

Active and passive smoking and depression among Japanese workers [☆]

Akinori Nakata ^{a,*}, Masaya Takahashi ^b, Tomoko Ikeda ^c, Minoru Hojou ^d,
Jeannie A. Nigam ^a, Naomi G. Swanson ^a

^a National Institute for Occupational Safety and Health, USA

^b National Institute of Occupational Safety and Health, Japan

^c Department of Nursing, School of Health Sciences, Ibaraki Prefectural University of Health Sciences, Ibaraki, Japan

^d Ota Regional Occupational Health Center, Tokyo, Japan

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Abstract

Objective. To assess the relation of passive and active smoking to depressive symptoms in 1839 men and 931 women working in a suburb of Tokyo in 2002.

Method. Self-reported smoking history and exposure to passive smoking (no, occasional, or regular) at work and at home. Depressive symptoms according to the Center for Epidemiologic Studies Depression Scale, with a cut-off point of 16.

Results. Compared to never smokers unexposed to passive smoking, never smokers reporting regular and occasional exposure to passive smoking at work had increased depressive symptoms. The adjusted odds ratios (aORs) were 1.92 (95% confidence interval (CI) 1.14, 3.23) for regular exposure and 1.63 (95% CI 1.08, 2.47) for occasional exposure. Current smokers had significantly increased depressive symptoms (aOR ranging from 2.25 to 2.38) but former smokers had only marginal increases of depressive symptoms (aOR ranging from 1.43 to 1.55). Gender did not modify the effects of active/passive smoking on depressive symptoms.

Conclusion. Passive smoking at work and current smoking appear associated with higher levels of depressive symptoms.
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Keywords: Passive smoking; Smoking; Depressive symptoms; Working population; Epidemiology; Occupational health

Introduction

A number of epidemiological studies have demonstrated a link between cigarette smoking and depressive symptoms/disorders (DSD) (Paperwalla et al., 2004). Although the nature of this relationship remains unclear, several hypotheses have been proposed to explain the association. DSD may cause smoking by increasing the likelihood that individuals will self-medicate negative emotions by smoking (Breslau et al., 1991; Escobedo et al., 1998; Fergusson et al., 2003; Patton et al., 1998). Smokers with a history of DSD are more likely to initiate smoking and progress to regular or heavy smoking (Anda et al., 1990; Escobedo et al., 1998; Patton et al., 1998), become

nicotine dependent (Breslau et al., 1998), are less successful with smoking cessation (Breslau et al., 1993; Covey et al., 1997; Glassman et al., 1990), and are at greater risk of developing a new episode of depression when abstaining from smoking (Glassman et al., 2001; Tsoh et al., 2000). An alternative hypothesis is that smoking itself leads to DSD (Brook et al., 2004; Brown et al., 1996; Choi et al., 1997; Goodman and Capitman, 2000; Johnson and Breslau, 2006; Kinnunen et al., 2006; Klungsoyr et al., 2006; Korhonen et al., 2007; Lam et al., 2005; Steuber and Danner, 2006; Wu and Anthony, 1999). This concept was supported by the fact that adolescents who smoke are at increased risk of subsequently developing DSD (Brook et al., 2004; Brown et al., 1996; Choi et al., 1997; Goodman and Capitman, 2000; Lam et al., 2005; Steuber and Danner, 2006; Wu and Anthony, 1999). Reciprocal mechanisms have also been proposed to explain the smoking–DSD association (Breslau et al., 1998; Fergusson et al., 2003; Windle and Windle, 2001). By contrast, there is the so-called ‘non-causal’ hypothesis, which asserts that shared genetic or environmental factors predispose

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* Corresponding author. Division of Applied Research and Technology, MS-C24, National Institute for Occupational Safety and Health, 4676 Columbia Parkway, Cincinnati, OH 45226, USA.

E-mail address: cji5@cdc.gov (A. Nakata).

Table 1
Characteristics of participants by smoking status in a suburb of Tokyo, Japan, 2002 (*n*=2770)^a

Characteristics	Smoking status			<i>p</i> value ^b
	Never	Former	Current	
Number of respondents (%) ^c	1168 (42.2)	315 (11.4)	1287 (46.5)	
Gender:				<.001
Men	529 (28.8)	238 (12.9)	1072 (58.3)	
Women	639 (68.6)	77 (8.3)	215 (23.1)	
Depressive symptoms:				<.001
Total respondents				
CES-D score ≥ 16	421 (39.1)	102 (9.5)	553 (51.4)	
CES-D score < 16	747 (44.1)	213 (12.6)	734 (43.3)	
Mean [SD]	14.8 [8.1]	14.0 [7.9]	15.9 [8.8]	<.001
Women				.033
CES-D score ≥ 16	222 (59.8)	30 (8.1)	119 (32.1)	
CES-D score < 16	417 (74.5)	47 (8.4)	96 (17.1)	
Mean [SD]	14.6 [8.2]	15.0 [9.2]	17.0 [9.6]	.003
Men				.012
CES-D score ≥ 16	199 (28.2)	72 (10.2)	434 (61.6)	
CES-D score < 16	330 (29.1)	166 (14.6)	638 (56.3)	
Mean [SD]	15.0 [7.9]	13.7 [7.4]	15.7 [8.6]	.001
Age groups:				.001
16 to 29	284 (49.9)	42 (7.4)	243 (42.7)	
30 to 39	282 (38.4)	79 (10.8)	373 (50.8)	
40 to 49	145 (34.0)	54 (12.7)	227 (53.3)	
50 to 59	306 (43.5)	81 (11.5)	316 (45.0)	
60 and over	151 (44.7)	59 (17.5)	128 (37.9)	
Mean [SD]	43.9 [14.4]	47.0 [13.8]	43.0 [12.8]	<.001
Marital status:				.001
Married	676 (39.1)	213 (12.3)	840 (48.6)	
Not married	492 (47.3)	102 (9.8)	447 (42.9)	
Highest education:				<.001
Junior high school	192 (40.2)	51 (10.7)	235 (49.2)	
High school	469 (40.7)	98 (8.5)	585 (50.8)	
Vocational/college/university	507 (44.5)	166 (14.6)	467 (41.0)	
Alcohol consumption (g ethanol/day):				<.001
Non-drinker (0.0)	469 (55.0)	95 (11.1)	289 (33.9)	
0.01 to 4.9	303 (58.3)	46 (8.8)	171 (32.9)	
5.0 to 14.9	212 (37.5)	66 (11.7)	288 (50.9)	
15.0 to 24.9	102 (24.3)	56 (13.4)	261 (62.3)	
25.0 and over	82 (19.9)	52 (12.6)	278 (67.5)	
Mean [SD]	6.2 [10.7]	11.9 [14.7]	13.6 [14.5]	<.001
Caffeine intake (cups of coffee or tea/day):				<.001
Almost none	138 (56.3)	35 (14.3)	72 (29.4)	
1 to 2	556 (43.6)	158 (12.4)	562 (44.0)	
3 or more	474 (38.0)	122 (9.8)	653 (52.3)	
Body Mass Index (kg/height (m) ²) (in quintiles):				.064
<20 th	234 (42.9)	53 (9.7)	259 (47.4)	
≥20 th –<40 th	235 (42.1)	57 (10.2)	266 (47.7)	
≥40 th –<60 th	235 (42.6)	51 (9.2)	266 (48.2)	
≥60 th –<80 th	228 (40.8)	75 (13.4)	256 (45.8)	
≥80 th	236 (42.5)	79 (14.2)	240 (43.2)	
Mean [SD]	22.6 [3.3]	23.0 [3.3]	22.6 [3.3]	.065
Number of chronic disease:				<.001
None	788 (41.0)	164 (8.5)	970 (50.5)	
1	347 (45.7)	129 (17.0)	284 (37.4)	
2 or more	33 (37.5)	22 (25.0)	33 (37.5)	
Job title:				<.001
Professional	140 (53.4)	48 (18.3)	74 (28.2)	
Managerial/clerical	378 (48.4)	94 (12.0)	309 (39.6)	
Sales/service	57 (27.8)	30 (14.6)	118 (57.6)	
Production/Technical	457 (39.5)	110 (9.5)	591 (51.0)	
Other	136 (37.4)	33 (9.1)	195 (53.6)	

Table 1 (continued)

Characteristics	Smoking status			<i>p</i> value ^b
	Never	Former	Current	
Industry sector:				<.001
Manufacturing	939 (41.6)	232 (10.3)	1,085 (48.1)	
Service	173 (50.3)	71 (20.6)	100 (29.1)	
Transportation	44 (32.1)	7 (5.1)	86 (62.8)	
Construction	12 (36.4)	5 (15.2)	16 (48.5)	

Note. SD, standard deviation; CES-D, Center for Epidemiologic Studies Depression Scale.

^a Data may not total 100% due to rounding.

^b *p* value derived from χ^2 test for categorical variables and analysis of variance for continuous variables (comparison within smoking status).

^c Percent within smoking status.

individuals to both increased risk of smoking and DSD (Breslau et al., 1991; Dierker et al., 2002; Duncan and Rees, 2005; Fergusson et al., 2003; Kendler et al., 1993; Son et al., 1997), although the evidence has been equivocal (Breslau et al., 1991, 1993; Fergusson et al., 2003; Martini et al., 2002). Such contradictions may partly be explained by the evidence that severe forms of depression are primarily affected by genetic factors while mild to moderate forms of depression are primarily affected by environmental factors (Lyons et al., 1998; McCaffery et al., 2003).

Despite a significant body of literature associating cigarette smoking with DSD, a critical issue remains unresolved, that is the effect of passive smoking on DSD. It is conceivable that if smoking induces DSD, non-smokers who are exposed to a high level of passive smoking may also experience DSD as a result. If this hypothesis is true, previous findings associating smoking and DSD could be underestimated because the referent (control) group in most studies of cigarette smoking included non/never smokers who are potentially exposed to passive smoking.

In Japan, a nationwide survey conducted in 2002 reported that 33.1% of non-smokers were exposed to passive smoking at work almost every day, and 39.7% were exposed occasionally (Statistical Database, Ministry of Health, Labor, and Welfare, Japan, 2002). This estimate indicates that three-quarters of Japanese non-smoking workers are exposed to passive smoking on a daily basis.

The purpose of this report is to examine the association of exposure of never smokers to different levels of passive smoking with depressive symptoms as well as the association of current and former smoking with such symptoms among the full-time working population.

Methods

Study sample and procedures

This cross-sectional study was conducted in a suburb of Tokyo, Japan, from June to December 2002. The study design has been described elsewhere (Nakata et al., 2006). Briefly, we randomly selected 391 enterprises (4351 full-time workers). Representation of the types of small and medium-scale enterprises (SMEs) was weighted according to the number of each type of SME in the area. Members of the study staff contacted each enterprise by telephone to request participation of their workers in completing a self-administered questionnaire

survey. A total of 3262 workers (2141 men, 1121 women) from 285 enterprises responded by providing informed consent and completing the questionnaire (response rate 75.0%). We excluded 492 respondents because of missing responses with regards to demographics, lifestyle factors (including smoking status), and depressive symptoms. Consequently, the questionnaire of 1839 men and 931 women were retained for the final analyses. The questionnaire elicited information on demographics, lifestyle, height and body weight, number of chronic diseases, job title, and industry sector (Table 1). We also obtained information on number of total workers in each enterprise.

The Medical Ethical Committee of the University of Tokyo and the National Institute of Occupational Safety and Health, Japan approved the study. Written informed consent was obtained from all participants.

Smoking status

Smoking status was assessed by the following 3 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 3) how many years have you been smoking? Exposure to passive smoking in never smokers was assessed by the following question: Are you currently exposed to cigarette smoke from other people? Exposure at work and home were asked separately, and responses were classified into 3 levels for each location: no exposure, occasional exposure, and regular exposure (Kawachi et al., 1997).

We validated smoking status questionnaire using a dataset from our previous study (not published). We measured urinary cotinine concentrations to determine smoking status among 340 white-collar workers. Gas chromatography/mass spectrometry method was used to measure urinary cotinine concentrations. We used a creatinine-adjusted cotinine (cotinine–creatinine ratios, CCR) concentration cut-off of 100 µg/g creatinine to distinguish current smokers from non-smokers as suggested in a previous study (Haufoed and Lison, 1998). We found that sensitivity and specificity were 96.8% and 94.7%, respectively (kappa coefficient=.886). Spearman's rank correlation coefficients were calculated to examine the relationships between CCR and daily cigarette consumption in current smokers as well as exposure levels to passive smoking at work, at home, and either at work or at home in never smokers. The results suggested that CCR correlated highly with daily cigarette consumption ($r_s=.500$, $p<.001$) and correlated modestly with passive smoking exposure at work ($r_s=.260$, $p=.019$), at home ($r_s=.230$, $p=.057$), and either at work or at home ($r_s=.253$, $p=.010$), similar to what was reported in previous studies (Kawachi and Colditz, 1996).

Depressive symptoms

Depressive symptoms were measured by a Japanese version of the Center for Epidemiologic Studies Depression (CES-D) scale (Shima et al., 1985). The 20-item depressive symptom scale measures the level of depressive symptoms experienced in the past week. The CES-D scale cut-off score is 16, which differentiates between those exhibiting high levels of depressive symptoms (score ≥ 16) from those with lower levels of such symptoms (score < 16) (Radloff, 1977). The internal consistency of the CES-D scale for the study sample was 0.84.

Covariates

Lifestyle factors and physical condition were assessed as follows. Alcohol consumption was assessed by the number of alcoholic drinks consumed per day, with one drink estimated as about 9 g of pure ethanol. Caffeine intake was estimated as the number of cups of tea/coffee per day. Body mass index (BMI) was calculated as weight in kilograms divided by the square of height in meters. For chronic disease, participants were asked if they were currently under treatment for any of the following diseases: hypertension, hyperlipidemia, diabetes mellitus, major depression, menopausal disorder, or other diseases. If the participants reported 'other diseases', they were asked to specify the condition. Participants reported various diseases including cardiovascular disease, cerebrovascular disease, cancer, liver disease, renal disease, peptic ulcer, gastrointestinal diseases, neurological diseases, hyperthyroidism, prostaticomegaly, rheumatism, musculoskeletal disorders, panic disorder, and allergy.

Statistical analyses

The risks of high levels of depressive symptoms associated with active and passive smoking were estimated using univariate and multivariate logistic regression with ORs and 95% CIs as measures of association. The model was adjusted for all potential confounders (as listed on Table 1) in addition to organizational factors stratified by workplace (size of enterprise by number of total workers, participation rate to survey, male rate per enterprise, and smoking rate per enterprise). Since gender did not significantly modify the effect of active/passive smoking on depressive symptoms, analyses were computed for all participants together. Tests for linear trends were performed by treating the ordinal exposure variable as continuous in the linear regression model. 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

Descriptive statistics

Descriptive statistics of participants stratified by smoking status are shown in Table 1. Roughly 58% of men were current, 13% were former, and 29% were never smokers while 23% of women were current, 8% were former, and 69% were never smokers. Women (39.8%) had a higher level of depressive symptoms (CES-D ≥ 16) than men (38.3%), but the difference was not statistically significant ($\chi^2=.267$, $df=1$, $p=.605$). Average scores for CES-D were 15.8 (SD=8.7) in women and 15.3 (SD=8.3) in men ($p>.05$). Current smoking women and men had higher percentages of depressive symptoms as well as higher average CES-D scores than never and former smokers among both women and men.

Relationship between smoking status and depressive symptoms

The multivariate logistic regression analysis found that compared to never smokers (referent group), increased odds of high levels of depressive symptoms were found in current smokers but not in former smokers (Table 2).

Table 2

Odds ratios for depressive symptoms in former and current smokers compared to never smokers in a suburb of Tokyo, Japan, 2002

Depressive symptoms:	Smoking status		<i>p</i>	Current OR (95% CI)	<i>p</i>	<i>P</i> _{trend}
	Never	Former OR (95% CI)				
Women and men combined ^a	<i>n</i> =1168	<i>n</i> =315		<i>n</i> =1287		
Unadjusted odds ratio	1.00	0.85 (0.65, 1.11)	.226	1.34 (1.14, 1.57)	.001	<.001
Adjusted odds ratio ^b	1.00	1.10 (0.79, 1.53)	.571	1.65 (1.30, 2.08)	<.001	.003

Note. OR, odds ratio; CI, confidence interval.

^a Gender did not significantly modify the relationship between smoking and depressive symptoms.

^b Adjusted for sex, age in 10-year increments, marital status, educational level, alcohol consumption, caffeine intake, BMI, chronic diseases, job title, industry sector, enterprise size, participation rate, male rate per enterprise, and smoking rate per enterprise.

Table 3
Odds ratios for depressive symptoms according to smoking status and smoke exposure by differential exposure setting stratified by workplace, in a suburb of Tokyo, Japan, 2002

Depressive symptoms:	Smoking status										
	Never					Former		Current			
	Exposure to passive smoking										
	No exposure ^a	Occasional OR (95% CI)	<i>p</i>	Regular OR (95% CI)	<i>p</i>	<i>P</i> _{trend}	OR (95% CI)	<i>p</i>	OR (95% CI)	<i>p</i>	<i>P</i> _{trend}
Exposure at work											
Women and men combined ^b	<i>n</i> =428	<i>n</i> =339		<i>n</i> =134			<i>n</i> =315		<i>n</i> =1287		
Unadjusted odds ratio	1.00	1.56 (1.13, 2.14)	.006	1.88 (1.26, 2.80)	.002	.007	1.13 (0.82, 1.57)	.460	1.67 (1.29, 2.15)	<.001	<.001
Adjusted odds ratio ^c	1.00	1.63 (1.08, 2.47)	.021	1.92 (1.14, 3.23)	.015	.012	1.43 (0.93, 2.18)	.101	2.25 (1.58, 3.21)	<.001	.001
Exposure at home											
Women and men combined ^b	<i>n</i> =428	<i>n</i> =138		<i>n</i> =67			<i>n</i> =315		<i>n</i> =1287		
Unadjusted odds ratio	1.00	1.45 (0.95, 2.20)	.084	1.03 (0.59, 1.78)	.931	.533	1.13 (0.82, 1.57)	.460	1.67 (1.29, 2.15)	<.001	<.001
Adjusted odds ratio ^c	1.00	1.43 (0.82, 2.47)	.206	1.78 (0.91, 3.49)	.092	.197	1.55 (1.00, 2.38)	.048	2.53 (1.75, 3.65)	<.001	.001
Exposure at work or at home											
Women and men combined ^b	<i>n</i> =428	<i>n</i> =428		<i>n</i> =163			<i>n</i> =315		<i>n</i> =1287		
Unadjusted odds ratio	1.00	1.51 (1.12, 2.05)	.008	1.74 (1.19, 2.52)	.004	.023	1.13 (0.82, 1.57)	.460	1.67 (1.29, 2.15)	<.001	<.001
Adjusted odds ratio ^c	1.00	1.62 (1.10, 2.40)	.015	2.09 (1.29, 3.38)	.003	.007	1.44 (0.95, 2.20)	.088	2.27 (1.59, 3.22)	<.001	.002

Note. OR, odds ratio; CI, confidence interval.

^a Neither exposed at work nor at home.

^b Gender did not significantly modify the relationship between active/passive smoking and depressive symptoms.

^c Adjusted for sex, age in 10-year increments, marital status, educational level, alcohol consumption, caffeine intake, BMI, number of chronic diseases, job title, industry sector, enterprise size, survey participation rate, male rate per enterprise, and smoking rate per enterprise.

Prevalence of passive smoking exposure among never smokers

Among the 1,106 never smoking participants, the proportions of no, occasional, and regular passive smokers at work were 38.7%, 30.7%, and 12.1%, respectively, while corresponding figures for exposure at home were 38.7%, 12.5%, and 6.1%, respectively. Never smokers were more often exposed at work rather than at home ($\chi^2=1086.0$, $df=4$, $p<.001$). The proportions of no, occasional, and regular passive smokers either at work or at home were 38.7%, 38.7%, and 14.7%, respectively, and corresponding figures for exposure both at work and home were 38.7%, 4.4%, and 3.4%, respectively.

Relationship between never smokers exposed to passive smoking, former or current smoking and depressive symptoms

Never smokers who were regularly and occasionally exposed to passive smoking at work (and either at work or home) had significantly higher levels of depressive symptoms compared to never smokers who were unexposed to passive smoking (referent group) (Table 3). In contrast, no significant associations were found in never smokers exposed to passive smoking at home. A dose-response gradient was apparent between frequency of passive smoking at work and depressive symptoms among never smokers. Compared to the referent group, current smokers had significantly higher odds of depressive symptoms, while former smokers only had a weak association.

Discussion

This study had 3 major findings. First, 61.4% of never smokers reported exposure to passive smoking, but they were

more often exposed at work (42.8%) rather than at home (18.6%). Second, never smokers ‘regularly’ and ‘occasionally’ exposed to passive smoking at work, but not at home, were more likely to experience higher levels of depressive symptoms (as measured by a CES-D score of 16 or higher) when compared to never smokers who were not exposed to passive smoking. This suggests that exposure at work was a major source of increased depressive symptoms. Third, current smokers had higher levels of depressive symptoms than never smokers (OR = 1.65), but this association was stronger when the analyses considered the effects of passive smoking among never smokers (ORs ranging from 2.25 to 2.53). Although the results of this study should be interpreted cautiously in light of the self-reporting and cross-sectional design, these data suggest that never smokers exposed to passive smoking at work are at greater risk of high depressive symptoms.

We observed that exposure to passive smoking at work was associated with a higher prevalence of depressive symptoms. This may provide indirect evidence of a causal link from cigarette smoking to depressive symptoms. Since passive smoking is not an active behavior, it is less likely that an effect in the opposite direction, i.e., depressive symptoms leading to passive smoking, could logically take place. At present, to our knowledge there is no direct evidence for the relationship of passive smoking exposure to depressive symptoms in adults. However, a series of prospective studies of adolescents indicated that active smoking induces high depressive symptoms (Brook et al., 2004; Choi et al., 1997; Goodman and Capitman, 2000; Lam et al., 2005; Steuber and Danner, 2006; Wu and Anthony, 1999). For example, Wu et al. investigated that cigarette smoking predicted a high level of depressed mood in a 4-year follow-up study among 8- to 14-year-old youths (Wu and

Anthony, 1999). Goodman and Capitman (2000) reported that current smoking at baseline was the strongest predictor of developing a high level of depressive symptoms (OR, 3.9) in a 1-year follow-up analysis of adolescents who were not depressed at the beginning of the study. These reports clearly indicated that active smoking predicts high depressive symptoms, which is supportive of our hypothesis.

This study demonstrated that the association of current smoking and depressive symptoms was intensified when the reference group excluded exposure to passive smoking. The OR for high levels of depressive symptoms among current smokers increased from 1.65 to at least 2.25 when the reference group consisted of never smokers unexposed to passive smoking (Tables 2 and 3). This suggests that if the past studies had considered the effect of passive smoking, the relationship between smoking and depressive symptoms could have been stronger. However, more evidence is needed to confirm this finding.

In the current study, the prevalence of high depressive symptoms did not differ between men (38.3%) and women (39.8%) ($\chi^2 = .596$, $df = 1$, $p = .440$). Studies have indicated that depression is about 2 times more prevalent in women than in men (Kessler et al., 1993; Nolen-Hoeksema, 1987; Piccinelli and Wilkinson, 2000), although recent reports suggested that the patterns for Asian countries are different than those for western countries (Inaba et al., 2005; Kawakami et al., 2004; Kim et al., 2007). For instance, Inaba et al. (2005) reported that the average scores of CES-D were 15.8 for men and 16.6 for women in the Japanese National Family Research survey. In an urban community in Japan, the prevalence of lifetime major depression was 3.1% for men and 2.8% for women with no significant difference between both genders (Kawakami et al., 2004). These studies support our finding that the prevalence of depressive symptoms is nearly equally distributed across gender.

We found that being married, non-drinking, and drinking fewer caffeinated beverages were moderately associated with depressive symptoms (data not shown). We did not detect any occupational/organizational factors that relate to depressive symptoms in this sample. As a result, active/passive smoking was the strongest factor associated with depressive symptoms in this study. Although we adjusted for a variety of confounders, we could not exclude the possibility that unadjusted/unknown confounders, i.e., diet, physical exercise, psychosocial work environment, and social factors such as socioeconomic status, social network, living arrangement, and health care availability may explain the present finding.

Strengths and limitations of the study

A key strength of our study is that to our knowledge this may be the first study that has considered the effect of passive smoking on depressive symptoms. We differentiated exposure to passive smoking at work and at home to make sources of exposure more clear. Limitations of our study are as follows: First, both smoking status and depressive symptoms were self-reported, which may introduce recall/reporting bias. Second, the data in this study came from a single survey of workers

from a limited geographic area, which was not representative of the entire Japanese workforce, making generalizability less definitive. Third, response bias may have occurred if non-respondents differed from respondents with respect to smoking status and depressive symptoms. Fourth, the inherent nature of the cross-sectional design does not permit the conclusion that passive or active smoking causes depressive symptoms. Finally, a relatively small sample size among passive smokers may have limited our power of detecting differences between groups.

Conclusion

Notwithstanding these limitations, our investigation suggests that never smokers exposed to passive smoking at work and current smokers are more likely to have associated with a higher degree of depressive symptoms than those without such exposure. Further research is needed to confirm that an elimination of exposure to passive smoking at work will lead to a reduction in depressive symptoms among never smokers.

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