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Original Investigation

Bruxism Is Associated With Nicotine Dependence: A Nationwide Finnish Twin Cohort Study

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Abstract

Objectives: To investigate the association of smoking with bruxism while controlling for genetic and environmental factors using a co-twin-control design. Especially, the role of nicotine dependence was studied in this context.

Methods: The material derives from the *Finnish Twin Cohort* consisting of 12,502 twin individuals who responded to a questionnaire in 1990 (response rate of 77%). All were born in 1930–1957, the mean age being 44 years. The questionnaire covered 103 multiple choice questions, 7 dealing with tobacco use and 22 with sleep and vigilance matters, including perceived bruxism. In addition, a subsample derived from the Nicotine Addiction Genetics Finland Study containing 445 twin individuals was studied.

Results: In age- and gender-controlled multinomial logistic regression, both monthly and rarely reported bruxism associated with both current cigarette smoking (odds ratio [*OR*] = 1.74 and 1.64) and former cigarette smoking (*OR* = 1.64 and 1.47). Weekly bruxism associated with current smoking (*OR* = 2.85). Current smokers smoking 20 or more cigarettes a day reported weekly bruxism more likely (*OR* = 1.61–1.97) than those smoking less. Among twin pairs (N = 142) in which one twin was a weekly bruxer and the cotwin a never bruxer, there were 13 monozygotic pairs in which one twin was a current smoker and the other twin was not. In all cases, the bruxer was the smoker (p = .0003). Nicotine dependence associated significantly with bruxism.

Conclusions: Our twin study provides novel evidence for a possible causal link between tobacco use and bruxism among middle-aged adults. Nicotine dependence may be a significant predisposing factor for bruxism.

Introduction

A limited number of epidemiological reports, varying in the study design, sample size, definition of smoking, assessment of bruxism, and control for covariates, show some degree of association between tobacco use and bruxism (J. Ahlberg, Savolainen, Rantala, Lindholm, & Kononen, 2004; K. Ahlberg et al., 2005; Johansson et al., 2004; Lavigne, Lobbezoo, Rompre, Nielsen, & Montplaisir, 1997; Molina et al., 2001; Ohayon, Li, & Guilleminault, 2001; Rintakoski et al., 2010), providing evidence that cigarette smokers may have higher rates of bruxism. None of these studies, however, have formally evaluated nicotine dependence and its association with bruxism.

A possible underlying mechanism exists to explain the association between smoking and bruxism: In smokers, nicotine accumulates in the body during the time spent awake, decreasing gradually during sleep. Nicotine induces acetylcholine and glutamate synaptic transmission and enhances dopamine release (Li, Mao, & Wei, 2008). In turn, this may influence the genesis of bruxism in a dose-dependent manner and further, higher levels of smoking, leading to increased levels of nicotine and dopamine release, could be more strongly related to bruxism.

Recently, based on a representative population-based data set, we reported an association of the dose–effect relationship of tobacco use and bruxism in young adults (Rintakoski et al., 2010). However, the association between these two may also arise from other factors common to both, such as the genetic variability known to underlie both smoking (Rose, Broms, Korhonen, Dick, & Kaprio, 2009) and bruxism (Hublin, Kaprio, Partinen, Heikkilä, & Koskenvuo, 1998). Thus, the aim of the present study was to examine smoking behavior and nicotine

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dependence as potential risk factors for bruxism and to study whether the association is accounted for by such shared genes.

Materials and Methods

Material

The material of the present study derives from the *Finnish Twin Cohort*. In 1990, 12,502 twin individuals responded to a questionnaire (response rate of 77%). All twins were born in 1930–1957, the mean age was 44 years, and all twins resided in Finland in 1987 as described earlier (Hublin, Kaprio, Partinen, Heikkilä, & Koskenvuo, 1997; Hublin et al., 1994). Zygosity was determined by an accurate and validated questionnaire method (Sarna, Kaprio, Sistonen, & Koskenvuo, 1978), which leaves some about 7% of twin pairs unclassified and has a misclassification probability as low as 1.7%. The validity of the questionnaire was further studied in a subsample, using 11 blood markers (Sarna et al., 1978). The ethical committee of the Department of Public Health, University of Helsinki, approved the study protocol. Subjects were informed of the study aims and all provided consent.

The questionnaire sent to the twins consisted of 103 multiple choice questions, of which 7 about tobacco use and 22 about sleep and vigilance matters, including perceived bruxism (Hublin & Kaprio, 2003). The frequency of tooth grinding during sleep was assessed as follows: "weekly," "monthly," "occasionally," "never," or "I do not know." The group of "never bruxers" was used as the reference category in the analyses, while those who did not know (n = 1,817) were excluded. Three outcomes were defined: weekly bruxism, monthly and rarely bruxism (i.e., occasionally), and never bruxism—the reference category.

Tobacco use was evaluated as follows: Never-smoker status was determined by asking "Have you smoked more than 5–10 packs of cigarettes during your entire life?". Participants who had smoked less than 5–10 packs (i.e., 100–200 cigarettes) were categorized as never-smokers. Former smokers indicated that they had smoked regularly, that is, daily or almost daily. Subjects who had never smoked regularly but could not be classified as never-smokers were categorized as occasional smokers. Current smokers were asked to report the number of smoked cigarettes per day. The response options were (a) none, (b) less than 5, (c) 5–9, (d) 10–14, (e) 15–19, (f) 20–24, (g) 25–39, and (h) more than 40. We classified current smokers as light smokers (less than 10), smokers of 10–19 cigarettes, smokers of 20–24 cigarettes daily, and heavy smokers (at least 25 cigarettes daily). We asked the age when they had started smoking regularly. Among former smokers, we asked the age of cessation and the amount smoked prior to quitting, creating the same four categories as for current smokers. Lifetime pipe or cigar smoking was used as a dichotomy and defined as someone reporting having ever smoked at least 50 cigars, 75 cigarillos, or more than 3–5 packages of pipe tobacco (Hukkinen, Kaprio, Broms, Koskenvuo, & Korhonen, 2009).

Subsample With Nicotine Dependence Data

The Nicotine Addiction Genetics (NAG; an international consortium among Finland, Australia, and United States) Finland Study forms a subsample that is based on earlier health questionnaires of the Finnish Twin Cohort Study, including also opposite-sex twin pairs (Kaprio & Koskenvuo, 2002). Ever smoking twin pairs, concordant for heavy smoking, were recruited to NAG Finland Study and were interviewed by telephone during 2001–2005 (Broms et al., 2007; Loukola et al., 2008). Diagnostic *DSM-IV* nicotine dependence, major depressive disorder, and alcohol dependence (American Psychiatric Association, 1994) were assessed by a psychiatric diagnostic interview, the SSAGA (Semi-Structured Assessment for the Genetics of Alcoholism; Bucholz et al., 1994), with the section of nicotine use and dependence based on the Composite International Diagnostic Interview (Cottler et al., 1991). The Finnish NAG subsample contains 445 twin individuals, of whom data on bruxism were available from the 1990 questionnaire. The mean age of the participants in the NAG Finland Study was 53.7 years (SD = 4.6). The ethical committee of the Hospital District of Helsinki and Uusimaa approved the study protocol in 2001.

In addition to the standard epidemiological analyses of the relationship of smoking with bruxism in individuals (see below), we utilized the twin data to analyze the risk of weekly bruxism using twin pairs discordant for smoking status, viz., examining the ratio of the number of pairs in which a smoking twin reports bruxism (at least weekly), while the co-twin neither smokes nor experiences bruxism, contrasted with the number of pairs in which the opposite was true: A smoking twin does not report bruxism, while the co-twin does not smoke but experiences bruxism. We identified 142 twin pairs discordant for bruxism, among whom the distribution of smoking status was examined.

Statistical Methods

We studied associations between smoking and bruxism using cross-tabulations and the Pearson chi-square test of independence, corrected for clustered sampling of twins within pairs, which is expressed as an F ratio (Rao, 1984). The association of tobacco use and bruxism was assessed using multinomial logistic regression models (Hosmer & Lemeshow, 2000) that controlled for age and sex as there were three outcome categories (at least weekly, monthly, and rarely) and never bruxism as the reference category. Odds ratios (*OR*s) of all models were adjusted for correlated observations within twin pairs by means of the statistical software package Stata 9.0 (StataCorp, 2005), using a robust estimator of variance. Conditional logistic regression models were used to obtain *OR*s for the risk of bruxism in relation to tobacco use and nicotine dependence in twin pairs discordant for bruxism. In the absence of covariates, this is equivalent to a McNemar chi-square test for matched pairs.

Results

Of the current smokers, 27.8 % of males and 19.9 % of females smoked 20 or more cigarettes daily, while 15.4% of males and 36.7% of females smoked less than 10 cigarettes daily. Bruxism was more frequent among cigarette smokers in both genders. Similarly, bruxism was more frequent among current heavy smokers than among current light smokers (Table 1).

In the age- and gender-controlled multinomial logistic regression, both monthly and rarely reported bruxism associated with both current cigarette smoking (*OR* = 1.74 [95% *CI* = 1.37–2.22] and1.64 [95% *CI* = 1.44–1.86], respectively) and former smoking

Table 1. Percentages of Cigarette Smoking Status and Amount Smoked in Adulthood For "Weekly," "Monthly," "Rarely," and "never" Bruxing in Males and Females

	Bruxism in males (%)				Bruxism in females (%)			
	Weekly	Monthly	Rarely	Never	Weekly	Monthly	Rarely	Never
	n = 188	*n* = 224	*n* = 967	*n* = 3,250	*n* = 224	*n* = 216	*n* = 979	*n* = 3,907
Smoking (*n* = 9,955)								
Never	2.4	4.1	16.9	76.6	3.3	3.1	16.1	77.5
Occasional	1.1	2.3	18.2	78.4	1.3	4.5	22.4	71.8
Former	3.5	5.0	23.1	68.5	3.5	5.8	20.6	70.2
Current	6.8	5.9	23.8	63.5	7.5	5.0	22.0	65.4
Cigarettes per day among current smokers (*n* = 2,604)								
<10	4.1	5.0	25.2	65.8	7.3	4.5	21.6	66.7
10–19	6.3	5.2	22.2	66.3	6.6	3.8	22.8	66.9
20–24	8.2	6.3	22.3	63.2	8.9	10.1	21.9	59.2
≥25	8.0	7.6	28.7	55.8	14.3	4.8	17.5	63.5

(*OR* = 1.64 [95% *CI* = 1.27–2.11] and 1.47 [95% *CI* = 1.29–1.67], respectively), while weekly bruxism was associated with current smoking (*OR* = 2.85 [95% *CI* = 2.26–3.61]; Table 2, model I). There was no significant effect of gender, while age, adjusted for smoking status, was associated with monthly bruxism. Daily pipe smokers also had a higher risk for weekly bruxism (*OR* = 1.65, 95% *CI* = 1.04–2.64; Table 2, model II). Current smokers smoking 20 or more cigarettes per day were 1.61 (95% *CI* = 1.02–2.54) to

Table 2. Multinomial Logistic Regression: Independent Effects of Cigarette, Pipe, and Cigar Smoking Habits on "Weekly," "Monthly," and "Rarely" Reported Bruxism Compared With Never Bruxism as the Reference Category. Adjusted by Age and Gender

	Model I: cigarette smoking (*n* = 9,955)			Model II: cigarette, cigar, and pipe smoking (*n* = 9,762)		
	Odds ratio	95% *CI*	*p* Value	Odds ratio	95% *CI*	*p* Value
Weekly						
Sex (female)	1.12	0.91–1.38	.27	1.16	0.94–1.43	.17
Age	0.99	0.98–1.01	.22	0.99	0.98–1.01	.32
Smoking status:						
Never smoker	1.00			1.00		
Occasional	0.41	0.15–1.13	.09	0.42	0.15–1.14	.087
Former	1.33	0.99–1.77	.06	1.32	0.99–1.77	.060
Current	2.85	2.26–3.61	<.001	2.78	2.18–3.54	<.001
Pipe/cigar	—	—	—	1.65	1.04–2.64	.035
Monthly						
Sex (female)	0.87	0.71–1.07	.18	0.86	0.70–1.06	.166
Age	0.97	0.96–0.99	<.001	0.98	0.96–0.99	<.001
Smoking status						
Never smoker	1.00			1.00		
Occasional	0.92	0.49–1.73	.81	0.76	0.38–1.51	.431
Former	1.64	1.27–2.11	<.001	1.65	1.27–2.13	<.001
Current	1.74	1.37–2.22	<.001	1.76	1.38–2.25	<.001
Pipe/cigar	—	—	—	0.95	0.53–1.70	.856
Rarely						
Sex (female)	0.92	0.83–1.02	.12	0.92	0.82–1.02	.120
Age	1.00	0.99–1.00	.19	1.00	0.99–1.00	.242
Smoking status:						
Never smoker	1.00			1.00		
Occasional	1.24	0.93–1.64	.14	1.23	0.93–1.64	.152
Former	1.47	1.29–1.67	<.001	1.47	1.29–1.68	<.001
Current	1.64	1.44–1.86	<.001	1.64	1.44–1.86	<.001
Pipe/cigar	—	—	—	1.10	0.82–1.48	.525
Never bruxism	1.00	reference category		1.00	reference category	

1.97 (95% *CI* = 1.16–3.37) times more likely to report bruxism weekly than the lightest smokers. Smokers smoking 20–24 cigarettes/day also had 1.71 (95% *CI* = 1.01–2.90) times more often monthly bruxism (Table 3).

Pairwise analyses and conditional logistic regression indicated that within twin pairs ($n = 142$: monozygotic pairs: $n = 49$, dizygotic pairs: $n = 86$, seven pairs were of uncertain zygosity and were omitted from zygosity-specific analyses), smoking was associated with bruxism. Among the monozygotic pairs, in which one twin was a weekly bruxer and the other twin never bruxed, there were 13 pairs in which one twin was a current smoker and the other twin was not. In all cases, the bruxer was the smoker (McNemar chi-square test, $p = .0003$).

In the NAG data, consisting of 445 ever-smokers, those with a *DSM-IV* diagnosis of nicotine dependence were two and half times more often weekly bruxers compared with those ever-smokers without such a diagnosis (*OR* = 2.50, 95% *CI* = 1.06–5.87). Further adjustment for a diagnosis of alcohol dependence weakened the relationship with weekly bruxism but strengthened the relationship with rarely bruxism (Table 4). Due to the selection procedure of the NAG sample, no comparison with never-smokers could be made. Analyses were age and sex adjusted, showing no significant effect of either covariate. Because of the relatively small sample size, we also analyzed the presence of any bruxism (vs. none) in relation to a diagnosis of nicotine dependence. This yielded an *OR* of 1.57 (95% *CI* = 1.05–2.35, $p = .028$), and after adjustment for depression and alcohol dependence, the association remained (*OR* = 1.56, 95% *CI* = 1.20–2.38, $p = .040$).

Discussion

This study provides an assessment of a unique and large-scale epidemiological dataset. The frequencies of tobacco use, the amount smoked, and perceived bruxism were overall associated as has been reported earlier (Rintakoski et al., 2010). Independent of genetic background, smoking, and bruxism were associated with identical twin pairs. Further, in the present study, nicotine dependence was associated with more frequent bruxism, even in the presence of a history of major depression and alcohol dependence.

The few earlier studies on this topic have all used self-reports of bruxism obtained by questionnaires or interviews for the epidemiological analyses (J. Ahlberg et al., 2004; K. Ahlberg et al., 2005; Johansson et al., 2004; Lavigne et al., 1997; Molina et al., 2001; Ohayon et al., 2001), with a more detailed sleep laboratory examination only in the study of Lavigne et al. (1997), albeit only on 15 bruxing subjects. Using questionnaires may cause difficulties in defining the actual prevalence of bruxism: It may be even more common among populations than surveys

Table 3. Multinomial Logistic Regression: Independent Effects of Amount Smoked (cigarettes per day, CPD) Among Current Smokers on "weekly," "Monthly," and "Rarely" Reported Bruxism Compared With Never Bruxism as the Reference Category. Adjusted by Age and Gender

n= 2,604 current smokers	*OR*	95% *CI*	*p* Value
Weekly			
Sex (female)	1.20	0.87–1.65	.26
Age	0.98	0.96–1.00	.06
CPD			
1–9	1.00		
10–19	1.09	0.72–1.65	.68
20–24	1.61	1.02–2.54	.041
>24	1.97	1.16–3.37	.013
Monthly			
Sex (female)	0.90	0.61–1.32	.58
Age	0.97	0.94–0.99	.011
CPD			
1–9	1.00		
10–19	0.98	0.61–1.58	.95
20–24	1.71	1.01–2.90	.046
>24	1.79	0.98–3.25	.057
Rarely			
Sex (female)	0.93	0.76–1.14	.49
Age	1.00	0.99–1.02	.56
CPD			
1–9	1.00		
10–19	0.97	0.76–1.23	.79
20–24	1.01	0.76–1.35	.95
>24	1.29	0.92–1.80	.14
Never	1.00	reference category	

Note. Trend test for CPD categories was significant for weekly ($p = .004$) and monthly ($p = .011$) bruxism. *OR* = odds ratio.

Table 4. Multinomial Logistic Regression: Effect of Lifetime *DSM-IV* Nicotine Dependence (yes/no, with no as the reference category) on "weekly," "Monthly," and "Rarely" Reported Bruxism Among Ever-Smokers Evaluated for Nicotine Dependence in the Nicotine Addiction Genetics sample. Adjusted by Age and Gender (Model I) and for Age, Gender, *DSM-IV* Diagnoses of Major Depression and Alcohol Dependence (Model II)

$n = 445$ ever smokers	Odds ratio	95 % *CI*	*p* Value
Weekly			
Model I	2.50	1.06–5.87	.036
Model II	2.10	0.86–5.08	.102
Monthly			
Model I	1.15	0.52–2.55	.726
Model II	0.93	0.43–2.22	.957
Rarely			
Model I	1.51	0.95–2.39	.080
Model II	1.62	1.00–2.62	.050
Never	1.00	reference category	

indicate but not recognized as a behavior by individuals because of its potential subconscious nature. However, any underreporting is unlikely to be associated with smoking status or nicotine dependence.

It is commonly agreed that sleep bruxism, defined as a stereotyped movement disorder occurring during sleep and characterized by tooth grinding and/or clenching, is in normal subjects detected in about 8% of the adult population (Lavigne, Manzini, & Kato, 2005) The prevalence for bruxism in our study is within the range, as also reported earlier (Hublin et al., 1998). Bruxism has been associated with stress, anxiety, orofacial pain, and sleep problems. It may also damage teeth and lead to costly treatments. On the other hand, bruxism may as part of sleep arousal mirror reflux disease or sleep apnea (Lavigne et al., 2005). Nevertheless, the pathophysiology of bruxism has remained far from clear.

Bruxism has been diagnosed for research purposes using multiple axes: subjective perception by questionnaires and interviews (including a bed partner report of grinding sounds) and objective assessment by extraoral and intraoral examination for secondary clinical signs of bruxism (e.g., masseter hypertrophy, pain on palpation of the masticatory muscles, tooth wear facets, and/or shiny spots on restorations) and/or by electromyographic (EMG) or polysomnographic (PSG) recordings. Clinical signs of bruxism, however, may reflect a problem in the past rather than the present, and even EMG and PSG may only provide a timely indication of a fluctuating phenomenon (Lavigne et al., 2005; Van der Zaag, Lobbezoo, Visscher, Hamburger, & Naeije, 2008). It is commonly accepted that PSG is necessary when physiological events adherent to bruxism episodes are investigated, but it should be borne in mind that most of the epidemiological data on bruxism are gathered from subjects by questionnaire or interview. Nevertheless, because sleep studies in large numbers of individuals are not feasible, self-report currently reflects the best available data for epidemiological surveys.

A Swedish cross-sectional study among 50-year-old males and females, based on a questionnaire ($n = 6{,}343$) and clinical examinations ($n = 941$) for validating and qualifying responses, showed a significant association in a multivariate model (with many covariates) between self-reported bruxism and daily tobacco use (either cigarette smoking or smokeless tobacco; Johansson et al., 2004). No difference in the prevalence of bruxism was found by tobacco use status prior to adjustment for covariates, which is opposite to our findings.

A 1-year follow-up study among Finnish 30- to 55-year-old workers in a media company ($n = 211$) revealed a significant association between tobacco use and bruxism. Smokers reported bruxism 2.4 (95% *CI* = 1.2–4.9) times more likely than non smokers. Bruxism was based on responses to baseline and follow-up surveys. All types of tobacco use (including cigars, pipe, and smokeless tobacco) were categorized as smoking (J. Ahlberg et al., 2004). In comparison, in the present study, the *OR* for weekly bruxism was 2.5 for heavy smokers compared with never-smokers. Another survey in the same company ($n = 874$) showed that increasing smoking frequency and frequent bruxism were slightly associated (K. Ahlberg et al., 2005). This association was, however, not significant.

In a multicenter telephone interview in the United Kingdom, Italy, and Germany ($n = 13{,}057$, females 52%, age range: 15–100 years), 8.2% reported tooth grinding during sleep at least weekly. Comparable proportions of males (4.1%) and females (4.6%) further met with the International Classification of Sleep Disorders (American Academy of Sleep Medicine, 2005) criteria for sleep bruxism. Subjects with various sleep problems, stress, or anxiety as well as heavy alcohol drinkers, caffeine drinkers, and smokers were at higher risk of reporting sleep bruxism (Ohayon et al., 2001). The crude *OR*s were 1.6 for smoking both less and more than 20 cigarettes daily compared with nonsmokers. After adjustment for multiple variables, however, the *OR* for heavier smokers was 1.0, while that for light smokers was 1.3. Thus, no evidence for a dose–response relationship was found in that study, in contrast to the present study, in which heavy smokers and dependent smokers were at higher risk.

In a survey of 2,019 Canadians on sleep disorders, Lavigne et al. (1997) found a significant *OR* of 1.9 for a smoker to report bruxism. Sampling subjects from that survey, they also found in the sleep laboratory that smokers (mean age: 29, *SD* = 5 years)

had five times more bruxism episodes during sleep than non smokers (mean age: 25, *SD* = 4 years), consistent with the implications of our own study.

Our recent study showed a clear association among 3,124 young adults between both cumulative cigarette smoking (*OR* = 1.9) and use of smokeless tobacco (*OR* = 2.1) with more frequent self-reported bruxism, which association held even after adjustment for known confounders. Since the associations were found with both forms of tobacco and a dose–response relationship was found, the results supported the hypothesis of a link between nicotine intake and bruxism (Rintakoski et al., 2010).

However, all other aforementioned epidemiological studies on bruxism and smoking have ignored the possible confounding effects of genes. Twin studies indicated that both smoking (Rose et al., 2009) and bruxism (Hublin et al., 1998) have significant genetic components. Thus, the association between the two may be due to underlying genetic effects in common, that is, pleiotropic effects of genes resulting in two different phenotypes. Familial aggregation may be due to family members sharing genes or sharing environments. The twin study on bruxism (Hublin et al., 1998) did not find a shared familial effect, so the association with smoking cannot be due to shared family effects in common to these two phenotypes. The discordant pair analysis suggests that the association between smoking and bruxism exists even when family background is taken into account, but a formal analysis of the contribution of genes and environment would require multivariate quantitative genetic modeling.

The Finnish Twin Cohort data have several strengths. Representativeness of bruxism is adequate in the present study population and database as other studies have given similar prevalence (Partinen & Hublin, 2000). The data are also representative of the smoking behavior of Finnish population. Lung cancer incidence is an excellent indirect measure of smoking behavior in a population and among the Finnish Twin Cohort; lung cancer incidence did not differ from that in the population (Verkasalo, Kaprio, Koskenvuo, & Pukkala, 1999), indicating that data represent well Finnish smoking population. The NAG study is focused more specifically on smoking and nicotine dependence but is based on the Finnish Twin Cohort. As in almost all surveys, the heaviest smokers were somewhat underrepresented in the NAG study (Broms et al., 2007). We had the opportunity to deepen the assessment of causality of tobacco use with respect to bruxism by using discordant twin pairs as matched cases and controls.

Smoking is decreasing in western societies but it is still rather common and detrimental for several aspects. A high proportion of smokers are dependent on nicotine (Fagerström & Furberg, 2008). In the present study, we used the psychiatric diagnostic scheme *DSM-IV* to diagnose nicotine dependence in a relatively small subset of our twins. The association between nicotine dependence and bruxism held even after adjustment for a lifetime history of another dependence, namely alcohol dependence, as well as major depression. Nicotine dependence plays a central role in maintaining smoking, and nicotine affects the dopaminergic system. On the other hand, some evidence exists suggesting that disturbances in the dopaminergic system would play a role in the genesis of bruxism (Chen, Lu, Lui, & Liu, 2005; Lobbezoo, Van Der Zaag, & Naeije, 2006), and there may thus be a common mechanism underlying the increased risk of bruxism among smokers. On the other hand, controlled trials among healthy subjects have not been performed, and the exact cerebral source generators of bruxism are still unknown (Lavigne et al., 2007).

The present results support our hypothesis that links both nicotine intake and dependence with bruxism. Our twin study also provides novel evidence that this association is independent of possible shared genes, which implies the need to establish causality between the two in further studies.

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Declaration of Interests

None declared.

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