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Smoking and Sleep

Cigarette Smoking as a Risk Factor or an Exacerbating Factor for Restless Legs Syndrome and Sleep Bruxism

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Summary: Cigarette smoking has been associated with sleep disturbances. However, little is known about how smoking affects restless legs syndrome (RLS) and sleep bruxism, two movement disorders associated with sleep. From a nationwide survey of 2,019 Canadian adults, we estimated the prevalence of smoking to be 36%. Although there was no difference between smokers and nonsmokers for RLS prevalence, almost twice as many smokers (12%) as nonsmokers (7%) were aware of experiencing sleep bruxism. The estimated risk of a smoker suffering from RLS was nonsignificant. On the other hand, the risk of a smoker grinding his or her teeth was moderate (odds ratio = 1.9). Analysis of sleep laboratory findings revealed no differences in motor RLS and periodic leg movements in sleep (PLMS) indices between smoking and nonsmoking patients; after adjustment for age, there were no differences in sleep efficiency, latency, number of awakenings, or the arousal index for the RLS/PLMS patients. Among those suffering from bruxism, smokers had more tooth-grinding episodes than did nonsmokers (35.0 vs. 7.0; $p = 0.056$); none of the sleep variables differentiated sleep bruxism smokers from nonsmokers. It appears that cigarette smoking does not influence RLS/PLMS, whereas the risk that smoking and tooth grinding are concomitant is moderate. Smoking was not significantly associated with more motor activity in RLS/PLMS, but more grinding was noted in sleep bruxism. **Key Words:** Smoking—Nicotine—Restless legs syndrome—Periodic limb movements during sleep—Bruxism—Tooth grinding.

In a recent survey of 2,019 Canadian adults, we found very high prevalences of restless legs syndrome (RLS) (10–15%) and sleep bruxism (8%) (1). However, the effects of cigarette smoking on both RLS and sleep bruxism are unknown. Although nicotine has been associated with an enhancement of acetylcholine and glutamate synaptic transmission and an increase in cortical arousal, smoking has been associated with problems in falling asleep, staying asleep, and daytime sleepiness (2–5). In light of this evidence, we hypothesized that smoking would exacerbate both RLS and sleep bruxism by increasing arousal—specifically, that it would be associated with higher-than-normal motor activity during sleep. Two sets of data were examined to test this hypothesis. One was a national survey that estimated the prevalence risk ratio of smokers and nonsmokers reporting either RLS, periodic leg movements in sleep (PLMS), or sleep bruxism. The other consisted of retrospective analyses of sleep and motor measures of RLS/PLMS and bruxism collected from patients who slept in our laboratory.

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METHODS

Survey

The epidemiological survey consisted of interviews with 2,019 English- and French-speaking Canadian adults from all 10 provinces. The methodology is described in a previous publication (1). Two questions were used to estimate the prevalence of restless legs syndrome, and two questions concerned awareness of sleep bruxism and frequency of cigarette smoking. 1) *Bedtime leg restlessness*: At bedtime, does restlessness in your legs very often, often, occasionally, or never delay your falling asleep? 2) *Leg muscle unpleasantness during sleep*: When you wake up during the night, do you very often, often, occasionally or never feel

unpleasant sensations in your leg muscles that require you to move your legs or to walk in order to be more comfortable? 3) *Tooth grinding*: *Do you very often, often, occasionally, or never grind your teeth during your sleep?* 4) *Smoking*: *Over the past two weeks, have you smoked cigarettes*? (Yes or No.) This information was collected by the private company Environics Research Group in Toronto, Canada. The error margin of the survey was estimated to be ±2.2%.

TABLE 1. *Odds ratios (and 95% confidence intervals) for a representative survey of Canadian adults* (n = *2,019*)

Survey questions	Risk of a smoker having RLS or bruxism
Bedtime leg restlessness	1.22 (0.95–1.57) NS
Leg muscle unpleasantness during sleep	1.25 (0.94–1.67) NS
Tooth grinding	1.90 (1.37–2.63)*

RLS, restless legs syndrome; NS, not significant.
* significant.

Sleep laboratory

The sleep and motor variables were collected in our sleep laboratory from two French-speaking populations; protocols were approved for study by local ethics committees. The presence of smoking was scored as positive if the patient reported that he or she smoked at least one cigarette per day. The RLS/PLMS patients were recruited on the basis of their clinical histories according to standard criteria for these disorders (6). Specifically, diagnoses were based on a "desire to move the extremities, often associated with paresthesia/dysesthesia; motor restlessness; worsening of symptoms at rest with at least temporary relief by activity; and worsening of symptoms in the evening or night". In RLS/PLMS nonsmokers, mean age was 49.0 years (SD = 12.8; range 22–73), with a gender distribution of 20 women and 19 men; among RLS/PLMS smokers, mean age was 45.3 years (SD = 11.4; range 30–66), with a gender distribution of 11 women and 15 men.

The sleep bruxism population was selected according to criteria proposed by the American Sleep Disorders Association (7), i.e. grinding sounds reported by a bed partner, observation of tooth wear, and reports of jaw fatigue, tenderness, or pain upon awakening. Furthermore, all those who suffered bruxism had more than four bruxism episodes per hour or 25 bruxism bursts per hour of sleep, with at least two episodes of grinding per night. The validity of these criteria has been demonstrated elsewhere (8). None of the patients exhibiting bruxism had apnea, RLS, or PLMS history. Analyses of sleep and motor variables were conducted according to standard criteria for our laboratory (8–10). Among sleep bruxism nonsmokers, the mean age was 28.6 years (SD = 4.7), with a gender distribution of four women and four men; among sleep bruxism smokers, the mean age was 24.5 years (SD = 4.3), with a gender distribution of four women and three men.

Chi-square (χ^2) and Mann-Whitney *U* tests were used to evaluate nonparametric variables, whereas analysis of variance and *t* tests were used to assess normally distributed variables. Significance levels were set at $p < 0.05$. Relative risks were estimated using odds ratios. An odds ratio was considered significant when its 95% confidence interval did not contain 1.0. SPSS statistical software (Chicago, Illinois) was used for all analyses.

RESULTS

The overall prevalence of smoking in the general Canadian population was 36%. No gender difference was noted [males, 38%; females, 35%; $\chi^2(1) = 2.58$; $p = 0.108$]. However, prevalence of smoking varied with age [$\chi^2(6) = 101.17$; $p < 0.001$], with the proportion of smokers decreasing from 40% for ages 18–25 to 17% for those over 65.

Complaints of bedtime leg restlessness were reported by 15% of the population. Among the 36% of the population who smoked, 16.4% (120/732) reported bedtime leg restlessness. Among nonsmokers, this symptom was present in 13.8% (176/1,273) [$\chi^2(1) = 2.24$; $p = 0.13$]. Complaints of leg muscle unpleasantless during sleep were reported by 10.6% of the subjects. Among the smokers, this symptom was reported by 12.0% (88/733), while among nonsmokers, it was reported by 9.8% (125/1,271) [$\chi^2(1) = 2.08$; $p = 0.15$). The estimated risk of a smoking subject having RLS/PLMS were low and nonsignificant for both the bedtime leg restlessness and leg unpleasantness questions (Table 1).

Sleep bruxism was reported by 8% of the population; tooth grinding prevalence was significantly higher for smoking (12%; 82/682) than for nonsmoking (6.7%; 80/1192) subjects [$\chi^2(1) = 14.83$; $p < 0.001$]. The risk of a smoker reporting tooth grinding was 1.9. Since the 95% confidence intervals do not include 1.0 (Table 1), we reject the null hypothesis that the risks are the same for smokers and nonsmokers.

In Tables 2 and 3, the motor and sleep variables are presented in relation to smoking for RLS/PLMS and sleep bruxism subjects. For RLS/PLMS sleep laboratory subjects, no differences between smokers and nonsmokers were noted for sleep and motor variables. After plotting the data distribution by age, one variable—sleep efficiency—showed an age effect that was

TABLE 2. *Motor variables recorded in the sleep laboratory for RLS/PLMS and sleep bruxism patients*[a]

Motor variable	Nonsmokers	Smokers	Statistics
RLS/PLMS			
n	36	24	
RLS index	35.2 ± 40.7	25.7 ± 21.5	$t = -1.18$, p = 0.24
n	39	26	
PLMS index/n	17.0 (0.0–109.0)	22.0 (0.0–60.0)	$U = 546.0$, p = 0.59
Sleep bruxism			
n	8	7	
No. of bruxism episodes with grinding	7.0 (2.0–26.0)	35.0 (3.0–45.0)	U = 11.5, p = 0.056
No. of bruxism episodes per hour of sleep	5.6 ± 2.4	6.9 ± 1.7	$t = -1.15$, p = 0.27
No. of bursts per episode	7.8 ± 1.9	9.5 ± 2.9	$t = -1.3$, p = 0.22

RLS, restless legs syndrome; PLMS, periodic leg movements in sleep.
[a] For normally distributed variables, *t* tests and means ± standard deviation are shown; for non-normal variables, Mann-Whitney *U* tests and medians (5th–95th percentiles) are shown.

qualified by a significant interaction with smoking [$F(1,61) = 5.84$; p = 0.02]. The group of patients under 60 years old (30 nonsmokers and 23 smokers) showed a nonsignificant effect for sleep efficiency (p = 0.13), while the patients over 60 years old (nine nonsmokers and three smokers) showed a significant effect (p = 0.002). However, the latter effect is based on too small a sample size to be considered reliable. For sleep bruxism, a trend was noted for the number of bruxism episodes with grinding: there were five times more grinding episodes in smokers (35.0) than in nonsmokers (7.0) ($U = 11.5$, p = 0.056) (Table 2).

DISCUSSION

The results suggest that cigarette smoking is a low risk factor for the production of RLS/PLMS symptoms and sleep abnormalities (Tables 1 and 2). However, for patients with sleep bruxism, there is a moderate risk that smoking and grinding will coexist (Table 1) and a tendency for smokers to have more tooth-grinding episodes than nonsmokers (Table 2).

This study does not definitively rule out the possibility that nicotine, as a neurochemically active substance, has no effect on RLS/PLMS or sleep bruxism. In fact, a major limitation of the study is that our measures of smoking do not take into account nicotine dose, duration of the habit, or degree of dependence. Specifically, the national survey estimated the prevalence of smoking only over the 2 weeks prior to the survey, whereas the laboratory smokers were self-reported "daily" smokers, but without further elaboration.

Ideally, the nicotine hypothesis should be tested either by using nicotine chewing gum or patches in a controlled challenge with nonsmokers or by provoking nicotine deprivation in smoking subjects. The possible cognitive and physiological effects of nicotine on sleep, arousal, and cardiovascular activities (11–14) also must be discriminated from similar changes as-

TABLE 3. *Sleep variables recorded in the laboratory for RLS/PLMS and sleep bruxism patients*[a]

Sleep variable	Nonsmokers	Smokers	Statistics
RLS/PLMS			
n	39	26	
Sleep latency (minutes)	14.0 (2.0–53.0)	17.0 (3.0–64.0)	$U = 548.0$, p = 0.58
% Sleep efficiency	80.7 ± 15.1	87.1 ± 10.0	$F = 2.33$, p = 0.13
No. of awakenings	6.0 (0.0-22.0)	4.5 (0.0–12.0)	$U = 407.5$, p = 0.18
Arousal index	12.6 ± 8.7 (n = 25)	12.1 ± 6.3 (n = 16)	$t = -0.21$, p = 0.83
Sleep bruxism			
n	8	7	
Sleep latency (minutes)	6.7 (0.3–29.0)	10.3 (7.0–50.7)	$U = 13.0$, p = 0.08
% Sleep efficiency	97.4 (86.0–99.3)	97.1 (94.8–98.7)	$U = 29.5$, p = 0.86
No. of awakenings	21.5 ± 18.0	22.4 ± 13.3	$t = -0.11$, p = 0.91
Movement arousal	74.6 ± 25.8 (n = 5)	66.0 ± 33.6 (n = 5)	$t = 0.46$, p = 0.66

RLS, restless legs syndrome; PLMS, periodic leg movements in sleep.
[a] For normally distributed variables, *t* tests and means ± standard deviation are shown; for non-normal variables, Mann-Whitney *U* tests and medians (5th–95th percentiles) are shown. An analysis of variance was performed for sleep efficiency to test for an interaction with age (see Results section).

sociated with motor activities in sleep. For example, sleep bruxism has been associated with electroencephalogram arousal and changes in heart rate (15). Moreover, nicotine has been associated with an increase in dopamine activity (16). The latter is an important variable to consider when quantifying RLS/PLMS and sleep bruxism interactions with nicotine because both of these sleep movement disorders are modulated by dopamine (15,17–19). To complicate the picture, smokers report higher anxiety and are more likely to have type A personalities (20,21). A Medline search of the last 30 years' literature failed to find evidence that RLS/PLMS patients have such personality features. On the other hand, it has been argued that sleep bruxism is associated with both anxiety and type A personality (see review in 15). Consequently, it is possible that the moderated risk for smoking and sleep bruxism results from a concomitant type A personality. This needs to be tested with appropriate personality tests and objective measures of both motor activity in sleep and biological nicotine concentrations.

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