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Sleep Bruxism and Obstructive Sleep Apnea – Shared Pathophysiology or Coincidence?

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ABSTRACT

Introduction: Sleep bruxism (SB) is a jaw-muscle activity during sleep while obstructive sleep apnea (OSA) is a disorder of recurrent upper airway collapse during sleep [5, 1]. Recent research has noted frequent co-occurrence of SB in OSA patients.
Aim: This narrative review examines the relationship between SB and OSA to determine if SB and OSA share underlying mechanisms or simply coexist by chance.
Materials and Methods: A literature review was conducted using the databases such as Pubmed and Google Scholar.
State of knowledge: SB was prevalent in about one-third to one-half of OSA patients across studies [5, 6]. Mild-moderate OSA is associated with significantly higher SB activity than severe OSA [6, 12]. Both conditions share risk factors, and OSA itself has been identified as an independent risk factor for SB [6, 3]. Notably, treating OSA can lead to significant reductions in SB episode frequency [15, 11].
Conclusions: SB and OSA frequently co-occur and exhibit intertwined pathophysiology in certain patients. Awareness of this association is important: OSA may be one of the most frequent modifiable triggers of SB [6], and treating OSA can alleviate SB in those cases [11, 13]. An interdisciplinary approach involving dentists and sleep physicians is recommended to identify and manage coexisting SB and OSA [14].

Keywords: *sleep bruxism; obstructive sleep apnea; sleep arousal; rhythmic masticatory muscle activity*

Introduction

Bruxism is a repetitive jaw-muscle activity characterized by clenching or grinding of the teeth, and it may occur during wakefulness (awake bruxism) or sleep (sleep bruxism). Sleep bruxism (SB), the focus of this review, is reported in approximately 8–15% of adults [6] and is now understood as a sleep-related movement disorder with a multifactorial etiology involving complex multisystem processes [1, 2]. Rather than a mere oral habit, SB episodes are typically associated with transient autonomic and cortical arousals during sleep [1]. Obstructive sleep apnea (OSA), on the other hand, is a prevalent sleep-related breathing disorder characterized by repeated partial or complete blockage of the upper airway during sleep, leading to oxygen desaturation and fragmented sleep [5]. OSA affects roughly 9–38% of the adult population (with higher rates in older and male individuals) [5] and is well known to cause snoring, hypoxemic stress, and frequent brief arousals. Given that both OSA and SB

manifest during sleep and involve autonomic activation, it is perhaps not surprising that they can co-occur in the same patient.

Notably, multiple clinical reports over the past two decades have pointed to a frequent coexistence of SB in patients with OSA [5, 13]. Patients evaluated in sleep laboratories for OSA have often been observed to grind their teeth or clench their jaws during apneic events, and some studies have documented a temporal association between apnea termination (arousal) and SB rhythmic masticatory muscle activity [1, 2]. Such observations have led to the hypothesis that the two conditions might be pathophysiologically linked. One theory posits that SB events may be triggered by the micro-arousals and autonomic surges that terminate OSA episodes, perhaps as a reflex to reopen the airway via jaw movement [5, 6]. Another hypothesis is that SB and OSA share common predisposing factors or neural pathways (for example, imbalances in central neurotransmitter systems or abnormal airway anatomy) that cause them to arise in parallel [7]. If true, these scenarios would mean SB is at least partly a symptom or byproduct of OSA ("shared pathophysiology"). Alternatively, the co-occurrence might be coincidental – for instance, stress or lifestyle factors could independently contribute to both teeth grinding and poor upper airway tone, making SB and OSA appear together without a direct causal link. Distinguishing between these possibilities has important clinical implications: if SB is often a consequence of OSA, then treating the sleep apnea could reduce bruxism activity, whereas if it is coincidental, then management of one condition may not impact the other.

Despite growing interest, the relationship between SB and OSA remains controversial. Earlier epidemiologic studies yielded conflicting results – some finding that OSA patients did not significantly differ in bruxism frequency compared to controls [12], and others suggesting OSA is a significant risk factor for SB [3]. More recent investigations employing polysomnography (PSG) have strengthened the case for a connection, identifying distinct subsets of OSA patients with high SB activity [5]. At the same time, a comprehensive scoping review in 2022 concluded that in adults "it is not possible to confirm that there is a relationship between SB and OSA" given the heterogeneity of findings and generally modest associations [12]. This narrative review will therefore critically examine the evidence to date regarding SB and OSA. We review how frequently SB and OSA co-occur, what physiological interactions have been observed (or refuted), and discuss whether these point toward a shared pathophysiology or mere coincidence. The scope is limited to adult populations, although pediatric considerations are noted briefly. By synthesizing data from clinical studies and reviews, we aim to clarify under what circumstances SB and OSA are linked and provide guidance for clinicians encountering patients who may suffer from both conditions.

Current state of knowledge

1. Prevalence of Co-Occurrence and Risk Factors

SB prevalence in OSA patients: A consistent finding across many studies is that a substantial subset of OSA patients exhibit sleep bruxism. Using overnight polysomnography to detect rhythmic masticatory muscle activity (RMMA), Tan et al. (2019) reported that approximately one-third (33.3%) of adults with OSA had concomitant SB as defined by >4 bruxism episodes per hour [5]. Similarly, a large 2024 clinical study found an SB prevalence of 37.1% in an OSA patient cohort [14]. Higher percentages have been observed in some samples: Martynowicz et al. (2019) noted SB (RMMA-based) in 50% of their OSA patients [6]. The variability in reported prevalence (ranging roughly from one-third to one-half) likely reflects differences in SB detection methods and patient characteristics. Nonetheless, these figures are markedly above the ~8–13% SB prevalence estimated for the general adult population [6], suggesting that OSA patients are enriched for sleep bruxism compared to the norm.

Importantly, the co-occurrence rate appears to depend on OSA severity. Evidence indicates that SB is more common in mild to moderate OSA and may become less frequent in severe OSA. In the 110-patient PSG study by Martynowicz et al., the bruxism episode index (BEI) was significantly higher in patients with mild/moderate OSA (AHI <30) than in those with severe OSA (AHI ≥30) – 5.50±4.58 vs. 1.62±1.28 episodes/hour, $p<0.05$ [6]. They observed a positive correlation between AHI and BEI within the mild-moderate range, but no correlation in severe OSA, where overall SB activity was low [6]. In that study, only 35.3% of severe OSA patients had any SB, compared to ~63% of mild/moderate OSA patients [12]. This pattern suggests a non-linear association: bruxism may manifest as a compensatory response in less severe OSA but can be "overwhelmed" or suppressed in advanced OSA [6]. Severe OSA entails more profound oxygen desaturations and often blunted arousal responses; patients may rely on stronger respiratory-effort mechanisms to terminate apneas, rather than oromotor activity, thus showing fewer bruxism events [6]. In contrast, when airway obstructions are moderate, the body

might recruit arousal-related jaw movements (i.e. SB) to help reopen the airway [6]. This hypothesis aligns with a 2022 study by Smardz et al., which found that OSA patients whose apneas were predominantly positional (typically milder OSA occurring mainly when supine) had a significantly higher incidence of SB than those with non-positional or more severe OSA phenotypes ($p < 0.05$) [10]. Thus, OSA "phenotype" and severity are key factors influencing SB prevalence.

OSA prevalence in bruxism patients: Fewer studies have addressed the inverse question (how many SB patients have OSA), but available data also point toward an above-chance overlap. An exploratory genetic study of 100 patients (74 with SB, 28 with OSA, some overlap) by Wieckiewicz et al. (2020) noted that 28% of their SB group were diagnosed with OSA, though that sample was not epidemiological [7]. In a population-based context, the association is more equivocal. Maluly et al. (2020) investigated bruxism and OSA in a general population sample of São Paulo and found no statistically significant difference in OSA occurrence between self-reported bruxers and non-bruxers [8]. Specifically, in that large survey with clinical follow-up (n=620), about 39% of those identified as having SB (by questionnaire + ≥2 RMMA/hour on PSG) also had OSA, which was not far from the OSA prevalence in the overall sample [8]. This indicates that in an unselected population, having SB does not always imply OSA is present. The divergence from clinical samples could be due to age (community bruxers skew younger and leaner than typical OSA patients) and the fact that many population "bruxers" are mild cases detected by self-report, some of which may be false positives [6]. Nevertheless, the absolute co-occurrence rate in that study (39% of bruxers had OSA) is well above what pure chance would predict given general prevalence rates, hinting that a subset of bruxers do in fact have undiagnosed OSA [8].

Shared risk factors: SB and OSA share certain epidemiological risk factors, which might contribute to their co-occurrence. Foremost among these is male sex. OSA is far more common in men than women until about age 60, and some studies have also found higher SB prevalence or severity in men [6]. In the mild/moderate OSA group studied by Martynowicz et al., male gender emerged as an independent predictor of elevated bruxism episode index [6]. Another overlapping risk factor is age, though in opposite directions: bruxism is generally more prevalent in younger adults and declines with age, whereas OSA prevalence increases with age [5]. This age discordance might actually reduce the observable overlap in the general population (older OSA patients may have "aged out" of frequent bruxism) [6]. Indeed, it has been speculated that the lower bruxism seen in severe OSA could partly be an age effect, since severe OSA patients tend to be older and may have less active bruxism reflexes [6, 1].

Certain health conditions have been linked to both disorders. Obesity, a key driver of OSA, has not been strongly linked to primary bruxism in the literature, but type 2 diabetes and metabolic syndrome (often coexisting with OSA) may have connections to SB. Martynowicz et al. identified diabetes as a novel independent risk factor for higher bruxism activity in OSA patients [6]. They postulated that diabetic autonomic neuropathy (causing sympathetic overactivity and dry mouth) could promote bruxism, and indeed dry mouth (xerostomia) is a known issue in both diabetes and OSA. Another condition of interest is gastroesophageal reflux disease (GERD), which has been associated with both OSA and SB. Reflux episodes at night can trigger arousals and jaw movements; chronic reflux was reported as a risk factor for bruxism in some studies [6], and OSA-related physiology (negative intrathoracic pressures) promotes reflux. Similarly, anxiety and stress levels tend to be higher in bruxism patients, and untreated OSA itself can heighten sympathetic stress responses and insomnia, potentially leading to awake bruxism or exaggerating SB. A 2018 comprehensive review of bruxism risk factors by Kuhn & Türp concluded that, among numerous variables, "sleep apnea syndrome" was a significant risk factor for bruxism in adults (pooled odds ratio ~4.0, 95% CI 1.0–15.2) [3]. In that review, OSA ranked alongside alcohol, tobacco, caffeine, and psychological stress as important factors contributing to bruxism [3]. This epidemiological linkage supports the notion that OSA and SB do not cluster purely by coincidence; rather, OSA may predispose individuals to bruxism or vice versa (or a third factor predisposes to both).

2. Sleep Physiology and Pathophysiological Links

Sleep stage and arousal pattern: One of the most striking commonalities between SB and OSA is their relationship with sleep micro-arousals. SB episodes typically occur during light sleep (NREM stage 2) or during transitions between deep and lighter sleep, often in tandem with brief cortical arousals [1, 2]. OSA events by definition terminate in arousals when breathing restarts. Research has shown that up to 80% of SB episodes are associated with transient arousals – characterized by a shift to shallower sleep and a burst of alpha EEG activity – and often occur in a cluster following the cyclic alternating pattern of arousal fluctuation in NREM sleep [1]. De la Hoz et al. (2011) described the typical sequence leading to an SB event: a gradual rise in sympathetic cardiac activity ~8 minutes before, a marked increase in EEG frequency ~4 seconds before, then a sharp heart rate acceleration and often an increase in breathing effort ~1 second before, followed by activation of jaw-

opening muscles and finally the phasic burst of jaw-closing (tooth grinding) activity [1]. Notably, they point out an "increase in respiratory and cardiac frequency" just prior to the bruxism burst [1], highlighting a potential role of breathing changes.

In OSA, a very similar arousal sequence is observed at apnea termination: sympathetic output and blood pressure surge as the brain transitions briefly to lighter sleep, heart rate rises, and often a recovery breath (deep inhalation) occurs with activation of airway-opening muscles. It is during this recovery/arousal phase that SB events are often noted. Kato et al.(as cited by Klasser et al. 2015) found that most SB episodes occur immediately following respiratory events or snores, suggesting SB could be part of a complex arousal reaction to airway compromise [2]. In support of this, SB episodes have been found to coincide with elevations in inspiratory effort and airflow. For example, one summary noted that "RMMA tends to occur with large breaths, and oral appliances used to improve airway patency help to reduce SB-RMMA frequency" [2]. The role of respiration is further hinted by the fact that masseter EMG bursts in SB are often preceded (0.8 s prior) by activation of suprahyoid (jaw-opening) muscles [1], which likely serve to open the airway or reposition the jaw forward [2]. This temporal choreography strongly suggests a coordinated reflex: as the brain senses suffocation and micro-arousal ensues, it triggers autonomic and motor responses including deep breathing and jaw movements, with the latter manifesting as SB when the reflex overshoots into teeth clenching/grinding.

Evidence of airway-protective reflex: Several studies support the concept that SB in OSA may be an airway-protective mechanism. The 2019 study by Tan et al. provides direct clinical evidence: they found OSA patients with bruxism had significantly more respiratory-related arousals (respiratory arousal index, RAI) and more oxygen desaturations (ODI) than OSA patients without bruxism [5]. The SB+OSA group's average RAI was notably higher (meaning a greater tendency to arouse in response to breathing events) [5]. Logistic regression in that study showed that the RAI was positively associated with SB (odds ratio ~1.05 per arousal) while the spontaneous (non-respiratory) arousal index was slightly protective [5]. These findings imply that OSA patients who are prone to arousals from respiratory disturbances are also the ones manifesting SB – consistent with SB being part of the arousal response to respiratory stress. Furthermore, Tan et al. observed that most bruxism in OSA was phasic SB (rhythmic bursts) rather than tonic clenching [5], which aligns with the phasic RMMA nature of arousal-linked bruxism rather than sustained clenching. They concluded that a phenotypic subtype of OSA patients with predominantly phasic SB exists, alluding to a possible protective role of these jaw movements during sleep-disordered breathing [5]. In essence, these patients' sleep bruxism might be an unconscious strategy to alleviate airway obstruction – for instance by protruding the jaw or increasing upper airway muscle tone, as has been hypothesized in other works [6].

Martynowicz et al. also found physiological links between OSA severity and bruxism: in their study, the degree of nocturnal hypoxemia correlated with bruxism indices. Specifically, the oxygen desaturation index (ODI) and minimum O_2 saturation showed a significant correlation with phasic SB frequency (more desaturations linked to more bruxism) [6]. Transient hypoxia itself is thought to possibly trigger bruxism episodes [6]. This aligns with the notion that as an apnea worsens and blood O_2 drops, the ensuing arousal and stress response might precipitate a bruxism event.

However, it must be noted that association is not proof of intent – just because SB often occurs when OSA events end does not necessarily mean it is functionally "opening the airway." It could be an epiphenomenon; for example, a brainstem arousal oscillator could activate both the tongue/airway muscles and jaw muscles in parallel without one causing the other [1]. SB is also seen in other contexts of arousal, such as in some parasomnias and even with acid reflux events, as de la Hoz et al. pointed out [1]. Thus, while SB might assist airway patency in OSA, it might also simply be a byproduct of the central arousal circuits firing.

Neurochemical and genetic factors: Intriguingly, emerging research suggests there may be genetic underpinnings linking SB and OSA, indicating a shared pathophysiological basis at the neurotransmitter level. The 2020 genetic association study by Wieckiewicz et al. examined polymorphisms in serotonin and dopamine pathway genes in patients with SB and OSA [7]. They found that a variant in the dopamine D1 receptor gene (DRD1 rs686 G allele) was overrepresented in their combined SB/OSA patient group compared to healthy controls [7], suggesting a predisposition to one or both conditions via dopaminergic pathways. More tellingly, they discovered that in individuals homozygous for a certain serotonin 2A receptor gene variant (HTR2A rs2770304 TT), there was a significant positive correlation between the bruxism episode index and the apnea–hypopnea index [7]. In other words, among those with that serotonin receptor genotype, the severity of SB tracked with the severity of OSA. This implies a common modulator – likely serotonin signaling – that can influence both breathing stability and motor arousal phenomena. Serotonergic neurons in the brainstem are indeed involved in both airway muscle tone regulation and the modulation of rhythmic jaw movements. The authors concluded that

these genetic results "might contribute to the association between SB and OSA", pointing to a possible genetic predisposition for a combined SB–OSA phenotype [7]. While this is a single study and needs replication, it provides a fascinating hint that SB and OSA could share some "brain wiring" or neurochemical pathways. If certain people are genetically wired to have a low arousal threshold or exaggerated arousal responses (serotonergic mechanisms), they might be prone to both apneas (due to low airway muscle tone during sleep) and bruxism (as an exaggerated motor response on arousal) [7].

Additionally, both OSA and SB are associated with activation of the sympathetic nervous system. SB episodes are preceded by increased sympathetic activity (e.g., tachycardia and blood pressure spikes) [1], and OSA is characterized by chronic sympathetic overactivation from nightly hypoxia. It has been suggested that individuals with inherently high sympathetic reactivity might experience both conditions – the sympathetic surges in OSA might facilitate SB, and conversely the physiological stress of SB (if frequent) might contribute to cardiovascular strain seen in OSA patients [6]. Indeed, one study noted that hypertension and cardiovascular comorbidities, common in OSA, were also present in many SB patients (though whether due to OSA or SB or both is hard to dissect) [6].

Overlapping conditions: Conditions that are known to provoke arousals can provoke both OSA and SB. For instance, as mentioned, GERD can cause nocturnal arousals with coughing or choking that mimic OSA events and can also trigger SB episodes as a reflex (possibly via discomfort or acid in the esophagus) [4]. The narrative overview by Wetselaar et al. noted that OSA, oral dryness (mouth breathing), GERD, and sleep bruxism are all inter-associated conditions – each can influence the others [4]. They concluded that the "dental sleep disorders" (like SB, orofacial pain, xerostomia) are interlinked with OSA such that their consequences are difficult to disentangle [4]. For example, OSA causes mouth breathing and dry mouth, which could exacerbate bruxism (as saliva lubricates and the body might jaw-move to stimulate saliva) [1]. Conversely, heavy bruxism could theoretically contribute to orofacial anatomical changes (e.g. inflammation or temporomandibular joint issues) that disturb sleep or breathing. However, current evidence leans more towards OSA influencing SB than the reverse. The presence of temporomandibular disorders (TMD) pain in OSA patients has been observed; a recent follow-up study found moderate/severe OSA was associated with increased orofacial pain and higher tooth wear scores over time [13]. This suggests untreated OSA can aggravate jaw muscle pain and tooth grinding damage, again implying a causal path from OSA to bruxism (and related TMD symptoms). It also reinforces that SB in OSA is not purely an "oral habit" but part of a systemic condition, as argued by Sambale et al., who found OSA patients with bruxism had more jaw muscle tenderness and limitations than those without bruxism [14].

Despite these links, it is crucial to mention that not all studies find a strong physiological coupling of SB and OSA. Some polysomnographic works have failed to show temporal association in every case. For instance, one frequently cited early study (Okeson et al. 1991) reported no significant difference in number of bruxism events between OSA patients and controls overnight [12]. They noted OSA patients had plenty of arousals but did not necessarily brux more than those without OSA. It is possible that only certain arousals (above a certain intensity or specific type) trigger bruxism. Moreover, many methodological factors (e.g. what cutoff is used to score a bruxism event, whether a concurrent leg movement might mask or coincide with a bruxism event, etc.) can influence detection. The 2022 scoping review by Pauletto et al. highlighted "marked methodological variability" among studies [12]. Some studies relied on self-reported grinding noises or tooth wear examinations to identify SB, which are far less reliable than masseter EMG in PSG [6]. Self-report likely underestimates true SB or misclassifies awake bruxism as sleep bruxism, diluting observed associations with OSA. Indeed, Pauletto's review noted that most adult studies did not find a statistically significant association between SB and OSA unless detailed polysomnography was used [12]. They found a possible association in children (26% of pediatric OSA patients had SB, which might be related to tonsillar hypertrophy causing both issues), but in adults, the evidence was insufficient to confirm a consistent link [12]. Thus, while clear overlaps in physiology exist, the presence of confounding factors and heterogeneity in study designs mean that SB is not an inevitable consequence of OSA in all patients – other modulating factors (like arousal threshold, anatomy, age, etc.) determine whether an OSA event will culminate in a bruxism event.

3. Impact of Interventions and Clinical Observations

If SB and OSA are pathophysiologically connected, treating one condition might influence the other. Several intervention studies, albeit mostly small, have explored this possibility, especially focusing on whether OSA therapy reduces bruxism. The results generally indicate that addressing OSA can significantly reduce SB activity in patients who have both.

Mandibular advancement devices (MADs): One conservative OSA treatment is a custom oral appliance that holds the jaw forward during sleep to prevent airway collapse. Interestingly, such devices might also mechanically affect bruxism (by altering occlusion or jaw position). Wojda et al. (2022) conducted a prospective trial on 8 patients diagnosed with both OSA and SB to test the effect of a mandibular advancement device [11]. After one week of MAD usage during sleep, follow-up polysomnography showed that, on average, there was a favorable decrease in OSA severity (significant reduction in AHI and oxygen desaturation index) along with a decrease in bruxism episode frequency [11]. Notably, the number of phasic SB episodes per hour dropped significantly with the MAD (this was the only SB metric with statistical significance, whereas total bruxism index decreased non-significantly) [11]. This suggests that when the airway was mechanically stented open (thus reducing apneas and arousals), the reflex phasic bruxism events also diminished. In other words, improved airway patency led to less bruxism, supporting a causal relationship whereby OSA was triggering SB. The authors concluded that treating OSA with MADs "has a beneficial effect on manifestations of SB, even though only the number of phasic bruxism episodes was statistically significantly reduced" [11]. This partial improvement might be because phasic SB is the type closely tied to arousals, whereas tonic bruxism (sustained clenching) might be influenced by other factors (e.g. stress) not addressed by MAD. Nonetheless, this finding is clinically meaningful – in these patients, an oral appliance intended for OSA served dual-duty by also mitigating sleep bruxism.

Respiratory muscle training: A novel randomized controlled trial by Cavalcante-Leão et al. (2024) tested whether improving a patient's own respiratory muscle function could impact both OSA and SB [15]. They enrolled patients with concurrent SB and mild OSA and had one group perform intensive inspiratory and expiratory muscle exercises (using Threshold IMT and PEP devices) for 12 weeks, versus a placebo exercise group [15]. The results were promising: the treatment group showed significant improvement in OSA metrics – notably, a reduction in the frequency of awakenings (suggesting better sleep continuity, $p \leq 0.05$) [15]. Crucially, the number of masseter muscle contractions (a proxy for SB events) dropped by 67% in the inspiratory-training group compared to placebo [15]. In 80% of the treated patients, awakening levels (arousal measures) improved alongside that bruxism reduction [15]. The authors concluded that strengthening the breathing muscles (and thus mitigating OSA events) "improved awaking levels and [reduced] the number of masseter contractions" relative to sham therapy [15]. This RCT provides high-level evidence that targeting the OSA aspect can indeed ameliorate SB. It's notable that even without an oral appliance or CPAP, just physiotherapy to enhance airway patency during sleep was enough to significantly quiet the bruxism. This underscores the role of breathing-related arousals in triggering SB: make breathing easier, and the brain no longer fires off the sequence culminating in grinding.

Bruxism treatments and OSA: What about the reverse direction – does treating bruxism influence OSA? There is less data on this, but it is generally thought that typical bruxism treatments (like tooth guards or stress reduction) do not worsen or improve OSA substantially, with one exception: certain oral splints. A full-coverage occlusal splint (night guard) that dentists often prescribe for bruxism could theoretically alter jaw position. Some reports have warned that a traditional flat-plane splint might slightly retroposition the mandible and potentially exacerbate OSA in susceptible individuals [2]. On the other hand, a well-titrated mandibular advancement splint, as used in OSA therapy, can both protect the teeth from grinding and improve the airway – essentially serving as a combined therapy. One included study, Ning et al. (2023), followed OSA patients over 6 months and noted that after OSA treatment (which in that cohort could include CPAP, weight loss, or surgery), tooth wear scores did not increase further [13], whereas in the untreated interval, patients with moderate/severe OSA had shown progressive tooth wear and orofacial pain [13]. This suggests that effective OSA management halted the degradation of dentition likely caused by SB, again pointing to OSA as a driver of the bruxism/tooth wear. In that study, moderate-to-severe OSA was found to significantly aggravate tooth wear and orofacial pain compared to mild OSA [13] – essentially an "exposure–outcome" relationship where heavier OSA burden led to more bruxism effects (wear facets, jaw pain). Treating the OSA (the exposure) was beneficial in reducing those oral consequences.

It is worth noting that no specific medication is proven for primary SB, but certain drugs (like SSRIs or stimulants) can modulate SB frequency [9]. If a patient's SB were truly secondary to OSA arousals, one would expect that merely suppressing muscle activity (e.g. with muscle relaxants or anxiolytics) might not fully eliminate SB unless the OSA is also addressed. Conversely, if one treated OSA but not the bruxism, as we saw, SB tends to diminish spontaneously. This asymmetric influence bolsters the argument that in SB–OSA concurrence, OSA is often the upstream condition.

Clinical identification and subtype: Several authors have begun to describe a clinical phenotype of patients in whom SB and OSA overlap. These tend to be middle-aged males who are perhaps not morbidly obese (if they

were, they might develop pure OSA without bruxism) but have moderate OSA, and they often complain of snoring, witnessed apneas and signs of bruxism (such as tooth grinding noises, morning jaw fatigue, or tooth wear) [5, 13]. In Sambale et al.'s OSA cohort, those with probable bruxism had higher incidence of jaw-muscle pain and dysfunction on awakening [14]. These patients might be the ones to particularly benefit from integrated treatment. Tan et al. referred to "OSA patients with predominantly phasic SB" as a subgroup [5]. From a diagnostic perspective, their work implies that polysomnographic monitoring of jaw EMG in OSA work-ups can reveal this phenotype, and dentists seeing unexplained severe tooth wear or TMD pain might consider undiagnosed OSA as an underlying cause. Indeed, given that OSA is vastly underdiagnosed, the presence of significant SB (especially with snoring or daytime sleepiness) could be a clue to screen for OSA. An interdisciplinary screening approach is supported by findings like those of Kuhn & Türp, where OSA was identified as a significant risk factor for bruxism [3] – they suggest including questions about sleep breathing in the history of bruxism patients [3]. Conversely, sleep physicians managing OSA patients may enquire about bruxism symptoms because coexisting SB could exacerbate morning symptoms (headaches, jaw pain) and because treating the OSA might alleviate those.

Controversies in outcomes: Despite the positive intervention studies mentioned, not every attempt to treat one condition will automatically fix the other. Some patients with SB might continue to brux out of habit or stress even after their apneas are resolved. And not every OSA patient with a mouthguard stops breathing – in fact, an improperly fitted dental splint could worsen apnea. The complexity of each condition means that individualized assessment is important. The general trend, however, is that when SB and OSA truly coexist (and are not just random), they behave as "communicating vessels" – relief of airway obstruction alleviates bruxism, and reduction of bruxism's harmful effects (via splints or therapy) improves patient comfort and may marginally aid sleep quality, though it won't cure OSA on its own.

The 2022 scoping review provides a cautious viewpoint: after reviewing 21 studies, the authors concluded that in adults there is still insufficient evidence to confirm a clear relationship [12]. They emphasized that many adult studies found no significant association, which might be due to methodological issuesas discussed (e.g. using self-reported bruxism introduces noise) [6]. They did find the association "plausible" in children [12], where enlarged tonsils/adenoids or allergies might simultaneously cause snoring and bruxism (children often show bruxism when uncomfortable during sleep). However, pediatric mechanisms might differ from adults (for example, children's bruxism may relate more to dental development or parasomnias). Thus, our focus on adult SB–OSA might not generalize to pediatrics, and vice versa.

In summary of results: the bulk of evidence indicates that SB and OSA often share a temporal and physiological connection mediated by sleep arousal mechanisms. A subset of patients display a clear coupling: OSA events trigger SB, and treating OSA dampens SB. In these cases, one could argue for a shared pathophysiology (common reflex pathways or risk factors). Yet, there are also individuals in whom the two conditions do not coincide despite expectations – reminding us that SB has multifactorial causes (including psychosocial stress, occlusal factors, etc.) and OSA has other manifestations (some patients' arousals manifest as limb movements or just EEG awakenings, not bruxism). Therefore, the relationship can be described as heterogeneous: sometimes causal, sometimes coincidental.

Discussion

The interplay between sleep bruxism and obstructive sleep apnea emerges from the results as a complex, yet significant, phenomenon. The evidence reviewed suggests that the co-occurrence of SB and OSA is more than chance in a considerable proportion of cases, pointing toward shared pathophysiological mechanisms – but this connection has important limits and conditions. We discuss here the degree of overlap, the hypothesized mechanisms linking the two disorders, the clinical implications of these findings, and the extent to which the current evidence answers the question of "shared pathophysiology or coincidence."

Shared pathophysiology – supporting arguments: Several key findings argue in favor of a shared mechanism linking SB and OSA in certain patients. First, the temporal association is compelling: SB episodes often happen at the moment an OSA-induced arousal occurs [1, 5]. This timing suggests SB could be part of the body's natural rescue response to airway obstruction. The jaw-opening and subsequent clenching movements in SB may help reposition the mandible and tongue, slightly enlarging the airway and allowing airflow – essentially functioning as a built-in "oral appliance." This hypothesis is reinforced by the protective effects observed: mild-moderate OSA patients (who can still generate arousals and SB) seem to maintain oxygenation better than severe OSA patients in some reports, possibly thanks to these arousal responses [6]. Tan et al.'s identification of an SB-prone OSA subtype with more respiratory arousals and desaturations hints that SB could be a marker of an active

arousal-defensive phenotype [5]. Rather than being a random occurrence, SB in these cases is intertwined with the pathogenesis of OSA episodes: the airway narrows, the brainstem triggers an arousal; simultaneously or milliseconds later, it triggers a rhythmic jaw movement (SB) as part of the arousal cascade. In this view, SB and OSA are two outputs of the same transient arousal event – a clear example of shared pathophysiology.

Second, autonomic nervous system activation is a common denominator. Both SB and OSA are characterized by bursts of sympathetic activity. It is plausible that certain individuals have an exaggerated autonomic reflex to airway obstruction that includes not just increased heart rate and blood pressure (which all OSA patients have on arousal) but also activation of the trigeminal motor system (manifested as bruxism). This could be due to central connectivity between respiratory and masticatory neural centers. Animal studies have shown that the brainstem chewing CPG (central pattern generator) can be influenced by reticular activating systems that are also involved in respiratory arousals [1]. Thus, a strong arousal might co-activate these networks. The consistent sequence – autonomic surge → arousal (EEG) → masseter EMG burst [1]– supports a cause-effect chain. It's telling that phasic SB (which is closely linked to arousal) is the type increased in OSA, whereas tonic bruxism (which might be more stress/ anxiety related) is not particularly tied to apnea events [5]. This specificity strengthens the argument for a physiologic link.

Third, the genetic and biochemical links provide a mechanistic bridge. The discovery that a serotonin receptor polymorphism correlates with both SB and OSA severity [7] suggests that the central serotonergic modulation of arousal and muscle tone could underlie both conditions concurrently. Serotonin plays a dual role in sleep: it stabilizes breathing (serotoninergic neurons help keep upper airway dilator muscles active) and modulates motor outputs during sleep (some bruxism treatments attempt serotonin modulation). If certain genotypes lead to low serotonin receptor function, one might have both a collapsible airway (predisposing to OSA) and an unstable arousal threshold (predisposing to bruxism as a motor manifestation of arousal). Dopamine, meanwhile, is implicated in the motor aspect of bruxism (some bruxers respond to dopaminergic drugs), and interestingly dopamine also interacts with ventilatory control. The shared genetic markers hint that we are not dealing with two completely separate disorders randomly colliding, but rather overlapping syndromes with some common upstream causes.

Fourth, common risk factors and comorbidities like male sex, GERD, and sympathetic hyperactivity conditions (e.g. hypertension, diabetes) suggest a clustering that is not random. If OSA and SB were entirely independent, one wouldn't expect them to consistently share these associations beyond what general poor sleep might cause. Yet, multiple sources identify OSA as a risk indicator for SB [3] and vice versa (e.g. bruxism noise is more often reported by partners of heavy snorers). The "Association Rule" mining in epidemiology places OSA among the factors linked to bruxism with a substantial effect size [3]. The fact that treating OSA results in decreased bruxism (MAD, exercise, likely CPAP) shows that the pathophysiology of some patients' bruxism is directly tied to their OSA: remove the stimulus (airway collapse), and the outcome (SB) diminishes. This therapeutic evidence is among the strongest arguments for a causal relationship, since it is effectively an experimental manipulation. In our review, two separate interventions – mandibular advancement [11] and muscle training [15] – each significantly reduced SB activity along with improving OSA. It is unlikely that such parallel improvement is coincidental; rather, it indicates a common mechanism (improved airway leads to fewer arousals, hence less bruxism).

Coincidence – counterpoints: On the other hand, not all instances of SB and OSA are connected, and several points illustrate that their association can be coincidental or indirect. One major counterpoint is the existence of many OSA patients who do not exhibit bruxism and many bruxers who do not have OSA. OSA has a high prevalence, and SB is not uncommon, so some overlap is expected by chance alone. The population study by Maluly et al. found that while there was some overlap, statistically OSA wasn't significantly more frequent in bruxers than in non-bruxers in the community [12]. This suggests that outside of specialized clinic samples, the two conditions can manifest independently. In such cases, a third factor (like stress, alcohol use, or reflux) might independently contribute to each. For example, high stress can cause bruxism and also fragment sleep, possibly worsening mild OSA – here the link is not a direct physiological one, but rather mediated by an external factor.

Another consideration is that SB has multiple etiologies. While some SB is arousal-driven (primary SB), other cases might be predominantly centrally driven or peripheral. For instance, malocclusion or dental factors are largely discounted now as primary causes, but certain peripheral sensory inputs (tooth interference, etc.) might play a minor role in triggering some bruxism episodes [1]. More importantly, psychological factors like anxiety, stress, or sleep disturbances (insomnia) can trigger SB even in the absence of OSA. Such SB would appear in someone who might also have poor sleep but not necessarily apnea. In epidemiology, bruxism correlates with poor sleep quality and insomnia symptoms [12]. It's conceivable that a person with insomnia might develop both

clenching habits (due to tension) and even pseudo-apneas (breath holding or upper airway resistance unrelated to classic OSA). Distinguishing true OSA-related SB from stress-related SB can be challenging without full sleep studies.

Moreover, the absence of correlation in severe OSA might indicate that bruxism requires a certain physiological window to occur. If apneas are very prolonged and oxygen drops severely, the brain may prioritize gasping and waking up fully rather than executing a chewing movement. In these cases, the patient might have OSA without any bruxism, not because they are unrelated, but because the OSA is so severe it "suppresses" the bruxism response (as Martynowicz et al. suggest, bruxism has a limited protective role that is insufficient in severe OSA) [6]. This creates a selection bias in some clinical studies: if one enrolls mostly severe OSA patients (e.g. at a tertiary sleep center), one might conclude SB and OSA aren't associated, whereas in a moderate-OSA sample one finds a correlation. Pauletto's scoping review noted exactly this potential issue – studies with different patient severity mixes reported different outcomes [6]. Thus, coincidence might be concluded in some studies simply because of how the sample was composed or how SB was measured. When rigorous PSG criteria are applied, subtle associations that were missed by questionnaire become apparent (like phasic bursts tied to apneas).

Diagnostic and clinical implications: From a clinical perspective, acknowledging a shared pathophysiology in many SB–OSA cases has practical benefits. Patients with severe tooth wear, TMJ pain, and morning headaches (classic bruxism sequelae) who also snore or are tired should be evaluated for OSA [13, 14]. This is important because treating OSA might kill two birds with one stone: improve the life-threatening aspect (apnea) and also reduce further dental damage. Conversely, dentists should be aware that simply giving a night guard to a bruxism patient who has undiagnosed OSA will protect the teeth but do nothing for the apnea – in fact, if it's a mandibular-repositioning splint, it could help the apnea, but if it's a flat relaxer splint, it might slightly worsen airway patency. Therefore, some experts recommend screening OSA patients for SB and vice versa. Our findings support this bidirectional screening. Sambale et al. conclude that bruxism in OSA means it's "not only an oral health problem" and urge interdisciplinary management [14]. In practice, this could mean sleep physicians collaborating with dentists: e.g., an OSA patient with bruxism might benefit from a combined oral appliance therapy that advances the jaw (to treat OSA) and has a surface that mitigates grinding forces. Likewise, a refractory TMD pain patient should be asked about snoring and apneas, as treating an occult OSA could relieve the nocturnal clenching contributing to their pain [14]. This kind of holistic approach stems from recognizing shared pathophysiology.

Resolving the question: So, are SB and OSA linked by shared pathophysiology or coincidence? The weight of evidence from our review leans toward shared pathophysiology in a subset of patients. It is not an all-or-nothing answer. In many patients (particularly those with moderate OSA and good arousal responses), SB appears as a secondary manifestation of OSA – a consequence of the nightly struggle to breathe. In these cases, the pathophysiological driver is OSA and SB shares in that pathway (hence, treating the driver fixes both). In other patients, SB might be "primary" and unrelated to OSA, or OSA might be so severe (or patient factors such as age so advanced) that the linkage doesn't manifest. Therefore, the association could be termed conditional or phenotype-specific. The concept of a "phenotypic subtype" of OSA with bruxism [5] is useful: it implies that not every OSA patient bruxes, only those with certain traits (perhaps a lower arousal threshold or particular genetic makeup). Similarly, among bruxism patients, those who brux largely due to autonomic arousals (as opposed to, say, habit during REM or stress in light sleep) are the ones who might have OSA triggers.

Controversies and future directions: A point of contention is quantifying how often SB is OSA-induced versus independent. Our review suggests possibly about one-third of OSA patients and perhaps a smaller fraction of bruxism patients have a true linkage. Future large-scale studies with simultaneous PSG scoring of both conditions are needed to refine these numbers. Moreover, investigations into neurophysiology (for example, measuring brain activity during apnea-related bruxism vs. non-apnea bruxism) could definitively show if the same neural patterns are involved. If shared pathophysiology is confirmed, one might even consider SB as a clinical indicator for a certain type of OSA (like an arousal-driven OSA, as opposed to a high-threshold OSA). Conversely, the absence of SB in a sleepy, heavy snorer might indicate a different phenotype (perhaps one with blunted arousal responses and higher risk of oxygen desaturation). Thus, bruxism could become part of OSA phenotyping – a concept hinted at by Smardz et al.'s phenotype study [10].

From a therapeutic angle, one intriguing possibility is leveraging the SB–OSA connection: for instance, could inducing slight jaw movements or maintaining a certain jaw position during sleep reduce OSA events? One might imagine a feedback device that stimulates jaw muscles when airway resistance increases, mimicking the natural SB response but in a controlled way. Conversely, will long-term use of OSA oral appliances

inadvertently reduce the brain's tendency to brux (since the airway is kept open, perhaps the brain "unlearns" the reflex)? These questions remain open.

In summary, our discussion finds that shared pathophysiology best describes the SB–OSA relationship in a meaningful subset of patients, evidenced by synchronous occurrence, common triggers, and reciprocal treatment responses. However, considerable individual variability exists, meaning the association can appear coincidental in others. Clinicians should thus neither outright dismiss SB in OSA as coincidence (and risk missing an integrative treatment opportunity) nor assume every bruxer has OSA. Instead, a prudent approach is to maintain a high index of suspicion for overlap and manage patients in a multidisciplinary fashion when co-occurrence is suspected. This balanced view acknowledges the growing evidence of linkage while respecting the complexities and other contributors to each condition.

Conclusion

Sleep bruxism and obstructive sleep apnea are two sleep-related phenomena that often intersect, revealing an intriguing blend of dentistry and sleep medicine. Based on the current body of evidence, it can be concluded that sleep bruxism and OSA share pathophysiological mechanisms in a substantial subset of patients, rather than their co-occurrence being purely coincidental. The key points supporting this conclusion include:

- Frequent Co-Occurrence: SB is considerably more prevalent among OSA patients than in the general population, particularly in those with mild to moderate OSA [6, 12]. More than one-third of OSA patients exhibit clinically significant bruxism activity during sleep in many studies [5, 14]. Conversely, OSA appears as a notable risk factor in adults with SB [3]. This epidemiological overlap exceeds what chance alone would predict.

- Shared Arousal-Mediated Pathways: The temporal and physiological link between apneic arousals and bruxism episodes strongly indicates a common pathway [1, 5]. Both conditions are characterized by sleep fragmentation and surges of autonomic activity. Bruxism episodes in OSA typically occur immediately following airway obstructions, as part of the arousal sequence to restore breathing [1, 5]. This suggests that SB in these cases is an integral component of the sleep-disordered breathing arousal response, potentially serving a protective role in reopening the airway [6, 5].

- Mutual Risk Factors and Genetic Links: OSA and SB share common risk factors such as male sex and certain comorbidities (e.g. GERD, diabetes) [3, 6]. Emerging genetic evidence (e.g. specific serotonin receptor polymorphisms) further supports a biologically intertwined etiology for some individuals [7]. These findings point to overlapping central neural mechanisms (involving neurotransmitters regulating both muscle activity and airway patency) that predispose individuals to both disorders.

- Reciprocal Impact of Treatments: Perhaps the most compelling evidence for a cause-effect relationship is that treating OSA can significantly reduce SB. Mandibular advancement therapy for OSA leads to a reduction in bruxism episode frequency [11], and targeted respiratory therapy has been shown to decrease nocturnal masseter muscle activity by two-thirds alongside improving apnea outcomes [15]. This improvement in SB with OSA treatment underscores that, at least in those patients, the SB was secondary to OSA-related factors. On the other hand, approaching SB in isolation (e.g. with a night guard alone) does not address the root cause if OSA is present, reaffirming that unrecognized OSA can drive bruxism.

At the same time, this review highlights that the SB–OSA relationship is not universal. In some patients, the two conditions may coexist independently. Severe OSA patients may have diminished bruxism due to blunted arousal responses [6], and many primary bruxism cases are unrelated to airway issues (stemming instead from stress, etc.). Therefore, the presence or absence of one condition does not guarantee the other. Clinicians should adopt a case-by-case, interdisciplinary approach. Specifically, dentists and sleep physicians are encouraged to collaborate: patients with sleep apnea should be evaluated for signs of bruxism (to prevent dental complications), and patients with significant bruxism (especially with snoring or daytime sleepiness) should be screened for occult sleep apnea [3, 14]. Such collaborative management is crucial because, as this review demonstrates, sleep bruxism in many OSA patients is not only an oral health issue but part of a systemic sleep disorder [14].

In conclusion, the evidence favors a model in which sleep bruxism and obstructive sleep apnea are often linked by shared pathophysiological threads – chiefly the mechanisms of sleep arousal and autonomic activation –

rather than being merely coincidental. This recognition carries important implications: addressing OSA can alleviate bruxism-related morbidity, and noting bruxism can aid in identifying sleep apnea. Future research should continue to unravel the neurophysiological underpinnings of this relationship and identify which patients are most at risk for combined SB–OSA pathology. In doing so, we will improve holistic care for those suffering from either or both of these conditions. An interdisciplinary mindset, combining the expertise of sleep medicine and dentistry, is the optimal path forward for effective diagnosis and management, ensuring that these patients receive comprehensive treatment for both a good night's sleep and a healthy masticatory system.

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CONFLICT OF INTEREST

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