Bruxism: An umbrella review of systematic reviews

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Abstract

Objectives: To synthesise available knowledge about both sleep (SB) and awake bruxism (AB) as depicted by previous published systematic reviews (SR).

Methods: SR investigating any bruxism-related outcome were selected in a two-phase process. Searches were performed on seven main electronic databases and a partial grey literature search on three databases. Risk of bias of included SR was assessed using the "University of Bristol's tool for assessing risk of bias in SR".

Results: From 1038 studies, 41 SR were included. Findings from these SR suggested that (a) among adults, prevalence of AB was 22%-30%, SB (1%-15%), and SB among children and adolescents (3%-49%); (b) factors consistently associated with bruxism were use of alcohol, caffeine, tobacco, some psychotropic medications, oesophageal acidification and second-hand smoke; temporomandibular disorder signs and symptoms presented a plausible association; (c) portable diagnostic devices showed overall higher values of specificity (0.83-1.00) and sensitivity (0.40-1.00); (d) bruxism might result in biomechanical complications regarding dental implants; however, evidence was inconclusive regarding other dental restorations and periodontal impact; (e) occlusal appliances were considered effective for bruxism management, although current evidence was considered weak regarding other therapies.

Conclusions: Current knowledge from SR was mostly related to SB. Higher prevalence rates were found in children and adolescents than in adults. Associated factors and bruxism effects on stomatognathic structures were considerably heterogeneous and inconsistent. Overall good accuracy regarding portable diagnostic devices was found. Interventions' effectiveness was mostly inconclusive regarding the majority of available therapies, with the exception of occlusal appliances.

Keywords
awake bruxism, bruxism, evidence-based dentistry, sleep bruxism, systematic review, umbrella review
1 | INTRODUCTION

According to an updated international consensus, bruxism is a masticatory muscle activity that may occur during sleep (characterised as rhythmic or non-rhythmic) and/or wakefulness (characterised as repetitive or sustained tooth contact and/or by bracing or thrusting of the mandible). In otherwise healthy individuals, bruxism should not be considered as a disorder, but rather as a behaviour that can be harmful or protective considering several health outcomes. However, in the presence of other clinical conditions such as sleep apnoea, further assessment is often recommended.¹

Even though high prevalence variability exists likely due to a lack of standardised diagnostic methods, epidemiological studies have shown that prevalence rates among adults may range from 10% to 13% for SB and 22%-31% for AB²; in younger populations, however, bruxism could be more frequent, affecting up to 40%-50% of studies' participants.³,⁴

It has been proposed that bruxism aetiology may be multifactorial and that several underlying mechanisms might play a role in its genesis, such as psychosocial (eg stress and anxiety), physiological (eg genetics) and exogenous factors (eg alcohol consumption, medication use, smoking).⁵,⁶ More importantly, although existing knowledge is still limited, associated factors are thought to be distinct regarding both circadian manifestations of bruxism. Whilst psychosocial aspects appear to have some influence on AB,⁷ autonomic/central nervous system activation might be the primary factors involved in SB genesis.⁸

The presence of bruxism might be identified by instrumental approaches (ie polysomnography or electromyography) or on self-report and/or clinical inspection.¹ In addition, 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.⁹

Both forms of bruxism might be harmful or not to the stomatognathic structures,¹⁰ and some of the most reported harmful effects includes abnormal tooth wear, mobile teeth, and problems with dental restorations, implants or fixed/removable prostheses.¹¹ 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.¹²

Nonetheless, in daily practice, clinicians are required to make decisions on the most suitable approach to manage bruxism, which includes recognising whether or not a treatment is needed.¹³,¹⁴ Therefore, although there is no definitive agreed upon treatment, some therapies might be useful in the management of this condition, including approaches like occlusal appliances, pharmacological treatments, behavioural therapies and other approaches.¹⁵ It must be pointed out that evidence regarding some of these therapeutic methods is often weak and therefore caution in their use is needed.¹⁶

Numerous SR focused on bruxism have been performed, especially during the last decade; however, an overall synthesis and appraisal of these reviews have not yet been performed. Such a summary would be a welcomed addition to the readership as it would synthesise what we know in a document. Therefore, the purpose of this umbrella review was to summarise available evidence and answer the following focused question: “What do we currently know so far about SB and/or AB regarding evidence available from systematic reviews?".

2 | MATERIALS AND METHODS

2.1 | Protocol and registration

A study protocol based on the Preferred Reporting Items for Systematic Reviews and Meta-Analyses Protocols (PRISMA-P)¹⁷ was elaborated and registered at Prospective Register of Systematic Reviews (PROSPERO),¹⁸ being made publicly available under the registration number CRD42018088560. In addition, the reporting of this study was based on the PRISMA checklist.¹⁹

2.2 | Eligibility criteria

SR with or without meta-analyses (MA) that investigated any bruxism-related outcome were considered eligible. Five major subgroups regarding outcomes investigated in SR (prevalence, associated factors, diagnosis, effects on the stomatognathic system and treatment effectiveness) were identified through a preliminary literature screening; however, it should be mentioned that they were used solely as a grouping method, not as eligibility criteria, as other subgroups would have been added if appropriate.

Studies were considered as SR if they matched the following description, as proposed by the Cochrane Collaboration’s Handbook (chapter 1.2.2)²⁰: “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: (a) SR in which outcomes were not directly related to sleep and/or awake bruxism; (b) studies that did not meet the minimum criteria for SR; (c) interventional studies, observational studies, laboratory research, abstracts, case reports, protocols, personal opinions, letters and posters, and (e) full-text not available.

2.3 | 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 partial grey literature search was conducted on Google Scholar, OpenGrey and ProQuest. All electronic database searches were performed from the initial coverage date through 21 May 2018. More information in regard to search strategies is provided in Appendix S1.
Reference lists of included SR were hand-searched to identify additional relevant papers, as proposed by Greenhalgh and Peacock. A computer software was used to manage references (EndNote X7, Thomson Reuters).

2.4 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). Afterwards, 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.

2.5 Data collection process and data items

Three reviewers (G. M.; J. D.; and P. P.) independently collected pertinent data. This information was then cross-checked for accuracy. 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 (GM) collected data regarding included primary studies within SR and information was summarised in five supplementary tables (Tables S1-S5).

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)
### Table 1: Summary of overall descriptive characteristics systematic reviews; prevalence (n = 3) and diagnostic accuracy (n = 2) subgroups

<table>
<thead>
<tr>
<th>Author (Year); Journal</th>
<th>Objectives or research question</th>
<th>Subgroup</th>
<th>Databases searched (Search date)</th>
<th>Included primary studies</th>
<th>Risk of bias assessment tools</th>
<th>Main results</th>
<th>Main conclusion</th>
</tr>
</thead>
<tbody>
<tr>
<td>Machado et al (2014); Dental Press Journal of Orthodontics</td>
<td>Prevalence of sleep bruxism in children</td>
<td>Prevalence rates</td>
<td>MEDLINE, Cochrane, EMBASE, PubMed, LILACS and BBO (from January 2000 to February 2013)</td>
<td>4 cross-sectional</td>
<td>Authors’ judgement (no specific tool)</td>
<td>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.</td>
<td>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 standardised and validated diagnostic criteria are necessary to assess the prevalence of SB in children more accurately.</td>
</tr>
<tr>
<td>Manfredini et al (2013); Journal of Oral Rehabilitation</td>
<td>Prevalence of sleep bruxism in children</td>
<td>Prevalence rates</td>
<td>PubMed, SCOPUS, Google Scholar and four journal Publishers’ website, including Elsevier, Wiley-Blackwell, Quintessence Publishing and Springer (August 2012)</td>
<td>8 cross-sectional</td>
<td>MORE checklist</td>
<td>The reported prevalence was highly variable between the studies (3.5–40.6%), with a commonly described decrease with age and no gender differences.</td>
<td>A very high variability in sleep bruxism prevalence in children was found, due to the different age groups under 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.</td>
</tr>
<tr>
<td>Manfredini et al (2013); Journal of Orofacial Pain</td>
<td>Prevalence of bruxism in adult populations</td>
<td>Prevalence rates</td>
<td>PubMed, SCOPUS and Google Scholar (February 2011)</td>
<td>7 cross-sectional</td>
<td>MORE checklist</td>
<td>Generically identified “bruxism” was assessed in two studies reporting an 8% to 31.4% prevalence, awake bruxism was 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%).</td>
<td>The present systematic review described variable prevalence data for bruxism activities. Findings must be interpreted with caution due to the poor methodological quality of the reviewed literature and to potential diagnostic bias related with having to rely on an individual’s self-report of bruxism</td>
</tr>
<tr>
<td>Casett et al (2017); Journal of Oral Rehabilitation</td>
<td>Which is the validity of questionnaires, clinical assessment and portable diagnostic devices in diagnosing SB, when compared to the reference standard PSG?</td>
<td>Diagnostic accuracy</td>
<td>EMBASE, LILACS, PubMed, ScienceDirect and Web of Science (August 2016)</td>
<td>8 diagnostic accuracy studies</td>
<td>QUADAS-2</td>
<td>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.</td>
<td>Questionnaires and the clinical assessment can be used as screening methods to identify non-SB individuals, although it is not that good in identifying subjects with SB.</td>
</tr>
</tbody>
</table>

(Continues)
2.6 | Risk of bias in individual studies

Risk of bias of included SR 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). This tool targets four domains through which bias may be introduced into a SR: (a) study eligibility criteria; (b) identification and selection of studies; (c) data collection and study appraisal; and (d) synthesis and findings. In addition, each domain presents 5-6 signalling questions, of which possible answers were as follows: “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 was determined by the authors, according to the following: “low risk” if all signalling 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: (a) low, if all four domains were judged as “low risk” or only one as “unclear risk”; (b) moderate, if two or more domains were judged as “unclear risk”; and (c) 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).

2.7 | Summary measures

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, standardised or weighted mean differences, RR and qualitative data.

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

2.8 | 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 SR.
3 | RESULTS

3.1 | SR selection

From a total of 2140 references identified on electronic databases searches, 1038 remained after duplicates had been removed. Papers identified 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, only 41 SR were finally included for qualitative synthesis; further information regarding reasons for SR exclusion is available in Appendix S2. Moreover, the complete process of studies’ identification and selection is provided in Figure 1.

3.2 | SR characteristics

Overall, three SR investigated prevalence rates among different populations,2,4 seventeen investigated associated factors,23,27,32,35,38,40,42,45,49,52,57,58 and fifteen evaluated effects on stomatognathic structures,40,44,45,48,52,55,56,62 statistical pooling of data using MA was available in 7 studies.33,34,40,41,45,46,59 Regarding language of publication, most reviews were published in English, one in German27 and one in Portuguese.27 All SR were published between 2007 and 2018. Overall characteristics of included SR are available in Tables 1-4.

With regard to primary studies found within SR, a total of 254 studies were identified, of which 151 were cited once, 62 were cited twice, and 7 were cited three times across SR. More information regarding primary studies is available in Tables S1-S5.

3.3 | Risk of bias within SR

Overall, nine SR were judged with low risk,23,25,29,41,46,50,53,54,59 seventeen with moderate risk,2,4,16,24,26,28-31,33,34,43,45,47,49,57,58 and fifteen with high risk of bias.3,27,32,35-38,40,43,44,48,51,52,55,56 Major concerns regarding risk of bias were observed, which included the following: (a) lack of a priori registration of the study protocol; (b) inappropriate range of database/electronic sources searched; (c) no risk of bias assessment; (d) study selection, data collection or bias assessment performed by only one reviewer; (e) no publication bias assessment or sensitivity analysis; (f) 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 are available in Figure 2 and Appendix S3.

3.4 | Results of individual SR

The majority of the SR did not specify if SB or AB were investigated separately. To highlight this, the term “generic bruxism” is used below.

3.4.1 | Prevalence rates

From 3 SR 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%.3,4 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%.2 It is worth mentioning that two SR reported that SB prevalence decreased with age.2,4

3.4.2 | Associated factors

Five SR investigated children and adolescents exclusively,24,28,30,33,24 of which one concluded that available evidence was considered insufficient to credit or discredit any association between tension-type headache and migraine with SB.28 Other SR, based on consistent evidence, proposed that generic bruxism was associated with second-hand smoke,24,34 sleep disturbances25 and psychosocial factors.30,34 It is worth mentioning that two SR investigated a wide range of sleep behaviours and risk factors and, based on consistent evidence, proposed that some were associated with generic bruxism in children, including snoring, mouth breathing, restless sleep and others.30,34

Association between bruxism and temporomandibular disorders (TMD) was assessed in 3 SR27,35,38; evidence was considered insufficient or plausible in all 3 SR. Manfredini et al27 suggested that investigations based on self-report or clinical bruxism showed a plausible association with TMD pain; however, potential bias and confounders at diagnostic level were major concerns in included studies. Later, Cunali et al27 concluded that evidence was insufficient to support an association between SB in particular and TMD, whilst Jiménez-Silva et al25 suggested that generic bruxism 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 al29 suggested that available evidence was insufficient to credit or discredit an association with SB. Similarly, a more recent SR (2018)36 concluded that there are not enough scientific data to define a clear causative link between obstructive sleep apnoea (OSA) and SB, although some clinical features appear to be common in both conditions.

Considering miscellaneous factors, significantly increased odds for SB were observed by Castroflorio et al25 with regard to gastro-oesophageal reflux disease, genetic polymorphisms, SB during childhood and others; dry mouth on awakening, on the other hand, was considered a protective factor, since significantly lower odds regarding SB were found. In addition, Feu et al suggested that that oesophageal acidification could induce SB, whilst smoking was also consistently associated with SB in a dose-dependent manner.21 Similarly, a more recent SR (2017)23 proposed that use of alcohol, caffeine and tobacco was also consistently
### TABLE 2 Summary of overall descriptive characteristics of systematic reviews; associated factors subgroup (n = 17)

<table>
<thead>
<tr>
<th>Author (Year); Journal</th>
<th>Objectives or research question</th>
<th>Subgroup</th>
<th>Databases searched (Search date)</th>
<th>Included primary studies</th>
<th>Risk of bias assessment tools</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bertazzo-Silveira et al (2016)\textsuperscript{23}; Journal of the American Dental Association</td>
<td>In adults, is there any association between SB and alcohol, caffeine, tobacco or drug abuse?</td>
<td>Associated factors</td>
<td>LILACS, PsycINFO, PubMed, ScienceDirect and Web of Science (April 2016)</td>
<td>2 cross-sectional studies 3 cohort studies 2 descriptive studies</td>
<td>MASTARI (different questionnaires according to study design)</td>
</tr>
<tr>
<td>Cruz et al (2016)\textsuperscript{26}; International Journal of Odontostomatology</td>
<td>Verify the existence of scientific evidence of association between the daytime and/or night-time bruxism and levels of salivary cortisol.</td>
<td>Associated factors</td>
<td>PubMed; OVID and VHL (Virtual Health Library, LILACS, IBECs; MEDLINE and Scielo (January 2016)</td>
<td>2 cross-sectional studies</td>
<td>Newcastle-Ottawa SCALE for cross-sectional studies modified by Herzog et al (2013) (reference in original article)</td>
</tr>
<tr>
<td>Cunali et al (2012)\textsuperscript{27}; Revista Dor</td>
<td>Verify the possible association between sleep bruxism and temporomandibular joint disorders</td>
<td>Associated factors</td>
<td>MEDLINE, Cochrane, EMBASE, PubMed, LILACS, and BBO (January 2000 to August 2012)</td>
<td>3 cross-sectional studies 1 longitudinal study</td>
<td>No risk of bias assessment</td>
</tr>
<tr>
<td>De Luca Canto et al (2014)\textsuperscript{28}; Headache</td>
<td>Evaluate and synthesise the possible association between the most common primary headaches disorders (TTH and migraine) with SB.</td>
<td>Associated factors</td>
<td>The Cochrane Library, MEDLINE, EMBASE, PubMed, LILACS and Google Scholar (January 2014)</td>
<td>2 cross-sectional studies</td>
<td>QUIPS</td>
</tr>
<tr>
<td>De Luca Canto et al (2014)\textsuperscript{29}; Journal of Orofacial Pain</td>
<td>Evaluate the association between SB and sleep-disordered breathing</td>
<td>Associated factors</td>
<td>MEDLINE, PubMed, EMBASE, the Cochrane Library and LILACS (October 2013)</td>
<td>1 experimental bruxism study</td>
<td>Qu-ATEBS</td>
</tr>
<tr>
<td>De Luca Canto et al (2015)\textsuperscript{30}; Clinical Pediatrics</td>
<td>Evaluate whether SB is associated with psychosocial factors in children and adolescents</td>
<td>Associated factors</td>
<td>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)</td>
<td>4 case-control studies 3 other studies</td>
<td>QUIPS</td>
</tr>
</tbody>
</table>
## Main results

**In 1 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 times for those who were current smokers. The abuse of methylenedioxymethamphetamine associated with SB remained without sufficient evidence.**

One randomised 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 SB (OR = 3.3, CI = 1.6-6.6). Parafunctional behaviours (OR = 2.3, CI = 1.2-4.3) had a moderate association.

Among the nine analysed articles, associations between SB and gastro-oesophageal reflux disease (GERD) (OR = 6.6, CI = 1.4-30.9) was found in one randomised 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, CI = 1.8-7.8) as 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.

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.

Evaluated studies were unable to establish a positive relationship between SB and TMD when keywords sleep bruxism, temporomandibular disorders and polysomnography were crossed; however, they reinforce the need for referring TMD patients with sleep disorders to polysomnographic evaluation.

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.

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 apnoeic events in any of the evaluated patients. In addition, masseter activity was not observed during apnoeic episodes.

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.

**Main conclusion**

SB was associated positively with alcohol, caffeine and tobacco. The association between the studied drugs could not be discredited; however, there is still a need for stronger evidence based on studies with greater methodological rigour.

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 recommended to increase the quality of future studies.

History of SB during childhood, gastro-oesophageal reflux disease and genetic polymorphisms seem to be important risk factors associated with SB in adults. Dry mouth on awakening seems to be a protective 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.

There is no conclusive evidence of association between bruxism and salivary cortisol.

Not enough evidence to support an association between SB and TMD.

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.

There is not sufficient scientific evidence either to confirm or discredit the association between SB and sleep-disordered breathing.

The current available evidence suggests an association between sleep bruxism and psychological factors in children older than 6 years.

(Continues)
### TABLE 2 (Continued)

<table>
<thead>
<tr>
<th>Author (Year); Journal</th>
<th>Objectives or research question</th>
<th>Subgroup</th>
<th>Databases searched (Search date)</th>
<th>Included primary studies</th>
<th>Risk of bias assessment tools</th>
</tr>
</thead>
<tbody>
<tr>
<td>Feu et al (2013)[32]; Journal of Orthodontics</td>
<td>To examine whether risk factors for bruxism can be identified in children and adults.</td>
<td>Associated factors</td>
<td>Cochrane Library, MEDLINE and EMBASE (1980 to 2011)</td>
<td>1 double-blind clinical trial 1 crossover, randomised, single-blinded trial 3 longitudinal studies</td>
<td>Cochrane Collaboration risk of bias tool</td>
</tr>
<tr>
<td>Garret et al (2018)[32]; Neurology Clinical Practice</td>
<td>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.</td>
<td>Associated Factors</td>
<td>PubMed (Search date not reported)</td>
<td>37 case reports</td>
<td>No risk of bias assessment</td>
</tr>
<tr>
<td>Guo et al (2017)[33]; Sleep &amp; Breathing</td>
<td>What sleep behaviours are associated with bruxism in children?</td>
<td>Associated factors</td>
<td>PubMed, Excerpta Medica Database (EMBASE), Cochrane Library database, Web of Science, Chinese National Knowledge Infrastructure (CNKI), Chinese Biomedical Literature Database (CBM) and Wanfang Data (WF) (September 2016)</td>
<td>11 case-control studies 3 cross-sectional studies</td>
<td>1. Newcastle-Ottawa Scale on case-control studies 2. Criteria of the cross-sectional/prevalence study quality (reference in original article)</td>
</tr>
<tr>
<td>Guo et al (2018)[34]; Archives of Oral Biology</td>
<td>The risk factors related to bruxism in children</td>
<td>Associated factors</td>
<td>PubMed, EMBASE, Cochrane Library database, Web of Science, CNKI, CBM, and WF (October 2016)</td>
<td>18 case-control studies 2 cohort studies 1 RCT</td>
<td>1. Newcastle-Ottawa Scale on cohort and case-control studies 2. Cochrane risk of bias table</td>
</tr>
<tr>
<td>Jiménez-Silva et al (2017)[35]; Acta Odontologica Scandinavica</td>
<td>Sleep and awake bruxism in adults and its relationship with temporomandibular disorders</td>
<td>Associated factors</td>
<td>PubMed, Cochrane Library, MEDLINE, EMBASE, BIREME, Lilacs and Scielo (from 2003 and 2014)</td>
<td>34 case-control studies 5 cohort studies</td>
<td>Newcastle-Ottawa Scale for case-control and cohort studies</td>
</tr>
<tr>
<td>Jokubauskas et al (2017)[36]; Journal of Oral Rehabilitation</td>
<td>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?</td>
<td>Associated factors</td>
<td>PubMed, ScienceDirect, Wiley Online Library, SAGE Journals and EBSCOhost (January 2006 to September 2016)</td>
<td>3 experimental bruxism studies</td>
<td>Qu-ATEBS</td>
</tr>
</tbody>
</table>
Main results

There is some evidence that:
1. Disturbances in the central dopaminergic system are implicated in the aetiology of bruxism;
2. SB can be induced by oesophageal acidification.
3. An important dose-dependent relationship exists between smoking and bruxism, and this is a behaviour 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.

Antidepressant-associated bruxism may occur in paediatric 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 pharmacologic agent. The incidence of this phenomenon is unknown.

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 (1.70, 1.0-2.39, 0.003) and inadequate sleep time (2.56, 1.48-4.43, 0.001).

Gender, age, gene, mixed position, anxiety, the nervous, second-hand 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, birthweight, occupation of family head, maternal marital status, hyperactivity, family income seemed to have statistical significance from the present systematic review and meta-analysis.

Thirty-nine studies (n = 39) were analysed in this review. According to bruxism diagnosis, articles were grouped as follows: 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.

Two studies gave evidence that OSA is associated with the occurrence of SB events: (a) SB events frequently occur during micro-arousal events consequent on apnoea-hypopnoea (AH) events, and (b) 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.

Main conclusion

There is convincing evidence that (sleep-related) bruxism can be induced by oesophageal 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 aetiology of bruxism.

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.

Snoring, mouth breathing, restless sleep, drooling, stomach position during sleep and lack of sleep were the risk factors related to bruxism in children.

The risk factors related to bruxism were as follows: Male, gene, mixed position, moves a lot, anxiety, the nervous, psychological reactions, responsibility, second-hand smoke, snore loudly, restless 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.

The evidence based on PSG was not as conclusive as the studies that used surveys and clinical examination 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.

There are not enough scientific data to define a clear causative link 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.

(Continues)
associated with SB. With regard to stress-related outcomes, one SR proposed that increased distress in everyday life, as generically described by the authors, could be a plausible associated factor for SB. Moreover, salivary cortisol levels (ie hormones related to stress) were investigated by Cruz et al. Furthermore, two SR evaluated the possible association between psychoactive medications and presence of sleep bruxism?

Furthermore, two SR evaluated the possible association between bruxism and use of several psychotropic medications. Garret et al, based on insufficient evidence from case reports, suggested that antidepressant-associated bruxism may plausibly occur in paediatric and adult patients and that fluoxetine, sertraline and venlafaxine were the most commonly reported agents. Moreover, Melo et al 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.

### 3.4.3 Diagnostic accuracy

Two SR were identified regarding diagnostic accuracy of SB assessment tools. Manfredini et al 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, Casset et al updated existing literature about portable devices and additionally 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

<table>
<thead>
<tr>
<th>Author (Year); Journal</th>
<th>Subgroup</th>
<th>Databases searched (Search date)</th>
<th>Included primary studies</th>
<th>Risk of bias assessment tools</th>
</tr>
</thead>
<tbody>
<tr>
<td>Kulis et al (2008)37; Schweizer Monatsschrift für Zahnmed*</td>
<td>What variables have been identified as risk factors for sleep and/or awake bruxism in adults?</td>
<td>PubMed, MEDPILOT.DE (URL: <a href="http://www.medpilot.de">www.medpilot.de</a>), publisher database the German doctors Publishing (URL: <a href="http://www.dzz.de">www.dzz.de</a>), publishing database of Quintessence Publishing (URL: <a href="http://www.quintessenz.de">www.quintessenz.de</a>) and Google Scholar (June 2007)</td>
<td>6 cross-sectional studies 1 longitudinal study</td>
<td>No risk of bias assessment</td>
</tr>
</tbody>
</table>
Main results

1. Three variables—severe stress experience; age between 25 and 44 years; age between 45 and 64 years—were grouped into 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.

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 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.

Overall, one study was categorised 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, 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), whilst 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).

Main conclusion

Considering the risk factors in categories A and B, it is apparent that the only modifiable risk factor is a very stressful life. It follows the recommendation 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 epidemiological and clinical studies should be planned and carried out with the help of our knowledge deepens on this subject.

Investigations based on self-report or clinical bruxism diagnosis showed a positive association with TMD pain, but they are characterised by some potential bias and 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.

Based on limited number of included papers, medications such as duloxetine, paroxetine, venlafaxine, barbiturates and methylphenidate may exhibit a positive association with the presence of SB.

3.4.4 | Effects on stomatognathic structures

Three SR investigated the effects of generically identified bruxism regarding dental implants.40,42,45 Manfredini et al42 suggested that generic 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,40 on the other hand, concluded that the effects of generic bruxing habits on the osseointegration and survival of endosteal dental implants are still not well established. Moreover, Zhou et al45 suggested that generic bruxism is a plausible contributing factor to dental implant technical/biological complications and plays a role in dental implant failure.

Two SR assessed the effects of bruxism on dental restorations. Schmitter et al44 concluded there is a lack of information about the effect of generic bruxism on the incidence of technical failure of veneered zirconia restorations. Melo et al41 concluded that available evidence did not credit or discredit any association between SB and increased odds of failure for ceramic restorations.

A single SR investigated possible harmful effects of generic bruxism on the periodontium.43 The authors (based on scarce quantity and quality of available literature) concluded that current evidence points out that generic bruxism cannot cause periodontal damage per se, although more and better studies were recommended to further clarify this issue.
TABLE 3  Summary of overall descriptive characteristics of systematic reviews; effects on stomatognathic structures subgroup (n = 6)

<table>
<thead>
<tr>
<th>Author (Year); Journal</th>
<th>Objectives or research question</th>
<th>Subgroup</th>
<th>Databases searched (Search date)</th>
<th>Included primary studies</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chrcanovic et al (2015)40; Implant Dentistry</td>
<td>In patients being rehabilitated with dental implants, what is the effect of bruxism on the implant failure rates, postoperative infection and marginal bone loss?</td>
<td>Effects on stomatognathic structures</td>
<td>PubMed, Web of Science and the Cochrane Oral Health Group Trials Register (June 2014)</td>
<td>2 controlled clinical trials 3 prospective non-controlled trials 5 retrospective anal</td>
</tr>
<tr>
<td>De Souza Melo et al (2017)41; Journal of Prosthetic Dentistry</td>
<td>Is sleep bruxism associated with an increased frequency of ceramic restoration failures?</td>
<td>Effects on stomatognathic structures</td>
<td>EMBASE, Latin American and Caribbean Health Sciences (LILACS), LIVIVO, PubMed (including MEDLINE), ScienceDirect, the Cochrane Library and Web of Science</td>
<td>8 retrospective cohort studies</td>
</tr>
<tr>
<td>Manfredini et al (2014)42; Clinical Implant Dentistry and Related Research</td>
<td>Role of bruxism as a risk factor for the different complications on dental implant-supported restorations</td>
<td>Effects on stomatognathic structures</td>
<td>MEDLINE for English-language articles (May 2012)</td>
<td>21 studies</td>
</tr>
<tr>
<td>Manfredini et al (2015)43; Journal of Periodontology</td>
<td>Is there any evidence that bruxism may cause periodontal damage per se?</td>
<td>Effects on stomatognathic structures</td>
<td>MEDLINE and SCOPUS for English-language articles (January 2014)</td>
<td>1 case-control study 5 cohort studies</td>
</tr>
<tr>
<td>Schmitter et al (2014)44; The International Journal of Prosthodontics</td>
<td>Investigate the influence of patient-related factors on restoration survival as well as to report the methods used to collect these factors.</td>
<td>Effects on stomatognathic structures</td>
<td>MEDLINE (via PubMed), Cochrane library and OpenSIGLE (July 2012)</td>
<td>No bruxism-related included study</td>
</tr>
<tr>
<td>Zhou et al (2016)45; Clinical Implant Dentistry and Related Research</td>
<td>Does bruxism contribute to dental implant failure?</td>
<td>Effects on stomatognathic structures</td>
<td>MEDLINE (PubMed) and EMBASE (November 2013)</td>
<td>7 cohort studies</td>
</tr>
</tbody>
</table>

Abbreviations: CI, confidence interval; GRADE, Grading of Recommendations, Assessment, Development and Evaluation; HR, hazard ratio; MASTARI, Meta-Analysis of Statistics Assessment and Review Instrument; OR, odds ratio; SB, sleep bruxism.
### TABLE 3

<table>
<thead>
<tr>
<th>Risk of bias assessment tools</th>
<th>Main results</th>
<th>Main conclusion</th>
</tr>
</thead>
<tbody>
<tr>
<td>Newcastle-Ottawa Scale</td>
<td>Ten publications were included with a total of 760 implants 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 )).</td>
<td>These results cannot suggest that the insertion of dental implants in bruxers affects the implant failure rates due to a limited number of published studies, all characterised 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.</td>
</tr>
<tr>
<td>MASTARI</td>
<td>Eight studies were included for qualitative synthesis, but only 5 for the meta-analysis. Three studies were categorised 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 favour or disfavour 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.</td>
<td>The overall result from the meta-analysis did not favour any association between SB and increased odds of failure for ceramic restorations.</td>
</tr>
<tr>
<td>Authors’ judgement (no specific tool)</td>
<td>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 to implant failures in six papers, whilst 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.</td>
<td>Bruxism is unlikely to be a risk factor for biological complications around dental implants, whilst there are some suggestions that it may be a risk factor for mechanical complications.</td>
</tr>
<tr>
<td>CASP cohort study checklist</td>
<td>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 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.</td>
<td>Despite the scarce quantity and quality of the literature prevents from drawing sound conclusions on the causal link between bruxism and the periodontal problems assessed in this review, it seems reasonable to suggest that bruxism cannot cause periodontal damage per se, but it is also important to emphasise that due to methodological problems regarding particularly SB assessment, more and better studies should be performed in order to further clarify this issue.</td>
</tr>
<tr>
<td>Not applicable</td>
<td>Not applicable</td>
<td>There is a lack of information about the effect of bruxism on the incidence of technical failure of veneered zirconia restorations because all available studies failed to use suitable instruments for diagnosis of bruxism.</td>
</tr>
<tr>
<td>Newcastle-Ottawa Scale for cohort studies</td>
<td>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 non-bruxers for all subgroups was 4.72 (95% CI: 2.66-8.36, ( P = 0.07 )). In group B, the total pooled OR of bruxers versus non-bruxers for all subgroups was 3.83 (95% CI: 2.12-6.94, ( P = 0.22 )).</td>
<td>This meta-analysis was performed to evaluate the relationship between bruxism and dental implant failure. In contrast to non-bruxers, 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 dental implant failure.</td>
</tr>
</tbody>
</table>
### TABLE 4 Summary of overall descriptive characteristics systematic reviews; interventions’ effectiveness subgroup (n = 13)

<table>
<thead>
<tr>
<th>Author (Year); Journal</th>
<th>Objectives or research question</th>
<th>Subgroup</th>
<th>Databases searched (Search date)</th>
<th>Included primary studies</th>
</tr>
</thead>
<tbody>
<tr>
<td>De la Torres Canales et al (2017)&lt;sup&gt;a&lt;/sup&gt;; Clinical Oral Investigations</td>
<td>Is there enough evidence to use botulinum toxin injections for bruxism management?</td>
<td>Therapy effectiveness</td>
<td>PubMed, SCOPUS, Web of Science, EMBASE, Cochrane, Scielo and Lilacs on English-language articles (1980 to March 2016)</td>
<td>2 RCT 3 before-after studies</td>
</tr>
<tr>
<td>Jokubauskas et al (2017)&lt;sup&gt;b&lt;/sup&gt;; Journal of Oral Rehabilitation</td>
<td>What is the effect of oral appliances on various treatment outcomes in adult patients with SB</td>
<td>Therapy effectiveness</td>
<td>Cochrane Library and MEDLINE (via PubMed) (January 2017)</td>
<td>7 before-after studies 7 RCTs 2 RCTs (crossover)</td>
</tr>
<tr>
<td>Jokubauskas et al (2018)&lt;sup&gt;c&lt;/sup&gt;; Journal of Oral Rehabilitation</td>
<td>Assessing the most recent literature and providing a comprehensive summary of the efficacy of any biofeedback treatment approach for the reduction or control of SB.</td>
<td>Therapy effectiveness</td>
<td>MEDLINE (searched via PubMed), EMBASE (searched via ScienceDirect), System for Information on grey literature in Europe, The Cochrane Library (Cochrane Central Register of Controlled Trials) and LILACS (January 2018)</td>
<td>4 RCTs 2 uncontrolled before-after studies</td>
</tr>
<tr>
<td>Lang et al (2009)&lt;sup&gt;d&lt;/sup&gt;; Research in Developmental Disabilities</td>
<td>This review involved a systematic analysis of studies that focused on the treatment of bruxism in individuals with developmental disabilities.</td>
<td>Therapy effectiveness</td>
<td>Education Resources Information Center (ERIC), MEDLINE, Psychology and Behavioural Sciences Collection and PsycINFO (December 2008)</td>
<td>11 studies</td>
</tr>
<tr>
<td>Long et al (2012)&lt;sup&gt;e&lt;/sup&gt;; International Dental Journal</td>
<td>The objective of this study was to assess the efficacy of botulinum toxins on bruxism.</td>
<td>Therapy effectiveness</td>
<td>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 on grey literature in Europe)</td>
<td>2 RCT 2 controlled before-after studies</td>
</tr>
</tbody>
</table>
### TABLE 4
Summary of overall descriptive characteristics systematic reviews; interventions' effectiveness subgroup (n = 13)

<table>
<thead>
<tr>
<th>Study &amp; Description</th>
<th>Main results</th>
<th>Main conclusion</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Lang et al (2009)</strong></td>
<td>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 randomised clinical trials were double-blinded, with a control group using saline solution. Two studies used polysomnography/electromyography for 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.</td>
<td>Despite the paucity of works on the topic, BoNT-A seems to be a possible management option for sleep bruxism, minimising symptoms and reducing the intensity of muscle contractions, although further studies are necessary especially as far as the treatment indications for bruxism itself is concerned.</td>
</tr>
<tr>
<td><strong>Jokubauskas et al (2018)</strong></td>
<td>Analysis of the included articles revealed a high variability of study designs and findings. Generally, the risk of bias was low to unclear for RCTs and 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 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.</td>
<td>Although many positive studies support the efficiency of OA treatment 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.</td>
</tr>
<tr>
<td><strong>De la Torres Canales et al (2017)</strong></td>
<td>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 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.</td>
<td>One of the biofeedback modalities, CES, is effective in reducing SB-related motor activities after a short-term treatment period. However, evidence of long-term effects is lacking. Further longitudinal studies with larger samples are necessary to acknowledge the clinical application of biofeedback.</td>
</tr>
</tbody>
</table>

**GRADE criteria**

- No risk of bias assessment
  - Across the 11 studies, intervention was provided to a total of 19 participants aged 4-43 years. Assessment procedures included dental screening under sedation and interviews with caregivers. Intervention approaches included prosthodontics, dental surgery, injection of botulinum toxin-a, behaviour modification, music therapy and contingent massage. Positive outcomes were reported in 82% of the reviewed studies.
  - Overall, the evidence base is extremely limited and no definitive statements regarding treatment efficacy can be made. However, behaviour modification and dental or medical treatment options (eg prosthodontics) seem to be promising treatment approaches. At present, a two-step assessment process, consisting of dental screening followed by behavioural assessment, can be recommended.

- 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 of < 100 U are safe for otherwise healthy patients.
  - Botulinum toxin injections are effective on bruxism and are safe to use. Therefore, they can be used clinically for otherwise healthy patients with bruxism.

(Continues)
### Table 4 (Continued)

<table>
<thead>
<tr>
<th>Author (Year); Journal</th>
<th>Objectives or research question</th>
<th>Subgroup</th>
<th>Databases searched (Search date)</th>
<th>Included primary studies</th>
</tr>
</thead>
<tbody>
<tr>
<td>Macedo et al (2014)^54; Cochrane Database of Systematic Reviews</td>
<td>To evaluate the effectiveness and safety of pharmacological therapy for the treatment of sleep bruxism compared with other drugs, no treatment or placebo.</td>
<td>Therapy effectiveness</td>
<td>The Cochrane Central Register of Controlled Trials (CENTRAL) (Issue 8, 2014); MEDLINE (1966 to August 2014); EMBASE (1980 to August 2013); LILACS (1982 to August 2014).</td>
<td>7 RCTs (crossover)</td>
</tr>
<tr>
<td>Machado et al (2011)^55; Dental Press Journal of Orthodontics</td>
<td>The objective of this systematic literature review is to discuss, based on scientific evidence, treatment alternatives for the control and management of SB</td>
<td>Therapy effectiveness</td>
<td>MEDLINE, Cochrane, EMBASE, PubMed, Lilacs and BBO for articles in English, Spanish or Portuguese (January 1990 until July 2008)</td>
<td>11 RCTs</td>
</tr>
<tr>
<td>Manfredini et al (2015)^56; Journal of Oral Rehabilitation</td>
<td>The review focuses on the most recent literature on management of sleep bruxism (SB) in adults</td>
<td>Therapy effectiveness</td>
<td>PubMed for articles in English (March 2015)</td>
<td>12 RCTs 2 before-after studies</td>
</tr>
<tr>
<td>Risk of bias assessment tools</td>
<td>Main results</td>
<td>Main conclusion</td>
<td></td>
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<tr>
<td>Cochrane Collaboration's risk of bias tool for randomised controlled trials</td>
<td>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 significant differences between the occlusal splint and control groups were found in the meta-analyses.</td>
<td>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 regard to tooth wear. This systematic review suggests the need for further investigation in more controlled RCTs that pay attention to method of allocation, outcome assessment, large sample size and sufficient duration of follow-up. The study design must be parallel, in order to eliminate the bias provided by studies of crossover type. A standardisation of the outcomes of the treatment of sleep bruxism should be established in the RCTs.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cochrane Collaboration's risk of bias tool for randomised controlled trials</td>
<td>Results were imprecise and consistent with benefit, no difference or harm. These were the specific findings for each of the drugs according to specific outcomes: 1. Amitriptyline versus placebo for masseteric electromyography (EMG) activity per minute: standardised 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).</td>
<td>There was insufficient evidence on the effectiveness of pharmacotherapy for the treatment of sleep bruxism. This systematic review points to the need for more, well-designed, RCTs with larger sample sizes and adequate methods of allocation, outcome assessment and duration of follow-up. Ideally, parallel RCTs should be used in future studies to avoid the bias associated with crossover studies. There is a need to standardise the outcomes of RCTs on treatments for sleep bruxism.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>No risk of bias assessment</td>
<td>1. Occlusal splint seems to be an acceptable and safe treatment alternative in the short and medium terms, whilst 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. 3. Cognitive-behavioural therapies such as psychotherapy, biofeedback, physical exercise and lifestyle changes, which are aimed at stress reduction, may be auxiliary in the treatment of SB.</td>
<td>According to the literature analysis, 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.</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

1. Cochrane Collaboration's risk of bias tool for randomised controlled trials  
2. CASP checklist for cohort studies  

The studies' results suggest that (a) 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; (b) all tested pharmacological approaches (ie botulinum toxin (two papers), clonazepam (one paper) and clonidine (one paper)) may reduce SB with respect to placebo; (c) the potential benefit of biofeedback (BF) and cognitive-behavioural (CB) approaches to SB management is not fully supported (two papers); and (d) the only investigation providing an electrical stimulus to the masseter muscle supports its effectiveness to reduce SB.  

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 for SB treatment are recommended.
**TABLE 4** (Continued)

<table>
<thead>
<tr>
<th>Author (Year); Journal</th>
<th>Objectives or research question</th>
<th>Subgroup</th>
<th>Databases searched (Search date)</th>
<th>Included primary studies</th>
</tr>
</thead>
<tbody>
<tr>
<td>Manfredini et al (2017)(^5); Journal of Prosthetic Dentistry</td>
<td>The purpose of this systematic review was to evaluate the relationship between prosthetic rehabilitation and TMDs and bruxism</td>
<td>Therapy effectiveness</td>
<td>PubMed (July 2016)</td>
<td>No included study</td>
</tr>
<tr>
<td>Restrepo et al (2009)(^5); Quintessence International</td>
<td>To conduct a systematic review to assess and analyse the scientific evidence about the available therapies for bruxism in children.</td>
<td>Therapy effectiveness</td>
<td>MEDLINE, PubMed, Ovid, Biomed Central, EBSCOhost, ISI, Cochrane Library, EMBASE, LILACS, Scielo, Scirus (March 1985 to September 2007)</td>
<td>1 quasi-experimental study 1 RCT</td>
</tr>
<tr>
<td>Stapelmann et al (2008)(^5); BMC Oral Health</td>
<td>The aim of this systematic review was to appraise the currently available evidence regarding the efficacy and safety of the NTI-TSS splint.</td>
<td>Therapy effectiveness</td>
<td>The Cochrane Library, PubMed, TRIP database, MEDPILOT.DE, BIREME, Deutscher Arzte-Verlag database, Quintessenz Database, Google Scholar, Web of Science (December 2007).</td>
<td>2 bruxism-related RCTs</td>
</tr>
<tr>
<td>Wang et al (2014)(^5); Sleep &amp; Breathing</td>
<td>The aim of this systematic review was to evaluate the efficacy of any biofeedback treatment on sleep bruxism.</td>
<td>Therapy effectiveness</td>
<td>Cochrane Central Register of Controlled Trials, MEDLINE, EMBASE, ISI Web of Science, System for Information on grey literature in Europe, Chinese Biomedical Literature Database and PsycINFO (October 2012)</td>
<td>7 RCTs</td>
</tr>
</tbody>
</table>

Abbreviations: BF, biofeedback; BoNT-A, type A botulinum toxin; CB, cognitive-behavioural; CI, confidence interval; CASP, Critical Appraisal Skills Programme; CES, contingent electrical stimulation; EMG, electromyography; GRADE, Grading of Recommendations, Assessment, Development and Evaluation; MD, mean difference; NTI-TSS, nociceptive trigeminal inhibition tension suppression system; OA, oral appliance; RCT, randomised controlled trial; SB, sleep bruxism; SMD, standardised mean difference; TMD, temporomandibular disorder.

\(^a\)Only data regarding bruxism were considered.

### 3.4.5 | Interventions’ effectiveness

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

Regarding occlusal appliances, Macedo et al,\(^5\) in a Cochrane review, concluded that available evidence was insufficient to state that occlusal splint is effective for SB management. Moreover, Stapelmann et al\(^5\) proposed that nociceptive trigeminal inhibition tension suppression system (NTI-TSS) device might present plausible effectiveness on the management of bruxism. Furthermore, the
most recent SR on occlusal appliances reported that, although there is consistent evidence regarding 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.69 With regard to pharmacological therapies, Macedo et al.54 in a Cochrane review, suggested that evidence was still insufficient on the effectiveness of pharmacotherapy for the treatment of SB. Moreover, regarding botulinum toxin injections in particular, both SR of Long et al52 and De La Torres Canales et al48 suggested that this therapeutic method may reduce intensity of muscle contractions where applied in generic bruxism patients.

Concerning biofeedback therapies, Wang et al39 concluded that there was no powerful evidence to support the use of biofeedback...
technology on SB treatment. On the other hand, Jokubauskas et al.\textsuperscript{50} 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. No long-term effects were assessed.

**FIGURE 2** 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)
There was a group of two SR that evaluated multiple treatment methods simultaneously; Machado et al.\(^\text{55}\) concluded there are lot of treatment options for SB; however, many lack scientific support. Similarly, Manfredini et al.\(^\text{16}\) reported outcomes related to occlusal appliances, pharmacological approaches, biofeedback and cognitive-behavioural approaches, and electrical stimulus for masseter muscles. The authors concluded that there was not enough evidence to define a standard of reference approach for SB management, with the exception of occlusal appliances, in which there was consistent evidence of effectiveness.

Moreover, four SR assessed miscellaneous therapies with regard to bruxism. Restrepo et al.\(^\text{57}\) evaluated treatment of generic 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.\(^\text{51}\) 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.\(^\text{56}\) revealed an absence of RCTs on the topic, and therefore, prosthetic changes in dental occlusion were considered not yet acceptable strategies for bruxism management.

### 3.4.6 Risk of bias across studies

A great variability was observed across included SR Regarding bruxism classification, most SR investigated SB alone, several SR used the generic term "bruxism" or "parafunational habits," and a single SR investigated AB separately from SB.\(^\text{2}\) In addition, bruxism diagnostic criteria were greatly heterogeneous; the majority of primary studies included in SR have evaluated bruxism through questionnaires or clinical examinations, whilst few have adopted the use of PSG or EMG examinations to confirm the diagnosis.

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

### 4 DISCUSSION

#### 4.1 Summary of evidence

This umbrella review aimed to summarise and critically appraise current literature regarding bruxism-related SR Although evidence from SR 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.\(^\text{60}\) Therefore, caution should be exercised by healthcare practitioners and policymakers with regard to biomedical publishing and the need to improve standards in conducting and reporting SR is highlighted.

Having analysed 41 SR, which included over 250 primary studies, authors identified that approximately one-fourth of primary studies were cited more than one time. This finding should be considered carefully since it could indicate unnecessary duplication of SR. It should be noted, however, that replication of an existing SR to overcome methodological limitations, update findings or investigate different outcomes is considered appropriate and not necessarily a problem associated with resource waste.\(^\text{61}\)

Nonetheless, a recent commentary in the field of bruxism research indicated that approximately one-third of articles published during the years of 2016-2017 were reviews, including SR and meta-analyses. It was also suggested that the "publish or perish" mentality might lead researchers with less experience to conduct a SR without needed skills and clinical expertise, resulting in methodological flaws or publications of limited value.\(^\text{62}\) Although these topics were not explored in depth in this umbrella review, it was observed that there was a considerable number of SR with methodological limitations or bias of other nature. These findings provide an opportunity to reflect on current development of SR and which direction related evidence-based research should follow.

With regard to findings from included SR, 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 paediatric populations. Moreover, sample sizes were usually large, which might explain the lack of PSG and/or EMG examinations.\(^\text{63}\) Therefore, overall conclusions from epidemiological SR should be interpreted with caution.\(^\text{2,4}\)

Although a considerable number of factors investigated in included SR presented increased odds/risk for the presence of bruxism, it is proposed that bruxism could potentially act as a protective factor by reducing the likelihood of negative events for certain conditions.\(^\text{1}\) For example, by increasing salivation rate, SB might reduce the risk of detrimental chemical tooth wear in case of gastro-oesophageal reflux.\(^\text{64}\) Therefore, although gastro-oesophageal reflux might induce SB,\(^\text{25,31}\) this might act as a protective factor.\(^\text{65}\) It should be mentioned that SB may also arise as a consequence of sleep breathing disorders, as, for example, SB activity might act as a protective factor regarding obstructive sleep apnoea by restoring patency of the airways.\(^\text{65}\) Moreover, although evidence from included SR was considered insufficient regarding an association between SB and sleep breathing disorders among adults,\(^\text{29,36}\) there is some evidence of an association regarding children,\(^\text{33,34}\) which might have contributed to the high prevalence of SB observed considering younger populations. Since current evidence might be potentially biased due to inaccurate diagnostic methods, more studies are recommended to further explore these topics.

Regarding accuracy of diagnostic methods investigating AB in particular, current synthesis literature was considered non-existent, as no SR on the topic were found. Some SR investigated methods...
to assess SB, proposing that portable diagnostic devices presented overall high values of specificity and sensitivity compared to the reference standard PSG, but it must be pointed out that a clear definition regarding bruxism as a behaviour or a disorder is not yet well established. **Depending on future consensus updates, there may be a reappraisal of PSG criteria, which are currently used as reference for SB diagnosis.**

Several SR 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, restorations and the periodontium. **However, based on results found, it could be proposed that SB might present deleterious effects for dental implants in particular.** It should be noted that although teeth grinding incidents occurs in approximately one-third of SB events, SB patients have approximately 3 to 4 times higher number and durations of SB episodes and tooth contacts compared to non-SB individuals, which might be related to findings observed. In addition, since no SR investigated deleterious effects of AB in particular on stomatognathic structures, no conclusions on this topic could be supported. Appropriate follow-up times and reliable diagnostic methods are recommended to further investigate above-mentioned topics.

Current evidence regarding interventions for the management of bruxism is still inconclusive, as previously described by Lobbezoo et al. Effectiveness of occlusal appliances in managing SB signs and symptoms was consistent across included SR; however, it should be mentioned that primary studies with longer follow-up time spans are necessary to assess its effects on the long term. Moreover, whilst these devices might play a role in reducing hazardous effects of bruxism on stomatognathic structures, evidence of further beneficial effects regarding frequency and intensity of SB episodes is weak and somewhat scarce. There was not enough evidence to propose any recommendation regarding pharmacological treatment of bruxism, although some SR proposed that botulinum toxin injections might present plausible effects in reducing amplitude of muscle contractions, but not in the number of SB events. Thus, further studies are necessary to evaluate possible beneficial effects of botulinum toxin in bruxism management.

Evidence regarding biofeedback therapies was not strong enough to suggest real benefits on bruxism management, with the exception of plausible effectiveness of CES. 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. 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 randomised and double-blinded study designs.

Considering risk of bias evaluation, the ROBIS tools were selected over other available tools due to the extent of this umbrella review. Tools such as AMSTAR-2 were developed specifically for bias assessment of systematic reviews of interventions, which was only one of the topics investigated. ROBIS was structured to be as generic as possible yet focused at four broad categories of systematic reviews mainly within health care, covering interventions, diagnosis, prognosis and aetiology. Therefore, it was considered a more appropriate tool for a comprehensive bias assessment of all included SR.

Although SR 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 SR 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 minimise or prevent potential sources of bias.

### 4.2 Limitations

The authors of this umbrella review acknowledge that inclusion criteria regarding SR definition were broad to a moderate extent. Since older SR 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 SR. In addition, it must be pointed out that a risk of over generalisation of results might be present due to the nature of this review and the identified weaknesses in the included SR, and therefore, conclusions should be interpreted with caution.

### 5 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, oesophageal acidification and second-hand smoke. Several TMD signs and symptoms presented a plausible association with SB. In paediatric 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 support harmful effects regarding other dental restorations or periodontal damage.
5. Overall, occlusal appliances were considered consistently effective for bruxism management. No treatment recommendations regarding other pharmacological treatments and biofeedback therapy could be provided, with the exception of CES.
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CONFLICT OF INTEREST

Authors have no conflicts of interest to declare.

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**SUPPORTING INFORMATION**

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