

Bruxism is Unlikely to Cause Damage to the Periodontium. Findings from a Systematic Literature Assessment

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Aim: This paper systematically reviews the Medline and Scopus literature to answer the following question: Is there any evidence that bruxism may cause periodontal damage?

Materials and methods: Clinical studies on humans, assessing the potential relationship between bruxism and periodontal lesions (i.e., decreased attachment level, bone loss, tooth mobility/migration, altered periodontal perception) were eligible. Methodological shortcomings were identified by the adoption of the Critical Appraisal Skills Programme (CASP) quality assessment, mainly concerning the internal validity of findings due to an unspecific bruxism diagnosis.

Results: 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.

Conclusions: 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 emphasize that due to methodological problems regarding particularly SB assessment, more and better studies should be performed in order to further clarify this issue.

KEYWORDS:

Bruxism; Periodontium; Review

Bruxism is an umbrella term grouping different motor phenomena. Recently, an expert group reached consensus to define it as follows: 'Bruxism is 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)'.¹ Over the past few years, as part of an ongoing strategy to summarize the available findings on the argument, the potential clinical consequences of bruxism have been systematically reviewed, with focus on its effects on the temporomandibular joints (TMJs) and jaw muscles as well as on natural teeth and restored implant-supported dentitions.²⁻⁴

On the other hand, past theories suggested that bruxism may be also a potential risk factor for overload of the teeth-supporting tissues, viz., the periodontium.^{5,6} However, the literature on the topic has not yet been systematically reviewed. Nevertheless, over half a century, several studies on the periodontal effects of the so-called occlusal trauma have been conducted.^{7,8} Also, progressive modifications to the definition of occlusal trauma itself have been performed.⁹ The most updated edition of the Glossary of Prosthodontic Terms defined it as a "trauma to the periodontium from functional or parafunctional forces causing damage to the attachment apparatus of the periodontium by exceeding its adaptive and reparative capacities".⁹ Hence, when occlusal forces exceed the adaptive capacity of the periodontal tissue, injury results. Within this framework, primary occlusal

trauma is defined as the condition in which the pathologic occlusal forces are the main etiological factor for changes in the periodontium, whilst secondary occlusal trauma occurs when the periodontium is already compromised by inflammation and bone loss.

In general, the more recent periodontal literature suggests that excessive forces on the dental occlusion are not likely to provoke any long-standing damage to a healthy periodontium, thus questioning the existence of a pure primary trauma.^{10,11} Notwithstanding that, even though the presence of bacterial plaque as a co-factor seems to be considered a pre-requisite for the onset of periodontal lesions, also in the presence of occlusal trauma, some authors suggested that the literature seems to be not fully conclusive on the issue.¹²

A possible explanation is that, given the variability of the potential clinical conditions leading to occlusal trauma, there is a need to determine the effects of each specific factor that may cause an excessive load on the teeth and their supporting structures. Based on this view, it must be remarked that most past investigations dealt mainly with the artificial insertion of an occlusal supra-contact, either in human or animal models, and the *ex juvantibus* effects of its removal in presence/absence of facilitating cofactors such as bacterial plaque, lesser number of teeth, or reduced support.¹³ In theory, trauma to the periodontium may also be due to excessive occlusal forces exerted during bruxism activities,¹⁴ but it seems that such discrimination between trauma from bruxism in the natural environment and experimental trauma from artificial interferences has not yet been clearly demonstrated.

Considering that, the aim of the present review was to provide an answer to the clinical research question “Is there any evidence that bruxism may cause periodontal damage *per se*?” by means of a systematic assessment of the available literature on the issue of bruxism-related effects on the periodontal structures.

MATERIALS AND METHODS

Search Strategy

On January 20th, 2014, a systematic search in the medical literature was performed to identify all peer-reviewed English language papers that were relevant to the review’s aim. As a first step in the search strategy, the keyword term “bruxism” was used to start browsing the literature indexed in the two most qualified medical databases (i.e., National Library of Medicine’s Medline and Scopus) to retrieve lists of potentially relevant papers. Combination of terms, also including the words “clenching” or “grinding” alone and in association with the terms “periodontium” or “periodontitis” or “bone loss” or “tooth/teeth mobility” or “bone resorption” were adopted. Based on title and abstract assessment, the studies were selected for potential inclusion independently by two of the authors (D.M, R.M.), who also performed data extraction and quality assessment by consensus decision. All authors contributed to the search expansion by checking for additional papers in the Google Scholar database, in the reference lists of potentially relevant papers, and in their own personal databases and institutional libraries.

The criteria for admittance in the systematic review were based on the type of study, and the inclusion was restricted to clinical studies on humans or animals, assessing the potential role of bruxism, as diagnosed with clinical assessment, questionnaires, interviews, polysomnography, or electromyography, as a causal factor for periodontal damage.

Systematic Assessment of Papers

The methodological characteristics of the selected papers were assessed according to a format which enabled a structured summary of the articles in relation to four main issues, viz., ‘P’ - patients/problem/population, ‘I’ - intervention, ‘C’ - comparison, and ‘O’ - outcome (PICO), for each of which specific questions were constructed.¹⁵

For each article, the study population ('P') was described based on the criteria for inclusion, the demographic features of the sample, and the sample size. The study design was described in the section reserved to questions on the study intervention ('I'), and information was gathered on the approach to bruxism diagnosis. The comparison criterion ('C') was based on the assessment of periodontium-related issues, by reporting the outcome variables, and the statistical approaches adopted by the authors to assess the role of bruxism as a risk factor for periodontal lesions. The study outcome ('O') was evaluated in relation to the influence of bruxism to the presence of periodontal lesions.

Quality Assessment

In an attempt to increase the strength of this review, and in line with current needs to weigh the quality of the reviewed literature in systematic reviews, studies that were pertinent for inclusion underwent a quality assessment by adopting the Critical Appraisal Skills Programme (CASP) Cohort Study Checklist.¹⁶ The CASP tool uses a systematic approach based on 12 specific questions to appraise three broad areas: study validity, an evaluation of methodological quality and presentation of results, and an assessment of external validity (Table 1). Each of the questions can be answered with 'yes', 'no', or 'can't tell' and each study can have a maximum score of 12. The CASP scores were used to grade the methodological quality of each study assessed.

Verification of Causality Criteria

The selected literature on the bruxism-periodontal damage relationship was also critically assessed in relation to the answer to this review's question, viz.: Is there any evidence that bruxism may cause periodontal damage *per se*?

To verify whether there is enough evidence for a cause-and-effect link between the two disorders, the widely adopted Hill's criteria were adopted (Table 2).¹⁷ Such list and its modifications have been often used for discussing causation in the bruxism, TMD, and dental occlusion literature.^{18,19} Each paper was assigned one point for each criterion satisfied in favor of a positive bruxism-periodontal damage relationship, so totalizing a minimum score of 0 (no relationship between bruxism and periodontal damage) to a maximum of 9 points (absolute relationship between bruxism and periodontal damage).

RESULTS

The search allowed identifying 2835 and 3767 citations in the Medline and Scopus databases, respectively, of which 2562 were present in both databases. Thus, 4040 citations were screened for eligibility. As shown in Figure 1, after excluding the citations that were clearly not pertinent for the review's aim on the basis of their title and abstract, 9 papers were retrieved in full text and were assessed to reach consensus as to include/exclude the papers for/from systematic assessment.²⁰⁻²⁸ Consensus decision was to exclude 5 out of the 9 papers. Reasons for exclusion were the following: not dealing with bruxism in humans or animals (N=2);^{24,25} adopting unspecified strategy to provoke bruxism in monkeys (N=1);²⁶ presenting duplicated data of an included study (N=1);²⁷ presenting a preliminary version of an included study (N=1).²⁸ Thus, four papers were included in the review.²⁰⁻²³ Search expansion strategies allowed including two further papers,^{29,30} accounting for a total of six papers included in the review.^{20-23,29,30}

Structured reading of the included articles showed a high variability of topics. Two papers dealt with the influence of bruxism on periodontal perception by the assessment of interdental tactile threshold,^{21,29} whilst single papers investigated the prevalence of periodontal problems in individuals with different grinding patterns,²⁰ the association between self-reported bruxism and periodontal problems at the general population level,²² the prevalence of pathological tooth migration in a cohort sample of periodontal patients in relation to self-reported bruxism,³⁰ and the

differences in periodontal parameters between two cohorts of periodontal or bruxism patients.²³ Given the heterogeneity of study designs, meta-analysis of data could not be performed. Methodological features and main findings concerning the possible relationship between bruxism and periodontal problems are summarized in table 3. In general, the unique effect of bruxism on the periodontal structures seems to be an increase in periodontal sensation.

Quality assessment showed that methodology was less than optimal, with only half of the investigations satisfying more than half of the quality items.^{21,22,23} A common shortcoming to most studies was the self-reported approach to bruxism diagnosis, with the exception of a single paper providing electromyographic (EMG) measurement of the masseter muscle activity during sleep in the home environment.²¹ Another point of major limitation was the unclear consistency of single papers' findings with respect to the available evidence, given the very poor literature on each specific topic. Thus, on average, the quality of investigations on the bruxism-periodontal lesions relationship can be improved and is currently not enough to provide high-quality evidence on the argument. Quality assessment of the individual papers is summarized in table 4.

According to the analysis of Hill's criteria,¹⁷ whether a negative or positive causal relationship between bruxism and periodontal lesions was claimed by the authors of the individual papers, the conclusions on causation satisfied at least half of the criteria only in three papers.^{21,22,23} Common shortcomings to all papers were the absence of any information about the temporal relationship and on the gradient effect, viz., dose-response effect, due to the lack of any longitudinal observations. Also, given the paucity of literature on the topic, very little information could be retrieved as far as the consistency, coherence, and analogy criteria are concerned. Assessment of validity of causation conclusions for individual papers is summarized in table 5.

DISCUSSION

Debate on the role of trauma from dental occlusion in the etiology of periodontal disease has been attracting generations of researchers and dental practitioners for decades.^{7,8,31} Despite a general tendency to agree that occlusal factors alone cannot explain the onset of periodontal disease, which is instead inflammatory/infective in nature, the argument is still animating discussions and still worthy to be summarized in more recent reviews.^{10,32,33} Within the factors that may exert forces on the periodontium, bruxism might be hypothesized to be a possible cause of overload. Thus, in line with recent papers that summarized several aspects about the potential pathological consequences of bruxism,^{2,3,34} this review aimed to provide a summary of the literature on the effects of bruxism on the periodontium. Unfortunately, the review felt short in the attempt to provide sound conclusions due to the shortcomings that were identified in the literature on the topic.

First, it should be noticed that very few research papers on the argument were published, with only six papers admitted in the review.^{20-23,29,30} Such limitation in the quantity of the available literature puts serious concerns about the external validity of each individual paper.³⁵ The strategies adopted to assess the consequences of bruxism on the periodontium were very variable, and prevented any attempts to meta-analyze the data. Moreover, the specificity of bruxism diagnosis was, on average, poor, since it was based on a self-reported approach as the only diagnostic method in almost all papers. Unfortunately, such an approach is suitable to detect, at best, possible bruxism,¹ and causes serious concerns about the internal validity of an individual investigation on any bruxism issue.³⁶

Second, the quality of the reviewed literature was, on average, low. The failure to provide a validated bruxism diagnosis, the absence of multiple observation points, and the lack of multiple papers on a same topic (thus preventing to control papers for their consistency with the available evidence) are the main shortcomings identified with quality assessment. In particular, even two of the three highest-quality papers did not use the standard of reference diagnostic approach to bruxism diagnosis.^{22,23} Such a limitation should have been prevented by selecting only those papers

adopting bruxism measurement diagnosis, thus potentially avoiding any reviewers' bias in quality assessment. On the other hand, factors such as the very low number of PSG-based papers in the whole bruxism literature,^{2,3,34} and the fact that the usefulness of PSG itself to detect clinically meaningful bruxism is currently under validity appraisal,⁴ suggested us to include papers in this review independently by the diagnostic approach to bruxism.

Third, mainly as a consequence of the above, the validity of causation conclusions was, in general, limited. Again, the absence of an evaluation on the temporal relationship and dose-response effects between bruxism and periodontal lesions, as well as the poor specificity with respect to the study aims concerning bruxism, were identified as the critical factors that prevented from definitively confirming or refuting a causal link between the two conditions.

Moreover, in theory, the possibility that some part of the historical literature dealing with the generic topic of occlusal trauma should have actually focused on trauma from bruxism or parafunctions, thus being potentially worthy of inclusion in the review, cannot be ruled out. On the other hand, in practice, it is unlikely that such possibility occurred and the review's findings may have been influenced, given the very poor specificity for a bruxism diagnosis even in the reviewed papers themselves. The choice of excluding papers was particularly difficult in the case of some animal studies.^{24,26} Indeed, among the several investigations on the role of occlusal trauma in animal models, some claimed to assess the effects of bruxism, due to the purported bruxism-provoking effects of an artificially-inserted high occlusal restorations.^{24,26} On the contrary, human studies have dismantled the role of natural malocclusion and/or artificially-high restorations as causal factors for bruxism.^{37,38} Thus, such animal studies were excluded from the review because of their different a priori speculations with respect to the current knowledge on humans. Anyhow, it should be pointed out that they claimed the absence of any clinical or histological evidence that bruxism had caused a progression of gingivitis to destructive, chronic marginal periodontitis in monkeys.^{24,26} Such findings are in line with the experiences of artificially-created occlusal trauma in dogs,³⁹ thus supporting the concept that, whatever the origin of trauma on the periodontium (i.e., bruxism or occlusion) or the model under study (i.e., human or animal), excessive forces cannot be viewed as the solo factor that may determine periodontal damage.

Taking the above factors into consideration, findings from the reviewed literature seem to suggest the absence of a causal link between bruxism and periodontal damage. It seems reasonable to hypothesize that an increased periodontal perception is the only plausible bruxism consequence on the periodontium.²¹ The absence of any radiological investigation makes not possible to hypothesize whether such increased perception is due to an enlargement of the periodontal ligament or to any other factors. However, such issue has important clinical implications, especially with respect to the need for adopting prudent prosthodontics strategies in bruxers.⁴⁰

Based on the above, whilst it may be suggested that bruxism cannot cause periodontal damage per se, several clinical questions remain unanswered due to the very poor quantity and less than optimal quality of the papers included in this review. For instance, an interesting topic for future investigations might be the assessment of bruxism prevalence in periodontal patients, so as to investigate the other side of the coin. Indeed, from a theoretical viewpoint, it is also possible that teeth with a decreased periodontal support or a certain degree of mobility may act as a protective mechanism against bruxism via the same pathways that reduce bruxism activities in individuals with high restorations. The role of bruxism itself as a real source of trauma to the periodontium should be carefully appraised, especially in the light of increasing evidence that several different motor activities with potentially different etiologies are actually grouped under the umbrella term 'bruxism'. Thus, the effects on such trauma on the periodontium, if existing, should be studied separately based on to the possible consequences of teeth clenching or grinding. Also, the possibility that bruxism, even if not a cause of pure primary trauma, may precipitate conditions and jeopardize survival of migrated or periodontally migrated teeth has to be addressed. Finally, as a strong recommendation for the future, it is of paramount importance that homogeneous definitions

and strategies to diagnose bruxism as well as multidisciplinary approaches (i.e., periodontists and bruxism/orofacial pain experts) to this kind of investigations are adopted.

CONCLUSIONS

This paper systematically reviewed the available bruxism literature to answer the following question: Is there any evidence that bruxism may cause periodontal damage *per se*? Unfortunately, the scarce quantity and quality of the reviewed literature prevented from achieving sound conclusions. Despite no positive relationship between bruxism and the periodontal problems assessed in this review could be found, possibly suggesting that bruxism cannot cause periodontal damage *per se*, it is also important to emphasize that due to methodological problems regarding particularly SB assessment, more and better studies should be performed in order to further clarify this issue.

CONFLICTS OF INTEREST:

The authors declare they do not have any conflicts of interest

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Figure 1.

Flow-chart of the search strategy.

Table 1.

CASP items for quality assessment of the reviewed papers.

1.Study issue is clearly focused
2.Cohort is recruited in an acceptable way
3.Exposure (bruxism) is measured accurately
4.Outcome (perio variables) is measured accurately
5.Confounding factors are addressed
6.Follow up is long and complete
7.Results are clear
8.Results are precise
9.Results are “credible”
10.Results can be applied to the local population
11.Results fit with available evidence
12.There are important clinical implications

Table 2.

Hill’s criteria for the assessment of the causal link between two phenomena.

1.Strength
2.Consistency
3.Specificity
4.Temporal
5.Gradient effect
6.Plausibility
7.Coherence
8.Experimental evidence
9.Analogy

Table 3.**PICO features of the reviewed studies.**

Study first author, year	Population (P)	Intervention (I)	Comparison (C)	Outcomes (O)
Calderon, 2009 ²⁹	N=115 (a.r. 14-37, m.a. yrs) without severe TMD	Case-control design (bruxers vs non bruxers) –unspecified size per group	Minimum interdental threshold (MID)	MID 0.013-0.016 mm for both bruxers and non-bruxers; p=0.74
		Clinical bruxism diagnosis (tooth wear, shiny spots, masseter hypertrophy) – three examiners	Sensibility frequency to test with different-thickness foils	
			Multiple linear regression analysis (unclear strategy to manage data)	
Tokiwa, 2008 ²⁰	50 individuals with stable occlusion (21 males, a.r. 23-74, m.a. 41.2 yrs)	Unspecified cohort-like design	Attachment loss (AL)	Individuals with grinding patterns involving the molars have higher values of AL, TM, NCL, DH
		Assessment of grinding types (canine vs molar grinding)	Tooth mobility (TM)	No stats for group comparison
		One-sided color foil	Non-carious cervical lesions (NCL)	No comparison vs non-bruxers
			Dental hypersensitivity (DH)	
Measurement of variables in different types of bruxers				
Ono, 2008 ²¹	28 students (a.r. 21-30, m.a. 26.3 yrs.)	Case-control design (14 bruxers vs 14 non-bruxers)	Periodontal sensation by interocclusal tactile threshold (ITT)	Mean ITT in bruxers lower than controls (p<0.0001)
		Nocturnal masseter EMG	Tooth displacement	Same pattern of force voluntary clenching-induced tooth displacement, irrespective of bruxism status, but higher displacement in bruxers (p<0.05)
			Mann-Whitney test	
Bernhardt, 2006 ²²	2980 out of 4310 individuals recruited for the Study of Health In Pomerania (SHIP) (a.r. 20-79 yrs)	Cohort study	Probing depth (PD)	Bruxism not associated with PD or CA loss
		Self-reported bruxism (69% never; 23% sometimes; 8% often/always)	Clinical attachment (CA) loss	
			Plaque score (PS)	
			Regression analysis	
Martinez-Canut, 1997 ³⁰	825 periodontal patients (36% males, a.r. 19-72, m.a. 42.5 yrs)	Cohort study	Pathological tooth migration (PTM) – as diagnosed on developing diastema in the upper anterior sextant	PTM: 15% of bruxers vs 12% of non-bruxers (p=0.159); 26% of clenchers vs 28% non-clenchers (p=0.551)
		Self-reported bruxism (14%) or clenching (27%)	Wald coefficient	
		Attrition to confirm bruxism		
Hanamura, 1987 ²³	51 patients with moderate-to-severe periodontitis (26 males, a.r. 35-60, m.a. 48.2 yrs), and	Small, selected cohort study	Bone level (BL)	Higher BL in bruxers (88% of root length vs 72%; p<0.001)

	40 patients with bruxism-tooth wear (19 males, a.r. 37-62, m.a. 48.9 yrs)	Self-reported bruxism diagnosis (57% in the bruxism group; 24% in the perio group)	Attachment level (AL)	Loss of AL more pronounced in periodontal patients (p<0.001) – loss > 5mm= 27.2% of perio vs 4.9% of bruxer sites
			Tooth mobility (TM)	TM more prevalent in perio (p<0.001)
			Cross-tabulation stats	

Legends: TMD, temporomandibular disorders; a.r., age range; m.a., mean age; EMG, electromyography.

unedited

Table 4.**CASP quality assessment of the reviewed papers. Columns showed the twelve quality items. Reasons for negative endorsement are provided.**

Study first author, year	Item #1	Item #2	Item #3	Item #4	Item #5	Item #6	Item #7	Item #8	Item #9	Item #10	Item #11	Item #12	Total quality score (0-12)
Calderon, 2009 ²⁹	Yes	No (unclear recruitment strategy)	Yes (even if clinical, not measurement-based, diagnosis)	No (MIT measured with foils)	No (no evaluation of teeth-related factors)	No (experiment without multiple observation points)	Yes	No (outcome variable not measured accurately)	No (outcome variable not measured accurately)	Yes	Can't tell (other studies not available)	No (unclear implications)	4
Tokiw a, 2008 ²⁰	Yes	Yes	No (subjective strategy to assess grinding patterns – no calibrated examiners)	Yes	No (other causes of periodontal problems not assessed)	No (experiment without multiple observation points)	No (conclusions not clearly stated)	No (no statistical analysis)	No (no statistical analysis)	Yes	Can't tell (other studies not available)	No (unclear implications)	4
Ono, 2008 ²¹	Yes	Yes	Yes	Yes	Yes	No (experiment without multiple observation points)	Yes	Yes	Yes	Yes	Can't tell (other studies not available)	Yes	10
Bernhardt, 2006 ²²	Yes	Yes	No (self-reported bruxism)	Yes	Yes	No (cross-sectional)	Yes	Yes	Yes	Yes	Yes	Yes	10
Martinez-Canut, 1997 ³⁰	Yes	Yes	No (self-reported bruxism)	No (unspecified period problems and unvalidated PTM)	Yes	No (cross-sectional)	Yes	No (unvalidated diagnoses)	Yes	Yes	Can't tell (other studies not available)	No (unclear implications)	6

				diagnosis)									
Hana mura, 1987 ²³	Yes	Yes	No (self-reported bruxism)	Yes	Yes	No (cross-sectional)	Yes	Yes	Yes	Yes	Can't tell (other studies not available)	Yes	9

Legend: PTM, pathological tooth migration.

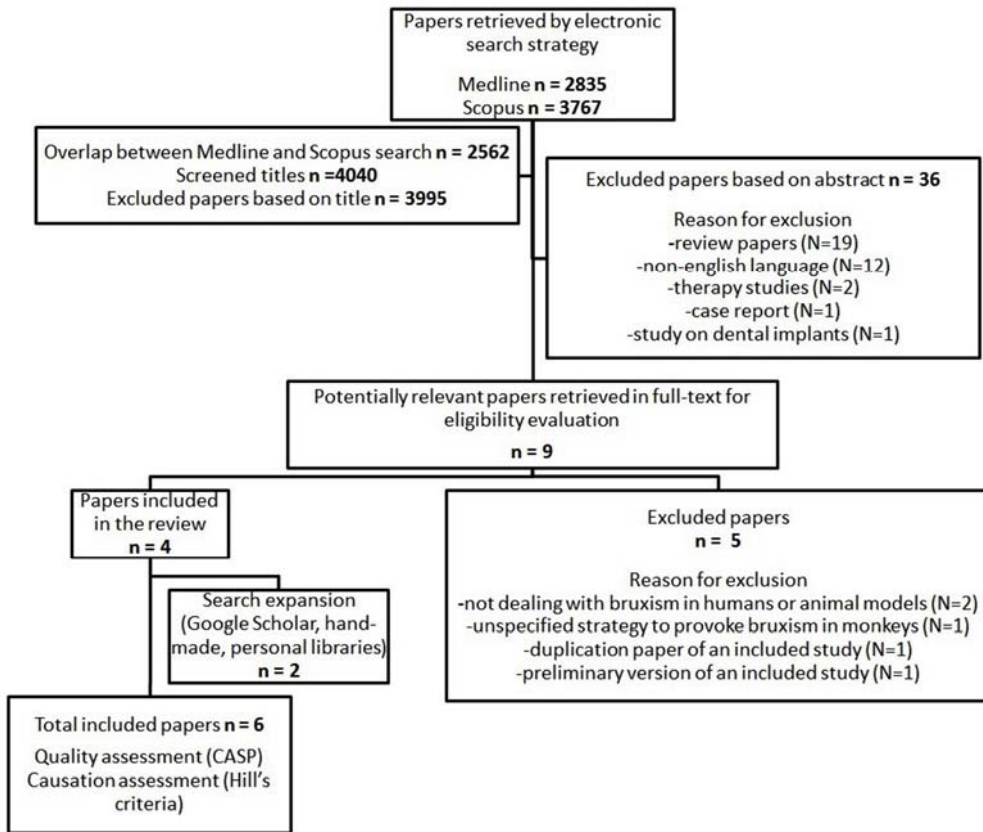
Table 5.

Assessment of validity of causation conclusions of the reviewed studies according to Hill's criteria.

Study first author, year	Main causation finding	Item #1	Item #2	Item #3	Item #4	Item #5	Item #6	Item #7	Item #8	Item #9	Total score for validity of causation conclusion
Calderon, 2009 ²⁹	Bruxism does not alter minimum interdental threshold	No (lack of internal validity)	n.a.	Yes	No (no longitudinal evaluation)	No (no longitudinal evaluation)	Yes	n.a.	No (not in line with other experiments)	No (different findings with respect to Ono's study)	2
Tokiwa, 2008 ²⁰	Grinding patterns involving the molar are associated with more periodontal problems	Yes	n.a.	Yes	No (no longitudinal evaluation)	No (no longitudinal evaluation)	Yes	n.a.	Yes (even if only experiment on the issue)	n.a.	4
Ono, 2008 ²¹	Bruxism reduces interocclusal tactile threshold	Yes	Yes	Yes	No (no longitudinal evaluation)	No (no longitudinal evaluation)	Yes	n.a.	Yes (even if findings are different from the study of Calderon et al)	Yes	5
Bernhardt, 2006 ²²	Bruxism is not associated with reduced CAL or increased PD	Yes	Yes	No (study of bruxism-perio relationship)	No (no longitudinal evaluation)	No (no longitudinal evaluation)	Yes	Yes	No (No experimental designs on the issue)	Yes	5

				p was not the primary aim)							
Martinez-Canut, 1997 ³⁰	Bruxism is not associated with pathological tooth migration	Yes	n.a.	No (unclear outcome variable)	No (no longitudinal evaluation)	No (no longitudinal evaluation)	Yes	n.a.	No (no experimental designs on the issue)	Yes	3
Hanamura, 1987 ²³	Bruxers have less perio problems than periodontal patients	Yes	n.a.	Yes	No (no longitudinal evaluation)	No (no longitudinal evaluation)	Yes	Yes	No (no experimental designs on the issue)	Yes	5

Legends: CAL, Clinical attachment level; PD, probing depth.



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