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Correlates and genetics of self-reported sleep and awake bruxism in a nationwide twin cohort

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Abstract

Background: Sleep bruxism (SB) and awake bruxism (AB) have been considered different entities, although co-occurrence between them has been shown. While genetic factors have a marked influence on phenotypic variance in liability to SB, this remains unclear for AB.

Aim: To examine the degree of co-occurrence of SB and AB, and whether they have common correlates and also twin similarity of SB and AB bruxism traits by zygosity and sex.

Methods: A questionnaire was mailed to all twins born 1945-1957 in Finland in 2012 (n = 11 766). Age and sex adjusted logistic regression models were used. Twin similarity was assessed using polychoric correlations, and crosstwin-crosstrait correlations were computed.

Results: The response rate was 72% (n = 8410). Any SB was reported by 14.8% and ≥ 3 nights weekly by 5.0%. Percentages for any AB were 18.4% and 6.3%, respectively. There was substantial co-occurrence (29.5%) between SB and AB, and several shared correlates were found. For SB, the polychoric intra-class correlation was 0.366 in monozygotic (MZ) and 0.200 in dizygotic (DZ) pairs, without gender difference. A twofold crosstwin-crosstrait correlation was observed in MZ twins compared to DZ twins.

Conclusions: The risk factor profiles of SB and AB were largely but not entirely similar. The higher correlation in MZ than in DZ pairs suggests the influence of genetic factors on both SB and AB. The higher crosstwin-crosstrait correlation in MZ than in DZ pairs suggests some degree of genetic influences shared by SB and AB.

KEYWORDS

awake bruxism, genetic epidemiology, sleep bruxism, twins

1 | INTRODUCTION

Lobbezoo et al[1] defined bruxism as a repetitive jaw-muscle activity characterised by clenching or grinding of the teeth and/or bracing or thrusting of the mandible. Bruxism has two distinct circadian manifestations: it can occur during sleep (indicated as sleep bruxism, SB) or during wakefulness (awake bruxism, AB). To operationalise the definition, the expert group proposed a diagnostic

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grading system of "possible," "probable" and "definite" SB or AB, the grade depending on the method used to assess bruxism. Since publication of this widely cited international consensus paper, its inclusion in both fourth and fifth editions of the Guidelines for Assessment, Diagnosis and Management of Orofacial Pain of the American Academy of Orofacial Pain,[2,3] as well as in the third edition of the International Classification of Sleep Disorders,[4] serves to confirm its rapid and broad adoption in the field. Recently, the consensus definition was revisited and updated by Lobbezoo et al as follows[5]:

1. Sleep bruxism (SB) is a masticatory muscle activity during sleep that is characterised as rhythmic (phasic) or non-rhythmic (tonic) and is not a movement disorder or sleep disorder in otherwise healthy individuals.
2. Awake bruxism (AB) is a masticatory muscle activity during wakefulness that is characterised by repetitive or sustained tooth contact and/or by bracing or thrusting of the mandible and is not a movement disorder in otherwise healthy individuals.

In addition, it was noted that both definitions begin with "masticatory muscle activity," a phrase intended to emphasise the role of the masticatory muscles during sleep and wakefulness, viz., muscle work as the source of potential positive or negative consequences.[6]

During the past two decades, "definite" SB has been diagnosed by means of polysomnography performed in sleep laboratories, or data have been gathered by ambulatory devices in the home environment. According to large epidemiological studies, "possible" SB is considered to be present in about 6%-8% in adult populations, with no significant gender difference and a slight decrease with age,[7,8] and half of the variation across populations has been suggested to be genetic in origin.[9-11] Also, SB reportedly has several underlying factors: psychological (eg dissatisfaction, stress sensitivity and anxiety),[12-14] behavioural and pharmacological (eg psychoactive medications, alcohol, tobacco and caffeine use),[15,16] and physiological (eg sleep disordered breathing and reflux disease).[17,18] These factors, with varying degrees of evidence for their role at present, are likely to act on an underlying genetic predisposition that is probably polygenic in nature given the lack of evidence for major genes, not to mention the lack overall of sufficiently powered genetic studies. Everyone has some degree of genetic predisposition and of triggering exposures, the relative mix varying for each patient. Yet, despite the many faces of SB, the problems involved in diagnosing it and the fact that it cannot be clearly linked to any pathophysiology or physiopathology,[19] it is commonly agreed that SB mostly manifests during light sleep and towards arousal.

AB in turn, with a reported prevalence up to 30% among adult populations,[20] has mainly been assessed by self-report, together with some specific clinical signs in the oral cavity (eg tongue scalloping, *linea alba* on buccal mucosa). Whereas SB appears to have a time-variant nature,[21] both that and the aetiology of AB have remained unclear. Therefore, in addition to the information derived from a single dental visit, patients are asked to keep a diary for 1-2 weeks to report their masticatory muscle activity (viz., teeth clenching, mandible bracing/thrusting even without tooth contact). For this purpose, a smart phone app with a so-called ecological momentary assessment function was recently introduced.[22-24] Based on repeated random alerts during wakefulness, the app seems promising for the study of real-time AB and its correlates on a larger scale, but may also serve as a possible tool for ecological momentary intervention in the management of AB by individuals.[24]

Even though SB and AB have commonly been considered as distinctly different entities, co-occurrence up to 20% has been reported.[25] In order to clarify this issue, we aimed, as part of a nationwide study among older Finnish twins, to examine whether self-reported SB and AB overlapped within individuals, and whether they had shared correlates. An additional goal was to analyse twin similarity by computing polychoric correlations of SB and AB bruxism traits by zygosity (monozygotic (MZ) and dizygotic (DZ) pairs) and sex. We also computed crosstwin-crosstrait correlations to explore genetic influences shared by SB and AB.

2 | MATERIAL AND METHODS

In the Finnish Twin Cohort (17), a fourth wave of data collection by postal questionnaire was conducted in 2011-2012. The questionnaire was mailed to all twins living in Finland who had been in the cohort in 1975, irrespective of response to earlier questionnaires. Of 11 766 twins born 1945-1957, 8410 (3753 men, 4657 women) returned the completed questionnaire. The response rate was 72%,[26] with mean age 60 years (range 53-67).

The domains examined covered multiple areas of health and risk factors (eg blood pressure and cholesterol measurements, diabetes, breathlessness, back pain, chronic diseases, use of common medications). Questions on sleep and bruxism (see below) were included. We derived smoking status based on detailed smoking history, and there were also questions on alcohol quantity, intake frequency and problematic drinking. Life events and traumatic experiences in childhood and adulthood were also asked about, as well as items on social interactions. We used a four-item life satisfaction scale and a brief assessment of extraversion and neuroticism, and also the Center for Epidemiologic Studies Depression (CES-D) depression scale.

Our analyses focused on four areas of interest: general health, health behaviours (viz., smoking habits, alcohol usage and coffee consumption), sleep issues and a broad sweep of psychosocial variables (viz., personality, depression, social interactions and history of traumatic experiences). The full questionnaire and items used in the analyses for the present study can be seen in detail at www.twinstudy.helsinki.fi. Ethical approval was obtained from the Ethics committee of the Helsinki and Uusimaa Hospital District. The study purpose was explained to the participants, and a returned questionnaire was taken as informed consent. All participants were aware they could withdraw from the study at any time without any consequence to themselves.

3 | STATISTICAL METHODS

We used logistic regression analyses to explore the associations between putative explanatory factors and the frequency of SB and AB, assessed using five-point questions as follows:

1. "Have you noticed or has someone told you (a person who sleeps with you) that you grind or clench your teeth while you're sleeping?" (1 = every night or almost every night, 2 = 3-5 nights weekly, 3 = 1-2 nights weekly, 4 = less than once a week, 5 = less than once a month or never).
2. "'Do you clench your teeth during wakefulness? (1 = every day or almost every day, 2 = 3-5 days weekly, 3 = 1-2 days weekly, 4 = less than once a week, 5 = less than once a month or never).

Four outcome variables were computed, two for SB and two for AB. We categorised and labelled them as (a) "any" sleep/awake bruxism (options 1-4) vs "rarely or none" (option 5), and (b) "frequent" sleep/awake bruxism (options 1 and 2) vs "rarely or none" (option 5). We standardised continuous variables to unit variances to enable comparisons of the strength of the associations across variables. The odds ratio thus provides an estimate of the change in odds per one standard deviation. Categorical variables were analysed using dummy binary variables, to give odds ratio estimates for each category relative to the chosen reference category.

Finally, we analysed twin similarity by computing polychoric correlations of the bruxism traits by zygosity and sex. Zygosity was determined by a validated questionnaire method.[27] Also, to examine whether AB and SB share a common genetic aetiology, we computed crosstwin-crosstrait correlations to determine the genetic correlation between them. This analysis allowed us to decompose the phenotypic correlation between AB and SB into genetic and environmental components and to examine to what extent shared genes accounted for the greater than expected co-occurrence between SB and AB prevalence.

4 | RESULTS

Any SB was reported by 14.8% (n = 1221) and frequent SB by 5.0% (n = 412). Percentages for any and frequent AB were 18.4% (n = 1532) and 6.3% (n = 522), respectively. Overall, both SB and AB were slightly more often reported by women, without significant difference between the sexes. The proportion of participants reporting both SB and AB frequently was 29.5% among all persons with either frequent SB or frequent AB or both. The corresponding co-occurrence for any AB and SB within individuals was slightly less.

Bruxism behaviours were not overall associated with sleep duration. However, perceived poor sleep quality was positively associated with both SB and AB, but showed higher ORs for AB than SB. Similarly, snoring, sleep apnoeas and insomnia were positively associated with both. However, the associations were stronger for sleep apnoeas with SB than AB, while ORs were higher for AB than SB in relation to insomnia. In addition, a clear evening chronotype was positively associated with all studied bruxism. Odds ratios and their corresponding 95% confidence intervals are shown in Table 1.

Several psychological variables related to mood (viz., depressive mood, neuroticism, negative life events and dissatisfaction with sex life) were positively associated with all studied SB and AB. Also, all various traumatic events reported were positively associated with reports of any SB and AB. These included serious accidents, either in childhood or in adulthood, violent crime in adulthood and sexual harassment. Parental divorce or separation showed an association with AB but not SB. Likewise, those participants who were satisfied with life reported SB and AB significantly less often. Extraversion showed a protective association with AB but no association with SB. Details are shown in Table 2.

Regarding social, lifestyle and health variables, we found that married participants reported SB and AB significantly less often, and those still in work life reported AB less often. Alcohol consumption and related negative consequences (heavy drinking, passouts and hangovers) were positively associated with all studied SB and AB. For alcohol problems, the associations appeared stronger for AB than SB. Similarly, smoking status and a measure of nicotine dependence were positively associated with both SB and AB, but not coffee or tea use. Psychotropic medication use (viz., sedatives and antidepressants) was positively associated with all studied SB and AB. Poor subjective health was associated with SB and somewhat more strongly with AB. Details are shown in Table 3.

Polychoric analyses examined twin similarity among MZ and DZ pairs for both SB and AB. For SB, the intra-class correlation was 0.366 in MZ pairs and 0.200 in DZ pairs, with very similar estimates in male and female pairs. The higher correlation in MZ than in DZ pairs suggests the influence of additive genetic factors on SB. For AB, the MZ correlation was 0.305, and 0.083 in DZ pairs, which is a finding consistent with the influence of both additive and non-additive genetic factors. However, given that MZ correlations were between 0.366 and 0.305, environmental factors (including measurement error) accounted for around two-thirds of the variation in liability to SB and AB. In addition, we observed a crosstwin-crosstrait correlation among MZ pairs twice as large as among DZ pairs, indicating a substantial genetic correlation between the two phenotypes (Table 4).

5 | DISCUSSION

Our main findings were the substantial correlates of behavioural and lifestyle factors with both SB and AB, with fairly similar risk factor profiles. There was also substantial co-occurrence between the conditions, such that nearly one-third of persons reporting either condition had both. The role of genetic factors, based on twin correlations, was fairly similar but relatively modest for both SB and AB. Also, SB and AB appear to share some genetic influences. This suggests that environmental factors play a major role in the aetiology of bruxism behaviours.

TABLE 1 Sleep and awake bruxism in relation to sleep related issues

	Sleep bruxism		Awake bruxism	
	OR (95% CI):			
	Any vs rarely or none	frequent vs rarely or none	Any vs rarely or none	frequent vs rarely or none
Sleep duration				
6 h or less	1.05 (0.83-1.32)	1.29 (1.05-1.59)	1.03 (0.71-1.45)	1.60 (1.17-2.20)
6.5 h	1.02 (0.82-1.28)	1.23 (1.05-1.58)	0.80 (0.55-1.45)	1.09 (0.78-1.54)
7 h	0.91 (0.75-1.09)	1.08 (0.91-1.23)	0.84 (0.69-1.02)	0.95 (0.71-1.27)
7.5 h	1.00	1.00	1.00	1.00
8 h	0.95 (0.78- 1.15)	0.97 (0.81-1.17)	0.95 (0.70-1.30)	1.05 (0.78-1.40)
8.5 h	0.91 (0.68-1.20	0.87 (0.67-1.15)	0.95 (0.61-1.47)	0.85 (0.55-1.33)
9 h	1.13 (0.82-1.55)	1.34 (1.00- 1.79)	0.87 (0.50-1.50)	1.38 (0.88-2.16)
9.5 h	1.80 (1.03-3.13)	1.16 (0.65-2.08)	1.08 (0.38-3.05)	1.59 (0.71-3.58)
10 h or plus	1.37 (0.82-2.26)	1.37 (0.82-2.26)	1.65 (0.80-3.41)	1.25 (0.59-2.68)
Sleep quality				
Good	1.00	1.00	1.00	1.00
Somewhat good	1.18 (1.00-1.39)	0.93 (0.72-1.21)	1.52 (1.30-1.79)	1.15 (0.90-1.46)
Slighty poorly	1.78 (1.45-2.17)	1.60 (1.17-2.19)	2.37 (1.96-2.87)	2.15 (1.61-2.86)
Poor	1.92 (1.42-2.60)	1.67 (1.06-2.65)	3.22 (2.46-4.22)	2.90 (1.96-4.31)
Snoring				
Never	1.00	1.00	1.00	1.00
Sometimes	1.61 (1.26-2.07)	0.90 (0.62-1.29)	1.58 (1.27-1.96)	1.24 (0.89-1.73)
Often	2.74 (2.09-3.59)	2.07 (1.40-3.05)	1.82 (1.44-2.32)	1.62 (1.11-2.35)
Almost always	2.72 (2.00-3.70)	3.45 (2.27-5.23)	1.71 (1.30-2.26)	1.57 (1.03-2.42)
Sleep apnea				
Every or almost every night	1.98 (1.35-2.90)	3.69 (2.19-6.22)	1.28 (0.88-1.87)	1.72 (0.99-3.00)
3-5 nights per week	2.65 (1.74-4.04)	3.56 (1.90-6.22)	1.70 (1.11-2.60)	1.27 (0.58-2.80)
1-2 nights per week	2.34 (1.65-3.29)	1.36 (0.65-2.86)	1.50 (1.07-2.11)	1.32 (0.73-2.39)
<1 night per week	1.81 (1.35-2.44)	1.43 (0.8-2.56)	1.33 (1.00-1.76)	1.37 (0.86-2.19)
<1 night per month or never	1.00	1.00	1.00	1.00
Insomnia				
Every or almost every night	1.83 (1.43-2.34)	2.00 (1.38-2.88)	2.51 (2.00-3.14)	3.25 (2.37-4.46)
3-5 nights per week	1.77 (1.38-2.28)	1.49 (0.98-2.26)	2.73 (2.18-3.43)	2.82 (2.00-4.00)
1-2 nights per week	1.66 (1.38-1.98)	1.43 (1.06-1.92)	2.37 (2.00-2.81)	2.14 (1.64-2.80)
<1 night per week	1.82 (1.02-1.38)	1.02 (0.79-1.33)	1.81 (1.57-2.07)	1.53 (1.22-3.96)
<1 night per month or never	1.00	1.00	1.00	1.00
Chronotype				
Clearly morning	1.00	1.00	1.00	1.00
Somewhat morning	0.96 (0.82-1.13)	0.81 (0.61-1.06)	0.99 (0.85-1.15)	0.89 (0.70-1.14)
Somewhat night	1.16 (0.99-1.36)	1.05 (0.81-1.37)	1.21 (1.05-1.40)	1.12 (0.88-1.43)
Clearly night	1.25 (1.04-1.51)	1.36 (1.01-1.82)	1.23 (1.04-1.47)	1.38 (1.06-1.79)

Note: Logistic regression, adjusted by age and sex. Odds ratios and their 95% confidence intervals. Complete frequency distributions are available from the authors upon request.

TABLE 2 Sleep and awake bruxism in relation to psychological issues

	Sleep bruxism		Awake bruxism	
	OR (95% CI):			
	Any vs rarely or none	frequent vs rarely or none	Any vs rarely or none	frequent vs rarely or none
Serious traffic accident				
None	1.00	1.00	1.00	1.00
Child	0.91 (0.62-1.35)	0.82 (0.42-1.60)	1.26 (0.90-1.75)	1.67 (1.05-2.66)
None	1.00	1.00	1.00	1.00
Adult	1.19 (0.95-1.49)	1.16 (0.80-1.69)	1.43 (1.18-1.74)	1.32 (0.97-1.81)
Other serious accident				
None	1.00	1.00	1.00	1.00
Child	1.72 (1.26-2.36)	1.61 (0.96-2.67)	1.41 (1.05-1.89)	1.29 (0.79-2.10)
None	1.00	1.00	1.00	1.00
Adult	1.43 (1.14-1.80)	1.41 (0.97-2.04)	1.43 (1.17-1.75)	1.58 (1.16-2.15)
Fire or convulsion of nature				
None	1.00	1.00	1.00	1.0
Child	1.09 (0.77-1.54)	1.02 (0.59-1.77)	1.00 (0.73-1.35)	0.88 (0.52-1.51)
None	1.00	1.00	1.00	1.00
Adult	1.21 (0.91-1.61)	0.96 (0.58-1.57)	1.18 (0.91-1.52)	0.99 (0.64-1.55)
Injury by violence				
None	1.00	1.00	1.00	1.00
Child	1.51 (1.08-2.10)	1.38 (0.80-2.38)	1.79 (1.33-2.41)	2.30 (1.51-3.51)
None	01:00	1.00	01:00	01:00
Adult	1.59 (1.29-1.97)	2.10 (1.55-2.83)	1.69 (1.39-2.05)	1.87 (1.40-2.50)
Sexual harrassment				
None	1.00	1.00	1.00	1.00
Child	1.42 (0.96-2.09)	1.42 (0.81-2.47)	1.53 (1.06-2.19)	1.66 (0.98-2.79)
None	1.00	1.00	1.00	1.00
Adult	1.77 (1.39-2.26)	2.35 (1.66-3.33)	1.74 (1.38-2.20)	2.07 (1.48-2.88)
Violent crime with a gun, knife or else				
None	1.00	1.00	1.00	1.00
Child	0.63 (0.18-2.17)	0.57 (0.07-4.41)	1.17 (0.47-2.93)	2.26 (0.78-6.56)
None	1.00	1.00	01:00	1.00
Adult	1.36 (0.91-2.03)	1.43 (0.78-2.63)	1.53 (1.08-2.17)	1.96 (1.21-3.17)
Divorced or separated parents				
No	1.00	1.0	1.00	1.00
Child	1.05 (0.80-1.39)	1.34 (0.90-1.98)	1.27 (1.01-1.61)	1.16 (0.78-1.72)
No	1.00	1.00	1.00	1.00
Adult	1.06 (0.78-1.44)	1.01 (0.61-1.67)	1.32 (1.00-1.75)	1.65 (1.12-2.42)
Other shocking event				
None	1.00	1.00	1.00	1.00
Child	1.25 (0.96-1.62)	1.28 (0.85-1.92)	1.40 (1.10-1.76)	1.68 (1.19-2.37)
None	1.00	1.00	1.00	1.00
Adult	1.23 (1.05-1.45)	1.19 (0.92-1.54)	1.42 (1.23-1.64)	1.50 (1.20-1.89)

(Continues)

TABLE 2 (Continued)

	Sleep bruxism		Awake bruxism	
	OR (95% CI):			
	Any vs rarely or none	frequent vs rarely or none	Any vs rarely or none	frequent vs rarely or none
CES-D (depression) (continuous)	1.30 (1.23-1.38)	1.26 (1.15-1.38)	1.50 (1.42-1.58)	1.59 (1.48-1.71)
Extroversion (continuous)	0.96 (0.90-1.02)	0.94 (0.85-1.05)	0.85 (0.80-0.90)	0.80 (0.73-0.88)
Neuroticism (continuous)	1.31 (1.23-1.39)	1.30 (1.18-1.44)	1.55 (1.47-1.64)	1.80 (1.65-1.96)
Negative life events (continuous)	1.36 (1.28-1.44)	1.33 (1.21-1.46)	1.40 (1.32-1.48)	1.47 (1.36-1.60)
Life dissatisfaction (continuous)	1.23 (1.17-1.31)	1.22 (1.12-1.34)	1.35 (1.29-1.42)	1.42 (1.32-1.54)
Has support from friend				
Yes	1.00	1.00	1.00	1.00
No	0.87 (0.67-1.14)	0.67 (0.45-1.00)	0.91 (0.72-1.16)	0.74 (0.52-1.07)
Has someone to share feelings with				
Yes	01:00	01:00	01:00	01:00
No	0.85 (0.70-1.03)	0.93 (0.67-1.29)	0.79 (0.66-0.93)	0.72 (0.56-0.95)
Sex life				
Very satisfying	1.00	1.00	1.00	1.00
Somewhat satisfying	1.02 (0.85-1.22)	0.79 (0.59-1.05)	1.15 (0.97-1.36)	1.08 (0.81-1.42)
Neither satisfying nor unsatisfying	1.41 (1.61-1.71)	1.19 (0.88-1.61)	1.52 (1.27-1.82)	1.48 (1.10-1.98)
Somewhat unsatisfying	1.59 (1.24-2.05)	1.06 (0.69-1.64)	1.94 (1.54-2.45)	1.98 (1.37-2.86)
Very unsatisfying	1.81 (1.39-2.36)	1.94 (1.33-2.83)	2.05 (1.60-2.62)	2.54 (1.77-3.64)

Note: Logistic regression, adjusted by age and sex. Odds ratios and their corresponding 95 % confidence intervals (for continuous variables calculated by an additional standard deviation). Significant associations highlighted. Complete frequency distributions are available from the authors upon request.

Over two decades ago, Lavigne and Montplaisir[7] reported the prevalence of SB (at present considered as "possible" SB) to be about eight per cent, being at highest at 20-40 years, without significant difference between sexes, and declining with age. The commonly cited prevalence figures from this representative nationwide study among Canadians have been considered the most representative to date. According to a recent systematic review, AB is reportedly more common than SB, with a prevalence of about 22%-30%.[20] However, the review's authors pointed out that the figures should be interpreted with caution due to the poor methodological quality of the reviewed literature. Their criticism mainly targeted the use of self-reported data. As with a vast number of diseases, it should be noted that the prevalence of both SB and AB (bearing in mind their non-disease entities by definition) may vary with place, time and characteristics of the population, including age, sex, genetic liability and external influences.

In the present study, based on questionnaire data derived from 8410 twins aged 53-67 years, the prevalences of frequent SB and any AB were both in line with the previous literature.[7,20] Bearing in mind that the figures imply "possible" bruxism, according to the international consensus,[1] there is no other feasible method yet to gather data on bruxism behaviours on such a large scale other than using self-reports. Our study consisted of the nationwide Finnish twin cohort data with a comprehensive set of validated questions, and the study population is representative of the Finnish population at same age.[26,28,29]

TABLE 3 Sleep and awake bruxism in relation to health and lifestyle issues, marital status, work, and health. Logistic regression, adjusted by age and sex

	Sleep bruxism		Awake bruxism	
	OR (95% CI):			
	Any vs rarely or none	frequent vs rarely or none	Any vs rarely or none	frequent vs rarely or none rarely or none
Alcohol consumption (continuous)	1.14 (1.06-1.23)	1.13 (1.00-1.28)	1.13 (1.06-1.21)	1.12 (1.01-1.25)
Daily coffee consumption (continuous)	1.01 (0.94-1.07)	0.98 (0.88-1.09)	0.98 (0.92-1.04)	1.04 (0.95-1.13)
Daily tea consumption (continuous)	0.94 (0.87-1.01)	0.89 (0.79-1.00)	1.01 (0.95-1.08)	1.00 (0.90-1.10)
Heavy smoking index (HSI) (continuous)	1.18 (1.09-1.29)	1.23 (1.07-1.41)	1.20 (1.11-1.30)	1.26 (1.13-1.42)
Marital status				
Single	1.00	1.00	1.00	1.00
Married	0.76 (0.62-0.92)	0.68 (0.51-0.92)	0.68 (0.57-0.81)	0.58 (0.45-0.76)
Married again	0.83 (0.56-1.22)	0.94 (0.52-1.67)	0.74 (0.52-1.06)	0.74 (0.43-1.28)
Cohabiting	0.76 (0.58-0.99)	0.58 (0.37-0.91)	0.80 (0.63-1.02)	0.72 (0.50-1.05)
Divorced or separated	1.08 (0.86-1.36)	1.09 (0.76-1.55)	1.01 (0.81-1.25)	1.09 (0.79-1.49)
Widowed	0.89 (0.63-1.25)	0.76 (0.43-1.33)	0.80 (0.58-1.10)	0.72 (0.44-1.19)
Working				
No	1.00	1.00	1.00	1.00
Yes	0.96 (0.84-1.11)	0.86 (0.69-1.08)	0.86 (0.76-0.98)	0.65 (0.53-0.80)
Subjective health				
Very good	1.00	1.00	1.00	1.00
Somewhat good	0.92 (0.77-1.11)	0.93 (0.68-1.26)	1.30 (1.08-1.55)	1.29 (0.96-1.73)
Moderate	1.51 (1.25-1.83)	1.64 (1.20-2.24)	1.99 (1.65-2.40)	2.24 (1.65-3.03)
Somewhat poor	1.79 (1.35-2.36)	1.52 (0.94-2.45)	2.29 (1.75-2.99)	2.77 (1.84-4.17)
Poor	0.70 (0.24-2.03)	0.53 (0.07-3.95)	1.79 (0.85-3.76)	1.87 (0.55-6.36)
Diabetes (DM II)				
No	1.00	1.00	1.00	1.00
Yes	1.20 (0.96-1.51)	1.36 (0.96-1.94)	1.35 (1.10-1.66)	1.48 (1.08-2.02)
Elevated blood pressure				
No	1.00	1.00	1.00	1.00
Yes	1.09 (0.97-1.24)	1.02 (0.83-1.25)	1.16 (1.04-1.30)	1.26 (1.05-1.51)
Psychotropic medication use				
Sedatives				
No	1.00	1.00	1.00	1.00
Yes	1.69 (1.45-1.96)	1.82 (1.43-2.33)	1.85 (1.61-2.11)	2.40 (1.96-2.94)
Antidepressants				
No	1.00	1.00	1.00	1.00
Yes	2.15 (1.77-2.62)	2.55 (1.91-3.40)	2.15 (1.79-2.58)	2.99 (2.32-3.85)
Smoking status				
Never	1.00	1.00	1.00	1.00
Occasionally	1.14 (0.87-1.50)	1.01 (0.64-1.60)	1.37 (1.08-1.74)	1.60 (1.08-2.36)
Former	1.27 (1.09-1.48)	1.38 (1.09-1.76)	1.39 (1.21-1.60)	1.90 (1.52-2.38)
Current	1.69 (1.43-2.00)	1.60 (1.22-2.11)	1.70 (1.45-1.99)	2.42 (1.89-3.11)

(Continues)

TABLE 3 (Continued)

	Sleep bruxism		Awake bruxism	
	OR (95% CI):			
	Any vs rarely or none	frequent vs rarely or none	Any vs rarely or none	frequent vs rarely or none rarely or none
Heavy drinking				
No	1.00	1.00	1.00	1.00
Yes	1.33 (1.15-1.54)	1.28 (1.00-1.63)	1.31 (1.15-1.49)	1.52 (1.23-1.86)
Passed out within past 12 mo				
Never	1.00	1.00	1.00	1.00
Once	1.57 (1.20-2.04)	1.94 (1.30-2.89)	1.53 (1.20-1.95)	1.71 (1.19-2.47)
2-3 times	1.40 (1.01-1.95)	1.41 (0.82-2.43)	1.61 (1.20-2.15)	1.24 (0.74-2.06)
4 times	1.10 (0.54-2.26)	2.02 (0.80-5.12)	1.36 (0.74-2.50)	1.47 (0.58-3.74)
7 or more times	2.09 (1.20-3.66)	2.76 (1.19-6.43)	2.48 (1.48-4.15)	4.50 (2.37-8.53)
Hangover within past 12 mo				
Never	1.00	1.00	1.00	1.00
Once	1.39 (1.16-1.67)	1.35 (1.01-1.81)	1.30 (1.09-1.55)	1.15 (0.86-1.53)
2-3 times	1.18 (0.97-1.44)	0.97 (0.69-1.37)	1.29 (1.08-1.53)	1.36 (1.02-1.81)
4 times	1.40 (1.02-1.92)	1.45 (0.87-2.42)	1.97 (1.52-2.57)	2.22 (1.49-3.31)
7 or more times	1.53 (1.13-2.06)	1.45 (0.87-2.41)	2.07 (1.59-2.69)	2.85 (1.95-4.14)

Odds ratios and their 95% confidence intervals (for continuous variables calculated by an additional standard deviation). Significant associations highlighted. Complete frequency distributions are available from the authors upon request.

TABLE 4 Intra-class and crosstwin-crosstrait correlations for sleep and awake bruxism by zygosity and sex

Trait	Zygosity	All pairs (N = 2771)	Male (N = 1123)	Female (N = 1648)
Sleep bruxism	MZ (n = 1009)	0.366	0.362	0.373
	DZ (n = 1775)	0.200	0.214	0.284
Awake bruxism	MZ (n = 1020)	0.305	0.277	0.328
	DZ (n = 1795)	0.083	0.107	0.068
Crosstwin-crosstrait correlations	MZ (n = 1005)	0.246	0.283	0.222
	DZ (n = 1766)	0.114	0.139	0.098

Note: Column numbers are for pairs in the crosstwin-crosstrait correlations. For sleep and awake bruxism intra-class correlations, the numbers are slightly higher due to some pairs with missing data on one trait but not the other.

SB and AB have usually been considered as different entities without mutual pathophysiology or physiopathology of the bruxism behaviours, not to mention the origin of these oromotor muscle activities in the brain.[1,5] However, our findings demonstrate that both behaviours share quite similar correlate profiles. For example, perceived sleep quality was significantly associated with both SB and AB (any vs. never or less than a month). However, sleep apnoeas were not associated with any AB, which has not been reported earlier. A closer relationship between SB and apnoeas may indicate a possible role of SB in enhancing airway patency during sleep arousals or comorbidity.[17,18]

The master biological clock, located in the bilaterally paired suprachiasmatic nucleus of the anterior hypothalamus, controls the timing of sleep and waking in humans and regulates the 24-hour (ie circadian) behavioural, physiologic and biologic rhythms.[30] There are two extremes in the so-called chronotype profiles: morningness (individuals who wake up early, are at their most active early, and go to sleep early) and eveningness (individuals who wake up late, are most active later, and go to sleep late). These two profiles may not always be balanced with social activities and work duties, for example. In addition, several psychological factors may be associated with chronotype profiles.[31] Nevertheless, chronotype profiles reportedly become less distinct by age and morningness becomes more common with increasing age among adults.[32] However, in the present study among twins aged 53-67 years, the evening type still seemed to associate with both studied bruxism behaviours.

It is noteworthy that a wide array of psychosocial measures (viz., depression, neuroticism and negative life events) was significantly associated with all studied bruxism behaviours. This is in line with previous findings.[12,14] That the use of psychotropic medicines was significantly associated with all bruxism behaviour also accords with existing literature.[15,16] Interestingly, serious traumatic events both in adulthood and childhood were associated with bruxism behaviours overall. This should be noted; such events may become stored in the amygdala, the limbic system of the brain, and may have a life-long influence on bruxism behaviour too—an issue that needs to be borne in mind as a possible underlying factor when encountering a patient with a possible bruxism-related problem.

Furthermore, the results confirm previously reported significant associations between SB and smoking, and alcohol consumption.[16] In the present study, heavy drinkers, especially those with alcohol-related problems, also seemed to be at risk of more severe AB. All of which should be taken into account during a patient visit in terms of any treatment planning.

The present study summarised results from numerous age and sex adjusted logistic regression models. Corresponding large scale findings have not been reported earlier. Despite the models not being corrected for multiple testing, the reader is asked to focus on the strength of the found associations. It should also be noted that multiple correlates for both SB and AB were found to be very similar, but we also saw some non-shared items between studied variables and bruxism behaviours.

Interestingly, a substantial co-occurrence between SB and AB emerged, which raises the question of whether they also share a common genetic liability. The twin analyses suggested moderate genetic effects on both SB and AB and that there is a substantial genetic correlation between the two phenotypes.

Notwithstanding the evidence for genetic liability, external factors appear to play a major role in the aetiology of bruxism. However, it should be noted that the aetiology of bruxism could be unrelated to clinical consequences,[33] and moreover that other constructs (viz., risk or trigger factors to bruxism) should be borne in mind when a patient complains of symptoms that could be linked in involuntary masticatory muscle work, viz., bruxism.

In conclusion, SB and AB may be two phenotypes with similar origin. In addition to shared genetics, both are markedly associated with several risk factors. Some of the negative outcomes of bruxism behaviours related to the latter could perhaps be reduced by lifestyle changes. Psychological characteristics and a history of traumatic events should be taken into account in clinical work when facing a patient with a possible bruxism-related problem.

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CONFLICTS OF INTEREST

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AUTHORS' CONTRIBUTIONS

JA and JK drafted the manuscript on the basis of the statistical the analyses by JK. All other expert authors critically revised the text and made comments on the manuscript.

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