muscle massages 2. Occlusal splint 3. Massage + occlusal splint 4. Soft occlusal splint	Massage therapy and the use of an occlusal splint had no significant influence on EMG activity of the masseter or anterior

								temporal muscles. A combination of treatments led to a reduction in the intensity of signs and symptoms in subjects with severe TMD and SB.
	Madani et al. (2013)[14]	RCT	20	28.9 (NR)	2 months	PSG in sleep laboratory	Occlusal splint + gabapentin (100mg) for the first 3 nights, then 200mg/night for the next 3 nights, thereafter 300 mg/night continued for 2 months	Significant reduction in most SB variables in both groups after treatment.

	Arima et al. (2012)[15]	RCT (cross-over)	11	25.9 (3.1)	1 week each	Portable EMG device		1. Maxillary and mandibular oral appliance which restricted mandibular movement/maxillary 2. Mandibular oral appliance with no restrictions and conventional maxillary oral appliance	The total number of phasic EMG episodes and bursts per hour of sleep is significantly reduced during any combination of oral appliance compared with baseline values. The restriction of mandibular movements with oral appliance does not have any major influence on

								jaw muscle activity during sleep.
	Landry-Schonbeck et al. (2009)[16]	RCT (cross-over)	12	26.0 (1.5)	2 weeks each	Polygraphy (plus AV) in sleep laboratory	Occlusal splint/ mandibular advancement appliance in 25%/ mandibular advancement appliance in 75% advancement position for 2 weeks each	MAA is more effective than OS to reduce SB. The short-term use of a robust MAA is associated with a significant SB decrease (no difference between the two positions was noted, yet 75% was superior). The OS did not reach

								any statistical significance.
	Ommerborn et al. (2007)[17]	RCT	57	29 (4.8)	6 months	Bite plate-like device measuring abrasion degree	Occlusal splint/cognitive behaviour therapy for 12 weeks	Significant reduction in SB activity, self-assessment of SB activity and psychological impairment, as well as an increase of positive stress-coping strategies in both groups. The effects were small, and no between-group differences were seen.

	Solanki et al. (2017)[18]	Before-after study	30	18-40	30 days	Ambulatory PSG	MAA in 50% advancement position	Statistically significant reductions in SB bursts and episodes per hour, and PSQI scores were found after 15 and 30 days; significant reduction in occlusal force after 15 days.
	Mainieri et al. (2014)[19]	Before-after study	19	39.9 (12.9)	3 months	Portable EMG device and PSG (plus AV) in sleep laboratory	MAA in 50%–75% of maximum protrusive position	MAA treatment resulted in the reduction of SB activity, SB signs and symptoms, occlusal force, and sleep scores

							(improvement in sleep quality). In 24% of patients treatment had to be stopped due to TMJ/muscle pain and/or discomfort.
Sjholm et al. (2014)[20]	Before-after study	14	27.5 (NR)	8 weeks	Polygraphy in sleep laboratory	Occlusal splint	43% of bruxists increased activity, while 36% decreased, and in 21%, there was no change in the level. OS does not have significant feedback inhibition on masseter

							muscle motor activity during sleep. However, OS may increase slow wave sleep.
Amorim et al. (2012)[21]	Before-after study	15	26.30 (3.0)	1 night	Eight-channel EMG system (diurnal)	Occlusal splint	Use of OS reduces EMG activity in the masseter and anterior temporalis muscles immediately after the insertion in patients with SB related to occupational stress.

	Sauressig et al. (2010)[22]	Before-after study	28	42.9 (12.0)	30 days	Portable EMG device	MAA in 50%–75% of maximum protrusive position	The soft thermoplastic had a positive effect on SB and sleep scores, and did not increase any traditional signs and symptoms of TMD.
	Amorim et al. (2010)[23]	Before-after study	15	26.5 (3.0)	30 days	Eight-channel EMG system (diurnal)	Occlusal splint	OS reduced the EMG activity of the right and left masseters in situation of mandibular rest and maximal isometric muscle contraction, showing its

									myorelaxing effect.
		Nascimento et al. (2008)[24]	Before-after study	15	22.13 (2.72)	60 days	Eight-channel EMG system (diurnal)	Occlusal splint	TMD signs and symptoms decreased significantly, but there was no significant difference in EMG records.
Jokubauskas et al. (2018);[25] Journal of Oral Rehabilitation	Assessing the most recent literature and providing a comprehensive summary of the efficacy of any biofeedback	Gu et al. (2015)[12]	RCT	24	25.65 (NR)	12 weeks	Clinical diagnostic criteria proposed by AASM	A maxillary OS + vibratory feedback/a maxillary OS without vibration	A significant decrease was observed in the biofeedback group when vibratory feedback was applied compared with the control after 6 and 12

	treatment approach for the reduction or control of SB.							weeks of treatment.
		Conti et al. (2014)[26]	RCT	15	34.6 (3.8)	10 ays	Questionnaire, clinical assessment	CES/blank control (placebo)	Significant differences were found in EMG episode/hour reduction for the biofeedback group after treatment (35% lower EMG level) and follow-up (38.4% lower EMG level) compared with baseline, but not for the control group.
		Sato et al. (2015)[27]	RCT	12	26.8 (2.5)	2 days	Clinical assessment and nighttime	Audio feedback during daytime	A significant decrease was

					EMG monitoring	clenching/blank control	observed in tonic EMG events compared with baseline, both daytime, and nighttime, in the biofeedback group.
Jadidi et al. (2013)[28]	RCT	11	37.0 (3.0)	6 weeks	Questionnaire, patient history, clinical assessment	CES/blank control (placebo)	After a 6-week treatment, the effect of CES showed a significant change in EMG episodes/hour with a reduction of 48-51% in contrast to a smaller

								nonsignificant decrease of 36% after the 4-week follow-up session. No changes were observed in the control group.
	Sumiya et al. (2014)[29]	Before-after study	10	26.7 (3.5)	2 nights	Nighttime EMG monitoring	Contingent electrical stimulation (CES)	Significant decrease in the numbers of SB events per night and per hour to approximately 45% of baseline values. Additionally, bursts per event and duration of events were suppressed

							to approximately 60% of baseline values on the two nights when CES was applied.
Raphael et al. (2013)[30]	Before-after study	14	34.9 (11.5)	6 weeks	Prior PSG evaluation, patient history	CES	CES was associated with a reliable reduction in EMG events after a 6-week treatment, but the frequency of these events returned to baseline levels during the 2-week follow-up.

Lang et al. (2009);[31] Research in Developmental Disabilities	This review involved a systematic analysis of studies that focused on the treatment of bruxism in individuals with developmental disabilities.	Alpoz et al. (1999)[32]	Experimental	1 with Rett Syndrome	5	NR	Dental screening under sedation found no occlusal abnormalities. Bruxism was diurnal	Soft acrylic prosthodontic was created that fit to upper jaw and prevented bruxing.	Authors stated treatment was effective but did not display data or discuss measures.
		Bebko et al. (1988)[33]	Experimental	2 with autism	10 and 11	6 months	Bruxism was audible and diurnal	When bruxism was heard, the participants were told ‘‘No grinding’’ and the therapist prompted to open mouth for 10 s by placing an index finger lightly on jaw outside of the cheek. After 10 s participant	Bruxism reduced from 86.6% of the time to 22.7% for participant 1 and from 62.8% to 27.9% of the time for participant 2. Similar effects were found across two school environments. At 6-month

						was praised for not grinding and released. The authors stress this was not punitive but gentle.	follow-up participant 1's bruxing had increased slightly to 40% and participant 2 no longer engaged in bruxing.
Blount et al. (1982)[3][4]	Experimental	2 with mental retardation	32 and 16	NR	Dental screening under sedation found no occlusal abnormalities. Bruxism was audible and diurnal	When bruxism was heard, ice was applied to the face near the cheek or jaw as a contingent punisher.	Bruxism reduced from 63% of the time to 8.4% of the time for participant 1 and from 60.6% to 11.4% for participant 2. Results generalized to times of day in which treatment

							was not conducted.
Caron et al. (1996)[35]	Experimental	4 with mental retardation	26-41	NR	Bruxism was audible and diurnal	Music Therapy: Participants were placed near a stereo and New Age music was played for 20 min.	No effect for any of the participants.
Ford (1999)[36]	Experimental	1 with mental retardation	NR	NR	Bruxism was audible and diurnal	Music Therapy: Participant was given headphones to listen to music, an electronic keyboard to play music, and was allowed to play in a tub of water (no electronics were placed in the water).	No effect.

	Gross et al. (1982)[37]	Experimental	2 with cerebral palsy and mental retardation	4	3 months	Bruxism was audible and diurnal	Praise was given contingent upon 10 s periods without bruxing. When bruxing occurred participants were physically prompted to exercise for 2 min as a contingent punisher.	Bruxism reduced from 75% of the time to 16% for participant 1 and from 85% to 8% for participant 2. At 3 month follow-up bruxing was 0% for both participants.
	Kramer (1981)[38]	Not-experimental	1 with mental retardation	8	3 weeks	Bruxism was audible and diurnal	When bruxing began the teacher said, ‘‘No’’ and blocked the bruxing by placing finger firmly on jaw	The participant averaged 18 incidents of bruxing in baseline and 10 during intervention. At 3-week follow-up

						outside of cheek.	bruxing occurred 0–3 times per day.
Monroy et al. (2006)[39]	Not-experimental	1 with autism and Bannayan-Zonana syndrome	12	60 days	Dental screening under sedation found no occlusal abnormalities. Description of the type of bruxism was not reported	Injection of botulinum toxin-a into each masseter while under general anesthesia for routine dental care.	Immediate and steady decrease in bruxing until cessation that last 60 days. At 60 days previous high levels of bruxing resumed (per phone interview with parent).
Muthu et al. (2008)[40]	Not-experimental	1 with mental retardation	4	1 year	Dental screening under general anesthesia found extensive	Full mouth rehabilitation consisting of stainless steel crowns on all molars,	Parents report substantial reduction and then elimination of bruxism

					damage to teeth likely causing considerable pain. Description of the type of bruxism was not reported	extraction of the maxillary right primary central incisor, and oral prophylaxis.	monitored for 1 year.
Romer et al. (1998)[41]	Not-experimental	1 with mental retardation	6	NR	Dental screening under general anesthesia found extensive damage to teeth likely causing considerable pain. Description of the type of bruxism was not reported	Five treatments were given in succession. **Treatment 1:** Repaired teeth with composite resin and prescribed antibiotics (Cefalexin suspension, 250 mg/5 ml, 500 mg BID for 10 days).	**Effects of treatment 1:** Bruxism and hand biting where reduced but tongue biting increased. **Effect of treatment 2:** Tongue biting was eliminated and mouth was healing however,

	Treatment 2: Removal of the tooth being used to bite the tongue and prescribed Amoxicillin suspension (250 mg q6h for 10 days). **Treatment 3:** Arm board used to keep hand out of mouth. **Treatment 4**: A mouth guard was placed to prevent bruxing. **Treatment 5:** Behavior modification (not described)	hand biting increased. **Effect of treatment 3:** Hand biting eliminated, but dramatic increase in bruxing causing sufficient damage to warrant hospitalization. **Effect of treatment 4:** Participant broke mouth guard and used broken edge to cut upper lip.

						Effect of treatment 5: Elimination of oral self-injury and mouth healed. Funding for behavior modification ended and treatment was withdrawn. After withdrawal bruxism and other self-injury returned. Behavior modification was not reintroduced

		Rudrud et al. (1981)[42]	Not-experimental	1 with mental retardation	43	NR	Bruxism was audible and diurnal	When bruxing began a 1-min massage was given by the residential facility's staff. The massage consisted of rubbing the participant's masseter muscles (around jaw line).	Bruxism reduced from 78.9% of the time in baseline to 28.7% of the time during intervention.
Lino et al. (2017);[43] Oral Diseases **	The aim of this systematic review was to search for scientific evidence of the efficacy of	Etzel et al. (1991)[44]	Placebo cross-over design	8	33.6 (NR) 22-47	3x8 days First 8 days baseline period	Patients diagnosed as chronic bruxers, with a history of nocturnal bruxing characterized by facial pain and/or	Tryptophan: 50 mg/kg of body weight	No significant treatment differences in bruxism levels were found, suggesting that Ltryptophan supplement

	antidepressant drugs for the treatment of oral problems.					restricted mandibular motion on awakening, excessive tooth wear, and grinding heard within the past 2 weeks (bedroom report).			ation in the absence of dietary manipulation is ineffective for the treatment of nocturnal bruxism. Reduction of muscle activity: 38% of the patients in the tryptophan group and 63% of patients in the placebo group (P>0.05).
		Mohamed et al. (1997)[4][5]	Placebo cross-over design	10	35.0 (12.0)	2x1 weeks with wash-out	Nocturnal grinding and/or clenching of		Amitriptyline / 25 mg amitriptyline per night	Small doses of amitriptyline cannot be recommend

					period	the teeth must be reported by the subject, and at least some occlusal tooth wear (attrition/ abrasion) must be present.		ed for the control of sleep bruxism and associated discomforts.
	Raigrodski et al. (2001a) [46]	Placebo cross-over design	10	39.0 (7.0) 31-54	2x4 weeks	Form and portable Masseteric Electromyography (EMG) for cumulative myoelectrical activity.	Amitriptyline: 25 mg per night	The administration of amitriptyline did not significantly (P>0.05) reduce nocturnal masseteric activity (40% of patients) and did not

								significantly (P>0.05) augment the duration of sleep.
	Raigrodski et al. (2001b)[47]	Placebo cross-over design	10	18 or older	2x4 weeks	The subject had to respond with a positive answer to at least one of the following questions: do you keep your teeth together; do you clench or grind your teeth together? The subject also had to agree not to consume alcohol for the duration	Amitriptyline: 25 mg per night	Amitriptyline did not significantly (P>0.05) reduce pain intensity levels but did significantly (P<0.05) reduce the level of stress in bruxers.

							of the study.			
Long et al. (2012);[48] International Dental Journal	The objective of this study was to assess the efficacy of botulinum toxins on bruxism.	Lee et al. (2010)[4]	RCT	12	20-30	12 weeks	NR		**Group 1:** each masseter [80 U Dysport (0.8 mL)] **Group 2:** each masseter (0.8 mL saline)	A significant decrease in bruxism frequency compared with saline group.
		Guarda-Nardini et al. (2008)[5]	RCT	20	25-45	6 months	NR		**Group 1:** each masseter (30 U Botox); each anterior temporalis (20 U Botox) **Group 2:** saline placebo	Significant decrease in pain on chewing and improvement in subjective efficacy compared with saline group.

	Sener et al. (2007)[4][9]	Controlled before-after study	13	NR	6 months	NR	**First stage (0–2 months):** nocturnal oral splint for 2 months. **Second stage (2–4 months):** wash out period. **Third stage (4–6 months):** 60 U Botox into masseters	Significant decrease in pain, sensitivity and weakness for both Botox and splint after treatment The two are equally effective.
	Bolayir et al. (2005)[6]	Controlled before-after study	12	18-35	3 months	NR	50 U Dysport into masseters	Subjectively reported less frequency of bruxism after injection VAS pain scores decreased significantly

									after treatment.
Macedo et al. (2007);[50] Cochrane Database of Systematic Reviews	To evaluate the effectiveness of occlusal splints for the treatment of sleep bruxismwith alternative interventions, placebo or no treatment.	Alvarez-Arenal et al. (2002)[51]	RCT (cross-over)	11	NR	1 month and a half washout period between treatments	Anamnese and/or a questionnaire, clinical examination, and tooth grinding reported by partner.	**Group A:** Occlusal splint (n = 11). They wore their splint 24 hours a day except for eating, for a 45-day period. **Group B:** Transcutaneous electric nerve stimulation (n = 11). Each transcutaneous electric nerve stimulation session lasted 45-60 minutes and	Patients treated with splint had a lower risk of clicks in TMJ during oral opening and closing when compared to the TENS group, but without statistical significance (risk ratio (RR) 0.60 (95% CI 0.19 to 1.92)). No statistically significant differences between groups were

							each patient underwent 15 sessions (1 every 2 days).	found in the clicks in TMJ, whether opening or closing the mouth (RR 1.00 (95% CI 0.33 to 3.02)).
	Dube et al. (2004)[52]	RCT	9	23.7 (0.9) 20-29	2 weeks for each proposed intervention	History of tooth grinding for at least 3 nights per week during the last 6 months reported by partner and polygraphic exam confirmed at least 4 episodes of sleep bruxism per	**Group A:** Occlusal splint (n = 9). **Group B:** Palatal splint (n = 9).	No statistically significant differences between intervention and control groups were found in the episodes with grinding noise (Outcome 1.4) (WMD 0.90 (95% CI -10.19 to 11.99)). No

		hour of sleep and at least 2 episodes with tooth grinding sound; presence of tooth wear showed at least the degree of exposed dentine (grade 2) and/ or masseter muscle hypertrophy upon voluntary clenching and/or symptons of morning orofacial jaw muscle fatigue			statistically significant differences between the groups were found in the awakenings during sleep (Outcome 1.5) (WMD 0.40 (95% CI -2.51 to 3.31)). No statistically significant differences between groups were found in sleep efficiency (Outcome 1.6) (WMD -2.40 (95% CI 8.36 to 3.56)).

	Hachmann et al. (1999)[53]	Quasi-randomized controlled trial	9	3-5	6 months	Tooth grinding and tooth grinding sounds during sleep reported by parents, abnormal tooth wear and jaw muscle discomfort	**Group A:** Occlusal splint (n = 5) only at night for 2 months with adjustments weekly. **Group B:** No treatment (n = 4).	No statistically significant difference between the groups (RR 0.20 (95% CI 0.03 to1.15)) was observed regarding increase in the size of wear facets outcome. The same results were found after the follow up of 6 months
	Landry et al. (2006)[54]	RCT (cross-over)	13	24.0 (NR)	2 weeks	History of tooth grinding for at least 3 nights per week and polygraphic	**Group A:** Occlusal splint (n = 13). **Group B:** Mandibular advancement	The participants preference resulted in a higher proportion of

		confirmation of a minimum of 4 episodes of sleep bruxism per hour of sleep and a minimum of 2 episodes with tooth grinding sound		device in 25% advancement position (n = 13). **Group C:** Mandibular advancement device in 75% advancement position (n = 13). **Group D:** Mandibular advancement device free (n = 13), 2 weeks of treatment duration.	benefitted participants in the occlusal splint group (12/13) as compared to the proportion in the other groups (1/13). Results were sent by the authors: sleep bruxism episodes per hour for the MAD max (mean difference = 5.9; standard deviation (SD) = 1.68; P < 0.001

								paired t-test); pain during the night for the MAD max and MAD min (8/13); oral dryness for MAD min (7/13); comfort (median - VAS 100 mm) occlusal splint = 79 mm, MAD free = 41 mm, MAD min = 15 mm, MAD max = 12 mm.
	Van der Zaag et al. (2005)[5] [5]	RCT	21	34.8 (12.2) 18-68	4 weeks	Tooth grinding sounds during sleep for at least	**Group A:** Occlusal splint (n = 11) with 4 men and 7	Number of bruxism episodes per hour of sleep

		3 nights per week during the last 6 months reported by partner, tooth wear to at least the degree of exposed dentine (grade 2)		women with mean age of 34.2 years (SD = ± 13.1; range = 21-68 years). **Group B:** Palatal splint (n = 10) with 1 men and 9 women with mean age of 34.9 years (SD = ± 11.2; range = 18-55 years). They wore their splint 24 hours a day, except for eating for 4 weeks.	(Epi/h) (Outcome 01) resulted in no statistically significant differences between the groups, as expressed by its confidence interval and significance test (WMD 0.54 (95% CI -10.95 to 12.93)). No statistically significant differences between groups were found in regards to the total sleep time (Outcome

										1.3) (WMD -8.60 (95% CI 96.17 to 78.97)).
Macedo et al. (2014);[56] Cochrane Database of Systematic Reviews	To evaluate the effectiveness and safety of pharmacological therapy for the treatment of sleep bruxism compared with other drugs, no treatment or placebo.	Etzel et al. (1991)[44]	RCT (cross-over)	8	36.6 (NR) 22-47	8 days	History of nocturnal bruxism characterized by facial pain or restricted mandibular motion on awakening (or both), excessive tooth wear, grinding sounds within the past 2 weeks	**Group A:** L-tryptophan 50 mg/kg body weight for 8 days, 8 participants **Group B:** placebo - lactose (colour and size matched) for 8 days, 8 participants Order of medication randomly assigned		There were no statistically significant differences in masseteric EMG between tryptophan and placebo.
		Huynh et al. (2006)[57]	RCT (cross-over)	25	24.4 (NR) 23-31	2 nights	History of tooth grinding for at least 3 nights/week during the	**Group A:** propranolol 120 mg (participants received an oral dose of		No statistically significant difference was found for the sleep

		last 6 months associated hypertrophy of masseter muscles or presence of tooth wear (or both)	long-acting drug at 7:00 PM), 10 participants **Group B:** placebo, 10 participants **Group C:** clonidine 0.3 mg (participants received an oral dose, 1 hour before bedtime), 16 participants **Group D:** placebo, 16 participants Comparing: group A versus group B and group C versus group D Regimen: each		bruxism index when comparing propranolol with placebo and clonidine with placebo.

							participant spent at least 4 nights at the sleep laboratory (night 1 for habituation, night 2 for sleep diagnosis, nights 3 and 4 for interventions). 1 person participated in both interventions , with an interval period of 6 months	
	Lavigne et al. (2001)[58]	RCT (cross-over)	7	28.4 (6.1) 23-39	2 weeks	History of tooth grinding for at least 5 nights/week and polygraphic	**Group A:** bromocriptine - start dose 1.25 mg to 7.5 mg (6 days to increase up	There were no statistically significant differences in the number of

		confirmation of a minimum of 4 episodes of sleep bruxism per hour of sleep and a minimum of 4 episodes with tooth grinding sound	the dose) and 7.5 mg maintained for the next 8 days, 7 participants **Group B:** placebo (capsules had the same colour as bromocriptine), 2 weeks, 7 participants Domperidone administrated 30 minutes before bromocriptine or placebo to reduce adverse effects Regimen: each participant spent 4		bruxism episodes per hour, bruxism bursts per hour, bruxism bursts per episode or the number of episodes with grinding noise in participants taking bromocriptine versus placebo.

							nights at the sleep laboratory for habituation, sleep diagnosis and interventions	
	Lobbezoo et al. (1997)[59]	RCT (cross-over)	10	27.5 (5.4) 19-36	2 nights	History of tooth grinding sounds during sleep for at least 5 nights/ week during the last 6 months reported by partner; tooth wear with a minimum score of 1	**Group A:** levodopa 100 mg plus benserazide 25 mg to reduce adverse effects, 10 participants **Group B:** placebo. 2 oral doses: the first dose 1 hour before bedtime and the second dose 4 hours after the first	No statistically significant differences were reported for sleep bruxism variables (bruxism episodes per hour of sleep and bruxism bursts per episode).

							dose, 10 participants Regimen: each participant spent 3 nights at the sleep laboratory for habituation, sleep diagnosis and interventions	
	Mohamed et al. (1997)[4][5]	RCT (cross-over)	10	35 (12)	1 week	History of tooth grinding or clenching (or both), presence of some occlusal tooth wear	**Group A:** amitriptyline 25 mg/night for 1 week, 10 participants **Group B:** placebo 25 mg/night for 1 week, 10 participants	The individual analysis of the studies, as well as the meta-analysis, found no statistically significant differences between the placebo and

								the medication groups.
	Raigrodski et al. (2001a) [46]	RCT (cross-over)	10	39 (NR) 31-54	4 weeks	History of tooth grinding or clenching (or both), presence of some occlusal tooth wear	**Group A:** amitriptyline 25 mg/night during 4 weeks, 10 participants **Group B:** placebo 25 mg/night during 4 weeks, 10 participants	The individual analysis of the studies, as well as the meta-analysis, found no statistically significant differences between the placebo and the medication groups.
	Raigrodski et al. (2001b) [47]	RCT (cross-over)	10	> 18 years	4 weeks	History of tooth grinding or clenching (or both), presence of temporomandibular disorder	**Group A:** amitriptyline 25 mg/night for 4 weeks, 10 participants **Group B**: placebo 25 mg/night for	The individual analysis of the studies, as well as the meta-analysis, found no statistically

							symptoms, such as facial pain or headaches from the jaw muscles or temporomandibular joint (or both)	4 weeks, 10 participants	significant differences between the placebo and the medication groups.
Machado et al. (2011);[60] Dental Press Journal of Orthodontics	The objective of this systematic literature review is to discuss, based on scientific evidence, treatment alternatives for the	Dube et al. (2004)[52]	RCT (cross-over)	9	NR	4 weeks	NR	Efficacy and safety of an occlusal splint and a palatal splint in the reduction of the muscle activity and teeth clenching	There was a statistically significant reduction in the number of episodes of SB with the use of both treatments, with no differences due to the design of the devices.

	control and management of SB	Van der Zaag et al. (2005)[55]	RCT	21	NR	4 weeks	Polysomnographic evaluations were performed, one conducted before the beginning of therapy and another after a treatment period of four weeks	Effects of occlusal and palatal splints in the management of SB	Study results showed that neither the occlusal splint, nor the palatal splint had an influence on the SB or in relation to patient sleep.
		Harada et al. (2006)[61]	RCT (cross-over)	16	NR	6 weeks	Muscle activity was evaluated by anm electromyographic portable device	Effects of a stabilization splint and a palatal splint in the management of SB	The results of this study showed that both the occlusal splint and the palatal splint reduced the masseter muscle activity during the

								night immediately after appliance installation. However, no effects were observed after 2, 4 and 6 weeks of use, and no differences were noted due to the splints designs.
	Landry et al. (2006)[54]	RCT (cross-over)	13	NR	NR	Polysomnographic examination, with diagnosis of SB	1. Mandibular advancement device 2. Traditional occlusal splint	The authors concluded that short-term temporary use of the mandibular advancement device is associated

								with a notable reduction in motor activity of SB, and to a lesser order the occlusal splint also found a reduction of SB. However, the use of mandibular advancement device in eight patients caused adverse effects, such as pain and discomfort.
	Etzel et al. (1991)[44]	RCT	8	NR	8 days each	Portable electromyography device		Tryptophan (50 mg/kg) or placebo	The study results showed no significant

								differences between therapies, suggesting that supplementation with L-tryptophan is ineffective in the treatment of SB.
	Mohamed et al. (1997)[45]	RCT	10	NR	1 week each	NR	25 mg of amitriptyline and 25 mg of placebo for one week each	The results showed that neither the intensity nor location of pain, and electromyographic activity of the masseter muscle were significantly affected by the tricyclic

								antidepressant therapy
	Raigrodski et al. (2001a)[46]	RCT	10	NR	4 weeks each	Portable electromyography device	Amitriptyline (25 mg/night) and inactive placebo (25 mg/night)	The results showed that administration of amitriptyline did not significantly decrease the activity of the masseter muscle, neither significantly increase sleep duration
	Lobbezoo et al. (1997)[59]	RCT (cross-over)	10	NR	3 nights	Evaluated in a sleep laboratory	Two doses (100 mg) of L-dopa or placebo	It was found that the use of L-dopa resulted in a decrease in the average number of bruxism episodes per hour of

								sleep, but this reduction proved to be modest, being only of the order of 26%.
	Lavigne et al. (2001)[58]	RCT (cross-over)	7	NR	2 weeks each 1 week washout	Polysomnography	1. The doses of bromocriptine ranged from 1.25 mg to 7.5 mg (six days) up to 7.5 mg dose (8 days). 2. Placebo	Examining the results, bromocriptine did not reduce the frequency of episodes of bruxism during the night or the amplitude of contractions of the masseter muscle.
	Huynh et al. (2006)[57]	RCT (cross-over)	25	NR	NR	History and diagnosis of SB (unspecifie	1. Clonidine (0.3mg)	The results showed that propranolol (n = 10) did

						d) Polysomnigraphic examination	2. Propranolol (120mg) 3. Placebo	not affect the SB, whereas clonidine (n = 16) decreased sympathetic tone in the minute preceding the onset of SB, reducing the SB by preventing activation of the sequence of autonomic and motor events characteristics of the same.
	Ommerborn et al.	RCT	57	NR	12 weeks treatment	SB (unspecified criteria)	1. Occlusal splint	The findings showed a significant

		(2007)[17]				6 months follow-up		2. Cognitive behavioral therapy	reduction in activity of the SB in the two groups, but the effects were small. Moreover, the cognitive behavioral therapy group had a tendency to return to baseline of the study when compared to occlusal splint.
Manfredini et al. (2015);[62] Journal of Oral Rehabilitation	The review focuses on the most recent	Valiente et al. (2015)[63]	RCT	16	39.9 (10.8) 24-62	4 week	Recent history of TG sounds for at least three nights per week during the	1**. Test group (4M,4F):** sleep hygiene instructions and	For both the control group and the experimental group, no significant

	literature on management of sleep bruxism (SB) in adults					last 6 months and grade 2 tooth wear	Jacobson's relaxation techniques (20-min CD recorded by a psychologist) 4-week protocol PSG 2. **Control group (4M,4F):** information on the condition of SB	differences could be observed between the PSG-SB outcome measures obtained before and after the 4-week period.
	Matsumoto et al. (2015)[9]	RCT	20	28.9 (NR) 24-38	29 nights	Clinical/ anamnestic American Academy of Sleep Medicine (AASM) criteria	1. **Test group (C):** continuous use of SA covering the occlusal surfaces of the maxillary dental arch during sleep 29-night protocol EMG	The intermittent use of stabilisation splints may reduce SB activity for a longer period compared with that of continuous use.

							activity of the masseter muscle on one side (portable EMG recording unit) 2. **Control group (I):** intermittent use of SA (every other week, that is, at the 1st to 7th, 15th to 21st and 29th nights)	
	Sato et al. (2015)[27]	RCT	13	26.8 (2.5) 22-31	3 weeks	Subjective awareness of awake bruxism	1. **Test group (BF, n = 7):** auditory biofeedback (BF) alert signals to remind the subjects of clenching	The number of tonic EMG events during sleep in the BF group significantly decreased in weeks 2 and 3, whereas

							were generated during the daytime 3-week protocol One-channel portable EMG-BF device (2-day, 5-h EMG recording periods during the daytime and sleeptime) 2. **Control group (n = 6):** only EMG recordings	that in the conytol group did not show any significant change throughout the recording period EMG-BF to improve AB tonic EMG events can also provide an effective approach to the regulation of SB tonic EMG events.
	Shim et al. (2014)[2]	RCT	24	20.2-38.7	4 weeks	Clinical diagnosis of SB	**1. Group A:** 10 subjects receiving bilateral BTX-A	BTX-A injection did not reduce the frequency,

							injections (25 U per muscle) into the masseter muscles only PSG **2. Group B:** 10 subjects receiving the injections into both the masseter and temporalis muscles	number of bursts, or duration for RMMA episodes in the two groups. The injection decreased the peak amplitude of EMG burst of RMMA episodes in the injected muscles (P < 0.001, repeated measure ANOVA) in both groups.
	Madani et al. (2013)[14]	RCT	24	28.3 (7.1) 18-50	2 months	Complaint of SB (ICSD criteria)	**1. Group A:** Jard SS covering the maxillary dental arch	Significant reduction in most SB variables in both groups

						2-month protocol PSG **2. Group B:** Gabapentin – 1 capsule (100 mg) orally at bedtime for the first 3 nights, then 200 mg/night for the next 3 nights, thereafter 300 mg/night continued for 2 months	after treatment.
Takahashi et al. (2013)[64]	RCT	23	22.2 (NR)	3 days	NR (healthy volunteers)	**1. Test group:** SS covering the occlusal surfaces of the maxillary dental arch Crossover design with two weeks	The number of MMA events per hour decreases significantly with SS.

							washout between phases One-channel EMG **2. Control group:** PS not covering the maxillary teeth	
	Arima et al. (2012)[15]	RCT	11	M 25.3 (3.2) F 25.9 (3.1)	30 nights	Self-reported SB	**1. Test group:** restrict-MMOA that prevented from performing mandibular movements 30-night protocol Crossover design with one of the three types of appliances (1 week each)	The total number of phasic EMG episodes and bursts per hour of sleep is significantly reduced during any of the three combinations of oral appliances when compared with

							Bilateral masseter home-EMG **2. Control group**: free-MMOA that allowed normal mandibular movements; or free-MOA Bilateral masseter-EMG	baseline values. The restriction of mandibular movements with oral appliances does not have any major influence on jaw-muscle activity during sleep.
Carra et al. (2010)[6][5]	RCT	16	24.5 (NR) 21.31	4 nights	PSG		1. Test group: single dose of clonidine (0.3 mg by mouth) 1 h before bedtime 4-night protocol	RMMA/SB decreases under clonidine.

							PSG Crossover design 2. Control group: single dose of placebo	
	Lee et al. (2010)[4]	RCT	12	M 25 (2.3) F 24 (0.8)	12 weeks	Nocturnal bruxism (unspecified criteria)	**1. Test group:** BTX-A into each subject's masseter muscles at three sites – 80U of BTX-A 12-week observation EMG of both masseter and temporalis muscles for three consecutive nights at home for an average of 6 hrs per night	The injection of botulinum toxin in the masseter muscle reduces the number of bruxism events during sleep for up to 12 weeks.

							2. Control group: Saline injection into each subjects' masseter muscles at three sites – 0.8 ml of saline	
	Saletu et al. (2010)[66]	RCT	21	45.1 (12.6)	3 nights	SB (ICSD criteria)	**1. Test group:** crossover study, with three consecutive (pre-drug night, placebo night and clonazepam - 1 mg night) PSG **2. Control group:** 21 sex and agematched	The bruxism index is significantly improved under 1 mg clonazepam (41% improvement with respect to placebo on individual change values).

						subjects without SB Non-randomised study	
Landry-Schonbeck et al. (2009)[16]	RCT	12	25 (1.5)	5 nights	Moderate to severe SB (unspecified)	**1. Test group:** MAA (25% or 75% advancement) 5-night crossover PSG **2. Control group:** MOS	MAA are more effective than MOS to reduce SB The short-term use of a robust MAA (75%) is associated with SB decrease.
Abekura et al. (2008)[67]	RCT	12	25.3 (NR)	2 nights with 5 nights washout between phases	NR (healthy volunteers)	**1. Test group:** Occlusal splints at 3 mm VDO increase worn for two nights	Splint with 3 mm increase in VDO is superior to 6 mm-splint in decreasing bruxism.

						One-sided masseter and temporalis muscle EMG **2. Comparison group:** Occlusal splints at 6 mm VDO increase worn for two nights	
	Mainieri et al. (2014)[19]	Before-after study	19	39.9 (12.9)	3 months	Clinical SB (unspecified)	MAD for 3 months; 50–75% advancement	33.7% reduction in EMG episodes per hour.
	Sumiya et al. (2014)[29]	Before-after study	10	26.7 (3.5)	NR	SB awareness	BF (masseter EMG stimulation after heart rate increase)	Electrical stimulation can reduce the number of SB events.

Manfredini et al. (2017);[68] Journal of Prosthetic Dentistry	The purpose of this systematic review was to evaluate the relationship between prosthetic rehabilitation and TMDs and bruxism	No included study	Not applicable	Not applicable	Not applicable	Not applicable	Not applicable	Prosthodontic treatment	This systematic review of publications revealed an absence of RCTs on the various topics concerning the relationship between TMD and bruxism and prosthodontics. Based on the best available evidence, prosthetic changes in dental occlusion are not yet acceptable as strategies for solving

									TMD symptoms or helping an individual stop bruxism.
Martin et al. (2012);[69] Interational Journal of Oral and Maxillofacial Surgery **	The present review was designed to investigate the evidence of the use of antidepressants in orofacial pain disorders.	Raigrodski et al. (2001b) [47]	RCT (cross-over)	10	NR	2x4 weeks	NR	1. Amitriptyline (25mg a day) 2. Placebo	No significant improvement in pain intensity reduction between the 2 groups. Positive significant in stress level.

	Which treatment modalities are effective for specific orofacial pain disorders or for orofacial pain in general.								
Restrepo et al. (2009);[70] Quintessence International	To conduct a systematic review to assess and analyze the scientific evidence about the available therapies for	Restrepo et al. (2001)[71]	Quasi-experimental	NR	3-6	NR	Bruxism was determined by indirect measurements	Efficiency of psychologic techniques to reduce the symptoms of bruxism in children	There is evidence for the positive effect of a combined technique of induced muscular relaxation and competence reaction in 3- to 6-year-old children

	bruxism in children.								with bruxism.
		DiFrancesco et al. (2004)[72]	RCT	69	2-12	NR	The classification of the children as bruxist was performed by a phonoaudiologist, whose training to do so was not described in the article	Efficacy of the adenotonsillectomy to reduce the signs and symptoms of bruxism was evaluated	A significant proportion of parents ceased to report bruxism after adenotonsillectomy.
Stapelmann et al. (2008);[73] BMC Oral Health	The aim of this systematic review was to appraise	Baad-Hansen et al. (2007)[74]	RCT (cross-over)	10	23-39	7-8 weeks	1. Self-reported toothgrinding during sleep, confirmed	NTI-TSS device (n = 10) vs. flat occlusal stabilization splint (OS)	A strong and lasting inhibition of EMG activity in masseter

**	the currently available evidence regarding the efficacy and safety of the NTI-tss splint.	by bedpartner 2. Reports of muscle soreness on awakening. 3. Signs of tooth wear.	(n = 10) worn at night	muscles during sleep was caused by wearing the NTI splint but not the OS. However, this was not directly related to the short-term clinical outcome measures.

	Kavakli et al. (2006)[75]	RCT	30	31.0 (NR) 14-52	4 months	1. Self-reported tooth clenching and tooth grinding for at least 6 months 2. Grinding sounds during sleep for at least 3 nights per week as confirmed by bedpartner 3. Jaw muscle discomfort 4. Abnormal tooth wear 5. Masseter hypertrophy 6. Diagnosis of sleep	NTI-tss device (n = 11) vs. Michigan-type stabilization splint (SS) (n = 9) worn at night	1. Both splint designs do not stop sleep bruxism activity as shown by polysomnographic evaluation. 2. The SS does not reduce the frequency, duration or intensity of the sleep bruxism. 3. The NTI-tss device reduces the intensity of bruxism. 4. Due to its positive effect on sleep

							bruxism in a sleep laboratory		bruxism and its easy adapatability, the NTI-tss device is recommended if regular check-ups by a dentist are possible.
Wang et al. (2014);[76] Sleep and Breathing	The aim of this systematic review was to evaluate the efficacy	Kardachi et al. (1978)[77]	RCT	I=4/4 C=4/4/4	18-39	1 week	A portable EMG device	Occlusal adjustment/audio feedback/mock occlusal adjustment/control	A reduction of approximately 70% was reported in all the subjects in the

	of any biofeedback treatment on sleep bruxism.						feedback/non bruxer	biofeedback group. Between-groups comparison was lacking.
		Casas et al. (1982)[78]	RCT	I=4/4/4 C=4	29	2 weeks	A portable EMG device	Stress-reduction behavioral counseling/audio feedback/stress-reduction behavioral counseling+audio feedback/blank control	Audio feedback was superior to the blank control, but the difference between audio feedback and stress reduction behavioral counseling was not significant.
		Pierce et al. (1988)[79]	RCT	I=20/20/20/20 C=20	38	2 weeks	An EMG unit	Diurnal biofeedback (relaxation)/nocturnal audio	The EMG-measured SB episode decreased significantly

							feedback/massed negative practice/splint/blank control	in the nocturnal biofeedback and splint groups, while there was no significance in other groups.
	Wiesel mann-Penkner et al. (2001)[80]	RCT	20	22-58	3 weeks	A computer aided biofeedback system	TENS/EMG biofeedback (visual)	Tendencies of decreased mean-EMG levels for both groups after the treatment sessions and higher EMG values in the TENS group than in the biofeedback group.
	Ommer born et al.	RCT	57	29	2 weeks	Bruxcore bruxism-monitoring	OS/CBT (partial audio feedback)	NR

	(2007)[17]					device (similar to bite plate)		
	Jadidi et al. (2008)[81]	RCT	28	24-60	6 weeks	A portable EMG device	CES/blank control	A significant difference in SB episode was displayed between CES and blank control (mean difference = −9.7, 95%CI = −18.94 to -0.46).
	Jadidi et al. (2011)[82]	RCT	28	32	1 night	A portable EMG device	CES/blank control	No significant difference in SB episode was displayed between CES and

							blank control (mean difference = −1.4, 95%CI = −5.49 to +2.69).

Legend: AASM: American Academy of Sleep Medicine; AB: Awake Bruxism; ANOVA: Analysis of Variance; AV: Audio-Video; BF: Biofeedback; BID: *Bis In Die* (twice a day); BoNT-A: Type-A Botulinum Toxin; BTX-A: Type-A Botulinum Toxin; C: Control; CBT: Cognitive Behaviour Therapy; CES: Contingent Electrical Stimulation; CI: Confidence Interval; EMG: Electromyography; F: Female; I; Intervention; M: Male; MAA: Mandibular Advancement Appliance; MAD: Mandibular Advancement Device; MMA: Masticatory Muscle Activity; MMOA: Maxillary and Mandibular Oral Appliance; MOA: Maxillary Oral Appliance; MOS: Mandibular Occlusal Splint; NA: Not Available; NR: Not Reported; NTI: Nociceptive Trigeminal Inhibition; NIT-TSS: Nociceptive Trigeminal Inhibition Tension Supression System; OR: Odds Ratio; OS: Occlusal Splint; PSG: Polysomnography; PSQI: Pittsburgh Sleep Quality Index; RCT: Randomized Controlled Trial; RMMA: Rhythmic Masticatory Muscle Activity; RR: Relative Risk; SB: Sleep Bruxism; SD: Standard Deviation; SS: Stabilization Splint; TENS: Transcutaneous Electric Nerve Stimulation; TMD: Temporomandibular Disorders; TMJ: Temporomandibular Joint; U: Unit; VAS: Visual Analogue Scale; VDO: Vertical Dimension of Occlusion; WMD: Weighted Mean Differences; (**) Data were colleted only from bruxism-related primary studies.

Supplementary table 5 - references

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ANEXOS

Anexo A - Artigos publicados durante o mestrado e incluídos na dissertação

THE JOURNAL OF PROSTHETIC DENTISTRY

SYSTEMATIC REVIEW

Association of sleep bruxism with ceramic restoration failure: A systematic review and meta-analysis

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ABSTRACT

Statement of problem. Ceramic restorations are popular because of their excellent optical properties. However, failures are still a major concern, and dentists are confronted with the following question: is sleep bruxism (SB) associated with an increased frequency of ceramic restoration failures?

Purpose. The purpose of this systematic review and meta-analysis was to assess whether the presence of SB is associated with increased ceramic restoration failure.

Material and methods. Observational studies and clinical trials that evaluated the short- and long-term survival rate of ceramic restorations in SB participants were selected. Sleep bruxism diagnostic criteria must have included at least 1 of the following: questionnaire, clinical evaluation, or polysomnography. Seven databases, in addition to 3 nonpeer-reviewed literature databases, were searched. The risk of bias was assessed by using the meta-analysis of statistics assessment and review instrument (MAStARI) checklist.

Results. Eight studies were included for qualitative synthesis, but only 5 for the meta-analysis. Three studies were categorized as moderate risk and 5 as high risk of bias. Clinical and methodological heterogeneity across studies were considered high. Increased hazard ratio (HR=7.74; 95% confidence interval [CI]=2.50 to 23.95) and odds ratio (OR=2.52; 95% CI=1.24 to 5.12) were observed considering only anterior ceramic veneers. Nevertheless, limited data from the meta-analysis and from the restricted number of included studies suggested that differences in the overall odds of failure concerning SB and other types of ceramic restorations did not favor or disfavor any association (OR=1.10; 95% CI=0.43 to 2.8). The overall quality of evidence was considered very low according to the GRADE criteria.

Conclusions. Within the limitations of this systematic review, the overall result from the meta-analysis did not favor any association between SB and increased odds of failure for ceramic restorations. (J Prosthet Dent 2018;119:354-62)

The increasing demand for esthetic procedures has led to the development of esthetic restorative materials that can withstand occlusal forces. Dental ceramics composed predominantly of glass particles lack adequate fracture resistance for posterior applications, unless combined with metal frameworks.[1] However, metal exposure in the cervical area can affect the esthetics of the prosthesis. Therefore, high-strength crystalline ceramic restorations have become popular.[2,3]

Initially, high-strength ceramics were veneered with feldspathic porcelain[4]; however, chipping of the veneering porcelain has been reported.[5-9] Subsequently, restorations fabricated from a single reinforced ceramic block have gained popularity. These may have

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REVIEW

WILEY Journal of Oral Rehabilitation

Association between psychotropic medications and presence of sleep bruxism: A systematic review

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Summary

The purpose of this study was to systematically review the literature for studies that investigated the association between use of psychotropic medications and presence of sleep bruxism (SB). Observational studies were selected in a two-phase process. Searches were performed on six electronic databases, and a grey literature search was conducted on three databases. SB diagnosis was based on questionnaires or clinical examinations; no polysomnography examinations were performed. Risk of bias was assessed using the Joanna Briggs Institute Critical Appraisal Checklist for Analytical Cross-Sectional Studies. Overall quality of evidence was evaluated according to the Grading of Recommendations Assessment, Development and Evaluation criteria. Five analytical cross-sectional studies were included, evaluating antidepressants, anticonvulsants and psychostimulants. One study was judged as low risk of bias, three as moderate risk and one high risk. Antidepressants were evaluated in adult populations only; duloxetine (Odds Ratio [OR] = 2.16; 95% Confidence Interval [95% CI] = 1.12-4.17), paroxetine (OR = 3.63; 95% CI = 2.15-6.13) and venlafaxine (OR = 2.28; 95% CI = 1.34-3.86) were positively associated with SB risk. No increased odds of SB were observed considering use of citalopram, escitalopram, fluoxetine, mirtazapine and sertraline. With regard to anticonvulsants, only barbiturates were associated with SB in children (OR = 14.70; 95% CI = 1.85-116.90), while no increased odds were observed for benzodiazepine, carbamazepine and valproate. The only psychostimulant evaluated was methylphenidate, and an association with SB was observed in adolescents (OR = 1.67; 95% CI = 1.03-2.68). Findings from this SR suggested that medications such as duloxetine, paroxetine, venlafaxine, barbiturates and methylphenidate might be associated with SB; however, overall quality of evidence was considered very low, and therefore, caution is recommended.

KEYWORDS
bruxism, mental disorders, neuropharmacology, psychotropic drugs, sleep bruxism, systematic review

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Anexo B - Artigos publicados durante o mestrado e não-incluídos na dissertação

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REVIEW

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Effects of glucosamine supplements on painful temporomandibular joint osteoarthritis: A systematic review

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Summary

The purpose of this study was to systematically review the literature for studies that assessed the effects of glucosamine supplements (GS) on pain and maximum mouth opening (MMO) restriction compared to other therapies, placebo or no intervention on painful temporomandibular joint osteoarthritis (TMJ OA). Randomised controlled trials were selected in a two-phase process. Seven electronic databases, in addition to three grey literature databases, were searched. Risk of bias was assessed using the Cochrane Collaboration's tool for assessing risk of bias in randomised trials. Twelve potentially eligible studies were identified, from which three were finally included. Furthermore, two were categorised at low risk and one at high risk of bias. Intervention groups were treated with glucosamine-sulphate, while controls were treated with placebo or ibuprofen. In two studies, GS were equally effective regarding pain reduction and mouth opening improvement compared to ibuprofen taken two or three times a day over 12 weeks; however, one study did not find significant differences in follow-up evaluations concerning these clinical variables in both glucosamine and placebo groups administered over six weeks. There is very low evidence regarding GS therapeutic effects on TMJ OA. Considering a follow-up of 12 weeks, GS were as effective as ibuprofen taken two or three times a day. However, over six weeks of medication intake, GS were not superior to placebo. Still, included studies presented major drawbacks, and therefore, conclusions must be interpreted with caution.

KEYWORDS
evidence-based dentistry, glucosamine, osteoarthritis, systematic review, temporomandibular joint, temporomandibular joint disorders

1 | INTRODUCTION

Glucosamine is a common metabolic product, acting as a preferred substrate for the biosynthesis of glycosaminoglycan chains, and therefore playing an important role in the metabolism of joint cartilages.[1] Because of its high concentration in joint tissues, it has been hypothesised that glucosamine supplements (GS) could provide symptomatic relief to articular diseases and associated conditions.[2] Moreover, previous studies have investigated its therapeutic properties on degenerative joints diseases, especially on the knee, with minimal or no reported side effects.[2,3]

Temporomandibular disorders could be described as a cluster of conditions affecting the temporomandibular joint (TMJ), masticatory muscles and their surrounding tissues.[4] Classically reported signs and symptoms include the following: pain in TMJ and/or masticatory muscles; articular sounds, such as clicking or crepitation; and functional limitations, including restriction, deviation or deflection of mouth opening movement.[5,6] Regarding joint-related

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Research Article

Intraoral Potentially Malignant Disorders in a Brazilian Oral Pathology Service: Epidemiological, Clinical, and Histopathological Findings

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The aim of this study was to investigate the characteristics of individuals with intraoral potentially malignant disorders (IOPMD) in an oral pathology service in Brazil. Cases were screened based on clinical diagnosis of leukoplakia (LKP), erythroleukoplakia (ELKP), and erythroplakia (EP). Clinical data and information regarding associated factors were gathered from biopsy reports. Histological diagnoses were collected from histopathological records. Among 208 IOPMD cases, 84.13% involved LKP; 11.1%, ELKP; and 4.8%, EP. The most affected sites were the gingiva and buccal mucosa. Histologically, epithelial dysplasia was present in 66.8% of the lesions, acanthosis and hyperkeratosis without epithelial dysplasia were present in 27.9%, and squamous cell carcinoma was present in 2.9%. Most patients were males, fair-skinned, with mean age of 53.4 years. Chronic smokers represented 73% of subjects, of which 30% also consumed alcohol. Smokers and drinkers were mostly males ($p < 0.001$). EP and ELKP represented histologically more severe degrees of epithelial dysplasia than LKP ($p < 0.001$). In conclusion, individuals with IOPMD were more frequently fair-skinned men in the sixth decade of life, with smoking habit. Special attention is required to clinical diagnoses of ELKP and EP since the prevalence of severe epithelial dysplasia, in situ carcinoma, and squamous cell carcinoma is higher than in LKP.

1. Introduction

The most frequent malignant neoplasm in the oral cavity is the oral squamous cell carcinoma (OSCC), a multifactorial disease in which smoked and/or smokeless tobacco is the main associated etiological factor [1, 2]. OSCC etiology varies worldwide; in Asian populations, the use of smokeless tobacco is highly associated with the development of OSCC [2]. On the other hand, in Brazil, the use of smokeless tobacco is rare and, therefore, the main etiological factor associated with OSCC development is consumption of the smoked form of tobacco [3, 4].

Clinically, intraoral potentially malignant disorders (IOPMD), such as leukoplakia (LKP), erythroplakia (EP), or mixed red and white lesions (erythroleukoplakia (ELKP) or speckled LKP), may precede the OSCC [5]. The diagnosis of IOPMD is based on clinical and histopathological characteristics. The clinical characteristics of LKP in particular may be misleading; therefore, clinicians must be able to rule out other oral white patches [6]. Histologically, these lesions can present some kind of epithelial alterations, such as epithelial dysplasia, hyperplasia, or in situ carcinoma (ISC); thus, biopsy and histopathological evaluation should be considered [7, 8]. More severe degrees of epithelial dysplasia, in which the epithelium is not organized in layers and presents with intense cellular atypia, are usually observed in red lesions, such as ELKP and EP, and in comparison with LKP, these lesions are most likely to be histologically diagnosed as in situ or invasive carcinomas [9].

In western countries, patients with IOPMD are usually fair-skinned males, around the fifth and sixth decades of life, with a history of chronic consumption of cigarettes and/or

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Review

Maté consumption association with upper aerodigestive tract cancers: A systematic review and meta-analysis

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ABSTRACT

Maté is a beverage regularly consumed by Latin American populations. Upper aerodigestive tract (UADT) cancers are frequent in this region and are suspected to be associated with maté consumption. The aim of this systematic review and meta-analysis was to answer a focused question: "Is there an association between maté consumption and occurrence of the UADT cancer?". Studies investigating any association between maté consumption and occurrence of UADT cancer were included. Out of the 569 studies, 18 met the inclusion criteria for qualitative and 15 for quantitative analysis. An increased odds was observed regarding maté consumption and overall occurrence of UADT cancer (OR = 2.24; 95%CI = 1.74–2.87). Consistent evidence of a positive association was found for all UADT subsites, oral, pharynx, esophagus and larynx. No differences in effect were found between consumption of cold/warm and hot/very hot mate (OR = 1.08; 95%CI = 0.83–1.41). Consumption of more than one liter of maté per day was associated with increased odds of having UADT cancer compared to an intake of less than one liter per day (OR = 1.72; 95%CI = 1.47–2.01). According to published data, regardless of the temperature, maté consumption significantly increased the odds of occurrence of UADT cancer.

Introduction

Malignancies of the upper aerodigestive tract (UADT) are one of the most common worldwide [1,2]. UADT malignancies include oral, pharyngeal, laryngeal, and esophageal cancers. Together, UADT account for more than one million new cases of cancer and more than eight hundred thousand deaths globally every year [3]. These malignancies are multifactorial diseases and several factors are associated with their development, such as excess of alcohol consumption and tobacco use, viral infections, and some occupational exposures [4–8]. A meta-analysis (MA) of observational studies reposted that the odds of having esophageal cancer were significantly increased by consumption of hot beverages and foods (Odds Ratio [OR] = 1.82; 95% confidence interval [CI] = 1.53–2.17), especially in Asian (OR = 2.06; 95% CI = 1.62–2.61) and South American populations (OR = 1.52; 95% CI = 1.25–1.85) [9].

South American and Caribbean populations present a high prevalence of UADT cancer, especially among males [10]. This might be explained by certain lifestyle factors, such as daily consumption of maté [11,12]. Maté is a tea-like infusion of *Ilex paraguariensis,* which is a popular drink in South America, especially Southern Brazil, Chile, Argentina, Uruguay, and Paraguay. Hot maté is consumed in an aqueous infusion by repeatedly adding hot water to approximately 50 g of dried *Ilex paraguariensis* leaves. Maté has gained recognition for its stimulant and antioxidant properties [13–15]. Other properties of maté include diuretic, central nervous system stimulant, hypocholesterolemic and hepatoprotective [16,17]. On the other hand, maté consumption is also suspected to be associated with an increased risk for developing several types of malignancies, including UADT cancer [18–20].

A previous systematic review (SR) and MA published in 2010 investigated the association between maté consumption and oral/oropharyngeal cancer in humans. The authors reported a weak association and remarked a need for more studies before a definite conclusion can be drawn [21]. Another MA published in 2013 assessed the association between maté consumption and occurrence of esophageal squamous cell carcinoma (ESCC); this study found significantly increased odds of ESCC occurrence (OR = 2.57; 95% CI = 1.66–3.98), especially comparing high vs. low consumption of maté (OR = 2.76;

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