Smoking as a Risk Factor for Sleep-Disordered Breathing

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Background: Recent evidence indicates that the prevalence of sleep-disordered breathing is remarkably high (24% for men and 9% for women) and that the public health burden attributable to sleep-disordered breathing is substantial. This investigation examines current and former cigarette smoking as potential risk factors for sleep-disordered breathing.

Methods: Data were from 811 adults enrolled in the University of Wisconsin Sleep Cohort Study, Madison. The Sleep Cohort Study is a longitudinal, epidemiologic study that uses nocturnal polysomnography to investigate sleep-disordered breathing and other disorders of sleep. The presence and severity of sleep-disordered breathing was quantified by the frequency of apneas and hypopneas per hour of sleep.

Results: Logistic regression analyses were used to control for potential confounding factors. Compared with never smokers, current smokers had a significantly greater risk of snoring (odds ratio, 2.29) and of moderate or worse sleep-disordered breathing (odds ratio, 4.44). Heavy smokers (≥40 cigarettes per day) had the greatest risk of mild sleep-disordered breathing (odds ratio, 6.74) and of moderate or worse sleep-disordered breathing (odds ratio, 40.47). Former smoking was unrelated to snoring and sleep-disordered breathing after adjustment for confounders.

Conclusions: Current cigarette smokers are at greater risk for sleep-disordered breathing than are never smokers. Heavy smokers have the greatest risk while former smokers are not at increased risk for sleep-disordered breathing. Thus, smoking cessation should be considered in the treatment and prevention of sleep-disordered breathing.

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SLEEP-DISORDERED breathing is characterized by repeated episodes of breath cessation (apneas) and reduced ventilation (hypopneas) during sleep.[1] Recent evidence indicates that the prevalence of sleep-disordered breathing is remarkably high (24% for men and 9% for women).[2] Moreover, the report of the National Commission on Sleep Disorders Research has underscored the devastating impact of sleep disorders on public health,[3] and the public health burden attributable to sleep-disordered breathing per se is substantial. Increased cardiovascular morbidity and mortality,[4,5] neuropsychological impairment,[6] and psychiatric disturbance[7,8] are all associated with sleep-disordered breathing.

Although there is little information on the relationship between cigarette smoking and sleep-disordered breathing, there is evidence suggestive of such a relationship. For example, smoking is related to snoring,[9,10] considered to be a preclinical form of sleep-disordered breathing. Smoking has also been associated with concomitants of sleep-disordered breathing such as nocturnal hypoxemia[11] and complaints of disturbed sleep.[12]

Evidence for a relationship between smoking and sleep-disordered breathing would have important clinical and public health implications. If smoking is causally related to sleep-disordered breath-

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SUBJECTS AND METHODS

SUBJECTS

Data were collected in two stages using a probability sampling strategy. In the first stage, surveys were distributed by mail to all adults employed at three state agencies with diverse work settings in Dane County, Wisconsin. The survey instrument consisted of a four-page questionnaire assessing demographic and sleep characteristics. Surveys were returned by 3516 individuals, representing a response rate of 82%. There were no significant differences between responders and nonresponders on age, gender, or occupational category.

In the second stage, subjects were recruited for overnight studies using a weighted sampling procedure designed to oversample individuals at risk for sleep-disordered breathing. Further details on the study methods are reported elsewhere.[2] Study eligibility was assessed via telephone contact. Exclusion criteria included pregnancy; unstable or decompensated cardiopulmonary disease, including myocardial infarction; lung and upper-airway malignancies; recent upper-airway surgery; and tracheostomy. Ten individuals failed to meet eligibility requirements. All eligible subjects were offered $50 as compensation for participation and the response rate was 64%. The inconvenience of sleeping away from home was the most common reason for refusing to participate. All study procedures were approved by the Institutional Review Board of the University of Wisconsin.

There was no evidence of a selection bias in the sampling procedures that would compromise the generalizability of the study results. Participants and nonparticipants were similar on survey responses of age, sex, snoring, and body mass index, although participants were slightly more educated than nonparticipants.

INTERVIEW DATA

Following their normal daily activities, subjects reported to the Sleep Cohort Research Laboratory located in the General Clinical Research Center at the University of Wisconsin Hospital. Prior to bedtime, body mass index was assessed using standard anthropometric techniques. Tobacco, alcohol, and caffeine consumption were assessed using a structured health history interview.

Current smokers, former smokers, and never smokers were identified by two questions: (1) Have you ever smoked tobacco regularly?; and, (2) Do you currently smoke? Data on quantity smoked were also assessed. To measure alcohol consumption, subjects were asked to indicate how many of the following they drank during a typical week: (1) cans or bottles of beer; (2) glasses of wine; and (3) mixed drinks or shots. These values were combined to yield a total number of drinks per week. Caffeine use was assessed with the following two questions: (1) How many cups of coffee (with caffeine), or tea (with caffeine) do you usually drink in a typical day? and (2) How many

ing, smoking cessation may become a critical component of sleep apnea syndrome treatment and prevention. Thus, this investigation examines the association between cigarette smoking and sleep-disordered breathing using data on 811 subjects enrolled in the University of Wisconsin Sleep Cohort Study, Madison. The Sleep Cohort Study is a longitudinal, epidemiologic study that uses nocturnal polysomnography to investigate sleep-disordered breathing and other disorders of sleep.

RESULTS

Characteristics of the study population by sleep-disordered breathing category are presented in **Table 1**. Unadjusted odds ratios and 95% confidence intervals for the association of smoking status with snoring and sleep-disordered breathing are presented in **Table 2**. Current smokers were more likely than never smokers to snore and to have moderate or worse sleep-disordered breathing. The relationship between current smoking and any sleep-disordered breathing was marginally significant ($P=.08$). Former smokers were more likely than never smokers to experience moderate or worse and any sleep-disordered breathing.

Odds ratios and 95% confidence intervals for the association of smoking status with snoring and sleep-disordered breathing, adjusted for sex, age, education, body mass index, caffeine use, and alcohol consumption, are presented in **Table 3**. Current smokers were more likely to snore than were never smokers, consistent with previous research.[9,10] In addition, current smokers were more likely than never smokers to experience any sleep-disordered breathing and were at particularly high risk of moderate or worse sleep-disordered breathing. Former smoking was not related to snoring or sleep-disordered breathing after adjustment.

A dose-response relationship was also evident (**Table 4**). Heavy smokers (≥40 cigarettes per day) had the greatest risk of mild, moderate or worse, and any sleep-disordered breathing. In fact, the odds of heavy smokers having moderate or worse sleep-disordered breathing were 10 times that of moderate or light smokers and 40 times that of never smokers. Moderate smokers (20 to 39 cigarettes per day) were at greater risk for snoring than were never smokers. Light smokers (<20 cigarettes per day) were at increased risk of experiencing any sleep-disordered breathing.

cans of cola or other soft drinks with caffeine do you usually drink in a typical day?

POLYSOMNOGRAPHIC DATA

Overnight 20-channel polysomnography was conducted in our sleep research laboratory at the University of Wisconsin General Clinical Research Center. Nocturnal polysomnography consisted of electroencephalography, electro-oculography, and electromyography to identify sleep stages, thermistry, and end-tidal carbon dioxide detection to measure oral and nasal airflow, oximetry to measure oxyhemoglobin saturation, and inductance plethysmography to detect respiratory effort. The resulting sleep records were scored by 30-second epochs. Sleep was staged using the method of Rechtschaffen and Kales.[13] Abnormal breathing events were scored when a 10-second total cessation of airflow was present (apnea) or when a discernible decrease in flow accompanied by a 4% drop in oxyhemoglobin saturation was observed (hypopnea). Sleep-disordered breathing was determined by the average number of abnormal events per hour of sleep (the apnea-hypopnea index [AHI]).

Five categories of sleep-disordered breathing were created: (1) *no sleep-disordered breathing* (no snoring and AHI, <5); (2) *snoring* (snoring and AHI, <5); (3) *mild sleep-disordered breathing* (AHI, 5 to 15); and (4) *moderate or worse sleep-disordered breathing* (AHI, >15). In addition, a fifth category combined the mild and moderate or worse sleep-disordered breathing categories to yield an *any sleep-disordered breathing* category (AHI, ≥5).

DATA ANALYSIS

The SAS computer software modules (SAS, Cary, NC) were used for all statistical analyses.[14] Results from the Sleep Cohort Study examining the relationship between sleep-disordered breathing and sex, age, and body habits have been reported previously.[2] Univariate analyses and multivariate logistic regression analyses were performed to calculate odds ratios and 95% confidence intervals for smoking status and risk of sleep-disordered breathing. All multivariate logistic regression analyses controlled for a number of known risk factors (sex, age, body mass index, and alcohol use)[2,15,16] and potential confounding factors (education and caffeine consumption). Smokers and former smokers were compared separately with never smokers and the sleep-disordered breathing categories of snoring (mild, moderate, or worse) and any sleep-disordered breathing were referenced to the no sleep-disordered breathing category. Results for men and women are presented together as the smoking status by sex interactions were nonsignificant in all models of sleep-disordered breathing. Results were considered statistically significant if P values were less than .05.

Data from a total of 40 subjects were excluded from all analyses, resulting in a sample size of 811 individuals. Data on smoking status were missing for eight subjects and on education, age, and/or body mass index for three additional subjects. Finally, to improve the validity of the smoking status classification, 17 cigar and pipe smokers and 12 former smokers who had quit smoking within the previous 6 months or who had missing data on the length of time since quitting were excluded as well.

COMMENT

This large, population-based epidemiologic study demonstrates that current cigarette smokers are at greater risk for sleep-disordered breathing than are never smokers. Moreover, a dose-response relationship indicated that heavy smokers have the greatest risk of sleep-disordered breathing. Although unadjusted odds ratios suggested that former smoking was associated with sleep-disordered breathing, the relationship appeared to be due to the confounding effects of age and sex. Former smokers were older and were more likely to be male than were current or never smokers.

This study cannot determine the direction of causality or the precise causal mechanisms involved in the relationship between smoking and sleep-disordered breathing. However, several hypotheses suggest that cigarette smoking plays a causal role in snoring and sleep-disordered breathing. First, studies of nicotine withdrawal have consistently reported that sleep disturbance is a common complaint.[17] It may be that sleep complaints during nicotine withdrawal are the result of withdrawal-induced increases in sleep-disordered breathing. For instance, nicotine withdrawal may cause sleep instability, which, in turn, is related to sleep-disordered breathing.[18] Similarly, the minor withdrawal experienced by smokers on a nightly basis may lead to or exacerbate sleep-disordered breathing via increased sleep instability, ie, sleep-disordered breathing among current smokers may reflect the decline in nicotine blood levels throughout the night.[19]

current cigarette smokers are at greater risk for sleep-disordered breathing than are never smokers

Second, some investigators have found a high prevalence of pulmonary function abnormalities in sleep apnea patients[20] and there are a number of detrimental pulmonary and respiratory effects attributable to smoking[21] that may result in an increase in sleep-disordered breathing. These effects include an accelerated loss of lung function; increased respiratory symptoms (cough, airway inflammation, phlegm production, wheeze, epithelial permeability); increased rates of respiratory illness and infection; and increased rates of obstructive airway diseases such as

Table 1. Characteristics of the Study Population by SDB Category*

Characteristics	No SDB (n=297)	Snoring AHI <5 (n=340)	Mild SDB, AHI 5 to 15 (n=102)	Moderate SDB, AHI >15 (n=72)
Sex, No. (%)				
Male	145 (49)	194 (57)	72 (71)	57 (79)
Female	152 (51)	146 (43)	30 (29)	15 (21)
Education, No. (%)				
≤High school	52 (18)	79 (23)	29 (28)	26 (36)
Some college	66 (22)	123 (36)	36 (35)	18 (25)
≥Undergraduate degree	179 (60)	138 (41)	37 (36)	28 (39)
Mean age (SD)	42.9 (7.3)	44.2 (8.0)	46.2 (7.8)	46.6 (7.8)
Mean body mass index, kg/cm^2 (SD)	25.4 (4.7)	27.9 (5.0)	30.5 (5.9)	33.5 (7.2)
Mean cans caffeinated soda per day (SD)	0.8 (1.0)	1.0 (1.3)	0.9 (1.4)	0.9 (1.1)
Mean cups caffeinated coffee or tea per day (SD)	2.5 (2.5)	3.1 (3.5)	3.2 (3.7)	2.8 (3.3)
Mean No. of alcoholic drinks per week (SD)	3.4 (5.2)	4.6 (7.8)	4.6 (8.0)	4.6 (7.1)
Smoking status, No. (%)				
Never smokers	148 (50)	131 (39)	41 (40)	20 (28)
Former smokers	106 (36)	123 (36)	45 (44)	39 (54)
Current smokers	43 (14)	86 (25)	16 (16)	13 (18)

SDB indicates sleep-disordered breathing; AHI, apnea-hypopnea index.

Table 2. Risk for SDB Associated With Current Smoking and Former Smoking Estimated by Unadjusted Odds Ratios (OR) and 95% Confidence Intervals (CI)*

	OR (95% CI)			
Smoking Status	Snoring, AHI<5	Mild SDB, AHI 5 to 15	Moderate SDB, AHI>15	Any SDB, AHI≥5
Former vs never	1.31 (0.92-1.86)	1.53 (0.94-2.50)	2.72 (1.50-4.93)	1.92 (1.27-2.91)
Current vs never	2.26 (1.46-3.49)	1.34 (0.69-2.63)	2.24 (1.03-4.86)	1.64 (0.94-2.86)

SDB indicates sleep-disordered breathing; AHI, apnea-hypopnea index. The reference category for SDB is no snoring and an AHI of less than 5.

emphysema, asthma, chronic bronchitis, and chronic obstructive pulmonary disease.

Finally, interactions among the stimulant effects of nicotine, nicotine withdrawal, and the pulmonary and respiratory effects of smoking may result in sleep-disordered breathing. Nicotine has been shown to stimulate upper airway musculature and decrease upper airway resistance in animals,[22] effects that would tend to reduce the occurrence of apneas. Consistent with this finding, a study of eight male patients with sleep apnea indicated that the administration of nicotine gum prior to sleep resulted in a decreased number of apneas during the first 2 hours of sleep,[23] a period when blood levels of nicotine are elevated. Thus, nicotine may actually reduce sleep-disordered breathing during the first few hours of sleep by decreasing upper airway resistance. However, as nicotine blood levels decline and upper airway resistance increases during the night, sleep-disordered breathing may then increase due to either nicotine withdrawal effects per se or the pulmonary and respiratory effects of smoking.

The hypotheses positing that smoking causes sleep-disordered breathing are also consistent with the lack of increased risk for former smokers. Nightly nicotine withdrawal as well as many of the respiratory and pulmonary effects of smoking would no longer affect the individual given an adequate period of time after smoking cessation. In addition, body mass index was not a confounding factor in the relationship between former smoking and sleep-disordered breathing. While former smokers had slightly larger body mass indexes than did current or never smokers, these differences were not statistically significant. Thus, our results suggest that smoking cessation may prove to be an important intervention for reducing sleep-disordered breathing.

The causal pathway may also operate in the opposite direction such that sleep-disordered breathing results in an increased likelihood of smoking. For example, individuals may smoke to cope with the consequences of sleep-disordered breathing such as hypersomnolence, depression, and cognitive impairment, ie, smoking may represent a form of self-medication.

Table 3. Risk for SDB Associated With Current Smoking and Former Smoking Estimated by Adjusted Odds Ratios (OR) and 95% Confidence Intervals (CI)*

	OR (95% CI)			
Smoking Status	Snoring, AHI<5	Mild SDB, AHI 5 to 15	Moderate SDB, AHI>15	Any SDB, AHI≥5
Former vs never	1.26 (0.86-1.85)	1.14 (0.62-2.09)	1.86 (0.83-4.15)	1.33 (0.77-2.30)
Current vs never	2.29 (1.41-3.72)	2.09 (0.90-4.82)	4.44 (1.52-13.01)	3.05 (1.44-6.44)

**SDB indicates sleep-disordered breathing; AHI, apnea-hypopnea index. The reference category for SDB is no snoring and an AHI of less than 5. Odds ratios have been adjusted for sex, education, age, body mass index, number of cans of caffeinated soda per day, number of cups of caffeinated coffee or tea per day, and number of alcoholic drinks per week using logistic regression.*

Table 4. The Dose-Response Relationship Between Cigarettes Smoked Per Day and SDB Estimated by Adjusted Odds Ratios (OR) and 95% Confidence Intervals (CI)*

	OR (95% CI)			
Smoking Status	Snoring, AHI<5	Mild SDB, AHI 5 to 15	Moderate SDB, AHI>15	Any SDB, AHI≥5
<20 cigarettes per day vs never	2.16 (1.00-4.66)	2.99 (0.91-9.86)	3.94 (0.92-16.97)	4.11 (1.41-11.99)
20-39 cigarettes per day vs never	2.24 (1.25-4.01)	1.02 (0.31-3.32)	3.25 (0.76-14.02)	1.66 (0.60-4.58)
≥40 cigarettes per day vs never	3.06 (0.86-10.88)	6.74 (1.20-37.89)	40.47 (2.37->50)	8.38 (1.68-41.94)

**SDB indicates sleep-disordered breathing; AHI, apnea-hypopnea index. The reference category for SDB is no snoring and an AHI of less than 5. Odds ratios have been adjusted for sex, education, age, body mass index, number of cans of caffeinated soda per day, number of cups of caffeinated coffee or tea per day, and number of alcoholic drinks per week using logistic regression.*

STUDY STRENGTHS AND LIMITATIONS

One limitation of this study is that the 95% confidence intervals for the dose-response relationship are broad and the precise magnitude of risk associated with varying levels of smoking is unclear. There is, however, little doubt that heavy smokers are at significantly increased risk of sleep-disordered breathing (odds ratios from 6.7 to 40.5).

highlights the need for clinicians to assess smoking status when considering treatment for sleep-disordered breathing

Another limitation of this study is the lack of biochemical confirmation of smoking status. However, other data indicate that there is excellent agreement between self-reported and biochemical indexes of smoking among individuals not attempting to quit smoking.[24] Furthermore, given that the current social climate discourages smoking, misclassification would most likely result from smokers reporting that they do not smoke, an effect that would weaken the association between smoking and sleep-disordered breathing.

The strengths of our study include the use of a population-based sample and assessment of sleep-disordered breathing via recommended and state-of-the-art polysomnographic techniques.[25] Moreover, sleep-disordered breathing measures were reproducible when assessed on separate overnight studies.[2] In addition, numerous known risk factors for sleep-disordered breathing were controlled for in our analyses as were several additional potential confounders.

CONCLUSIONS

This study establishes a link between current smoking and sleep-disordered breathing and provides a strong rationale for further research attempting to ascertain the precise relationship between smoking and sleep-disordered breathing as the results have important public health implications. The study also highlights the need for clinicians to assess smoking status when considering treatment for sleep-disordered breathing. The finding that former smokers are not at increased risk for sleep-disordered breathing suggests that smoking cessation may serve to eliminate or dramatically attenuate whatever risk is incurred by smoking. In fact, it may be that behavioral interventions (smoking cessation, weight loss, avoidance of alcohol) provide sufficient reduction of sleep-disordered breathing for many people such that more invasive and burdensome treatments (surgery, continuous positive airway pressure) need only be used after behavioral interventions prove ineffective, ie, in a stepped-care approach to treatment. Given the recent report of the National Commission on Sleep Disorders Research highlighting the public health burden of sleep disorders,[3] the iden-

tification of a modifiable health risk behavior such as smoking is an important step in reducing the impact of sleep-disordered breathing on limited health care resources.

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