Twin Research and Human Genetics
Volume 15 ▌ Number 6 ▌ pp. 714–719 ▌ © The Authors 2012 ▌ doi:10.1017/thg.2012.54

Genetic Factors Account for Half of the Phenotypic Variance in Liability to Sleep-Related Bruxism in Young Adults: A Nationwide Finnish Twin Cohort Study

Katariina Rintakoski,[1,2] Christer Hublin,[3] Frank Lobbezoo,[4] Richard J. Rose,[5] and Jaakko Kaprio[2,6,7]
[1] *Institute of Dentistry, University of Helsinki, Helsinki, Finland*
[2] *Department of Public Health, University of Helsinki, Helsinki, Finland*
[3] *Finnish Institute of Occupational Health, Helsinki, Finland*
[4] *Department of Oral Function, Academic Centre for Dentistry Amsterdam (ACTA), Amsterdam, The Netherlands*
[5] *Department of Psychological and Brain Sciences, Indiana University, Bloomington, IN, USA*
[6] *Department of Mental Health and Substance Abuse Services, National Institute for Health and Welfare, Helsinki, Finland*
[7] *Institute for Molecular Medicine FIMM, Helsinki, Finland*

Objectives: The aim of the present study was to examine the role of genetic and environmental factors in the phenotypic variance of bruxism in a large population-based cohort of young adult twins in Finland.

Methods: The material of the present study derives from the *FinnTwin16* cohort study consisting of five birth cohorts of twin pairs born in 1975–1979 who completed a questionnaire (at mean age 24, range 23–27 years) with data on frequency of sleep-related bruxism in 2000–2002. We used quantitative genetic modeling, based on the genetic similarity of monozygotic and dizygotic twins, to estimate the most probable genetic model for bruxism, based on decomposition of phenotypic variance into components: additive genetic effects (A), dominant genetic effects (D), and non-shared environmental effects (E).

Results: On average, 8.7% experienced bruxism weekly, 23.4% rarely, and 67.9% never, with no significant gender difference ($p = .052$). The best fitting genetic model for bruxism was the AE-model. Additive genetic effects accounted for 52% (95% CI 0.41–0.62) of the total phenotypic variance. Sex-limitation model revealed no gender differences.

Conclusions: Genetic factors account for a substantial proportion of the phenotypic variation of the liability to sleep-related bruxism, with no gender difference in its genetic architecture.

Keywords: Bruxism, genetic, twins

Bruxism is diurnal or nocturnal parafunctional activity that includes clenching, bracing, gnashing, and grinding of teeth (American Academy of Sleep Medicine, 2005) causing several problems like abnormal tooth wear, pain in the temporomandibular joint or jaw muscles, headaches and even social problems (Kato et al., 2001, Lavigne et al., 2005). Bruxism is further divided into two, possibly independent, disorders: awake and sleep-related bruxism (Manfredini & Lobbezoo, 2010). Sleep bruxism, defined as a stereotyped movement disorder occurring during sleep, is characterized by tooth grinding and/or clenching associated with sleep excessive (intense) micro-arousals (De Laat & Macaluso, 2002; Kato et al., 2001; Lavigne et al., 2003, 2005, 2008; Macaluso et al., 1998).

Originally bruxism was thought to be caused by peripheral (morphological) factors but nowadays its theory about central (pathophysiological and psychological) regulation overrides (Lavigne et al., 2008; Van der Zaag et al., 2008). Among others, disorders in the dopaminergic system, stress, sleep disturbances, orofacial pain, psychoactive substances like smoking, alcohol, and coffee, age, gender and genetic

RECEIVED 25 May 2012; ACCEPTED 8 August 2012. First published online 6 September 2012.

ADDRESS FOR CORRESPONDENCE: Katariina Rintakoski, PL 41, 00014 University of Helsinki, Helsinki, Finland. E-mail: katariina.koivumaki@helsinki.fi

factors are associated with bruxism (Chen et al., 2005; Lavigne & Manzini, 2000; Lavigne et al., 2001, 2008; Lobbezoo et al., 1997a, 1997b, 2006; Ohayon et al. 2001; Rintakoski et al., 2010a, 2010b). Prevalence of sleep bruxism is about 8% in the young adult population (Glaros, 1981; Lavigne & Montplaisir, 1994; Lavigne et al., 2005; Ohayon et al., 2001; Reding et al., 1966), although prevalence varies depending on the study design. Overall, the prevalence is highest in young adults and it gradually decreases with increasing age; in addition, no gender difference exists (Laberge et al., 2000; Lavigne & Montplaisir, 1994; Ng et al., 2005; Ohayon et al., 2001).

To date, a limited number of family studies have found some evidence for genetic background for SB (Lavigne et al., 2008). In fact, Hublin et al. (1998) reported a notable hereditary component for sleep-related bruxism in middle-aged (33–60 years, born 1930–1957) same-sex twin pairs, with a gender difference (39% in males and 53% in females), leaving residual variance in SB to be explained by non-shared environmental factors (i.e., not shared by the twins in a pair). However, in their quantitative genetic analyses using *Finnish Twin Cohort* data, Hublin et al. (1998) did not have access to information from opposite-sex pairs and, therefore, could not test whether genes specific to one sex affect the variation in bruxism.

Thus, to estimate the genetic contribution of bruxism in young adults and to reveal the possible sex-specific factors underlying the genetic contribution to bruxism, we studied bruxism in a large population-based cohort of young adult twins in Finland.

Methods

The material of the present study derives from the nationwide longitudinal *FinnTwin16* study (Kaprio et al., 2002). Twins received questionnaires assessing their lifestyle as well as general and mental health on four separate occasions (at ages 16 (baseline), 17, 18.5, and as young adults). The zygosity of the twins was determined using a validated questionnaire on physical similarity (Sarna et al., 1978). In a similar study of slightly younger twins (*FinnTwin12*), the zygosity was determined from DNA for 397 same-sex pairs: in 97% of the pairs, the questionnaire-based zygosity was confirmed. The study protocol was approved by the ethical committee of the Department of Public Health, University of Helsinki, and the Institutional Review Board of Indiana University. Subjects were advised about the study goals before they provided informed consent.

As part of the *FinnTwin16* study, twins born in 1975–1978 ($n = 4{,}283$) participated in the fourth wave of the study carried out in 2000–2002 (mean age 24 years, range 23–27), yielding a response rate of 88% (Kaprio, 2006). In the fourth wave of the study, the questionnaire included multiple health-related questions and also items on oral health (Rintakoski et al., 2010c). Twins born in 1979 did not answer items on oral health. Altogether 3,781 individuals responded to the question related to bruxism, and 649 of them responded that they did not know. Pairwise status was available from 1,141 twin pairs. Of the remaining pairs, there were 734 pairs where the co-twin did not answer the bruxism question, and 55 pairs whose zygosity was not certain.

Bruxism was assessed as an ordered variable in response to the following question: Do you grind your teeth? The options for the answers were: (1) every night, (2) weekly, (3) once in a while (meaning less often than every week), (4) never, and (5) I do not know. The options for answers create a clear understanding about its focus on sleep-related bruxism. We further classified those responding to the first two alternatives as "weekly bruxers," those responding to alternative #3 as "rarely bruxers." The group of "never bruxers" was our reference category, while we excluded those responding 'I do not know" from the analyses.

We calculated descriptive statistics and polychoric correlations with the Stata program (StataCorp, 2005). The comparison of monozygotic and dizygotic twins, based on the fact that monozygotic twins share 100% of their genes (at the sequence level) and dizygotic twins on average share only 50% of their segregating genes, provides estimates about the heritability. Twins were classified into five different zygosity-sex groups: monozygotic male, monozygotic female, dizygotic male, dizygotic female, and opposite-sex dizygotic twins. We used the quantitative genetic methods based on structural equation modeling (Neale & Cardon, 1992) to analyse the data.

Quantitative genetic modeling compares different models to permit estimation of variance components and provides statistical tests for the assumption that the twins are representative in the population (Posthuma et al., 2003; Rintakoski et al., 2010c). The estimated variance components are divided into four phenotypic variance components: additive genetic effects (A), dominant genetic effects (D), shared environmental effects (C) — that is all experiences affecting both twins similarly (such as childhood diet), and non-shared environmental effects (E) — that is experiences and exposures affecting only one member of the twin pair. E-effects also include measurement error. However, the component D and C cannot be simultaneously modeled with twin data alone and therefore, for the present study, we estimated three variance components.

The baseline model was the ADE model based on the pattern of intrapair correlations in monozygotic and dizygotic pairs. We tested the more restrictive two-parameter model (AE) and the pure E model to specify that no familial aggregation exists against the baseline model by using the χ^2 difference test and comparing degrees of freedom (*df*) between the more complex model and the more constrained model.

In addition, we used sex-limitation models (Neale & Cardon, 1992) that utilize the information from opposite-sex pairs to test whether genes specific to one sex, or

https://doi.org/10.1017/thg.2012.54 Published online by Cambridge University Press

TABLE 1
Prevalence (%) of Weekly, Rarely, and Never Bruxism by Gender

	Bruxism			
	Weekly	Rarely	Never	Total
Male	7.3 (106)	23.5 (339)	69.2 (1000)	100 (1445)
Female	9.8 (165)	23.4 (393)	66.8 (1123)	100 (1681)
Total	8.7 (271)	23.4 (732)	67.9 (2123)	100 (3126)

Note: Number of subjects is given in parenthesis.

TABLE 3
Pairwise Similarity Of Bruxism

	Correlation coefficient	95% confidence interval
All monozygotic pairs	0.55	0.44–0.67
All dizygotic pairs	0.20	0.09–0.30
Male monozygotic pairs	0.43	0.21–0.65
Female monozygotic pairs	0.61	0.48–0.74
Male dizygotic pairs	0.26	0.05–0.47
Female dizygotic pairs	0.10	−0.12–0.33
Opposite-sex dizygotic pairs	0.22	0.07–0.37

Note: Polychoric correlations by sex and zygosity

sex-specific additive genetic effects affect the variation bruxism. Sex-limitation models first test whether there are genes specific to one sex. If sex-specific genetic effects are not found, the model tests whether the magnitudes of A, D, and E effects can be set to be equal between sexes and whether this constraint weakens the fit of the model. Sex-specific effects for the given character are expected if the correlations within opposite-sex pairs are smaller than those for same-sex pairs.

Results

The prevalence of weekly, rarely, and never bruxism are shown in Table 1. On average, 8.7% experienced bruxism weekly, 23.4% rarely, and 67.9% never, with no significant gender difference ($p = .052$). The distribution of bruxism in monozygotic and dizygotic pairs is shown in Table 2. There were nine monozygotic pairs in which both were weekly bruxers and correspondingly 17 dizygotic pairs. Based on these three by three tables, polychoric correlations (r) for bruxism, computed for all pairs and separately by gender, were higher in monozygotic ($r = 0.55$, 95% CI 0.44–0.67) than in dizygotic twin pairs ($r = 0.20$, 95% CI 0.09–0.30). The correlation coefficient for monozygotic pairs was higher in female than in male pairs, and for dizygotic twin pairs higher in male and opposite-sex pairs than in female pairs, although not significantly so (Table 3).

Genetic modeling started with the ADE model (Chi-square 36.777, $p = .572$, Akaike's information criterion −41.223) based on 3 × 3 tables. We found no evidence of violations of the assumptions of the twin model, i.e., equal prevalence in MZ and DZ twins and in the first and second twin of a pair. Sex-limitation modeling indicated that the same genes are expressed in males and females and also that the variance components in men and women could be set to be equal. Non-additive genetic effects (D) were not needed and thus the best fitting model was the AE-model (Chi-square 45.025, $p = .471$, Akaike's information criterion −44.975). Under that model, 52% (95% CI 0.41–0.62) of the total phenotypic variance was explained by the additive genetic effects and the remaining 48% (95% CI 0.38–0.59) by non-shared environmental effects (Table 4). A model with only environmental effects (E) could be very clearly rejected as not fitting the data ($p < .001$).

Discussion

Our study revealed a substantial genetic component in regard to the variation of sleep-related bruxism, with no gender difference in genetic architecture in a population-based sample of young adult twin pairs.

Hublin et al. (1998) first reported a gender difference in the variation of sleep-related bruxism in an adult population (with genetic factors explaining 38.9% of the phenotypic variation in males and 53.2% in females). They also used data from Finnish twins, but their studied twins were older than those forming our sample and came from a separate cohort study. The best fitting model in that study as well, was the AE-model, but the authors were unable to test for the presence of sex-specific effects due to lack of opposite-sex twin pairs. In the present study, however, we could fully use the sex-limitation model and interestingly found no gender difference in the genetic variation of bruxism, suggesting a common genetic background of bruxism in men and women.

To date, few questionnaire-based studies or large-scale twin studies have found some genetic background for sleep-related bruxism (Hublin et al., 1998; Lavigne et al., 2005,

TABLE 2
The Pairwise Status for Bruxism in Monozygotic ($N = 380$) and Dizygotic ($N = 761$) Pairs

Monozygotic pairs:		Twin1			Dizygotic pairs:		Twin1		
		Weekly	Rarely	Never			Weekly	Rarely	Never
Twin2	Weekly	9	13	15	Twin2	Weekly	17	16	38
	Rarely	9	35	31		Rarely	19	44	114
	Never	7	43	218		Never	38	113	362

https://doi.org/10.1017/thg.2012.54 Published online by Cambridge University Press

TABLE 4

Model-Fitting Results for the Analysis of the Phenotypic Liablity To Sleep-Related Bruxism Among Monozygotic and Dizygotic Twins — Analysis for the Effects of Additive Genetic (A), Dominant Genetic (D), and Unique Environment (E)

Model	Chi-square value	*p*	Akaike's information criterion
Basic ADE model*	36.777	0.572	−41.223
ADE, but constrained to have same prevalence in men and women	41.003	0.47	−40.997
ADE, no sex-specific genetic component	41.003	0.515	−42.997
ADE, variance components constrained to be the same in men and women	43.023	0.513	−44.977
AE, dominance effects dropped from model	45.025	0.471	−44.975
E, no genetic effects	114.375	0.000	22.375

Note: The modeling progresses from top to bottom with additional model constraints added at each stage.
*Basic ADE model allows different prevalences in men, and women, a gender specific additive genetic component and the magnitude of variance components to differ by sex.

2008). Earlier reports of the hereditary component of bruxism from 1960s and 1970s were largely based on attrition patterns in dentition (Horowitz, 1963; Lindqvist, 1974) although the studies of Abe and Shimakawa (1966) and Reding et al. (1966) were based on self-report of sleep bruxism. In their twin studies, Horowitz (1963) and Lindqvist (1974) both evaluated bruxism based on attrition patterns and found some evidence of concordance rates being higher in monozygotic twins than in dizygotic twins. In their studies bruxism was only evaluated examining the attrition patterns. Reding et al. (1966) reported a significant association between current bruxism and reported bruxism of their blood relatives. According to the Abe and Shimakawa study (1966), children whose parents experienced sleep bruxism were more likely to suffer from bruxism compared to those whose parents did not experience bruxism. However, these early reports were completed when peripheral regulation of bruxism was the leading theory and the etiology of bruxism was virtually unknown. In our study, the focus was especially on sleep-related bruxism and its heritability. Our results were parallel to the previous findings and the study provides further information about the genetic architecture of the condition. Taken together with the results from Hublin et al. (1998), our study indicates that the relative role of genetics versus environment in bruxism is age-dependent. In young adults there are no gender differences, while in middle-aged adults there are likely to be gender-dependent genetic influences, or at least the relative magnitude of genetic effects varies by gender as adults become older.

In light of our results and the previous findings, the proportion of the genetic effects of total phenotypic variance in the liability to sleep-related bruxism is clearly higher than that found for the other conditions related to the temporomandibular joint (TMJ) area, such as temporomandibular disorder (TMD). It is, however, hypothesized that genetic factors affect TMD as well (Meloto et al., 2011). Still, only little evidence exists that TMD aggregate in families or has a strong genetic component (Smith et al., 2011). For example, Michalowicz et al. (2000) found no evidence in heritability of TMJ signs or symptoms while Matsuka et al. (2007) reported that genetic factors may affect TMD in an adolescent population, although their findings were not statistically significant. The observed substantial genetic components in liability to sleep-related bruxism compared to the rather minor genetic component of other disorders in the TMJ area gives important knowledge about the possible difference in the mechanism of these disorders. Notwithstanding, there is a need for research about the potential link between them. We are not aware of any twin or family study that has conducted a bivariate genetic analysis between bruxism and a TMJ disorder.

The prevalence of sleep bruxism, as frequently noted in the existing literature, occurs "often" or "very often" in about 8% of the young adult population (Lavigne & Montplaisir, 1994; Ohayon et al., 2001). Our prevalence (8.7%) for bruxism reported to be present "every night" or "weekly" (computed as weekly bruxism for the analyses) is consistent with that literature. Further, because it is known that the prevalence of bruxism changes over age the very restricted age-range in our study virtually eliminates confounding effects due to age. In addition, the present study comprised twin individuals with a high response rate and is thus very representative of the general population. A limitation of the study was the relatively high proportion of subjects who did know their sleep bruxism frequency. In spite of that, the large number of subjects and the high response rate support the use of questionnaire and the prevalence of bruxism similar with previous studies support the use of the method.

The International Classification of Sleep Disorders (American Academy of Sleep Medicine, 2005) defines sleep bruxism as "an oral parafunction characterized by grinding or clenching of the teeth during sleep that is associated with an excessive sleep arousal activity." Hence, it has been stated that current sleep bruxism can only be diagnosed with direct measurements from electromyography and polysomnography (De Leeuw, 2008). At present, bruxism is often evaluated using questionnaires, possibly a clinical examination and/or some direct measurement techniques. A limitation of questionnaire-based studies is that not all individuals are aware of their bruxism (for example, those living alone), which may yield some under reporting, also those suffering less severe bruxism may not know about their sleep bruxism habit.

https://doi.org/10.1017/thg.2012.54 Published online by Cambridge University Press

On the other hand, issues with sleep laboratory setting include the high price and logistic challenges of the analysis, the high requirements of sleep laboratories as well as the possible sleep disturbances caused by the atypical sleeping environment (Lavigne et al., 1996; Manfredini & Lobbezoo, 2010). A limitation of the present study is the lack of a validated method to evaluate bruxism and the lack of ICSD criteria-based diagnoses for bruxism. However, most of the epidemiological data on bruxism is still gathered from subjects by questionnaires or interviews because sleep studies using large sample sizes are not feasible due to high costs (Manfredini & Lobbezoo, 2010). Thus, self-report currently reflects the best available data for large epidemiological surveys. These serve to test hypotheses emanating from lab studies, but also to generate hypotheses that may be possible to test further in laboratory settings.

The classification of bruxism into three categories (weekly, rarely, never) in this study does not affect the heritability estimates in the method used. In genetic modeling we compared monozygotic and dizygotic twin pairs to each other regardless of the classification of the given phenomenon. Thus, the genetic model estimates the heritability based on the similarity of twin pairs. Hublin et al. (1998) previously suggested in their bivariate model that same genetic influences affiliate childhood and adulthood bruxism. Still, the mechanism behind the genetic factors remains unknown. However, this study adds important knowledge about the heritability of sleep bruxism and the role of gender in the heritability. There is still a need for further studies investigating the actual genes, genetic mechanisms, and the inheritance patterns relating to sleep bruxism. Our results, however, demonstrate that the phenotypic variation of bruxism can be explained by genetic factors (52%) and non-shared environmental factors (48%), common in both genders.

Conflict of Interests

Dr. Rintakoski reports no disclosures. Dr. Hublin was supported by the Finnish Work Environment Fund, gave expert statement for Valeant Canada, participated on congresses sponsored by and served on the scientific advisory board for Boehringer-Ingelheim, and has received honoraria for a lecture from UCB. Dr. Lobbezoo reports no disclosures. Dr. Rose has been supported by research grants from the National Institute on Alcohol Abuse and Alcoholism. He serves as an Associate Editor for the journals *Behavior Genetics* and *Psychiatric Genetics* and chairs the Advisory Board for NIAAA's *Genes, Environment and Development Initative* or GEDI. Dr. Kaprio was supported by the Academy of Finland Centre of Excellence on Complex Disease Genetics. He serves on the editorial boards of *Twin Research and Human Genetics, Psychiatric Genetics, International Journal of Molecular Epidemiology and Genetics* and *Addiction*, and has consulted for Pfizer on the genetics of nicotine dependence.

Acknowledgments

The authors of the manuscript wish to thank Prof. Heikki Murtomaa for his valuable contribution in data collection.

References

Abe, K., & Shimakawa, M. (1966). Genetic and developmental aspects of sleeptalking and teeth-grinding. *Acta Paedopsychiatrica*, *33*, 339–344.

American Academy of Sleep Medicine (AASM). (2005). *The international classification of sleep disorders* (2nd ed.). Westchester, IL: AASM.

Chen, W. H., Lu, Y. C., Lui, C. C., & Liu, J. S. (2005). A proposed mechanism for diurnal/nocturnal bruxism: Hypersensitivity of presynaptic dopamine receptors in the frontal lobe. *Journal of Clinical Neuroscience*, *12*, 161–163.

De Laat, A., & Macaluso, G. M. (2002). Sleep bruxism as a motor disorder. *Movement Disorders*, *17*, S67–S69.

De Leeuw, R. (2008). *Orofacial pain. Guidelines for assessment, diagnosis, and management* (4th ed.). Chicago, IL: Quintessence.

Glaros, A. G. (1981). Incidence of diurnal and nocturnal bruxism. *The Journal of Prosthetic Dentistry*, *45*, 545–549.

Horowitz, S. L. (1963). Clinical aspects of genetic research in dentistry. *Journal of Dental Research*, *42*, 1330–1343.

Hublin, C., Kaprio, J., Partinen, M., Heikkilä, K., & Koskenvuo, M. (1998). Sleep bruxism based on self-report in a nationwide twin cohort. *Journal of Sleep Research*, *7*, 61–68.

Kaprio, J. (2006). Twin studies in Finland, 2006. *Twin Research and Human Genetics*, *9*, 772–777.

Kaprio, J., Pulkkinen, L., & Rose, R. J. (2002). Genetic and environmental factors in health-related behaviours: Studies on Finnish twins and twin families. *Twin Research*, *5*, 366–371.

Kato, T., Rompre, P., Montplaisir, J. Y., Sessle, B. J., & Lavigne, G. J. (2001). Sleep bruxism: An oromotor activity secondary to micro-arousal. *Journal of Dental Research*, *80*, 1940–1944.

Laberge, L., Tremblay, R. E., Vitaro, F., & Montplaisir, J. (2000). Development of parasomnias from childhood to early adolescence. *Pediatrics*, *106*, 67–74.

Lavigne, G. J., Kato, T., Kolta, A., & Sessle, B. J. (2003). Neurobiological mechanisms involved in sleep bruxism. *Critical Reviews in Oral Biology & Medicine*, *14*, 30–46.

Lavigne, G. J., Khoury, S., Abe, S., Yamaguchi, T., & Raphael, K. (2008). Bruxism physiology and pathology: An overview for clinicians. *Journal of Oral Rehabilitation*, *35*, 476–494.

Lavigne, G. J., & Manzini, C. (2000). Bruxism. In M. H. Kryger, T. Roth & W. C. Dement (Eds.), *Principles and practice of sleep medicine* (3rd ed.). Philadelphia, PA: WB Saunders.

Lavigne, G. J., Manzini, C., & Kato, T. (2005). Sleep bruxism. In: M. H. Kryger, T. Roth & W. C. Dement (Eds.), *Principles and practice of sleep medicine* (4th ed.). Philadelphia, PA: Elsevier & Saunders.

Lavigne, G. J., & Montplaisir, J. (1994). Restless legs syndrome and sleep bruxism: Prevalenve and association among Canadians. *Sleep*, *17*, 739–743.

https://doi.org/10.1017/thg.2012.54 Published online by Cambridge University Press

Lavigne, G. J., Rompre, P. H., & Montplaisir, J. Y. (1996). Sleep bruxism: Validity of clinical research diagnostic criteria in a controlled polysomnographic study. *Journal of Dental Research*, *75*, 546–552.

Lavigne, G. J., Soucy, J., Lobbezoo, F., Manzini, C., Blanchet, P. J., & Montplaisir, J. Y. (2001). Double-blind, crossover, placebo-controlled trial of bromocriptine in patients with sleep bruxism. *Clinical Neuropharmacology*, *24*, 145–149.

Lindqvist, B. (1974). Bruxism in twins. *Acta Odontologica Scandinavica*, *32*, 177–187.

Lobbezoo, F., Lavigne, G. J., Tanguay, R., & Montplaisir, J. (1997a). The effect of the catecholamine precursor L-dopa on sleep bruxism: A controlled clinical trial. *Movement Disorders*, *12*, 73–78.

Lobbezoo, F., Soucy, J. P., Hartman, N. G., Montplaisir, J. Y., & Lavigne, G. J. (1997b). Effects of the D2 receptor agonist bromocriptine on sleep bruxism: Report of two single-patient clinical trials. *Journal of Dental Research*, *76*, 1610–1614.

Lobbezoo, F., Van Der Zaag, J., & Naeije, M. (2006). Bruxism: Its multiple causes and its effects on dental implants—An updated review. *Journal of Oral Rehabilitation*, *33*, 293–300.

Macaluso, G. M., Guerra, P., Di Giovanni, G., Boselli, M., Parrino, L., & Terzano, M. G. (1998). Sleep bruxism is a disorder related to periodic arousals during sleep. *Journal of Dental Research*, *77*, 565–573.

Manfredini, D., & Lobbezoo, F. (2010). Relationship between bruxism and temporomandibular disorders: A systematic review of literature from 1998 to 2008. *Oral Surgery, Oral Medicine, Oral Pathology, Oral Radiology & Endodontics*, *109*, e26–50.

Matsuka, Y., Nagamatsu, C., Itoh, S., Tomonari, T., Makki, A., Minakuchi, H., Maekawa, K., Kanyama, M., & Kuboki, T. (2007). Comparison of inter-twin concordance in symptoms of temporomandibular disorders: A preliminary investigation in an adolescent twin population. *Cranio : The Journal of Craniomandibular Practice*, *25*, 23–29.

Meloto, C. B., Serrano, P. O., Ribeiro-DaSilva, M. C., & Rizzatti-Barbosa, C. M. (2011). Genomics and the new perspectives for temporomandibular disorders. *Archives of Oral Biology*, *56*, 1181–1191.

Michalowicz, B. S., Pihlstrom, B. L., Hodges, J. S., & Bouchard, T. J. (2000). No heritability of temporomandibular joint signs and symptoms. *Journal of Dental Research*, *79*, 1573–1578.

Neale, M. C., & Cardon, L. R. (1992). *Methodology for genetic studies of twins and families*. Dordrecht, The Netherlands: Kluwer Academic Publishers.

Ng, D. K., Kwok, K., Cheung, J. M., Leung, S., Chow, P., Wong, W. H., Chan, C. H., & Ho, J. C. (2005). Prevalence of sleep problems in Hong Kong primary school children: A community-based telephone survey. *Chest*, *128*, 1315–1323.

Ohayon, M., Li, K., & Guilleminault, C. (2001). Risk factors for sleep bruxism in the general population. *Chest*, *119*, 53–61.

Posthuma, D., Beem, A. L., de Geus, E. J., van Baal, G. C., von Hjelmborg, J. B., Iachine, I., & Boomsma, D. I. (2003). Theory and practice in quantitative genetics. *Twin Research*, *6*, 361–376.

Reding, G. R., Rubright, W. C., & Zimmerman, S. O. (1966). Incidence of bruxism. *Journal of Dental Research*, *45*, 1198–1204.

Rintakoski, K., Ahlberg, J., Hublin, C., Broms, U., Madden, P. A., Könönen, M., Koskenvuo, M., Lobbezoo, F., & Kaprio, J. (2010a). Bruxism is associated with nicotine dependence: A nationwide Finnish twin cohort study. *Nicotine & Tobacco Research*, *12*, 1254–1260.

Rintakoski, K., Ahlberg, J., Hublin, C., Lobbezoo, F., Rose, R. J., Murtomaa, H., & Kaprio, J. (2010b). Tobacco use and reported bruxism in young adults: A nationwide Finnish twin cohort study. *Nicotine & Tobacco Research*, *12*, 679–683.

Rintakoski, K., Kaprio, J., & Murtomaa, H. (2010c). Genetic and environmental factors in oral health among twins. *Journal of Dental Research*, *89*, 700–704.

Sarna, S., Kaprio, J., Sistonen, P., & Koskenvuo, M. (1978). Diagnosis of twin zygosity by mailed questionnaire. *Human Heredity*, *28*, 241–254.

Smith, S. B., Maixner, D. W., Greenspan, J. D., Dubner, R., Fillingim, R. B., Ohrbach, R., Knott, C., Slade, G. D., Bair, E., Gibson, D. G., Zaykin, D. V., Weir, B. S., Maixner, W., & Diatchenko, L. (2011). Potential genetic risk factors for chronic TMD: Genetic associations from the OPPERA case control study. *The Journal of Pain*, *12*, T92–T101.

StataCorp. (2005). *STATA statistical software*. College Station, TX: Statcorp LP.

Van der Zaag, J., Lobbezoo, F., Visscher, C. M., Hamburger, H. L., & Naeije, M. (2008). Time-variant nature of sleep bruxism outcome variables using ambulatory polysomnography: Implications for recognition and therapy evaluation. *Journal of Oral Rehabilitation*, *35*, 577–584.

https://doi.org/10.1017/thg.2012.54 Published online by Cambridge University Press