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Original Article

Associations between sleep bruxism, sleep quality, and exposure to secondhand smoke in Japanese young adults: a cross-sectional study

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ABSTRACT

Objective: Sleep bruxism, a major sleep disorder that causes serious harm to oral health, is considered a multifactorial disease. Sleep bruxism can be induced by smoking, which also adversely affects sleep quality. The objective of present study was to clarify the associations between sleep bruxism, sleep quality, and exposure to secondhand smoke (SHS).

Methods: To assess the prevalence of sleep bruxism, sleep quality, and SHS exposure, we conducted oral examinations and self-report questionnaires on university students in Japan. Sleep bruxism and quality were screened using the Japanese version of the Pittsburgh Sleep Quality Index (PSQI) and the third edition of the International Classification of Sleep Disorders (ICSD-3). The inclusion criteria were adults aged between 18 and 19 years, non-smokers and non-alcohol drinkers. The exclusion criteria was failing to complete the questionnaire in full.

Results: We analyzed a total of 1781 Japanese young adults. Young adult females who had been exposed to SHS had worse sleep quality ($p = 0.019$) than those who had not. Young adult female with worse sleep quality showed a higher prevalence of sleep bruxism ($p = 0.034$) than those with better sleep quality. Using structural equation modeling, direct associations were identified between SHS exposure and poor sleep quality (standardized coefficients, 0.153; $p = 0.008$) and between sleep bruxism and poor sleep quality (standardized coefficients, 0.187; $p = 0.022$) in young adult females. However, no association was found among young adult males.

Conclusion: SHS exposure is indirectly associated with sleep bruxism through poor sleep quality in Japanese young adult females.

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1. Introduction

Sleep bruxism, a major sleep disorder that adversely affects oral health, is defined by the American Academy of Sleep Medicine as "repetitive jaw muscle activity characterized by the clenching or grinding of teeth or bracing or thrusting of the mandible" [1]. It can lead to tooth wear, tooth or restoration fracture, hypersensitive or painful teeth, loss of periodontal support, pain in the temporomandibular joint or jaw muscles, headaches, and masticatory muscle hypertrophy [2]. Therefore, dentists often encounter the effects of sleep bruxism in the clinical setting. Sleep bruxism is considered a multifactorial disease. Some studies have reported risk factors for sleep bruxism, including gender, age, alcohol drinking, gastroesophageal reflux disease, and disorders of the dopaminergic system [3]. However, the causes of sleep bruxism remain unclear.

Smoking is also known to be a risk factor for sleep bruxism [4] and to affect sleep quality adversely [5]. Tobacco smoke includes over 4000 chemicals, more than 200 of which are harmful [6]. Nicotine, the most harmful chemical in tobacco smoke, induces

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acetylcholine and glutamate synaptic transmission and enhances dopamine release in the human brain [4,7,8]. Dopamine can stimulate the central nervous system, lead to worse sleep quality, and cause sleeping disorders such as sleep bruxism.

Smokers harm not only their own health, but also that of their family. Especially, children can be easily damaged by exposure to secondhand smoke (SHS). Exposure to nicotine in SHS might worsen sleep quality in the same way. Based on our current knowledge, evidence that the avoidance of SHS exposure improves sleep bruxism in children has only been reported in one randomized controlled trial [9]. However, the interactions between SHS exposure, sleep bruxism, and poor sleep quality remain unclear. Therefore, we hypothesized the process that (1) SHS worsens sleep quality and sleep bruxism, and (2) poor sleep quality induces sleep bruxism. The aim of the present study was to investigate the interactions between SHS exposure, sleep quality, and sleep bruxism in young adults.

2. Methods

2.1. Study population

Data were obtained from first-year students who had undergone both a general and an oral health examination at the Health Service Center of Okayama University in April 2018. All students received oral examinations and filled out self-report questionnaires. The inclusion criteria were young adults between 18 and 19 years of age, non-smokers and non-alcohol drinkers. The exclusion criteria was failing to complete the questionnaire in full.

2.2. Ethical procedures and informed consent

The present study was approved by the ethics committees of Okayama University Graduate School of Medicine, Dentistry and Pharmaceutical Sciences and Okayama University Hospital (No. 1060). Written informed consent was obtained from all participants before the study began.

2.3. Assessment of sleep bruxism

Sleep bruxism was diagnosed using the third edition of the International Classification of Sleep Disorders (ICSD-3). Self-report questionnaires were used to assess teeth grinding during sleep, jaw muscle pain or fatigue, and temporal headaches, as shown below:

During the past three months,

Q1 Has anyone heard you grinding your teeth while you were asleep at night? (yes/no)
Q2 How often is your jaw fatigued or sore on waking in the morning? (frequently/sometimes/rarely/never)
Q3 How often do you have a temporal headache on waking in the morning? (frequently/sometimes/rarely/never)

We combined the “frequently” and “sometimes” responses into a single category of positive awareness, and “rarely” and “never” into a single category of negative awareness [10].

Nine dentists (D.E., T.Y., K.K., A.T-T., A.Y., D.F., Y.U., N.T., and H.S.) checked for abnormal tooth wear in oral examinations [11]. Abnormal tooth wear was defined as exposed dentin on the canine teeth or first or second premolars. The examiners were calibrated before the oral examinations (kappa value > 0.8).

The presence of sleep bruxism was based on the ICSD-3 criteria, in the case that (A) and (B) were met:

A) The answer to Q1 was “yes”.
B) The answer to Q2 or Q3 was “yes”, or students had abnormal tooth wear.

The diagnostic criteria are shown in a flowchart in Fig. 1.

2.4. Assessment of SHS exposure

Exposure to SHS was assessed using a self-report questionnaire. A previous study reported that 73% of nonsmokers living with smokers were exposed to SHS inside the home [12]. A law restricting smoking in public places was enacted in Japan in 2004, and since that time, SHS exposure in public places has become less common [13]. Therefore, we investigated SHS exposure in the home. The participants were asked, “Does anyone in your family smoke?” [14], and the answers were “current smoker, past smoker, and nonsmoker”. We defined “current smoker” as current SHS exposure, “past smoker” as previous SHS exposure, and “nonsmoker” as no SHS exposure.

2.5. Assessment of other factors

We also asked about other factors that could be associated with sleep bruxism, including gender, age, and smoking and drinking status, on the self-report questionnaires [15]. Sleep quality was assessed using the Japanese version of the Pittsburgh Sleep Quality Index (PSQI-J) [16]. A PSQI score >5 was considered “poor sleep quality”.

2.6. Statistical analysis

SPSS software (version 20; IBM, Tokyo, Japan) was used for all statistical analyses. Values of $p < 0.05$ were considered significant. Chi-squared tests was used to assess differences based on gender, age, sleep bruxism, SHS exposure, and sleep quality.

We then performed pathway analysis to clarify the process from age, SHS exposure, and sleep quality to sleep bruxism. Statistical analysis was carried out using structural equation modeling (SEM) accompanied by the weighted least square mean and variance adjusted (WLSMV) estimation method. Next, we devised a pathway based on our hypothesis (Fig. 3). To construct the final model, we kept only significant paths with p values < 0.30 [17]. We used Mplus software (version 8.2; Muthén & Muthén, Los Angeles, CA, USA) for path analysis. Model fitness was defined as good in the case of (1) a root mean square error of approximation (RMSEA) < 0.05; (2) a comparative fit index (CFI) > 0.9; (3) a Tucker–Lewis index (TLI) > 0.9; and (4) a standardized root mean square residual (SRMR) < 0.1.

3. Results

A flowchart of the recruitment process is shown in Fig. 2. Students who were aged ≥20 years, current smokers or alcohol drinkers, or did not fill out the questionnaire in full were excluded. Finally, 1781 (970 males [54.5%] and 811 females [45.5%]) of 2144 students were included in the analysis.

The distribution of SHS exposure, sleep quality, and sleep bruxism by gender is shown in Table 1. In total, 364 (20.4%) students were classified as having poor sleep quality (PSQI score > 5). Sleep bruxism was significantly more prevalent in females than in males ($p = 0.006$). However, no significant gender differences were seen in the ratio of SHS exposure and poor sleep quality ($p \geq 0.05$).

As the prevalence of sleep bruxism was significantly different between males and females, we performed a sub-group analysis separated by gender. The results indicated that sleep quality was

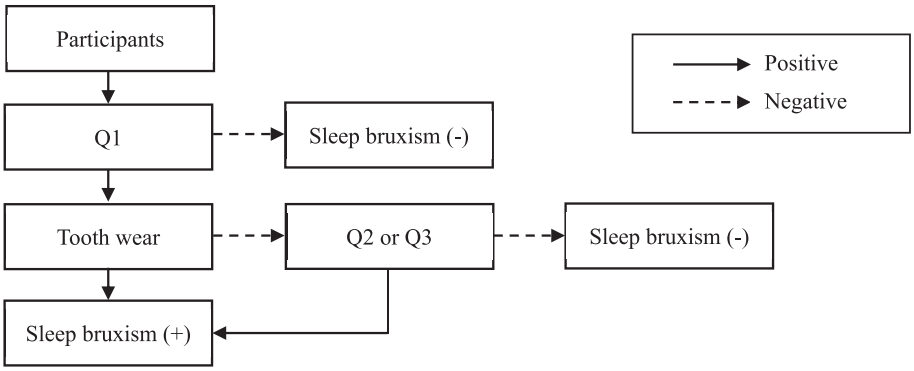

Fig. 1. Protocol for diagnosing sleep bruxism based on the International Classification of Sleep Disorders third edition. Q1: Has anyone heard you grinding your teeth while you were asleep at night?. Q2: How often is your jaw fatigued or sore on waking in the morning?. Q3: How often do you have a temporal headache on waking in the morning?.

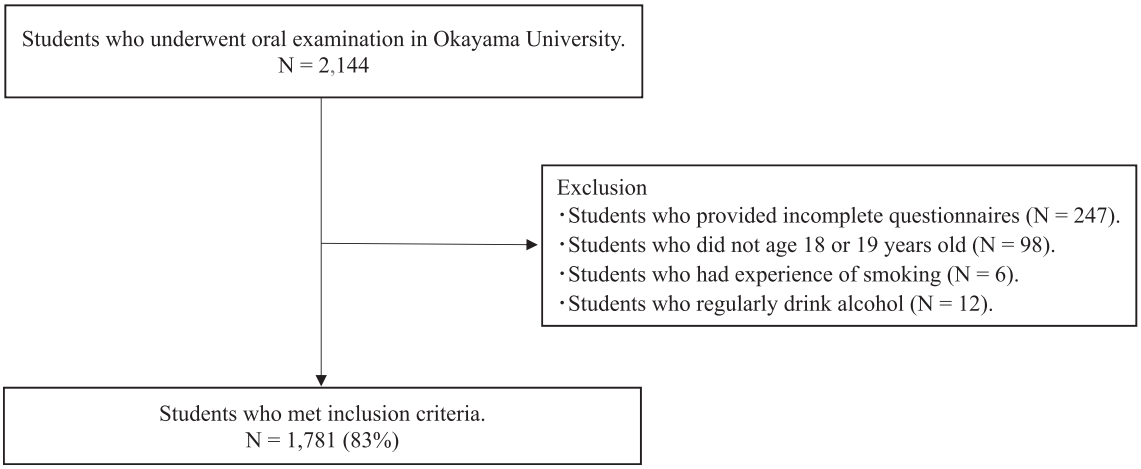

Fig. 2. Flowchart showing the protocol for selecting students for analysis from among who did not meet the exclusion criteria (age, current smoker or drinker).

associated with SHS exposure in females (Table 2). Female students exposed to SHS exhibited poor sleep quality ($p = 0.019$). On the other hand, no significant association was observed between sleep quality and SHS exposure in male students ($p \geq 0.05$). In addition, an association was found between sleep bruxism and sleep quality in females (Table 3). The prevalence of females with poor sleep quality was higher in those with than in those without sleep bruxism ($p = 0.034$).

The results of the pathway analysis among females indicated direct associations between SHS exposure and sleep quality (standardized coefficient [SC] = 0.153; standard error [SE] = 0.058; $p = 0.008$) and between sleep quality and sleep bruxism (SC = 0.187; SE = 0.081; $p = 0.022$) (Fig. 4). The model fit index showed good fit (RMSEA = 0.000, CFI = 1.000, TLI = 1.193, SRMR = 0.017). SHS exposure did not directly influence sleep bruxism (SC = −0.144, $p = 0.075$), and no associations were observed among any variables in males ($p > 0.05$, data not shown).

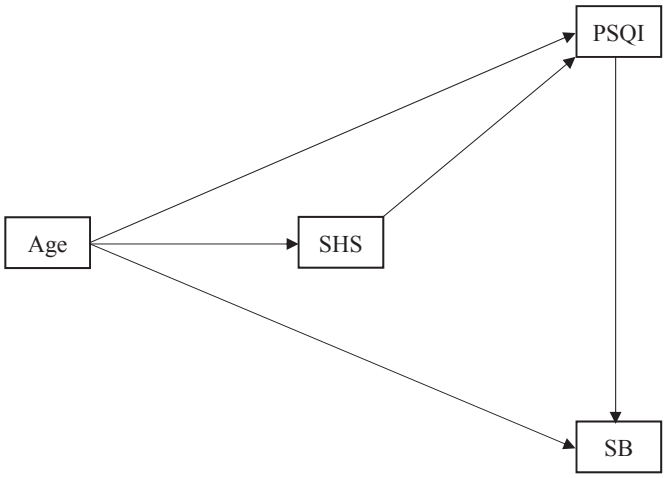

Fig. 3. Pathway based on our hypothesis showing the associations between sleep bruxism, sleep quality, and SHS exposure in relation to age. SHS, secondhand smoke. PSQI, Pittsburgh Sleep Quality Index. SB, sleep bruxism.

Table 1
Gender differences among the participants.

Variables	Categories	Male	Female	p[b]
		N = 970	N = 811	
Age (y)	18	799 (82.4)[a]	712 (87.8)	0.001
	19	171 (17.6)	99 (12.2)	
SHS exposure	Current	147 (15.2)	135 (16.6)	0.687
	Past	148 (15.3)	120 (14.8)	
	Never	675 (69.6)	556 (68.6)	
Sleep quality	Poor	197 (20.3)	167 (20.6)	0.883
	Good	773 (79.7)	644 (79.4)	
Sleep bruxism	Positive	47 (4.8)	65 (8.0)	0.006
	Negative	923 (95.2)	746 (92.0)	

SHS, secondhand smoke.
[a] N (%).
[b] Chi-squared test.

Table 2
Associations between sleep quality and other variables.

		Sleep quality		p[b]
		Good	Poor	
Male		N = 725	N = 245	
Age (y)	18	643 (83.2)[a]	156 (79.2)	0.189
	19	130 (16.8)	41 (20.8)	
SHS exposure	Never	549 (71.0)	126 (64.0)	0.155
	Past	113 (14.6)	35 (17.8)	
	Current	111 (14.4)	36 (18.3)	
Female		N = 598	N = 213	
Age (y)	18	562 (87.3)[a]	150 (89.8)	0.369
	19	82 (12.7)	17 (10.2)	
SHS exposure	Never	456 (70.8)	100 (59.9)	0.019
	Past	81 (14.1)	29 (17.4)	
	Current	97 (15.1)	38 (22.8)	

SHS, secondhand smoke.
[a] N (%).
[b] Chi-squared test.

Table 3
Association between sleep bruxism and other variables.

		Sleep bruxism		p[b]
		Positive	Negative	
Male		N = 47	N = 923	
Age (y)	18	38 (80.9)[a]	761 (82.4)	0.779
	19	9 (19.1)	162 (17.6)	
Sleep quality	Poor	10 (21.3)	187 (20.3)	0.866
	Good	37 (78.7)	736 (79.7)	
Female		N = 65	N = 746	
Age (y)	18	59 (90.8)[a]	653 (87.5)	0.445
	19	6 (9.2)	93 (12.5)	
Sleep quality	Poor	20 (30.8)	147 (19.7)	0.034
	Good	45 (69.2)	599 (80.3)	

[a] N (%).
[b] Chi-squared test.

4. Discussion

The results of the present study revealed direct associations between SHS exposure and sleep quality and between sleep quality and sleep bruxism. In addition, an indirect association was found between SHS exposure and sleep bruxism in Japanese young females (Fig. 4). To the best of our knowledge, this is the first study to report such interactions between sleep bruxism, sleep quality, and SHS exposure.

Nicotine could be related to both SHS exposure and poor sleep quality because it is a stimulant, and smokers have been reported to be at higher risk for poor sleep quality [5]. Nicotine binds to nicotinic cholinergic receptors, leading to dopamine release. As dopamine is also a stimulant [18], SHS exposure would be considered to promote poor sleep quality. In the present study, an association was found between SHS exposure and poor sleep quality. Morioka et al. [19] reported an association between SHS exposure and sleep disturbance in Japanese adolescents, which supports our results. However, we did not measure nicotine levels in the body. Further studies incorporating the measurement of nicotine levels may be needed to clarify further the association between SHS exposure and sleep quality.

Our results also identified a direct association between sleep bruxism and sleep quality in Japanese young female university students. Psychophysiological insomnia is characterized by a stress response, and individuals with bruxism are more sensitive to

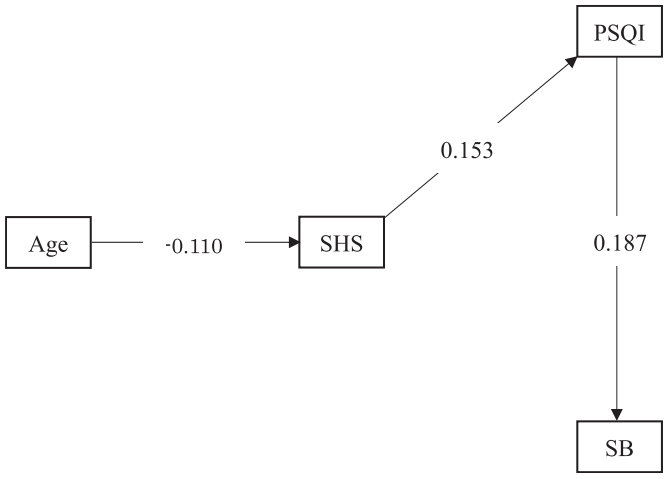

Fig. 4. Pathway based on structural equation modeling showing the associations between sleep bruxism, sleep quality, and SHS exposure in relation to age. There were the significant association between SHS, PSQI, and sleep bruxism in females. The values for single head arrows show standardized coefficient. SHS, secondhand smoke. PSQI, Pittsburgh Sleep Quality Index. SB, sleep bruxism.

psychological stress [20,21]. Therefore, sleep quality might be related to sleep bruxism. Solanki et al., Serra-Negra et al., and Kato et al. suggested that poor sleep quality was related to sleep bruxism [22–24], which supports the present results. In addition, micro-arousal may link sleep bruxism with sleep quality. Micro-arousal is measured during sleep cycles 2 and 3 in non-rapid eye movement sleep [24,25], and sleep bruxism occurs after micro-arousal [24]. Changyun et al. [26] reported that less and more frequent micro-arousal were associated with better and worse sleep quality, respectively. These reports suggest that poor sleep quality caused by micro-arousal is associated with sleep bruxism.

In the present study, associations between sleep bruxism, sleep quality, and SHS exposure were seen only in females. Thus, biological factors might explain the higher prevalence of poor sleep quality in females than in males. Mong et al. [27] suggested that biological sex and sex hormones were associated with the risk of poor sleep quality. Estrogen, a sex hormone produced mostly in the ovaries, has been found to affect sleep in females [28]. As chemicals in tobacco smoke are anti-estrogenic [29], females might be more susceptible to SHS exposure than males. Moreover, psychological stress reduces estrogen levels in young females [30], which could affect sleep quality more among females than among males.

The participants who took part in the present study were not considered a special group. Systematic reviews have reported that the prevalence of sleep bruxism ranges from 5.9 to 49.6% in children and from 1.1 to 15% in adults [31–33]. The prevalence of sleep bruxism in the present study was 6.3% (112 students; Table 1). In addition, the rate of exposure to SHS in the present study was 15.8%. Matsuyama et al. [34] reported that 14.6% of Japanese nonsmokers aged 20–39 years were exposed to SHS at home. On the other hand, the average PSQI score of the participants in the present study (4.1 points) was lower than that of participants in previous studies (data not shown). PSQI scores in the US or among other Japanese university students have ranged from 5.2 to 7.5 [35,36]. A lower PSQI scores means better sleep quality. As the students analyzed in the present study were nonsmokers and nondrinkers, their sleep quality might have been better than the participants in previous studies. Moreover, we conducted the present investigation within only two months after university entrance examinations, which should be considered because sleep deprivation tends to be more prevalent among preparatory students [37]. As the participants in

the present study had just passed their examinations, some of them might have had improved sleep quality and duration.

The present study did have some limitations. First, sleep bruxism was diagnosed based on self-report questionnaires and current oral status. The gold standard for diagnosing sleep bruxism is a full-night polysomnography (PSG) [38]. However, in population studies, using PSG is difficult because of technical and budgetary constraints [39]. Therefore, we used the ICSD-3 criteria to diagnose sleep bruxism. The concordance between the ICSD-3 and PSG diagnostic criteria has been reported to be moderate, with an area under the curve ranging from 0.55 to 0.75 [40]. The ICSD-3 criteria were therefore considered to be a useful screening tool for sleep bruxism in present study [41]. Second, we did not investigate SHS exposure from sources other than family members (eg, school, restaurant, street). As almost all of the participants had been living with their parents for long time, they might be the most susceptible to SHS exposure from their parents. Third, all the participants were recruited from the student population at Okayama University, which may limit the ability to extrapolate these findings to the general population of young adults. Fourth, as this was a cross-sectional study, causal relationships cannot be determined between sleep bruxism, sleep quality, and SHS exposure. Longitudinal studies are therefore needed. Finally, we should pay attention to confounders that may affect sleep quality and sleep bruxism. For example, sleep quality was related with atopic dermatitis [42], allergic rhinitis [43], neuroticism [44], and sleep apnea [45], and that sleep bruxism was related with reflex esophagitis [15]. However, there were no participants with sleep apnea and reflex esophagitis. Furthermore, even though we excluded the participants with atopic dermatitis, allergic rhinitis, and neuroticism, the conclusion was same. Thus, the effects of these factors may be neglected at least.

In conclusion, the results of the present study clarified the interactions between sleep bruxism, sleep quality, and SHS exposure among Japanese young females, and found that SHS exposure was indirectly related to sleep bruxism. However, additional studies using PSG are necessary to clarify these relationships further and help prevent the onset of sleep bruxism, improve sleep quality, and highlight the importance of SHS exposure for clinicians and parents.

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Ethical approval

All procedures involving human participants were carried out in accordance with the 1964 Helsinki declaration and its later amendments or comparable ethical standards, and with the standards of the ethics committees at Okayama University Graduate School of Medicine, Dentistry and Pharmaceutical Sciences and at Okayama University Hospital.

Informed consent

Informed consent was obtained from all participants.

Acknowledgments

None.

Conflict of interest

All authors declare that they have no conflicts of interest.

The ICMJE Uniform Disclosure Form for Potential Conflicts of Interest associated with this article can be viewed by clicking on the following link: https://doi.org/10.1016/j.sleep.2019.09.003.

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