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Prevalence and associated factors of sleep bruxism in Turkish schoolchildren with mixed dentition: a cross-sectional study

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ABSTRACT

Background: Factors associated with sleep bruxism (SB) in children with mixed dentition remain incompletely understood. This study examined the relationship between sleep bruxism and sleep behaviors, oral habits, temporomandibular disorders (TMD), and health status in Turkish schoolchildren during the mixed dentition stage.

Methods: In this cross-sectional study, a representative sample of 452 schoolchildren was assessed. Sleep bruxism was assessed by a subjective-based assessment (parental report of tooth grinding during sleep). During the clinical oral examination, tooth wear, free-way space, maximum mouth opening, and signs of TMD were recorded. Parents or caregivers completed a questionnaire on sociodemographic factors, general health, sleep behavior, oral habits, and recent life changes. Associations were analyzed using the χ^2 or Fisher's exact test, the Mann–Whitney U test, and logistic regression, with effect sizes (Cramér's V, odds ratios, and r).

Results: Sleep bruxism was identified in 22.6% of the 452 children, with no significant difference between genders ($p = .077$). Regarding health status, SB was more common among children with allergic rhinitis ($p < .001$; OR = 2.42, 95% CI 1.50–3.91). Regarding TMJ/jaw function and oral habits, SB was associated with head/jaw trauma, difficulty opening the mouth, TMJ pain, pain while chewing hard foods, biting hard foreign objects, chewing hard candies or ice, and leaning the jaw against the hand (all $p < .05$). Regarding sleep-related variables, SB was associated with snoring, drooling on the pillow, bedwetting, restless sleep, and mouth-breathing (all $p < .05$). Regarding family factors, SB was associated with parental and sibling grinding and with parental divorce (all $p < .05$). TMD symptoms were present in 22.5% of bruxism-positive children compared with 12.3% of bruxism-negative children ($p = .010$; OR = 2.08, 95% CI 1.18–3.65); all effect sizes were small (Cramér's V ≤ 0.21). No significant association was found between SB and tooth wear ($p = .282$). In multivariable analysis, allergic rhinitis, parental history of grinding, drooling on the pillow, restless sleep, and TMD remained independently associated with SB.

Conclusions: Sleep bruxism was associated with several sleep-related, respiratory, behavioral, and family-history factors and with TMD signs and symptoms, although all associations were of small magnitude. After multivariable adjustment, allergic rhinitis, parental history of grinding,

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drooling on the pillow, restless sleep, and TMD were independently associated with SB. Given the cross-sectional design, these findings indicate associations rather than causal or aetiological relationships.

Ethics: Approved by the Ethics Committee of Dokuz Eylul University (2024/13-18).

Keywords: Sleep bruxism, habits, mixed dentition, sleep behavior, TMD.

Background

Bruxism is a repetitive jaw-muscle activity characterized by clenching or grinding of the teeth and/or by bracing or thrusting of the mandible. It has two distinct circadian manifestations: it can occur during sleep (sleep bruxism, SB) or during wakefulness (awake bruxism, AB) [1]. Sleep bruxism is more common in children than in adults, and its reported prevalence in children ranges widely, from 3% to 49%, tending to decrease with age [2]. A recent global meta-analysis estimated the pooled prevalence of sleep bruxism at approximately 21% (and up to 43% when based on polysomnography); this wide range reflects methodological heterogeneity in assessment method, study design, and case definition rather than true differences in occurrence [3]. According to the current international consensus, SB is assessed—rather than diagnosed—using one or more of the three approaches defined in the Standardized Tool for the Assessment of Bruxism (STAB): subjective-based assessment (SBA; parent/guardian self-report), clinical-based assessment (CBA; clinical examination), and instrumental-based assessment (IBA; electromyography or polysomnography) [4]. These approaches are not hierarchical, as each may capture different aspects of the behaviour. While instrumental-based polysomnography remains the reference standard for measuring masticatory muscle activity during sleep, its use in pediatric populations is limited by feasibility and cost; consequently, most epidemiological studies rely on subjective-based (parent-reported) assessment.

Bruxism is considered to have a multifactorial and predominantly centrally mediated origin. Rather than being controlled by a single mechanism, it appears to arise from a complex interaction between central arousal and sleep regulation, neurotransmitter systems (e.g., dopaminergic, serotonergic, and noradrenergic pathways), individual predisposition, psychosocial stress, and environmental factors, including systemic conditions such as allergic rhinitis and asthma and the family environment [5, 6, 7]. Although evidence in children remains limited, findings in adults suggest that the psychological sphere and the broader environment in which individuals live—including depression and anxiety symptoms—are associated with bruxism [8]. Oral and behavioral factors, including sleep disturbances, oral habits, and temporomandibular disorders, have also been reported to be relevant [9, 10]. Although multiple factors have been associated with it, no definitive cause has yet been established [11].

As a motor behaviour, SB frequently co-occurs with sleep-related conditions and is predominantly associated with transient arousal events during sleep [12]. Sleep disturbances can impair children's physical, behavioral, and cognitive functioning, as children are highly sensitive to inadequate or poor-quality sleep [13]. SB and TMD have multifactorial origins; tooth grinding, tension-type headaches, pain or fatigue in the masticatory muscles and/or TMJ, limited jaw movements, and TMJ sounds are among the most common signs and symptoms in children and adolescents [14, 15]. A systematic review and meta-analysis reported an association between

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tooth grinding and TMD pain, although this association was subject to potential bias and confounding [16, 17].

Oral habits are common among schoolchildren but are often overlooked by caregivers [18]. Schoolchildren with a history of nail biting and object biting have been reported to show a higher prevalence of SB [19], whereas other studies found no significant links between SB and malocclusion or oral habits [14, 20]. Oral health status, particularly respiratory problems such as asthma and airway infections, has been identified as a factor associated with bruxism [7, 21], and associations have been reported between bruxism and conditions such as allergic rhinitis, sinusitis, ear infections, and mouth-breathing [21, 22]. Parenting style has also been identified as a factor associated with parent-reported SB, potentially influencing both the occurrence of the behaviour and the accuracy of parental reports [23]. Despite these studies, the factors associated with bruxism have not been clearly established in the mixed dentition. This study therefore aimed to investigate associations between sleep bruxism and oral habits, TMD, sleep disorders, and general health status in Turkish children with mixed dentition. This study was framed around a multifactorial conceptual model, in which biological, behavioral, psychosocial, and sleep-related domains were considered as potentially co-occurring correlates of sleep bruxism rather than isolated causal pathways. The null hypothesis (H0) was that there is no association between sleep bruxism and sleep behaviors, oral habits, temporomandibular disorders, or general health status in children with mixed dentition.

Methods

This study was approved by the Ethics Committee of Dokuz Eylul University (2024/13-18). Parents or legal guardians of the participating children provided written informed consent, and the study adhered to the Declaration of Helsinki. This school-based cross-sectional study was conducted in İzmir, Türkiye, the third-largest city in the country. In 2024, 19 719 students were reported to attend 44 primary schools in the central district [24]; a total of 14 819 students aged eight to ten (grades 2–4) met the age criterion. Of the 44 public schools, four were randomly selected using probability-proportional-to-size sampling. All selected schools were contacted, and authorization was obtained from the school principals and the district administration.

The sample size was determined based on an expected sleep bruxism prevalence of 35.3% [25], with a 95% confidence level and 5% absolute precision, yielding a minimum of 351 schoolchildren. To account for the school- and class-based cluster sampling design, a design effect of 1.2 was applied, and a further 20% was added for potential non-response, resulting in a target of 506 participants.

The study included parents/guardians and children aged eight to ten enrolled at a public school. Mixed dentition (the concurrent presence of primary and permanent teeth) was confirmed clinically during the oral examination, and children who were not in the mixed dentition stage were excluded. Children who were unwilling to participate, did not live with a parent or guardian, or had mental or physical disabilities were excluded, as were children with a prior history of SB treatment or orthodontic treatment.

Data were collected between October 2024 and February 2025. Questionnaires were delivered to children by their teachers one week before the clinical assessment and completed by parents/guardians at home. The questionnaire gathered parental information (educational level,

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marital status, occupation, family income) and child information (number of siblings, health status/systemic diseases, medication use, oral habits, sleep behavior, frequency of dental visits, grinding habits, life changes in the last year, and complaints regarding TMJ and jaw functions). Oral information, including tooth wear, signs and symptoms of TMD, and orofacial pain, was obtained during a classroom oral examination. Four dentists participated in training and calibration exercises with 20 children under the supervision of an experienced reference examiner one month before the study; intra-examiner agreement was 0.91, 0.87, 0.86, and 0.83, and inter-examiner agreement was 0.82 (Kappa statistic). The spread among the examiners' intra-rater values (0.83–0.91) indicates some measurement variability that should be considered when interpreting borderline tooth-wear classifications.

Sleep bruxism was assessed—not definitively diagnosed—by subjective-based assessment (SBA), in line with the STAB and the current international consensus [1, 4]: a child was classified as having sleep bruxism when the parent/guardian reported tooth grinding during sleep on at least three nights per week over the preceding 3–6 months [26]. Because instrumental-based assessment (IBA; electromyography or polysomnography) was not performed, SB was not instrumentally confirmed, and the possibility of misclassification is acknowledged. Tooth wear was assessed separately using the Smith and Knight Tooth Wear Index [27] and examined for its association with bruxism. Both primary and permanent teeth present in the mixed dentition were examined for tooth-wear recording. Only attrition-type facets (flat, glossy, matching the opposing occlusal contacts) were counted, whereas smooth, cupped or concave lesions with loss of surface anatomy, together with a reported history of gastric reflux, were considered erosive and excluded. Teeth with developmental enamel defects (e.g., molar–incisor hypomineralisation, enamel hypoplasia, or fluorosis) were not scored for wear, to reduce misclassification.

Temporomandibular disorder (TMD) signs were assessed by clinical examination according to the following criteria [28]: (1) an audible sound or a vibration palpable with the index fingers over the joint during at least two to three consecutive opening and closing movements; (2) tenderness on palpation of the right or left temporomandibular joint; and (3) tenderness on palpation of the right or left masseter and temporal muscles. TMD was considered present when two or more of these signs were detected. In addition, free-way space [29] and maximum mouth opening [30] were recorded as objective clinical indicators of masticatory-muscle and TMJ function that may be affected by bruxism, in order to explore whether they differed according to bruxism status.

Statistical analyses were conducted using IBM SPSS Statistics v25.0 (IBM Corp., Armonk, NY, USA). Normality was assessed with the Kolmogorov–Smirnov test; as the data were not normally distributed, non-parametric methods were used. Categorical associations were tested with the χ^2 or Fisher's exact test and continuous variables with the Mann–Whitney U test. In addition to significance testing, effect sizes were calculated following recent dentistry-specific guidance [31]: Cramér's V for χ^2-based associations (0.10 small, 0.30 medium, 0.50 large for 1 degree of freedom) and the effect-size correlation r ($|Z|/\sqrt{N}$) for Mann–Whitney U comparisons. Given the exploratory, hypothesis-generating nature of this cross-sectional analysis and the large number of bivariate comparisons, associations were interpreted with caution; to address the risk of Type I error inflation, p-values were additionally adjusted for multiple testing using the Benjamini–Hochberg false discovery rate (FDR) procedure as a sensitivity analysis. To identify factors independently associated with sleep bruxism after accounting for confounding and co-occurrence among exposures, multivariable binary logistic regression was performed using two

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complementary variable-selection strategies: in Model A, all variables significant in bivariate analysis were entered and reduced by backward stepwise (likelihood-ratio) elimination (retaining variables at $p < 0.10$); in Model B, the variables with a bivariate association at $p \leq 0.01$ were entered simultaneously. Multicollinearity was assessed using variance inflation factors (VIF), and model performance was summarised using the Nagelkerke R^2 and the likelihood-ratio test. Adjusted odds ratios (aOR) with 95% CIs were reported. All tests were applied at the 0.05 significance level.

Results

A total of 452 children participated (response rate: 89%); the primary reason for dropout was failure to return the questionnaire. The mean age was 9.02 ± 0.89 years; 53% were boys (n = 240) and 47% girls (n = 212). Sleep bruxism was observed in 22.6% (n = 102) of participants, with boys being 1.49 times more likely to have SB, although the difference was not statistically significant (p = .077, OR = 1.50, 95% CI = 0.96–2.35). No significant differences were found between SB and the mother's educational level, working status, father's educational level, working status, or family income (p = .608; p = .656; p = .328; p = 1.00; p = .836, respectively).

The relationship between systemic diseases and SB is presented in Table 1. SB was more common in children with allergic rhinitis ($p < .001$, OR = 2.42, 95% CI = 1.50–3.91, Cramér's V = 0.17). Diabetes mellitus, thyroid disease, and intestinal parasite categories had insufficient cell counts for meaningful inferential testing and were therefore presented descriptively only. No significant associations were found between SB and the other systemic diseases or the use of antihistamines, antianxiety medications, analgesics, or homeopathic drugs (all $p > .05$).

Regarding the head, TMJ, and jaw functions, SB was significantly more common in children with a history of head or jaw trauma, difficulty opening the mouth in the morning, TMJ pain, and pain while chewing foods (p = .020, Cramér's V = 0.12; p = .025, Cramér's V = 0.12; p = .037, Cramér's V = 0.12; p = .005, Cramér's V = 0.15, respectively), whereas no significant differences were detected for morning tension-type headache or earache (p = .193; p = .345). Oral habits such as biting hard foreign objects, chewing hard candies or ice, and leaning the jaw against the hand were also significantly associated with SB (p = .041, Cramér's V = 0.10; p = .001, Cramér's V = 0.16; p = .036, Cramér's V = 0.10, respectively) (Table 2).

The evaluation of sleep-related variables is presented in Table 3. No significant relationships were seen between SB and sleep duration, bedroom features, or nightmares (p = .327; p = .074; p = .134). However, snoring, drooling on the pillow, bedwetting, restless sleep, and mouth-breathing were significantly associated with SB (p = .001, OR = 2.45, 95% CI = 1.39–4.32, Cramér's V = 0.15; $p < .001$, OR = 2.52, 95% CI = 1.53–4.15, Cramér's V = 0.17; p = .031, OR = 4.46, 95% CI = 1.18–16.92, Cramér's V = 0.11; p = .017, OR = 5.96, 95% CI = 1.40–25.39, Cramér's V = 0.13; p = .003, OR = 2.26, 95% CI = 1.31–3.89, Cramér's V = 0.14, respectively). The bedwetting and restless-sleep associations were based on very small numbers of affected children (n = 9 and n = 8, respectively), yielding wide confidence intervals, and should therefore be interpreted with considerable caution.

A positive family history was associated with a higher SB prevalence, both in parents ($p < .001$, OR = 2.99, 95% CI = 1.80–4.95, Cramér's V = 0.21) and siblings (p = .032, OR = 2.43, 95% CI = 1.05–5.58, Cramér's V = 0.10). Parental divorce within the past year was also significant (p =

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.046, OR = 2.27, 95% CI = 1.00–5.17, Cramér's V = 0.09), whereas no relationships were found with the addition of a new sibling, relocation, or loss of a close family member (p = .258; p = .936; p = .504).

Bruxism was present in 34.8% of children with TMD signs versus 20.5% of those without TMD signs (p = .010, OR = 2.08, 95% CI = 1.18–3.65, Cramér's V = 0.12); conversely, TMD signs were present in 22.5% of children with SB versus 12.3% of those without. Among the TMD signs, no significant differences were found for joint sounds or masseter/temporal muscle tenderness, whereas significant differences were detected for TMJ tenderness and the presence of ≥ 2 clinical criteria (p = .001). No significant relationship was found between SB and maximum mouth opening or free-way space (p = .441, r = 0.04; p = .262, r = 0.05, respectively) (Table 4). No significant association was found between SB and tooth wear (p = .282).

To identify factors independently associated with sleep bruxism, two complementary multivariable logistic regression models were constructed (Table 5). All variance inflation factors were below 1.3, indicating no meaningful multicollinearity among the candidate predictors, including the respiratory/sleep-related variables (snoring, mouth-breathing, and allergic rhinitis). In the backward-elimination model (Model A; final model of 8 variables; Nagelkerke R^2 = 0.185), allergic rhinitis (aOR = 2.16, 95% CI 1.27–3.65), parental history of grinding (aOR = 2.40, 95% CI 1.39–4.14), drooling on the pillow (aOR = 2.00, 95% CI 1.16–3.45), restless sleep (aOR = 5.98, 95% CI 1.29–27.78), and TMD (aOR = 2.10, 95% CI 1.14–3.88) remained independently associated with sleep bruxism. The full-entry model restricted to variables with a bivariate association at $p \leq 0.01$ (Model B; Nagelkerke R^2 = 0.195) identified the same five factors, with closely comparable effect sizes. Several bivariately significant variables (e.g., snoring, mouth-breathing, head/jaw trauma, difficulty opening the mouth) were attenuated after adjustment and were no longer independently significant, indicating that part of their crude association was accounted for by co-occurring exposures. In a sensitivity analysis applying the Benjamini–Hochberg FDR correction (Q = 0.10) across all 31 bivariate comparisons, all 17 associations significant at the conventional threshold remained significant, indicating that the bivariate findings were unlikely to be attributable to Type I error inflation from multiple testing.

Overall, although several associations reached statistical significance, their effect sizes were small: Cramér's V ranged from 0.09 to 0.21 (highest for a parental history of grinding, V = 0.21) for the significant categorical associations and was negligible for the non-significant variables, while effect sizes for the continuous comparisons were negligible (r = 0.04–0.05). The detected associations were therefore statistically significant but of small magnitude.

Discussion

This study investigated the prevalence of sleep bruxism among schoolchildren and its association with sleep behavior, TMD, and health status. The 22.6% prevalence is consistent with prior reports and with a recent global meta-analysis, which estimated a pooled sleep bruxism prevalence of approximately 21% [3]; the wide 3–49% range reported across studies largely reflects methodological heterogeneity (assessment method, study design, and case definition) rather than true differences in occurrence [2, 32]. Because SB was assessed by subjective-based assessment (parental report) without instrumental (EMG/PSG) confirmation, our estimates are not directly comparable with studies using instrumental methods and may include children with masticatory activity of other origin.

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The association between bruxism and gender remains inconclusive; some studies report higher rates in boys [9, 19], others the opposite [33], and others no difference [20, 34, 35]. Our findings showed higher rates in boys, but the difference was not significant. Likewise, we found no association between SB and parental education or family income, consistent with some studies [34] but not others [19].

Respiratory conditions were associated with SB in our sample. Upper-airway problems such as rhinitis and sinusitis may cause nasal congestion and mouth-breathing during sleep, and it has been suggested that children with obstructed airways reposition the mandible to maintain airflow [7, 22, 36]. However, because of the cross-sectional design, the direction of this association cannot be determined; shared mechanisms such as sleep disturbance, autonomic arousal, and inflammatory pathways may underlie the co-occurrence of respiratory conditions and SB. Conversely, sleep bruxism has also been proposed to act as a protective factor in obstructive sleep apnea, as forward positioning of the mandible during rhythmic masticatory muscle activity may help to at least partially restore upper-airway patency [4]. Our finding that snoring, drooling, bedwetting, restless sleep, and mouth-breathing were associated with SB is in line with studies describing such symptoms as potential indicators of sleep-disordered breathing [37, 38, 39]. Evaluation of the upper respiratory tract by dentists may be a useful adjunct in the overall assessment of children with SB; however, respiratory findings should be regarded as potential contributing or comorbid factors rather than as diagnostic or management criteria for bruxism. Children in mixed dentition who mouth-breathe have been reported to show a 3.8 times higher prevalence of sleep bruxism [36]. Additionally, sleep disturbances such as sleeping with the light on and noise in the room, decreased sleep duration (less than 8 hours), and nightmares have been strongly associated with sleep bruxism [40, 41]; however, no such relationships were found in our study. In the literature, no relationship has been found between antihistamine drugs and SB except in a single case [42]; in our study, no association was found between antihistamines, analgesics, antianxiety medications, or homeopathic medications and SB.

Regarding bruxism and TMD, children with bruxism have been reported to be 2.97 times more likely to present TMD signs and symptoms in prior systematic reviews/meta-analyses [14, 16]; in our sample, the unadjusted association was OR = 2.08 (95% CI 1.18–3.65), and TMD remained independently associated with SB in the multivariable analysis (aOR = 2.10). This represents an association rather than a causal relationship; because of the cross-sectional design, the direction cannot be determined, and the influence of bruxism on TMD likely depends on factors such as loading duration, pain susceptibility, and psychosocial factors [43]. Headache has been reported as a factor associated with bruxism [44], although the precise mechanism remains unclear [45]; in our sample the effect sizes for the TMD-related associations were small (Cramér's $V \leq 0.15$).

With respect to tooth wear, we did not find a significant association between tooth wear and sleep bruxism ($p = .282$). This contrasts with Martins et al., who reported that the severity of self-reported sleep and awake bruxism was associated with the number of attrition tooth wear facets in children and adolescents [46]. The discrepancy may reflect differences in assessment (parental report of grinding rather than instrumental confirmation), the mixed-dentition stage, and the presence of physiological tooth wear, which can obscure a bruxism-specific signal. This finding also supports the current view that tooth wear should not be equated with bruxism.

Oral habits such as biting hard objects and chewing hard candies were associated with SB, whereas nail biting and finger sucking were not. Although thumb sucking and nail biting have been described as coping strategies for emotional stress, current evidence does not establish that

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these habits act as stress-reduction strategies leading to bruxism; they may instead reflect shared psychological or behavioural factors [9, 19, 47]. Environmental and familial factors were also relevant: a positive family history (parents and siblings) and parental divorce were associated with SB, consistent with reports linking family separation and parenting style to childhood bruxism [48]. As no genetic analyses were performed, these familial associations reflect familial aggregation and cannot be interpreted as evidence of a genetic mechanism. These associations, moreover, were of small magnitude and, given the cross-sectional design, should be interpreted as co-occurrence rather than causation.

This study has several limitations. First, its cross-sectional design measured exposures and outcomes simultaneously and therefore cannot establish causal relationships. Second, sleep bruxism was assessed by subjective-based (parental) report without instrumental confirmation, so some misclassification cannot be excluded. Third, awake bruxism was not assessed; as sleep and awake bruxism may co-occur and overlap in their self-report, focusing exclusively on SB may not fully capture the children's overall bruxism behaviour [49]. Fourth, TMD was evaluated through a sign-based clinical examination rather than a fully standardized protocol (e.g., DC/TMD), so TMD-related findings should be interpreted with caution. Fifth, data were collected using a study-specific questionnaire that was not formally validated, which may introduce measurement and recall bias, particularly for parent-reported items on systemic diseases, medication use, TMD symptoms, and oral behaviours; future studies should employ validated instruments such as the 3Q/TMD, the Symptom Questionnaire, and the Oral Behaviours Checklist, and the associations derived from these items should be interpreted with caution. Tooth wear, examined here as a separate variable, is itself multifactorial and may be influenced by acid erosion, diet, and developmental enamel defects; despite the exclusion criteria applied, residual misclassification of wear cannot be fully excluded. Sixth, some statistically significant associations were based on very small subgroups (particularly bedwetting and restless sleep), resulting in wide confidence intervals; these estimates should therefore be interpreted with particular caution. Finally, only public schools were included; children attending private schools in Türkiye may differ in socioeconomic status, lifestyle, and access to dental care, which may limit generalizability. A strength of the study is its representative, probability-based sample; accordingly, the prevalence estimate should be regarded as the primary finding, and the reported associations as exploratory. Given the large number of comparisons performed, some bivariate associations may represent false-positive findings; the multivariable models and the Benjamini–Hochberg FDR sensitivity analysis were therefore used to identify the most robust independent associations.

Based on these findings, the null hypothesis was partially rejected. It was rejected for the variables that showed significant associations with sleep bruxism—allergic rhinitis, specific oral habits (biting hard objects, chewing hard candies or ice, and leaning the jaw against the hand), sleep-related symptoms (snoring, drooling, bedwetting, restless sleep, and mouth-breathing), family history, and TMD signs—and retained for the variables that showed no significant association, including sociodemographic factors, medication use, sleep duration, bedroom features, nightmares, and tooth wear.

Conclusion

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This study investigated the prevalence of sleep bruxism and its associated factors among schoolchildren with mixed dentition. SB was associated with allergic rhinitis, several oral habits, family history, sleep-related symptoms, and TMD signs; however, all associations were of small magnitude, and no significant association was found with tooth wear. As this is a cross-sectional study, these findings indicate statistical associations rather than causal or aetiological relationships, and prospective studies are required to determine their direction and causality. Within these limits, the results underscore the value of a multidisciplinary approach—involving pediatric dentists, pediatricians, and otolaryngologists—particularly for children presenting with mouth-breathing or allergic conditions.

Abbreviations

AB: awake bruxism; aOR: adjusted odds ratio; CBA: clinical-based assessment; CI: confidence interval; EMG: electromyography; IBA: instrumental-based assessment; OR: odds ratio; SB: sleep bruxism; SBA: subjective-based assessment; STAB: Standardized Tool for the Assessment of Bruxism; TMD: temporomandibular disorders; TMJ: temporomandibular joint; VIF: variance inflation factor.

Declarations

Ethics approval and consent to participate

This study was approved by the Research Ethics Committee of Dokuz Eylul University (2024/13-18). Parents or legal guardians of all participating children provided written informed consent. The study adhered to the Declaration of Helsinki.

Consent for publication

Not applicable.

Availability of data and materials

The datasets generated and/or analysed during the current study are available from the corresponding author upon reasonable request.

Competing interests

The authors declare that they have no competing interests.

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Authors' contributions

G.B. designed the survey, collected the primary data, conducted the statistical analysis, and prepared the initial draft of the manuscript. G.K. contributed to data interpretation and critically revised the manuscript for important intellectual content. B.Ç. contributed to data interpretation and prepared the tables. S.E.G. contributed to the study design and assisted with statistical analyses. All authors reviewed and approved the final version of the manuscript.

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Clinical trial number

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Table 1. The relationship between systemic diseases and bruxism

Variable		Absence n (%)	Presence n (%)	p	Cramér's V

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Diabetes mellitus	No	349 (77.4)	102 (22.6)	—	—
	Yes	1 (100)	0		
Asthma	No	335 (77.9)	95 (22.1)	.298	0.050
	Yes	15 (68.2)	7 (31.8)		
Thyroid disease	No	349 (77.6)	101 (22.4)	—	—
	Yes	1 (50)	1 (50)		
Nutrition deficiency	No	346 (77.6)	100 (22.4)	.621	0.030
	Yes	4 (66.7)	2 (33.3)		
Reflux disease	No	342 (77.4)	100 (22.6)	1.000	0.009
	Yes	8 (80)	2 (20)		
Intestinal parasite	No	348 (77.3)	102 (22.7)	—	—
	Yes	2 (100)	0 (0)		
Allergic rhinitis	No	281 (81.4)	64 (18.6)	<.001*	0.172
	Yes	69 (64.5)	38 (35.5)		

*$p < 0.05$, statistically significant (χ^2 test). Cramér's V, effect size (0.10 = small, 0.30 = medium, 0.50 = large). Diabetes mellitus, thyroid disease, and intestinal parasite categories were presented descriptively only because of insufficient cell counts for meaningful inferential testing.

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Table 2. The relationship between oral habits and bruxism

Variable		Absence n (%)	Presence n (%)	p	Cramér's V
Biting hard foreign objects	No	332 (78.5)	91 (21.5)	.041*	0.096
	Yes	18 (62.1)	11 (37.9)		
Chewing hard candies or ice	No	296 (80.7)	71 (19.3)	.001*	0.160
	Yes	54 (63.5)	31 (36.5)		
Nail biting	No	286 (77.1)	85 (22.9)	.708	0.018
	Yes	64 (79.0)	17 (21.0)		
Opening bottles with teeth	No	340 (77.8)	97 (22.2)	.345	0.048
	Yes	10 (66.7)	5 (33.3)		

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Daily gum chewing	No	277 (78.2)	77 (21.8)	.431	0.037
	Yes	73 (74.5)	25 (25.5)		
Shifting the jaws in different planes	No	346 (77.8)	99 (22.2)	.193	0.061
	Yes	4 (57.1)	3 (42.9)		
Leaning the jaw against the hand	No	330 (78.6)	90 (21.4)	.036*	0.099
	Yes	20 (62.5)	12 (37.5)		
Biting lip and cheek	No	329 (77.4)	96 (22.6)	.965	0.002
	Yes	21 (77.8)	6 (22.2)		
Thumb sucking	No	339 (77.8)	97 (22.2)	.372	0.040
	Yes	11 (68.8)	5 (31.3)		

**p < 0.05, statistically significant (χ^2 test). Cramér's V, effect size (0.10 = small, 0.30 = medium, 0.50 = large).*

Table 3. The relationship between sleep-related variables and bruxism

Variable		Absence n (%)	Presence n (%)	p	Cramér's V
Sleep time at night	Less than 8 hours	42 (72.4)	16 (27.6)	.327	0.046
	More than 8 hours	308 (78.2)	86 (21.8)		
Features of the bedroom	Bright and noisy	33 (89.2)	4 (10.8)	.074	0.084
	Dark and quiet	317 (76.4)	98 (23.6)		
Snoring	No	311 (79.9)	78 (20.1)	.001*	0.149
	Yes	39 (61.9)	24 (38.1)		
Nightmares at night	No	325 (78.3)	90 (21.7)	.134	0.070
	Yes	25 (67.6)	12 (32.4)		
Drooling on the pillow	No	292 (81.1)	68 (18.9)	<.001*	0.174
	Yes	58 (63.0)	34 (37.0)		
Bedwetting	No	346 (78.1)	97 (21.9)	.031*	0.112
	Yes	4 (44.4)	5 (55.6)		
Restless sleep	No	347 (78.2)	97 (21.8)	.017*	0.128
	Yes	3 (37.5)	5 (62.5)		
Mouth-breathing	No	304 (80.0)	76 (20.0)	.003*	0.141

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	Yes	46 (63.9)	26 (36.1)		

**p < 0.05, statistically significant (Fisher's exact test). Cramér's V, effect size.*

Table 4. Bruxism in relation to maximum mouth opening, free-way space, and TMD

Variable		Absence n (%)	Presence n (%)	p	Cramér's V
Max mouth opening (mm)	Mean±SD (median; min–max)	47.44±5.12 (47.00; 33.0–63.5)	47.07±5.18 (46.50; 36–59)	.441	r=0.04
Free-way space (mm)	Mean±SD (median; min–max)	2.01±0.78 (2.00; 1–4)	2.10±0.75 (2.00; 1–4)	.262	r=0.05
TMD	Absent	307 (79.5)	79 (20.5)	.010*	0.121
	Present	43 (65.2)	23 (34.8)		

**: p<0.05; statistically significant. Effect size for continuous variables: r = |Z|/√N from the Mann–Whitney test (max mouth opening Z=−0.771, r=0.036; free-way space Z=−1.121, r=0.053; both negligible). Cramér's V reported for TMD.*

Table 5. Bivariate and multivariable logistic regression analyses of factors associated with sleep bruxism (n = 452)

Variable	Crude OR (95% CI)	p	aOR (95% CI) Model A	p	aOR (95% CI) Model B	p
Allergic rhinitis †	2.42 (1.50–3.91)	<0.001	2.16 (1.27–3.65)	0.004	2.03 (1.19–3.46)	0.009
Parental history of grinding †	2.99 (1.81–4.95)	<0.001	2.40 (1.39–4.14)	0.002	2.29 (1.32–3.99)	0.003
Drooling on the pillow †	2.52 (1.53–4.15)	<0.001	2.00 (1.16–3.45)	0.013	1.80 (1.02–3.18)	0.041
Restless sleep †	5.96 (1.40–25.39)	0.006	5.98 (1.29–27.78)	0.023	5.14 (1.07–24.68)	0.041
TMD symptoms †	2.08 (1.18–3.65)	0.010	2.10 (1.14–3.88)	0.018	1.99 (1.07–3.69)	0.029
History of head/jaw trauma	3.29 (1.30–8.33)	0.008	2.58 (0.93–7.18)	0.070	2.42 (0.85–6.89)	0.098
Difficulty opening the mouth	7.10 (1.28–39.35)	0.009	5.16 (0.86–30.95)	0.073	4.71 (0.76–29.26)	0.097
Mouth-breathing	2.26 (1.31–3.89)	0.003	1.81 (0.99–3.31)	0.055	1.61 (0.86–3.00)	0.137
Snoring	2.45 (1.39–4.32)	0.001	—	—	1.47 (0.77–2.82)	0.247
Pain while chewing hard foods	3.46 (1.52–7.83)	0.002	—	—	1.59 (0.63–4.01)	0.325
Chewing hard candies or ice	2.39 (1.43–3.99)	<0.001	—	—	1.22 (0.67–2.20)	0.520

OR, odds ratio; aOR, adjusted odds ratio; CI, confidence interval; TMD, temporomandibular disorder. Rows marked with † remained independently significant (p < 0.05) in both multivariable models.

Model A: backward stepwise (likelihood-ratio) elimination starting from all 17 variables significant in bivariate analysis; variables retained at p < 0.10 are shown (final model: 8 variables; Nagelkerke R^2 = 0.185; model χ^2 = 58.6, df = 8, p < 0.001). Variables eliminated during the procedure are shown as "—".

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Model B: simultaneous ("enter") entry of the 11 variables with a bivariate association at $p \leq 0.01$ (Nagelkerke $R^2 = 0.195$; model $\chi^2 = 61.8$, $df = 11$, $p < 0.001$).

Reference category for all predictors: absence of the characteristic. All variance inflation factors were < 1.3, indicating no meaningful multicollinearity. Crude odds ratios were derived from univariate binary logistic regression and may differ slightly from the χ^2/Fisher's exact test p-values in Tables 1–4 owing to the different statistical methods.

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