

Association of Active and Passive Smoking with Sleep Disturbances and Short Sleep Duration among Japanese Working Population

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Background: The relationship between passive smoking and sleep is uncertain. **Purpose:** To examine the association of passive/active smoking with sleep disturbances. **Method:** 732 women and 1,896 men, working in a suburb of Tokyo, were surveyed using a self-administered questionnaire. Information on smoking, passive smoking exposure, and sleep was elicited. Exposure levels to passive smoking were assessed separately at work and at home as no, occasional, or regular exposure. Risk of sleep disturbances according to smoking status was estimated using logistic regression with odds ratios (OR) and 95% confidence intervals (CIs) as measures of association. **Results:** Compared to never smokers, odds of difficulty awakening in the morning (DAM) in current smokers were significantly higher for women (OR 1.95) and men (OR 1.50), while increased difficulty initiating sleep (OR 1.88) and decreased early morning awakening (OR 0.31) were found only in women. Never smoking men occasionally exposed to passive smoking at work but not at home had increased odds (OR 1.81) of short sleep duration (SSD, <6 h) than unexposed counterparts. **Conclusions:** The analyses suggest that exposure to passive smoking at work is associated with SSD in men, while current smoking relates to various subtypes of sleep disturbances in both sexes.

Key words: active smoking, passive smoking, sleep disturbance, short sleep duration, Japan

Introduction

Sleep disturbance is one of the most commonly reported health complaints, with a prevalence of 16% to 38% in the working population (e.g., Doi,

2005; Jacquinet-Salord, Lang, Fouriaud, Nicouldet, & Bingham, 1993; Kuppermann et al., 1995; Nakata et al., 2000, 2004a), and active smoking has been suspected to be an important modifiable risk factor (Htoo, Talwar, Feinsilver, & Greenberg, 2004).

The relationship between smoking and sleep disturbance has been investigated in many previous studies, and it has been reported that active smokers compared with nonsmokers more often experience sleep disturbance such as difficulty initiating sleep (DIS) (Janson et al., 1995; Kaneita et al., 2005; Ohida et al., 2004; Phillips & Danner, 1995; Soldatos, Kales, Scharf, Bixler, & Kales, 1980; Wetter & Young, 1994; Zhang, Samet, Caffo, & Punjabi, 2006), difficulty maintaining sleep (DMS) (Lexcen & Hicks, 1993; Phillips & Danner, 1995), early morning awakening (EMA) (Kaneita et al., 2005; Lexcen & Hicks, 1993), and difficulty awakening in the morning (DAM) (Akerstedt, Knutsson, Westerholm, Theorell, Alfredsson, & Kecklund, 2002; Lexcen & Hicks, 1993), as well as short sleep duration (SSD) (Bale & White, 1982; Kaneita et al., 2005; Ohida et al., 2001, 2004; Palmer, Hjorns, & Harrison, 1980; Riedel, Durrence, Lichstein, Taylor, & Bush, 2004; Soldatos et al., 1980; Zhang et al., 2006), subjective insufficient sleep (Kaneita et al., 2005;

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Ohida et al., 2001, 2004), poor sleep quality (Bale & White, 1982; Lexcen & Hicks, 1993), excessive daytime sleepiness (Kaneita et al., 2005; Ohida et al., 2004; Phillips & Danner, 1995), snoring (Franklin et al., 2004; Khoo, Tan, Ng, & Ho, 2004; Shin, Joo, Kim, & Kim, 2003), sleep disordered-breathing (Khoo et al., 2004; Wetter, Young, Bidwell, Badr, & Palta, 1994), and poor sleep hygiene (Jefferson et al., 2005).

In contrast, some studies found no relationship between smoking and insomnia symptoms (Kim, Uchiyama, Okawa, Liu, & Ogihara, 2000; Phillips & Danner, 1995), poor sleep quality (Doi, Minowa, & Tango, 2003; Palmer et al., 1980), EMA (Janson et al., 1995; Wetter & Young, 1994), DIS (Lexcen & Hicks, 1993; Riedel et al., 2004), DMS (Kaneita et al., 2005; Riedel et al., 2004; Wetter & Young, 1994), and disturbed sleep (Akerstedt et al., 2002). One study found a significant positive effect by smoking, such as waking up less often during the night and reduced difficulty remaining asleep (Fabsitz, Sholinsky, & Goldberg, 1997).

A major concern related to these studies is that they did not take into account the contribution of passive smoking to sleep disturbance even though exposure to passive smoking is prevalent in many developed/developing countries (Janson et al., 2001; Nakata, Tanigawa, Araki, Sakurai, & Iso, 2004c; Statistical Database, Ministry of Health, Labor and Welfare, Japan, 2002). Thus, the findings must be interpreted with caution since most past studies of active smoking treated non/never smokers exposed to passive smoking as the referent (control) group. Such categorization could obscure the relationship between smoking and sleep. In addition, many previous studies did not adjust for lifestyle or sociodemographic factors that are closely related to both smoking and sleep. Smokers drink caffeinated beverages and consume alcoholic drinks more often than non-smokers, both of which are known to impair sleep (Istvan & Matarazzo, 1984; Urponen, Vuori, Hasan, & Partinen, 1988). Educational status is often negatively correlated with smoking prevalence and positively correlated with sleep quality (Centers for Disease Control and Prevention, 2005; Chen, Kawachi, Subramanian, Acevedo-Garcia, & Lee, 2005; Doi, Minowa, & Tango, 2003). Married people are reported to have better quality sleep than unmarried people (Akerstedt et al., 2002; Chen et al., 2005; Doi, Minowa, & Tango, 2003). Overweight is also a well-known factor related to sleep problems (Akerstedt et al., 2002; Chen et al., 2005; Ohida et al., 2001). Shift work is suspected to be a major occupational risk factor for impaired sleep (e.g., Takahashi et al., 2006). Taking into account the above various potential confounding factors may reveal a more clear relationship between smoking and sleep.

A number of studies have shown that passive smoking is adversely related to various health problems (e.g., Janson et al., 2001), but little attention has been given to its association with sleep. As far as we know, only one study has examined the relationship between passive smoking and sleep-related problems. A recent study by Franklin et al. (2004) revealed that passive smoking is a major risk factor for habitual snoring; never smokers who were exposed to passive smoking on a daily basis had a snoring rate 1.6 times higher than non-exposed counterparts, which was equivalent (1.8 times) to active smokers' consumption of 10 cigarettes a day.

Therefore, we designed this study to examine the association of passive and active smoking with sleep disturbance and sleep duration among 3,693 workers in small and medium-sized enterprises (SMEs), where smoking is prevalent. Exposure to passive smoking at work and at home was taken into account as major sources of passive smoking exposure, and a broad range of potential confounding factors were controlled in the statistical analyses.

Methods

Subjects and Procedure

The study design was cross-sectional and data were collected by a self-rated questionnaire from October to December 2002. Subjects were workers in SMEs, with 1 to 158 workers in Yashio City, Saitama, and Ohta ward, Tokyo. Yashio City has the highest percentage of manufacturing plants in Saitama prefecture. The Ohta ward, which is a so-called "industrial area" is unique for its number of SMEs. Questionnaires were distributed to 2,591 workers from 248 factories in Yashio City and 1,102 workers from 52 factories in the Ohta ward by visiting each factory ($n = 3,693$). Finally, responses were obtained from 2,884 workers from 296 enterprises, representing a response rate of 78.1%. Among 2,884 workers, we excluded 156 because of missing responses. Data on a total of 2,628 workers were subjected to the final analyses.

The questionnaire elicited information on demographics, lifestyle, height and body weight, work schedule, and history of physical/psychological disease(s). Sleep variables and smoking history were included under the topics of lifestyle and physical/psychological disease(s).

The Medical Ethical Committee of the University of Tokyo approved this study, and written informed consent was obtained from all participants.

Measurements

Smoking status. Smoking status and exposure to passive smoking were assessed by the following three questions: (1) Are you a current, former, or never smoker? (2) If you are a current smoker, how many

cigarettes a day do you smoke and how many years have you been smoking? and (3) Are you currently exposed to cigarette smoke from other people? We asked these questions separately for two locations, work and home, and responses were classified into three levels: no exposure, occasional exposure, and regular exposure (Kawachi et al., 1997; Nakata et al., 2006).

Sleep questions. The self-administered sleep questionnaire was based on our previous studies (Nakata et al., 2004a, 2004b, 2005), and five questions about daily sleep habits during the previous 1-year period were included as follows:

1. How long does it usually take you to fall asleep in bed? (SQ1) (0–10 min/11–30 min/31–59 min/1–2 hr / 2+ hr)
2. How often do you have difficulty staying asleep? (SQ2) (never or almost never/few times a year/more than once a month/more than once a week/more than three times a week/almost every day)
3. How often do you wake up too early and cannot fall asleep again? (SQ3) (never or almost never/few times a year/more than once a month/more than once a week/more than three times a week/almost every day)
4. Do you find it difficult to wake up in the morning? (SQ4) (not at all/rarely/not very/a little/somewhat/considerably)
5. On the average, how much sleep at night did you usually get during the last year? (SQ5) (less than 5 hr/5–6 hr/6–7 hr/7–8 hr/8–9 hr/9+ hr)

DIS was defined as present if the answer to the first question indicated “31–59 min,” “1–2 hr,” or “2+ hr.” DMS and EMA were indicated by the answer “more than 3 times a week” or “almost every day” in response to the second and third questions. DAM was indicated by an answer of “considerably” or “somewhat” to the fourth question. SSD was defined as present if the answer to the fifth question indicated “less than 5 hr” or “5–6 hr.”

The reliability of sleep questions used in this study was assessed by test-retest over 1 year in another set of samples. We analyzed data derived from the same sleep questions as used in the present study obtained from 132 white-collar workers collected during April 2002 and 2003. Spearman's rank correlation coefficient for each sleep question was calculated as follows: SQ1 .698; SQ2 .545; SQ3 .631; SQ4 .777; and SQ5 .739 (all *p* values were less than .001). Validity was estimated by calculating Spearman's rank correlation coefficients among age, sex, educational level, depressive symptoms (Center for Epidemiologic Studies Depression scale (CES-D)), alcohol drinking, caffeine intake, survey location, and sleep questions in the present sample (see Appendix). The results showed that all corre-

lations were in the expected direction as in previous studies (Chen et al., 2005; Nakata et al., 2000), and the sleep questions properly showed an almost zero correlation with a measure with which it was predicted to have no relationship (i.e., survey location).

Other covariates. Other covariates were age, educational level (junior high school, high school, vocational/college/university), marital status (married/not married), work schedule (shift or night/daytime), lifestyle, and history of physical/psychological diseases. Lifestyle factors included alcohol consumption (number of alcoholic drinks consumed/day, with one drink estimated as about 9 g of pure ethanol) and caffeine intake (cups of tea or coffee/day). Physical/psychological conditions included presence of self-reported physical/psychological disease(s) and body mass index (calculated as weight in kilograms divided by the square of height in meters, BMI). Types of physical/psychological diseases included hypertension, hyperlipidemia, diabetes mellitus, menopausal syndrome, and other diseases such as heart disease, cancer, liver disease, renal disease, peptic ulcer, gastrointestinal diseases, neurological diseases, musculoskeletal disorders, and psychiatric illnesses. We excluded from analysis 4 women and 12 men who had been diagnosed as having major depression.

Statistical Analyses

Differences in sleep disturbances and SSD by gender were calculated using the χ^2 test. Risk of each sleep variable by smoking status was estimated using univariate and multivariate logistic regression with odds ratios (ORs) and 95% confidence interval (CI) as measures of association. The multivariate model adjusted for age in 10-year increments, marital status, educational level, alcohol consumption (0, 0.01–4.9, 5.0–14.9, 15.0–24.9, 25.0 and over, g/d), caffeine intake (almost none, 1–2, or 3 or more cups of coffee or tea), BMI (<20.0, 20.0–22.5, 22.6–25.0, >25.0), disease(s) currently under treatment, work schedule, and other sleep variables (DIS, DMS, DAM, and/or SSD). Because DMS and EMA were highly correlated ($r > 0.5$), only DMS was included as a confounding variable in the multivariate model. Pearson correlation coefficients and partial correlation coefficients adjusting for confounders were used to examine the linear relationship between active/passive smoking and sleep variables. This was done for the never smoking subgroup as well as for all smoking subgroups, including never, former, and current smokers. The significance level for all statistical analyses was $p < 0.05$ (two-tailed test). All data were analyzed using the Statistical Package for the Social Sciences version 14.0 (SPSS Inc., Chicago, IL, USA).

Results

The characteristics of study participants stratified by smoking status and sex are shown in Table 1. Overall, 62.3% of men and 24.7% of women were current smokers. Prevalence of sleep disturbances in women was 16.9% for DIS, 8.7% for DMS, 7.1% for EMA, and 26.9% for DAM, and in men was 16.8% for DIS, 7.6% for DMS, 6.0% for EMA, and 25.6% for DAM. Prevalence of SSD was 41.5% for women and 38.6% for men. There were no significant differences between women and men in the prevalence of sleep disturbances or SSD, although such disturbances were slightly more prevalent in women than in men.

We examined the association between smoking status (current, former, and never) and sleep variables. Compared to never smokers (referent group), increased odds of DAM were found in current smoking women (OR 1.95, 95% CI 1.23, 3.09) and men (OR 1.50, 95% CI 1.12, 2.02) by multivariate logistic regression analysis (and also by univariate analysis). Current smoking women also had increased odds of DIS (OR 1.88, 95% CI 1.11, 3.20) but reduced odds of EMA (OR 0.31, 95% CI 0.11, 0.98) compared to never smoking women. In men, decreased EMA (OR 0.63, 95% CI 0.43, 0.94) in current smokers and decreased DAM (OR 0.48, 95% CI 0.31, 0.76) in former smokers were found by univariate analysis but not by multivariate analysis. No significant differences were found between other sleep variables and smoking status.

We calculated the prevalence of never smokers exposed to passive smoking at work and at home according to levels of passive smoking exposure. Overall, 64.8% of women and 59.4% of men reported exposure to passive smoking either at work or at home. Eighteen percent of women and 6% of men were occasionally exposed at home while 9.7% of women and 2.5% of men were regularly exposed at home. On the other hand, 25.3% of women and 37.5% of men were occasionally exposed at work while 11.9% of women and 13.3% of men were regularly exposed at work. Thus, women were more often exposed at home (27.7%) than men (8.5%), and men were exposed at work (50.8%) more often than women (37.2%).

Relationships between never smokers exposed to different levels of passive smoking and sleep, differentiated by sources of exposure, are shown in Table 2. First, we focused on the effect on sleep variables of exposure to passive smoking at work in never smokers (see left side of Table 2). Compared to never smokers unexposed to passive smoking either at work or at home (referent group), increased odds of DAM were found in never smoking women with regular exposure to passive smoking at work by univariate analysis (OR 2.16, 95% CI 1.15, 3.94), but the effect was insignificant after adjusting for confounders (OR 1.73, 95% CI 0.80, 3.73). In men, occasional exposure to passive smoking

at work in never smoking men resulted in significantly higher odds of SSD (OR 1.81, 95% CI 1.11, 2.94), while regular exposure was not related to a significant increase in SSD (OR 1.22, 95% CI 0.65, 2.30) compared to the referent group by multivariate analyses. Current smoking women (OR 2.12, 95% CI 1.12, 4.01) had significantly increased odds by multivariate logistic regression analysis (data not shown). There was a significant dose-dependent increase between smoking status and DAM, but this increase was insignificant when analyses were limited to never smokers exposed to different levels of passive smoking.

Second, we focused on the effect of passive smoking exposure at home on sleep variables (see right side of Table 2). We found no significant relationship between passive smoking and sleep in both women and men. In women, only current smoking was significantly associated with DAM (OR 1.96, 95% CI 1.03, 3.76) by multivariate analyses. In men, former smokers had significantly increased odds of SSD (OR 1.63, 95% CI 1.01, 2.62).

Third, analyses were also done to examine the effect of passive smoking exposure either at work or at home on sleep variables. As the results were almost identical to those of exposure at work, data are not described in the text.

Discussion

This study revealed that current smoking increases DAM in both men and women, and increases DIS and reduces EMA in women compared to never smokers. When never smokers were categorized into three groups according to their level of exposure to passive smoking, and never smokers unexposed to passive smoking comprised the referent group, only men occasionally exposed to passive smoking at work had significantly increased odds of SSD. Current smoking women had significantly increased odds of DAM compared to never smokers unexposed to passive smoking. The analyses suggest that passive smoking at work may be related to sleep loss in men, while current smoking relates to various subtypes of sleep disturbances in both sexes.

Occasionally exposure to passive smoking in men was significantly associated with SSD. The results revealed that exposure to passive smoking at work, but not at home, could be a contributing factor for SSD. However, we failed to find a significant relationship between men "regularly" exposed to passive smoking and SSD. Four explanations are possible for these findings: (1) Since exposure levels to passive smoking were measured subjectively, men may have underreported their levels of exposure. (2) Men occasionally exposed to passive smoking may be more sensitive than the regular exposure group because the occasional

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Table 1. *Characteristics of Survey Respondents According to Smoking Status Stratified by Sex^a*

Characteristics	Women Smoking Status			Men Smoking Status		
	Never	Former	Current	Never	Former	Current
Number of subjects (%) ^b	506 (69.1)	45 (6.1)	181 (24.7)	480 (25.3)	235 (12.4)	1,181 (62.3)
Age (years), mean (SD)	46.2 (14.0)	38.7 (10.9)	42.0 (12.9)	42.9 (14.1)	50.9 (12.5)	43.2 (12.6)
Age group						
16 to 29	91 (62.8)	11 (7.6)	43 (29.7)	90 (31.7)	14 (4.9)	180 (63.4)
30 to 39	74 (60.2)	14 (11.4)	35 (28.5)	108 (23.2)	32 (6.9)	325 (69.9)
40 to 49	71 (61.2)	12 (10.3)	33 (28.4)	75 (22.5)	39 (11.7)	220 (65.9)
50 to 59	184 (74.8)	6 (2.4)	56 (22.8)	127 (24.1)	83 (15.7)	317 (60.2)
60 and over	86 (84.3)	2 (2.0)	14 (13.7)	80 (28.0)	67 (23.4)	139 (48.6)
Marital status						
Married	339 (70.6)	26 (5.4)	115 (24.0)	297 (23.1)	180 (14.0)	810 (62.9)
Not married	167 (66.3)	19 (7.5)	66 (26.2)	183 (30.0)	55 (9.0)	371 (60.9)
Highest education						
Junior high school	108 (73.0)	4 (2.7)	36 (24.3)	115 (22.9)	62 (12.4)	325 (64.7)
High school	269 (67.9)	26 (6.6)	101 (25.5)	196 (43.8)	88 (19.6)	164 (36.6)
Vocational/college/university	129 (68.6)	15 (8.0)	44 (23.4)	169 (17.9)	85 (9.0)	692 (73.2)
Alcohol consumption (g ethanol/day)						
Non-drinker (0.0)	308 (77.2)	16 (4.0)	75 (18.8)	137 (30.3)	61 (13.5)	254 (56.2)
0.01 to 4.9	122 (69.3)	13 (7.4)	41 (23.3)	87 (36.4)	21 (8.8)	131 (54.8)
5.0 to 14.9	57 (57.0)	8 (8.0)	35 (35.0)	120 (28.2)	50 (11.7)	256 (60.1)
15.0 to 24.9	14 (41.2)	4 (11.8)	16 (47.1)	71 (18.6)	50 (13.1)	260 (68.2)
25.0 and over	5 (21.7)	4 (17.4)	14 (60.9)	65 (16.3)	53 (13.3)	280 (70.4)
Caffeine intake (cups of coffee or tea/day)						
Almost none	44 (81.5)	0 (0.0)	10 (18.5)	76 (43.4)	33 (18.9)	66 (37.7)
1 to 2	235 (73.2)	30 (9.3)	56 (17.4)	235 (26.5)	116 (13.1)	536 (60.4)
3 or more	227 (63.6)	15 (4.2)	115 (32.2)	169 (20.3)	86 (10.3)	579 (69.4)
Body Mass Index (kg/height (m) ²)						
< 20.0	129 (62.6)	11 (5.3)	66 (32.0)	53 (18.3)	26 (9.0)	211 (72.8)
20.0 to 22.5	177 (68.9)	16 (6.2)	64 (24.9)	144 (24.1)	60 (10.0)	394 (65.9)
22.6 to 25.0	116 (73.4)	11 (7.0)	31 (19.6)	153 (26.9)	81 (14.3)	334 (58.8)
> 25.0	84 (75.7)	7 (6.3)	20 (18.0)	130 (29.5)	68 (15.5)	242 (55.0)
Sleep disturbance (Yes)						
Difficulty initiating sleep	77 (62.1)	4 (3.2)	43 (34.7)	77 (24.1)	39 (12.2)	203 (63.6)
Difficulty maintaining sleep	44 (68.8)	4 (6.3)	16 (25.0)	41 (28.5)	22 (15.3)	81 (56.3)
Early morning awakening	38 (73.1)	3 (5.8)	11 (21.2)	34 (30.1)	18 (15.9)	61 (54.0)
Difficulty awakening in the morning	114 (57.9)	14 (7.1)	69 (35.0)	107 (23.4)	26 (5.7)	325 (71.0)
Habitual sleep duration						
Less than 5 hr	43 (67.2)	6 (9.4)	15 (23.4)	47 (27.3)	15 (8.7)	110 (64.0)
5 to 6 hr	162 (67.2)	15 (6.2)	64 (26.6)	131 (23.9)	72 (13.1)	346 (63.0)
6 to 7 hr	206 (70.8)	17 (5.8)	68 (23.4)	167 (23.1)	93 (12.9)	462 (64.0)
7 to 8 hr	77 (69.4)	7 (6.3)	27 (24.3)	113 (32.8)	38 (11.0)	193 (56.1)
> 8 hr	18 (72.0)	0 (0.0)	7 (28.0)	22 (20.2)	17 (15.6)	70 (64.2)
Disease(s) currently under treatment (Yes)	123 (72.4)	9 (5.3)	38 (22.4)	146 (27.8)	110 (20.9)	270 (51.3)
Hypertension	63 (81.8)	3 (3.9)	11 (14.3)	95 (33.5)	67 (23.6)	122 (43.0)
Hyperlipidemia	20 (76.9)	3 (11.5)	3 (11.5)	28 (30.8)	19 (20.9)	44 (48.4)
Diabetes mellitus	10 (66.7)	0 (0.0)	5 (33.3)	29 (31.5)	18 (19.6)	45 (48.9)
Menopausal syndrome	21 (75.0)	1 (3.6)	6 (21.4)	1 (14.3)	3 (42.9)	3 (42.9)
Other diseases ^c	78 (68.4)	12 (10.5)	2 (21.1)	60 (28.8)	40 (19.2)	108 (51.9)
Work schedule						
Daytime	481 (69.4)	43 (6.2)	169 (24.4)	456 (25.2)	228 (12.6)	1,129 (62.3)
Shift/night	25 (64.1)	2 (5.1)	12 (30.8)	24 (28.9)	7 (8.4)	52 (62.7)

^aData may not total 100% due to rounding.

^bPercent within smoking status.

^cOther diseases included heart disease, cancer, liver disease, renal disease, peptic ulcer, gastrointestinal diseases, neurological diseases, musculoskeletal disorders, and psychiatric illnesses.

Table 2. Odds Ratios for Sleep Disturbances and Short Sleep Duration in Never Smokers Exposed to Passive Smoking at Work or at Home Stratified by Sex

Sleep Variable	No Exposure ^a	At Work				At home			
		Occasional OR (95% CI)	p	Regular OR (95% CI)	p	Occasional OR (95% CI)	p	Regular OR (95% CI)	p
Women	n = 178	n = 128		n = 60		n = 91		n = 49	
Difficulty initiating sleep (yes vs. no)									
Unadjusted odds ratio	1.00	1.16 (0.65, 2.08)	.616	0.88 (0.41, 1.87)	.731	.874	.877	0.68 (0.28, 1.65)	.392
Adjusted odds ratio ^b	1.00	0.83 (0.38, 1.81)	.633	1.02 (0.40, 2.60)	.971	.584	.636	0.57 (0.19, 1.69)	.310
Difficulty maintaining sleep (yes vs. no)									
Unadjusted odds ratio	1.00	0.63 (0.29, 1.38)	.248	0.67 (0.26, 1.75)	.415	.292	.493	1.03 (0.41, 2.57)	.958
Adjusted odds ratio ^c	1.00	0.66 (0.22, 1.99)	.459	1.22 (0.36, 4.16)	.746	.957	.746	1.64 (0.52, 5.19)	.404
Early morning awakening (yes vs. no)									
Unadjusted odds ratio	1.00	1.04 (0.45, 2.44)	.923	0.90 (0.30, 2.64)	.842	.884	.873	1.43 (0.51, 2.73)	.497
Adjusted odds ratio ^c	1.00	1.44 (0.38, 5.54)	.595	2.61 (0.51, 13.3)	.248	.618	.989	2.13 (0.53, 8.51)	.284
Difficulty awakening in the morning (yes vs. no)									
Unadjusted odds ratio	1.00	1.57 (0.92, 2.68)	.096	2.16 (1.15, 3.94)	.016	.012	.467	1.71 (0.86, 3.37)	.124
Adjusted odds ratio ^d	1.00	1.06 (0.54, 2.07)	.872	1.73 (0.80, 3.73)	.160	.214	.832	1.65 (0.72, 3.78)	.237
Short sleep duration (yes vs. no)									
Unadjusted odds ratio	1.00	1.06 (0.68, 1.66)	.792	0.95 (0.55, 1.64)	.859	.932	.597	0.98 (0.54, 1.76)	.933
Adjusted odds ratio ^e	1.00	1.12 (0.65, 1.92)	.690	0.96 (0.50, 1.86)	.912	.827	.817	1.44 (0.72, 2.88)	.298

Table 2. Odds Ratios for Sleep Disturbances and Short Sleep Duration in Never Smokers Exposed to Passive Smoking at Work or at Home Stratified by Sex

Sleep Variable	No Exposure ^a	At Work				At home			
		Occasional OR (95% CI)	p	Regular OR (95% CI)	p for Trend	Occasional OR (95% CI)	p	Regular OR (95% CI)	p for Trend
Men	n = 195	n = 180	n = 64	n = 29	n = 12				
Difficulty initiating sleep (yes vs. no)									
Unadjusted odds ratio	1.00	0.99 (0.57, 1.71)	.965	1.15 (0.56, 2.35)	.700	1.35 (0.51, 3.58)	.547	2.03 (0.51, 8.07)	.317
Adjusted odds ratio ^b	1.00	0.98 (0.51, 1.90)	.954	1.10 (0.48, 2.53)	.826	0.63 (0.16, 2.42)	.499	2.39 (0.51, 11.2)	.268
Difficulty maintaining sleep (yes vs. no)									
Unadjusted odds ratio	1.00	0.83 (0.41, 1.66)	.595	0.39 (0.11, 1.34)	.134	0.65 (0.14, 2.95)	.577	—	.237
Adjusted odds ratio ^c	1.00	0.81 (0.35, 1.89)	.629	0.55 (0.15, 2.08)	.379	—	—	—	.469
Early morning awakening (yes vs. no)									
Unadjusted odds ratio	1.00	0.84 (0.39, 1.82)	.663	0.67 (0.21, 2.08)	.485	0.40 (0.05, 3.16)	.387	—	.196
Adjusted odds ratio ^c	1.00	0.84 (0.34, 2.11)	.713	0.94 (0.27, 3.21)	.918	—	—	—	—
Difficulty awakening in the morning (yes vs. no)									
Unadjusted odds ratio	1.00	0.87 (0.53, 1.43)	.576	1.45 (0.79, 2.67)	.235	1.83 (0.79, 4.21)	.156	2.09 (0.58, 7.49)	.092
Adjusted odds ratio ^d	1.00	0.58 (0.32, 1.06)	.075	1.03 (0.50, 2.12)	.943	0.85 (0.31, 2.37)	.759	0.72 (0.17, 3.07)	.658
Short sleep duration (yes vs. no)									
Unadjusted odds ratio	1.00	2.11 (1.39, 3.22)	.001	1.52 (0.86, 2.67)	.148	1.66 (0.75, 3.68)	.211	1.42 (0.40, 5.06)	.585
Adjusted odds ratio ^e	1.00	1.81 (1.11, 2.94)	.017	1.22 (0.65, 2.30)	.540	1.01 (0.39, 2.58)	.988	0.54 (0.12, 2.31)	.402

^aNeither exposed at work nor at home.

^bAdjusted for age in 10-year increments, marital status, educational level, alcohol consumption, caffeine intake, BMI, disease(s) currently under treatment, work schedule, habitual sleep duration, DMS, and DAM.

^cAdjusted for age in 10-year increments, marital status, educational level, alcohol consumption, caffeine intake, BMI, disease(s) currently under treatment, work schedule, habitual sleep duration, DIS, and DAM.

^dAdjusted for age in 10-year increments, marital status, educational level, alcohol consumption, caffeine intake, BMI, disease(s) currently under treatment, work schedule, habitual sleep duration, DIS, and DMS.

^eAdjusted for age in 10-year increments, marital status, educational level, alcohol consumption, caffeine intake, BMI, disease(s) currently under treatment, work schedule, DIS, DMS, and DAM.

exposure group was not used to such exposure. (3) The sample size of the regular exposure group may not have been sufficiently large to observe a relationship (exposure at work, $n = 64$; exposure at home, $n = 12$). (4) The findings may be by chance or by the effects of unadjusted/unknown confounding variables. Further studies are warranted to replicate this finding by introducing objective measures such as blood/urine cotinine concentrations to estimate passive smoking levels with a larger sample size of passive smokers.

Univariate analyses revealed that women regularly exposed to passive smoking at work had significantly increased odds of DAM. Although the association was attenuated to be insignificant after controlling for potential confounders, the OR remained at a moderately high level (OR 1.73). Never smokers exposed to passive smoking may have uncomfortable manifestations such as smoke-induced irritation, cough, wheeze, phlegm, runny nose, annoyance, and disturbed mood (Eisner & Blanc, 2002; Janson et al., 2001; Lam et al., 2000), which may cause DAM. These discomforts occur more often in women than in men since a national survey suggested that 55.3% of women but only 38.5% of men reported that they felt uncomfortable or sick by exposure to passive smoking (Statistical Database, Ministry of Health, Labor and Welfare, Japan, 2002). This evidence may partly explain why passive smoking is weakly related to DAM in women.

Current smoking was significantly associated with DAM compared to never smoking. Wetter & Young (1994) reported that current smokers had a 1.5–2.4 times greater risk of “very difficult to wake up” than never smokers both in men and women. Akerstedt et al. (2002) also reported a 1.75 times higher risk of “difficulties in awakening” in smokers, but that smoking was neither associated with “disturbed sleep” nor “not rested” among healthy working people. Lexcen & Hicks (1993) found that smokers “woke up tired” more often than nonsmokers, which may be related to DAM. Exposure to smoking may interrupt restoration of fatigue causing DAM.

A significant increase of DIS and a decrease of EMA among current smoking women compared to never smoking women were found in this study. However, after considering the effect of passive smoking exposure, these relationships turned out to be insignificant. Former smokers also had an insignificant decrease in DIS than those of unexposed never smokers (OR 0.42, 95% CI 0.11, 1.69). Former smoking women may have quit smoking because of DIS. A decrease in EMA was an unexpected finding, but current smoking women may use tobacco to reduce EMA associated with depressive symptoms (e.g., Johnson & Breslau, 2006).

Former smoking men had significantly higher odds of SSD than never smoking men unexposed to passive smoking. It is not clear why former smokers exhibit reduced sleep duration. However, former smok-

ing women also had an insignificant increase in SSD. Sleep disturbance, such as problems with awakening in the morning, may have disappeared through smoking cessation, but former smokers may continue the same duration of sleep as when they were active smokers.

Potential mechanisms underlying the present findings could be explained as follows. First, exposure to nicotine stimulates the brain to enhance/maintain alertness, which keeps smokers from initiating sleep (Benowitz, 1996; Soldatos et al., 1980; Takahashi et al., 2005). This results in a reduced total sleep time and may be decreased EMA. That transdermal nicotine decreases total sleep time and reduces sleep efficiency and the percentage of rapid eye movement sleep support the above assumption (Boutrel & Koob, 2004; Davila, Hurt, Offord, Harris, & Shepard, 1994). Next, the nicotine level in the brain gradually decreases during sleep and eventually induces smokers to exhibit nicotine deprivation leading to DAM. Because of insufficient sleep and DAM, smokers may use tobacco to increase alertness and help them wake up. In passive smokers who exhibit DAM, a similar mechanism may occur even though the exposure level to nicotine is much lower.

The results of the present study suggested that women's sleep was more affected by smoking than men's sleep. Several previous studies may help explain this difference. First, it was reported that women tend to develop bronchial hyper-responsiveness and respiratory symptoms more than men when exposed to tobacco smoke (Leynaert, Bousquet, Henry, Kiard, & Neukirch, 1997). Second, women who smoke have a 1.3–1.4 times higher risk of snoring than men who smoke (Franklin et al., 2004). Also, in the same study, never smoking women who were exposed to passive smoking every day exhibited a 1.6 times higher risk of snoring than their never exposed counterparts. Such smoking-induced snoring may result in insufficient sleep and increase the risk of sleep disturbance. Further studies are warranted to confirm the role of snoring and disturbed respiratory functioning on the relationship between smoking and sleep disturbance.

Another possibility that may help explain why women have more sleep problems than men because of smoking may be related to the higher social pressure not to smoke placed on women than on men in Japan (Mori et al., 2003). Women who persist in smoking despite such pressure may have more problems with psychosocial stress, quality of life, anxiety, and depressed mood, which may contribute to impaired sleep. Also, as a consequence of such social pressure, more women than men may try to smoke in private spaces, such as at home, rather than at the workplace or in public places to avoid exposure to such pressure. This may result in women smoking before bedtime and developing DIS.

Our results should be interpreted in light of at least five limitations. First, response bias may have occurred if the nonrespondents differed from the respondents with respect to smoking status and sleep symptoms. The nonresponding population may have more problems related to smoking or sleep. Of a total of 3,693 subjects, 1,065 (28.8%) did not participate in the survey. However, we could not examine this possible bias because detailed data on nonrespondents were unavailable. Second, the data in this study were derived from a single survey of workers in two geographically small areas (Yashio City and Ohta ward) with small subgroups of exposure to passive smoking, which may attenuate the power of statistical analysis. Third, data on smoking status and sleep were obtained by self-report, a means of obtaining information that could result in bias. However, several previous studies have observed that self-reported data on sleep were at least moderately associated with physiologic recordings of sleep (Carskadon et al., 1976; Wetter & Young, 1994). Self-reported passive smoking was also found to be modestly associated with salivary and urinary cotinine (Cummings, Markello, Mahoney, Bhargava, McElroy, & Marshall, 1990; Emmons et al., 1994). Another restriction of the finding is that the timeframe of the sleep questionnaire used in this study was 1 year, which may not reflect the seasonal effects on sleep habits. Although we need to be cautious in interpreting the results, our intention was to assess chronic sleep habits. Fourth, the inherent nature of a cross-sectional design does not permit the conclusion that current smoking or passive smoking causes sleep disturbance or short sleep duration. Fifth, some confounding variables that may have an impact on both smoking and sleep, such as working hours, habitual exercise, habitual nap, occupation, stress, and marital satisfaction were not included in this study.

Despite the fact that smoking status and sleep variables were assessed by self-report, as well as other limitations, these data suggest that passive smoking is associated with SSD in men and DAM in active smoking women. Current smoking affected sleep to a greater degree in women than in men, suggesting a gender difference in the effect. Further studies are warranted to replicate the present observations.

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Appendix. *Correlations among Sleep Questions, Age, Sex, Educational Level, Depressive Symptoms, Alcohol Drinking, Caffeine Intake, and Survey Location*

Variable	1	2	3	4	5	6	7	8	9	10	11	12
1. Time to fall asleep (Q1)												
2. Frequency of difficulty maintaining sleep (Q2)	.358***											
3. Frequency of early morning awakening (Q3)	.286***	.650***										
4. Frequency of difficulty awakening in the morning (Q4)	.131***	.075***	.025NS									
5. Habitual sleep duration (Q5)	-.101***	-.084***	-.071***	-.177***								
6. Age (in years)	-.067***	.110***	.144***	-.380***	.157***							
7. Sex (men, 1; women, 2)	-.020NS	-.024NS	-.058	.052**	-.039*	-.004NS						
8. Educational Level (lowest, 1; highest, 3)	-.042 *	-.055**	-.055**	.109***	-.153***	-.334***	-.060**					
9. Depressive symptoms (CES-D with sleep item)	.210***	.307***	.265***	.223***	-.116***	-.073***	-.011NS	-.055**				
10. Depressive symptoms (CES-D without sleep item)	.184***	.276***	.242***	.215***	-.104***	-.078***	-.014NS	-.051**	.994***			
11. Alcohol drinking (g ethanol/day)	-.010NS	.086***	.112***	-.078***	.098***	.095***	-.388***	-.023NS	-.024NS	-.022NS		
12. Caffeine intake (cups of tea or coffee/day)	-.004NS	-.024NS	-.017NS	.057**	-.076***	-.041*	.046*	.045*	-.042*	-.041**	-.055**	
13. Survey location (Yashio, 1; Ohta, 2)	.005NS	.001NS	.019NS	.004NS	-.107***	.005NS	-.167***	.109***	.007NS	.008NS	.095***	-.045*

Note: CES-D = Center for Epidemiologic Studies Depression Scale.

* $p < .05$; ** $p < .01$; *** $p < .001$; NS, non-significant (Spearman's rank correlation coefficients).