

The Relation between Cigarette Smoking and Sleep Disturbance¹

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Background. There is little epidemiological or clinical information on the relation between smoking and sleep disturbance, despite evidence suggestive of a relationship. The present study tested the hypothesis that cigarette smoking is associated with sleep disturbance.

Methods. Survey data from 3,516 adults were collected as part of a longitudinal, epidemiologic study of sleep-disordered breathing. Symptoms of insomnia, hypersomnia, and parasomnia were assessed using diagnostic criteria from the Diagnostic and Statistical Manual of Mental Disorders (3rd ed., revised).

Results. Among both males and females, smoking was associated with difficulty initiating sleep, and difficulty waking up. Excessive daytime sleepiness was related to smoking only for females while nightmares and disturbing dreams were related to smoking only among males.

Conclusions. Smoking was associated with difficulty initiating sleep and with a constellation of symptoms suggestive of sleep fragmentation. Sleep disturbance may be more prevalent among smokers due to the stimulant effects of nicotine, nightly withdrawal, an increased prevalence of sleep disordered breathing relative to nonsmokers, and/or an association with psychological disturbance. These results have important clinical and public health implications for reduction of the disease and disability associated with smoking and sleep disturbance. © 1994 Academic Press, Inc.

The recent report of the National Commission on Sleep Disorders Research highlighted the devastating impact of sleep disorders on public health (1). Sleep disturbance is associated with numerous deficits in physical and psychological functioning including an increased risk of mortality (2, 3), frequent hospitalizations and use of health-related services (4, 5), cognitive impairment (6), and affective disturbances (5, 7, 8). Thus, the identification of a modifiable risk factor such as cigarette smoking would be an important step in

reducing the public health burden attributable to sleep disturbance.

Nevertheless, there is little epidemiological or clinical information on the relation between smoking and sleep disturbance, despite evidence suggestive of a relationship. Limited polysomnographic data indicate that smokers have a longer sleep latency and spend more time awake in bed (9), a pattern consistent with insomnia. Most epidemiologic investigations of sleep disturbance have not examined smoking as a potential risk factor, however (4, 5, 10). In an epidemiologic study that did examine the impact of smoking on sleep (11), there was little evidence of an association although accurate estimates of this relationship may have been prevented by inadequate assessment of the frequency of sleep disturbance symptoms. In this study, subjects reported the frequency of sleep disturbance symptoms on a rating scale that did not specify the actual number of nights per week or month that subjects were affected by their symptoms, i.e., subjects reported symptoms as "never," "seldom," "sometimes," "often," or "always." Thus, a relationship may have been obscured by the lack of clear numerical definitions of each frequency category.

Evidence of a relationship between smoking and sleep disturbance would have important clinical and public health implications. If smoking is causally related to sleep disturbance, smoking cessation interventions have the potential to significantly reduce the occurrence of sleep disturbance and the associated decrements in functioning. With the reported prevalence of sleep disturbance ranging anywhere from 10 to 50% in the general population (4, 5, 10, 11), the number of individuals affected by such a relationship could be quite large.

Not only might smoking cause sleep disturbance, but sleep disturbance could play a role in the motivation to smoke. The symptoms of sleep disturbance—depression, fatigue, cognitive impairment—resemble those of nicotine withdrawal, and such symptoms may motivate the desire to smoke (12). In fact, sleep problems are commonly reported during tobacco withdrawal (13, 14), but it may be that sleep disturbance and the corresponding symptoms of such disturbance frequently

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antedate smoking cessation. Indeed, some symptoms attributed to smoking withdrawal might reflect a comorbid sleep disorder that is exacerbated by nicotine deprivation.

The purpose of the present epidemiologic investigation was to test the hypothesis that cigarette smoking is associated with symptoms of insomnia, hypersomnia, and parasomnia as defined by diagnostic criteria in the Diagnostic and Statistical Manual of Mental Disorders, 3rd ed. revised (DSM-III-R; 15).

METHODS

Subjects

Data were collected as part of the University of Wisconsin Sleep Cohort Study, a longitudinal, epidemiologic study of sleep-disordered breathing (16). Surveys were distributed by mail to all adults employed at two state agencies with diverse work settings in Dane County, Wisconsin. Surveys were returned by 3,516 individuals, representing a response rate of 82%. There were no significant differences between responders and nonresponders on age, gender, or occupational category. Respondent characteristics are shown in Table 1.

Survey Instrument

The survey instrument consisted of a four-page questionnaire assessing demographic and sleep characteristics. Symptoms of insomnia, hypersomnia, and parasomnia were derived from diagnostic criteria in DSM-III-R (15) and were assessed by questions similar to those commonly used in clinical sleep laboratories (17, 18).

Insomnia symptoms assessed were: (a) Difficulty getting to sleep; (b) Wake up during the night and have a hard time getting back to sleep; (c) Wake up repeatedly during the night; (d) Wake up too early in the morning and can't get back to sleep; and (e) Not feel rested during the day, no matter how many hours of sleep you had.

Hypersomnia symptoms assessed were: (a) Feelings of excessive daytime sleepiness (b) Very difficult to wake up in the morning; (c) Fall asleep or doze momentarily—watching TV, reading, etc.; and (d) Fall asleep or doze momentarily—at meetings, church, etc.

One symptom of parasomnia was assessed: (a) Nightmares or disturbing dreams.

For each symptom, respondents indicated the frequency with which it was experienced: never; rarely (1 day or night/month); sometimes (2–4 days or nights/month); often (5–15 days or nights/month); or almost always (16–30 days or nights/month). Individuals were considered to have a *very frequent* disturbance if that symptom occurred 16 or more days or nights per month, a *frequent* disturbance if it occurred 5–15 days or nights per month, an *infrequent* disturbance if it occurred 2–4 days or nights per month, and a *rare* dis-

turbance if it occurred 1 or fewer days or nights per month. DSM-III-R criteria for insomnia state that the disturbance must occur "at least three times a week for at least one month" and criteria for hypersomnia state that the disturbance must occur "nearly every day for at least one month, or episodically for longer periods of time." (15). No frequency criteria are specified for parasomnias. Thus, only individuals who have a *very frequent* disturbance would be likely to meet criteria for a clinical diagnosis of insomnia or hypersomnia.

Current smokers, former smokers, and never smokers were identified by two questions: (a) Do you currently smoke cigarettes? and (b) Have you ever been a regular smoker, that is smoked at least a pack a week? Data on the quantity smoked and the number of years of smoking were not available.

RESULTS

Univariate analyses and multiple logistic regression analyses were performed in order to calculate odds ratios and 95% confidence intervals. Smokers and former smokers were separately compared with never smokers. The sleep disturbance categories of very frequent, frequent, and infrequent were referenced to the rare category.

Potential confounding factors were also examined. Age and education were found to be related to both smoking and sleep difficulties and these variables were controlled for in the logistic regression analyses. Adjustment for age and education had little effect on the pattern of results. Two unadjusted odds ratios that were significant became nonsignificant after adjustment (infrequent falling asleep while watching TV, reading, etc. for males; very frequent nightmares and disturbing dreams for females) while three odds ratios became significant only after adjustment (infrequent difficulty waking up for adult males; very frequent and frequent falling asleep while watching TV, reading, etc. for females). In addition, because female gender has been consistently associated with greater rates of sleep disturbance (4, 5, 10, 11), as was also true in this study, all analyses were performed separately for males and females.

Characteristics of the sample are shown in Table 1. Odds ratios and 95% confidence intervals for the association of insomnia symptoms with current smoking, adjusted for age and education, are presented in Table 2. Current smoking was related to very frequent, frequent, and infrequent difficulty getting to sleep for males and to very frequent and frequent difficulty getting to sleep for females. Male and female smokers were more likely to have very frequent non-restorative sleep than were never smokers. Female smokers were also more likely to have frequent and infrequent non-restorative sleep than were female never smokers. Smoking was not associated with difficulties in sleep

TABLE 1
Characteristics of the Study Population

Characteristic	No. and (%) of Respondents	
	Males (<i>n</i> = 1666)	Females (<i>n</i> = 1840)
Age		
20-29	19 (1)	17 (1)
30-39	507 (31)	775 (42)
40-49	686 (42)	708 (39)
50-59	413 (25)	315 (17)
60-69	20 (1)	14 (1)
Education		
<High school	25 (2)	26 (1)
High school	736 (46)	1139 (63)
>High school	827 (52)	644 (36)
Smoking status		
Never smoker	754 (46)	893 (50)
Former smoker	555 (34)	458 (25)
Current smoker	339 (21)	452 (25)

maintenance, i.e., difficulty getting back to sleep after waking during the night, repeated awakenings, or difficulty getting back to sleep after early morning awakening.

Odds ratios and 95% confidence intervals for the association of hypersomnia and parasomnia symptoms with current smoking, adjusted for age and education, are presented in Table 3. Smoking was related to very frequent, frequent, and infrequent excessive daytime sleepiness only for females. Smoking was associated with very frequent difficulty waking up for both males and females, with frequent difficulty waking up for males, and with infrequent difficulty waking up for both males and females.

For females, smoking was inversely related to both very frequent and frequent falling asleep while watching TV, reading, etc. For males, smoking was associated with very frequent, frequent, and infrequent nightmares and disturbing dreams.

There was little evidence that former smokers were at increased risk of sleep disturbance relative to never smokers (data not shown). Analyses comparing former smokers with never smokers resulted in very few significant findings and no consistent pattern of relationship between former smoking and specific symptoms of sleep disturbance. Therefore, these data suggest that current smoking but not former smoking is associated with sleep disturbance.

We were concerned about the confounding effects of alcohol and caffeine consumption since both substances have been correlated with smoking (19) and sleep problems (20-22). Although data on alcohol and caffeine use were not available from the survey, detailed information (number of alcoholic drinks/week, number of cups of caffeinated coffee/day, number of cans of caffeinated soda/day) was collected by interview from a subset of survey participants who underwent polysomno-

graphic assessment (*n* = 602). Analysis of these data indicated that our findings do not appear to be due to the confounding effects of alcohol or caffeine use. First, the magnitude and significance of odds ratios for smoking and sleep difficulties obtained by logistic regression analyses were unaffected by adjustment for alcohol and caffeine use. Second, alcohol and caffeine use were largely unrelated to symptoms of sleep disturbance and therefore, these variables cannot be confounding factors.

DISCUSSION

This is the first population-based epidemiologic study of sleep disturbance to demonstrate that current cigarette smoking is related to specific sleep difficulties. Moreover, for those symptoms of sleep disturbance related to smoking, a trend was usually evident such that as the frequency of the symptom increased, the association with smoking tended to increase.

Smoking was associated with difficulty initiating sleep and with a constellation of symptoms suggestive of sleep fragmentation, i.e., excessive daytime sleepiness, non-restorative sleep, and difficulty waking up. Sleep fragmentation is characterized by a lower percentage of slow wave and rapid eye movement sleep and by more frequent arousals (23). Furthermore, periodic arousals and sleep state discontinuity are often imperceptible to the sleeping person even though deficits in daytime functioning such as hypersomnolence are a frequent result (18, 24). Thus, disruptions in sleep architecture may lead to increased complaints of excessive daytime sleepiness, non-restorative sleep, and difficulty waking up without necessarily increasing complaints of difficulty maintaining sleep.

Although the present study cannot determine the causal mechanisms involved, the pattern of results is consistent with several hypotheses. One hypothesis suggests that the stimulant effects of nicotine contribute to sleep disturbance. Nicotine blood levels tend to peak in the late afternoon and evening (25), consistent with the stimulant hypothesis and with the finding that smokers have more difficulty initiating sleep than do nonsmokers (9).

Another line of evidence suggests that the minor withdrawal experienced by smokers on a nightly basis may lead to or exacerbate sleep disturbance. Studies of nicotine withdrawal have consistently found sleep disturbance to be a common complaint (13, 14). Thus, for current smokers, symptoms of sleep fragmentation may reflect the drop in nicotine blood levels throughout the night (25).

Sleep-disordered breathing or "sleep apnea," characterized by repeated breath cessations and reduced ventilation during sleep, is surprisingly common (24% for men and 9% for women; 16) and may be even more common among smokers. Several studies have linked smoking with snoring (26-28), considered to be a pre-

TABLE 2

Risk for Insomnia Symptoms Associated with Current Smoking versus Never Smoking Estimated by Adjusted Odds Ratios (OR) and 95% Confidence Intervals (CI)^a

	Males						Females					
	Nonsmokers		Smokers		Adjusted OR	95% CI	Nonsmokers		Smokers		Adjusted OR	95% CI
	N	%	N	%			N	%	N	%		
Difficulty getting to sleep												
0-1 nights/month	440	59	154	46			449	51	194	43		
2-4 nights/month	203	27	110	33	1.44	1.05-1.98	297	34	145	32	1.01	0.77-1.32
5-15 nights/month	84	11	53	16	1.56	1.02-2.38	101	11	82	18	1.78	1.26-2.52
>15 nights/month	16	2	16	5	2.32	1.04-5.16	34	4	28	6	1.88	1.09-3.23
Difficulty getting back to sleep												
0-1 nights/month	415	56	168	51			421	48	226	50		
2-4 nights/month	214	29	115	35	1.24	0.91-1.70	292	33	139	31	0.82	0.63-1.59
5-15 nights/month	98	13	39	12	0.86	0.56-1.34	129	15	63	14	0.82	0.58-1.71
>15 nights/month	16	2	10	3	1.35	0.57-3.21	35	4	20	4	1.07	0.59-1.94
Repeated awakenings												
0-1 nights/month	426	58	180	54			446	51	235	52		
2-4 nights/month	161	22	85	26	1.01	0.72-1.42	207	23	105	23	0.87	0.65-1.54
5-15 nights/month	103	14	45	14	0.83	0.54-1.26	147	17	70	16	0.82	0.58-1.71
>15 nights/month	50	7	23	7	0.99	0.56-1.73	81	9	39	9	0.80	0.52-1.93
Early morning awakening												
0-1 nights/month	454	61	178	54			519	59	264	59		
2-4 nights/month	179	24	94	28	1.17	0.84-1.63	215	24	111	25	0.97	0.73-1.29
5-15 nights/month	88	12	48	15	1.28	0.84-1.95	114	13	57	13	0.93	0.65-1.33
>15 nights/month	19	3	11	3	1.09	0.49-2.44	32	4	16	4	0.86	0.45-1.69
Nonrestorative sleep												
0-1 nights/month	389	53	146	44			382	43	123	28		
2-4 nights/month	208	28	104	31	1.24	0.90-1.73	291	33	176	39	1.81	1.37-2.41
5-15 nights/month	114	15	58	17	1.25	0.84-1.87	161	18	105	23	1.73	1.24-2.40
>15 nights/month	25	3	24	7	2.20	1.15-4.19	47	5	43	10	2.61	1.62-4.19

^a Odds ratios have been adjusted for age and education using logistic regression. The reference category is 0-1 nights/month for each sleep disturbance symptom.

clinical phase of sleep-disordered breathing. Sleep-disordered breathing, in turn, is related to sleep fragmentation, complaints of disturbed sleep, and decrements in daytime functioning (17, 29). Likely causes of sleep-disordered breathing among smokers are the detrimental pulmonary and respiratory effects attributable to smoking (30). For instance, smoking-induced obstructive airways diseases (emphysema, asthma, very frequent bronchitis, very frequent obstructive pulmonary disease), respiratory illness and infection, or respiratory symptoms (cough, airway edema, phlegm production) may be mechanisms through which smoking affects sleep. Smoking clearly impairs pulmonary function (30, 31) and some researchers have found a high prevalence of pulmonary function abnormalities in patients with sleep-disordered breathing (32).

Moreover, these hypothesized causal mechanisms are not mutually exclusive. In fact, some sleep disturbance may result from complex interactions among the stimulant effects of nicotine, nicotine withdrawal, and the pulmonary and respiratory effects of smoking. For

example, a study of eight male sleep apnea patients demonstrated that the administration of nicotine gum prior to sleep resulted in a decrease in the number of apneas during the first 2 hr of sleep (33), i.e., when nicotine blood levels were elevated. This finding may be explained by studies showing that nicotine stimulates upper airway musculature and decreases upper airway resistance in animals (34), effects that would tend to reduce apneas. Thus, the stimulant effects of nicotine may not only increase sleep latency, but may 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 fragmentation and sleep-disordered breathing may then increase.

The causal pathway may also operate in the opposite direction such that very frequent sleep disturbance results in an increased tendency to smoke. Individuals may attempt to cope with the problems of non-restorative sleep, difficulty waking up, and excessive daytime sleepiness by smoking. Furthermore, the re-

TABLE 3

Risk for Hypersomnia and Parasomnia Symptoms Associated with Current Smoking versus Never Smoking Estimated by Adjusted Odds Ratios (OR) and 95% Confidence Intervals (CI)^a

	Males						Females					
	Nonsmokers		Smokers		Adjusted OR	95% CI	Nonsmokers		Smokers		Adjusted OR	95% CI
	N	%	N	%			N	%	N	%		
Excessive daytime sleepiness												
0-1 days/month	407	55	186	56			473	54	184	41		
2-4 days/month	223	30	98	30	0.94	0.69-1.46	265	30	163	36	1.47	1.12-1.91
5-15 days/month	85	12	40	12	1.02	0.65-1.61	118	13	76	17	1.48	1.04-2.09
>15 days/month	21	3	7	2	0.57	0.21-1.51	22	3	27	6	2.61	1.43-4.78
Very difficult to wake up												
0-1 days/month	501	68	192	58			522	59	211	47		
2-4 days/month	147	20	72	22	1.47	1.03-2.10	207	23	133	30	1.59	1.20-2.11
5-15 days/month	62	8	46	14	2.53	1.58-4.05	98	11	56	13	1.46	0.99-2.15
>15 days/month	32	4	23	7	2.34	1.25-4.39	54	6	48	11	2.37	1.51-3.72
Fall asleep watching TV, reading, etc.												
0-1 days/month	297	40	109	33			345	39	191	43		
2-4 days/month	219	30	113	34	1.23	0.88-1.73	251	28	131	29	0.86	0.64-1.15
5-15 days/month	179	24	85	26	1.06	0.73-1.54	204	23	97	22	0.70	0.51-0.96
>15 days/month	45	6	24	7	1.08	0.60-1.93	78	9	29	6	0.54	0.33-0.88
Fall asleep at meeting, church, etc.												
0-1 days/month	585	79	250	76			736	84	373	83		
2-4 days/month	115	16	55	17	1.10	0.75-1.61	97	11	51	11	0.98	0.68-1.43
5-15 days/month	30	4	20	6	1.35	0.72-2.51	33	4	20	4	1.11	0.62-2.00
>15 days/month	8	1	6	2	1.41	0.46-4.31	13	1	5	1	0.83	0.29-2.41
Nightmares or disturbing dreams												
0-1 nights/month	584	79	223	67			598	68	276	62		
2-4 nights/month	135	18	81	24	1.41	1.01-1.98	213	24	121	27	1.12	0.85-1.47
5-15 nights/month	19	3	22	7	2.85	1.42-5.74	58	7	35	8	1.29	0.82-2.04
>15 nights/month	1	0	5	2	11.87	1.27-110.61	10	1	13	3	2.14	0.90-5.10

^a Odds ratios have been adjusted for age and education using logistic regression. The reference category is 0-1 days or nights/month for each sleep disturbance symptom.

relationship between smoking and sleep disturbance may be reciprocal where smoking causes sleep disturbance and where sleep disturbance promotes smoking in response to fatigue, sleepiness, and disturbances in mood.

Finally, psychological characteristics may account for the relationship between smoking and sleep disturbance. Recent research has suggested that both smoking and sleep disorders are associated with psychiatric disorders and with personality patterns characterized by depressive and anxious affect (5, 7, 35-37).

Study Limitations

Reliance on self-report data is a limitation of the present study. There is no polysomnographic confirmation of sleep disturbance or biochemical confirmation of smoking status. However, numerous studies have indicated that complaints of sleep disturbance are relatively accurate with respect to comparison among individuals and that self-reported sleep disturbance

shows at least moderate agreement with polysomnographic findings (38-41).

Data on smoking status indicate that there is good agreement between self-reported and biochemical indices of smoking among individuals not attempting to quit smoking (42). In fact, self-report may even underestimate the prevalence of smoking, an effect that would reduce the association between smoking and sleep disturbance. Unfortunately, data on quantity smoked for current smokers and years since quitting for former smokers were not available. It may be that the effects of smoking and nicotine on sleep disturbance are highly dose-dependent and occur only among heavy smokers, an issue we could not address with the present data. Furthermore, the lack of relationship found for former smoking and sleep disturbance may be due to a "dose-response" relationship between sleep disturbance and time since smoking cessation, i.e., the effects of smoking on sleep may diminish with time since cessation. Again this was an issue we could not address in the current study. Future research will need

to carefully examine these issues in order to differentiate between alternative causal processes.

Although data on caffeine and alcohol use were not available for the entire sample, data from a subset of individuals suggests that these substances were not responsible for the study results. Finally, determining the influence of personality patterns and psychiatric disorders on the relationship between smoking and sleep disturbance will be of the utmost importance in future research.

Conclusions

The results of the present study have important clinical and public health implications for reducing the disease and disability associated with smoking and sleep disturbance. Moreover, the results provide a strong rationale for further research attempting to ascertain the precise relationship between smoking and sleep disturbance. In particular, the assessment of alternative causal processes will require: (a) epidemiologic investigations that allow for the determination of dose-response relationships and statistical control of psychiatric status, disease status, medication use, etc.; and (b) polysomnographic studies that continuously record data on sleep state and breathing. Finally, the study not only highlights the need for clinicians to assess smoking status when considering treatment for sleep disorders, as smoking cessation may prove to be beneficial, it also suggests that some individuals might benefit from treatment of sleep disturbance prior to and/or concomitant with smoking cessation.

REFERENCES

1. National Commission on Sleep Disorders Research. *Wake up America: A National Sleep Alert*. Washington, DC: Government Printing Office, 1993.
2. Kripke DF, Simons RN, Garfinkel L, Hammond EC. Short and long sleep and sleeping pills: Is increased mortality associated? *Arch Gen Psychiatry* 1979; 36:103-116.
3. Wingard DK, Berkman LF. Mortality risk associated with sleeping patterns among adults. *Sleep* 1983; 6:102-107.
4. Bixler EO, Kales A, Soldatos CR, Kales JD, Healey S. Prevalence of sleep disorders in the Los Angeles metropolitan area. *Am J Psychiatry* 1979; 136:1257-1262.
5. Ford DE, Kamerow DB. Epidemiologic study of sleep disturbance and psychiatric disorders. *JAMA* 1989; 262:1479-1484.
6. Berry DTR, Webb WB, Block AJ, Bauer RM, Switzer DA. Nocturnal hypoxia and neuropsychological variables. *J Clin Exp Neuropsychol* 1986; 8:229-238.
7. Kales A, Caldwell AB, Soldatos CR, Bixler EO, Kales JD. Biopsychobehavioral correlates of insomnia. II. Pattern specificity and consistency with the Minnesota Multiphasic Personality Inventory. *Psychosom Med* 1983; 45:341-356.
8. Rodin J, McAvay G, Timko C. A longitudinal study of depressed mood and sleep disturbance in elderly adults. *J Gerontol* 1988; 43:45-53.
9. Soldatos CR, Kales JD, Scharf MB, Bixler EO, Kales A. Ciga-

- rette smoking associated with sleep difficulty. *Science* 1988; 207:551-553.
10. Mellinger GD, Balter MB, Uhlenhuth EH. Insomnia and its treatment. *Arch Gen Psychiatry* 1985; 42:225-232.
11. Karacan I, Thornby JL, Williams RL. Sleep disturbance: A community survey. In: Guilleminault C, Lugaresi E, Eds. *Sleep/Wake Disorders: Natural History, Epidemiology, and Long-Term Evolution*. New York: Raven Press, 1983.
12. Wetter DW, Brandon TH, Baker TB. The relation of affective processing measures and smoking motivation indices among college-age smokers. *Adv Behav Res Ther* 1992; 14:169-193.
13. Hughes JR, Gust SW, Skoog K, Keenan RM, Fenwick JW. Symptoms of tobacco withdrawal: A replication and extension. *Arch Gen Psychiatry* 1991; 48:52-59.
14. Hughes JR, Hatsukami D. Signs and symptoms of tobacco withdrawal. *Arch Gen Psychiatry* 1986; 43:289-294.
15. American Psychiatric Association. *Diagnostic and Statistical Manual of Mental Disorders*, 3rd ed., revised. Washington DC: American Psychiatric Association, 1987.
16. Young T, Palta M, Dempsey J, Skatrud J, Weber S, Badr S. The occurrence of sleep-disordered breathing among middle-aged adults. *N Engl J Med* 1993; 328:1230-1235.
17. Guilleminault C. Clinical features and evaluation of obstructive sleep apnea. In: Kryger MH, Roth T, Dement W, Eds. *Principles and Practice of Sleep Medicine*. Philadelphia: WB Saunders, 1989:552-558.
18. Roehrs T, Zorick F, Wittig R, Conway W, Roth T. Predictors of objective level of daytime sleepiness in patients with sleep-related breathing disorders. *Chest* 1989; 95:1202-1206.
19. Istvan J, Matarazzo JD. Tobacco, alcohol, and caffeine use: A review of their interrelationships. *Psychol Rev* 1984; 95:301-326.
20. Curatolo PW, Robertson D. The health consequences of caffeine. *Ann Intern Med* 1983; 98:641-653.
21. Issa FG, Sullivan CE. Alcohol, snoring and sleep apnea. *J Neurol Neurosurg Psychiatry* 1982; 45:353-359.
22. Rundell OH, Lester BK, Griffiths WJ, Williams HL. Alcohol and sleep in young adults. *Psychopharmacologia* 1972; 26:201-218.
23. Carskadon MA, Dement WC. Normal human sleep: An overview. In: Dryger MH, Roth T, Dement W, Eds. *Principles and Practice of Sleep Medicine*. Philadelphia: WB Saunders, 1989:3-13.
24. Guilleminault C, Partinen M, Quera-Salva MA, Hayes B, Dement WC, Nino-Murcia G. Determinants of daytime sleepiness in obstructive sleep apnea. *Chest* 1988; 94:32-37.
25. Benowitz N., Kuyt F, Jacob P III. Circadian blood nicotine concentrations during cigarette smoking. *Clin Pharmacol Ther* 1982; 32:758-764.
26. Bloom JW, Kaltenborn WT, Quan SF. Risk factors in a general population for snoring. *Chest* 1988; 94:678-683.
27. Norton PG, Dunn EV. Snoring as a risk factor for disease: An epidemiological survey. *BMJ* 1985; 291:630-632.
28. Telakivi T, Partinen M, Koskenvuo M, Kaprio J. Snoring and cardiovascular disease. *Compr Ther* 1987; 13:53-57.
29. Lavie P. Sleep habits and sleep disturbances in industrial workers in Israel: Main findings and some characteristics of workers complaining of excessive daytime sleepiness. *Sleep* 1981; 4:147-158.
30. Public Health Service. *The Health Benefits of Smoking Cessation*. A Report of the Surgeon General. U.S. Department of Health and Human Services, 1990. DHHS Publication No. (CDC) 90-8416.
31. Higgins MW, Enright PL, Kronmal RA, Schenker MB, Anton-Culver H, Lyles M. Smoking and lung function in elderly men

- and women: The cardiovascular health study. *JAMA* 1993; 269:2741-2748.
32. Kales A, Cadieux RJ, Bixler EO, *et al.* Severe obstructive sleep apnea—I: Onset, clinical course, and characteristics. *J Chronic Dis* 1985; 38:419-425.
- Gothe B, Strohl KP, Levin S, Cherniack NS. Nicotine: A different approach to treatment of obstructive sleep apnea. *Chest* 1985; 87:11-17.
- Haxhiu MA, van Lunteren E, van de Graaff WB, Strohl KP, Bruce EN, Mitra J, Cherniack NS. Action of nicotine on the respiratory activity of the diaphragm and genioglossus muscles and the nerves that innervate them. *Respir Physiol* 1984; 57: 153-169.
35. Anda RF, Williamson DF, Escobedo LG, Mast EE, Giovino GA, Remington PL. Depression and the dynamics of smoking: A national perspective. *JAMA* 1990; 264:1541-1545.
36. Glassman AH, Helzer JE, Covey LS, Cottler LB, Stetner F, Tipp JE, Johnson J. Smoking, smoking cessation, and major depression. *JAMA* 1990; 264:1546-1549.
37. Hughes JR, Hatsukami DK, Mitchell JE, Dahlgren LA. Prevalence of smoking among psychiatric outpatients. *Am J Psychiatry* 1986; 143:993-997.
38. Bixler EO, Kales A, Leo LA, Slye EA. A comparison of subjective estimates and objective sleep laboratory findings in insomnia patients. *Sleep Res* 1973; 2:143.
39. Carskadon MA, Dement WC, Mitler MM, Guilleminault C, Zarcone B, Spiegel R. Self-reports versus sleep laboratory findings in 122 drug-free subjects with complaints of chronic insomnia. *Am J Psychiatry* 1976; 133:1382-1388.
40. Frankel BL, Coursey RD, Buchbinder R, Snyder F. Recorded and reported sleep in chronic primary insomnia. *Arch Gen Psychiatry* 1976; 33:615-623.
41. Hoch CC, Reynolds CF, Kupfer DJ, Berman SR, Houck PR, Stack JA. Empirical note: Self-report versus recorded sleep in healthy seniors. *Psychophysiology* 1987; 24:293-299.
42. Wagenknecht LE, Burke GL, Perkins LL, Haley NJ, Friedman GD. Misclassification of smoking status in the CARDIA study: A comparison of self-report with serum cotinine levels. *Am J Public Health* 1992; 82:33-36.

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