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Original Article

Fine Particulate Matter and Diabetes Prevalence in Okayama, Japan

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Many studies have shown an association between long-term exposure to particulate matter having an aerodynamic diameter of 2.5 μm or less ($PM_{2.5}$) and diabetes mellitus (DM), but few studies have focused on Asian subjects. We thus examined the association between long-term exposure to $PM_{2.5}$ and DM prevalence in Okayama City, Japan. We included 76,591 participants who had received basic health checkups in 2006 and 2007. We assigned the census-level modeled $PM_{2.5}$ data from 2006 and 2007 to each participant and defined DM using treatment status and the blood testing. $PM_{2.5}$ was associated with DM prevalence, and the prevalence ratio (95% confidence interval) was 1.10 (1.00-1.20) following each interquartile range increase (2.1 μg/m^3) in $PM_{2.5}$. This finding is consistent with previous results and suggests that long-term exposure to $PM_{2.5}$ is associated with an increased prevalence of DM in Okayama City, Japan, where the $PM_{2.5}$ level is lower than in other cities in Asian countries.

Key words: air pollution, diabetes mellitus, epidemiology, glycosylated hemoglobin, particulate matter

Many studies have shown associations between long-term exposure to air pollution, especially particulate matter with an aerodynamic diameter of 2.5 μm or less ($PM_{2.5}$), and various adverse health outcomes [1,2]. Recently, some studies reported possible adverse effects of $PM_{2.5}$ on diabetes mellitus (DM) [3-10], but most of those studies were conducted in Europe and North America. Because insulin sensitivity [11] and visceral adipose distribution [12,13] are different between Caucasian and Asian people [14], air pollution may have different influences on DM in Asians.

In Asian countries, several cross-sectional and cohort studies demonstrated adverse associations between $PM_{2.5}$ and DM. However, they were conducted mostly in areas with relatively high exposure to $PM_{2.5}$. For example, mean or median $PM_{2.5}$ concentrations were 72.6 μg/m^3 and 82.0 μg/m^3 in cross-sectional studies in China [15,16] and 35.8 μg/m^3 in a cohort study in Hong Kong [17]. Recently, one cohort study based on in Tokyo (18.5 μg/m^3) examined the association between $PM_{2.5}$ and DM prevalence [18], but more evidence is needed from Asian countries, especially in areas with low levels of $PM_{2.5}$.

In the present study, we aimed to examine the association between long-term low-level exposure to $PM_{2.5}$ and DM prevalence in Okayama City, Japan, where the $PM_{2.5}$ level is lower than in other cities in Asian countries.

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Subjects and Methods

Study design and participants. We employed a cross-sectional study design. We included 76,591 participants who had received basic health checkups in Okayama City from April 2006 through March 2008 (corresponding to fiscal years 2006-2007). Okayama City, in the western part of Japan, has a population exceeding 0.7 million. The basic health checkups were conducted under the Health and Medical Service Act for the Aged and were intended to prevent so-called life-style-related diseases, such as cardiovascular disease, among people aged 40 years and older. The details of the study setting are described elsewhere [19]. In the checkups, self-reported questionnaires were distributed to the participants (*e.g.*, current medical status and history, as well as alcohol/smoking and dietary habits), who underwent a physical examination (*e.g.*, height and weight) and blood testing (including fasting plasma glucose and/or hemoglobin A1c (HbA1c) JDS).

Air pollution exposure. We defined air pollution exposure as exposure to particulate matter having an aerodynamic diameter of 2.5 μm or less ($PM_{2.5}$). We utilized modeled annual $PM_{2.5}$ data from the Atmospheric Composition Analysis Group [20], which estimated ground-level $PM_{2.5}$ concentrations by combining aerosol optical depth with the GEOS-Chem chemical transport model; we then used geographically weighted regression to adjust the estimates to global ground-based observation of $PM_{2.5}$. These models have been well validated [4, 7-9]. We obtained global estimates of $PM_{2.5}$ after removing dust and sea salt components, with a spatial resolution of approximately 1×1 km. Using these estimates, we calculated the annual average $PM_{2.5}$ concentrations for each census area in Okayama City during the study period. Although we did not have the participants' current addresses, we obtained their census area information. The census area is the smallest area unit used for the Japanese National Census. The average size of the 773 census areas included in the present study was 0.19 km^2. We assigned the census-level modeled $PM_{2.5}$ data in 2006 and 2007 to each participant and used the mean of 2-year annual concentrations (2006 and 2007) as the main exposure indicator. After excluding a total of 1,031 participants who were assigned negative values, we analyzed 75,553 participants.

Diabetes mellitus. We used three different definitions of DM status. First, we defined DM by the current treatment status obtained from the self-reported questionnaire. Second, based on the blood test results, fasting plasma glucose > 126 mg/dl or casual > 200 mg/dl and HbA1c > 6.5% were considered to indicate DM following the screening criteria for type 2 DM [21]. Third, we combined these two definitions because some participants undergoing DM treatment had normal blood test findings, *i.e.*, we considered a participant to have DM if he or she met either the first or second definition.

Data analysis. After conducting a descriptive analysis, we estimated the adjusted prevalence ratios (PRs) for DM per one IQR (interquartile range) increase in $PM_{2.5}$ using Poisson regression with robust error variance.

We first adjusted for age (years) and sex, then adjusted for other individual factors, and finally adjusted for area-level socioeconomic status. We chose these covariates as potential confounders following the previous studies [3-9]. Individual factors were drinking (regular alcohol drinker or not), smoking (current smoker or not), regular exercise (regular exercise of more than 30 min per day for more than one year or not), consumption of vegetables (do some efforts or not), reduction of calorie intake (do some efforts or not), and body mass index (kg/m^2). All of these individual factors except body mass index were assessed in the self-reported questionnaire at the basic health checkups [22]. Body mass index was calculated by height and weight in the physical examination. As an indicator of area-level socioeconomic status, we enrolled a proportion of white-collar workers (*i.e.*, managerial, professional, or technical, and clerical workers) among residents aged 15 years or older in each census area obtained from the National Census in 2015.

To validate the linearity assumption between $PM_{2.5}$ and DM prevalence, we replaced the continuous $PM_{2.5}$ variable in the model with quartile $PM_{2.5}$. We estimated prevalence ratio for each quartile category, using the lowest quartile as the reference. In further analyses, we stratified by sex to examine the possible effect modification [4].

We used STATA/IC version 15 (Stata, College Station, TX, USA) for all statistical analyses. All confidence intervals (CIs) were calculated at the 95% level. This study was approved by the Okayama University Graduate School of Medicine, Dentistry and Pharma-

ceutical Sciences, and by the Okayama University Hospital Ethics Committee (No. K1801-034).

Results

Table 1 shows the demographic characteristics of the study participants in total and by the quartile $PM_{2.5}$ categories. The 2-year mean of the annual $PM_{2.5}$ level in 2006 and 2007 was 14.1 μg/m^3 and ranged from 12.1 to 15.3 μg/m^3. The participants who lived in the area with the lowest $PM_{2.5}$ level tended to be older, to be non-smokers, to get less regular exercise, and to consume more calories. In the lowest $PM_{2.5}$ level area, the proportion of white-collar workers was also lower.

In Table 2, we show the estimated PRs per IQR increase (*i.e.*, 2.1 μg/m^3) for DM according to the three definitions. In the fully adjusted model, the prevalence of DM was positively associated with $PM_{2.5}$ in the three definitions, albeit statistically significant result was observed only when DM was defined based on either treatment status or blood testing (PR: 1.10, 95% CI: 1.00-1.20).

When we entered the quartile $PM_{2.5}$ into the model, the associations between the $PM_{2.5}$ category and the DM prevalence ratio did not substantially depart from linearity (Fig. 1). Table 3 shows the PRs stratified by sex; the male participants had higher effect estimates than the female participants.

Discussion

In the present study, we examined the association of long-term low-level exposure to $PM_{2.5}$ with DM prevalence in Okayama, Japan. We found that the 2-year average $PM_{2.5}$ level was associated with the prevalence of DM.

The observed association between $PM_{2.5}$ and DM is consistent with the previous studies in Europe, North America, and Tokyo, where concentrations of $PM_{2.5}$ are relatively low [3,4,7,8,10,18]. The point estimates from our study were almost the same as those in Europe, North America, and Tokyo [10]. On the other hand, they were higher compared with those in high-exposure Asian countries. For each 1 μg/m^3 increase in $PM_{2.5}$, our estimated PR (95% CI) for DM defined by treatment status or blood testing (1.10; 1.00-1.20, in Table 2) was equivalent to 1.05 (1.00-1.09). This estimate is similar to that from the previous study in Tokyo

Table 1 Demographic characteristics of the study participants

	Total (n = 75,553)	Quartile $PM_{2.5}$ category[a]				*p*-value*
		Quartile 1 (n = 18,933)	Quartile 2 (n = 18,945)	Quartile 3 (n = 18,925)	Quartile 4 (n = 18,750)	
Individual characteristics						
Mean age ± SD	69.9 ± 11.2	71.4 ± 10.6	69.5 ± 11.0	69.1 ± 11.4	69.7 ± 11.6	<0.001
Female sex, n (%)	51,322 (67.9)	12,538 (66.2)	12,703 (67.05)	13,098 (69.2)	12,983 (69.2)	<0.001
Examination fiscal year, 2006 (%)	58,792 (77.8)	13,842 (73.1)	15,281 (80.7)	14,904 (78.8)	14,765 (78.8)	<0.001
Mean BMI ± SD	22.8 ± 3.41	22.9 ± 3.35	22.9 ± 3.41	22.8 ± 3.44	22.8 ± 3.44	<0.001
Alcohol drinking, n (%)	24,607 (32.6)	5,750 (30.4)	6,151 (32.5)	6,470 (34.2)	6,236 (33.3)	<0.001
Smoker, n (%)	7,998 (10.7)	1,809 (9.68)	1,959 (10.5)	2,032 (10.9)	2,198 (11.9)	<0.001
Regular exercise, n (%)	31,072 (42.4)	7,307 (39.9)	7,595 (41.3)	8,128 (44.2)	8,042 (44.2)	<0.001
Effort to consume more vegetables, n (%)	41,333 (54.7)	10,194 (53.8)	10,317 (54.5)	10,642 (56.2)	10,180 (54.3)	<0.001
Effort to reduce intake of callories, n (%)	19,770 (26.2)	4,391 (23.2)	4,841 (26.0)	5,301 (28.0)	5,237 (27.9)	<0.001
Area level characteristics						
Mean white collar proportion ± SD	0.38 ± 0.081	0.32 ± 0.067	0.37 ± 0.051	0.42 ± 0.092	0.39 ± 0.076	<0.001

BMI, body mass index; $PM_{2.5}$, particulate matter less than 2.5 μm; SD, standard deviation.
[a]PM2.5 exposure ranges from 12.1 to 15.3 μg/m^3 and quartiled PM2.5 categorization is as follows: quartile 1 (12.1–13.0 μg/m^3), quartile 2 (13.0–14.1 μg/m^3), quartile 3 (14.1–15.1 μg/m^3), and quartile 4 (15.1–15.3 μg/m^3). The interquartile range is 2.1 μg/m^3.
*Kruskal-Wallis rank sum test for continuous variables, Pearson χ^2 test for categorical variables to test $PM_{2.5}$ quartile groups.

Table 2 Prevalence and prevalence ratios of DM following IQR increase in $PM_{2.5}$

	Case, n (%)	Prevalence ratios (95% confidence intervals)		
		Age and sex adjusted model	Individual factors adjusted model[a]	Fully-adjusted model[b]
DM defined by treatment status	5,984 (7.9)	1.14 (1.02–1.27)	1.01 (0.90–1.13)	1.09 (0.96–1.23)
DM defined by blood screening[c]	8,156 (10.8)	1.09 (0.99–1.19)	1.03 (0.94–1.14)	1.09 (0.98–1.22)
DM defined by both treatment status or blood screening	10,197 (13.5)	1.10 (1.02–1.20)	1.03 (0.95–1.13)	1.10 (1.00–1.20)

DM, diabetes mellitus; IQR, interquartile range; $PM_{2.5}$, particulate matter less than 2.5 μm.
[a]Adjusted for age, sex, drinking, smoking, exercise, effort to consume more vegetables, effort to reduce intake of callories, and body mass index.
[b]Further adjusted for proportion of white collar worker at each census area.
[c]DM blood screening was considered positive if the participants had measured hemoglobin A1c >6.5, fasting plasma glucose >126 mg/dl, or two-hour glucose >200 mg/dl.

Table 3 Relative prevalence ratio of DM following IQR increase in $PM_{2.5}$ stratified by sex

	Prevalence ratio (95% confidence interval)[a]	
	Male	Female
DM treatment[b]	1.18 (0.99–1.41)	1.00 (0.84–1.19)
DM blood screening positive[c]	1.19 (1.02–1.39)	1.02 (0.88–1.18)
DM treatment or DM blood screening positive	1.16 (1.02–1.33)	1.04 (0.91–1.18)

DM, diabetes mellitus; IQR, intraquartile range; $PM_{2.5}$, particulate matter less than 2.5 μm.
[a]Adjusted for age, drinking, smoking, exercise, effort to consume more vegetables, effort to reduce intake of callories, body mass index, and proportion of white collar worker at each census area.
[b]DM treatment was obtained from the questionnaires.
[c]We defined a DM blood screening positive group measured HbA1c >6.5, fasting plasma glucose >126 mg/dl, or two-hour glucose >200 mg/dl.

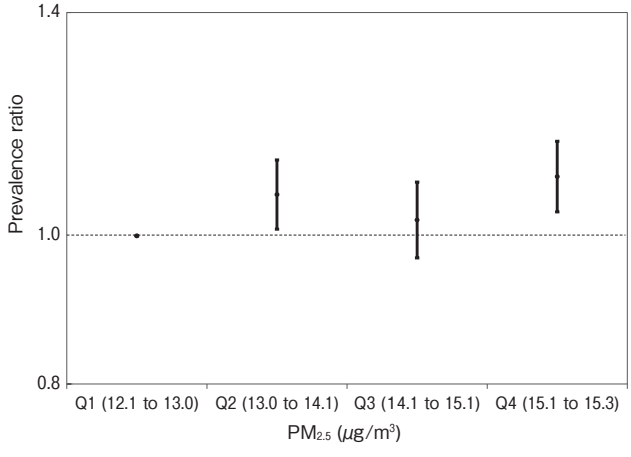

Fig. 1 Prevalence ratios for the association of quartile particulate matter less than 2.5 micrometer ($PM_{2.5}$) and diabetes mellitus. Error bars denote 95% confidence intervals.

[1.029 (1.003-1.055)], whereas the corresponding figures from the previous studies were 1.003 (1.001-1.004) [15], 1.005 (1.001-1.009) [16], and 1.018 (1.003-1.033) [17]. These differences may be explained in part by the different exposure levels from study area to study area. This finding aligns with previous mortality studies, which indicated that the point estimates for mortality due to long-term exposure to $PM_{2.5}$ were higher in countries with low-level exposure compared to those with high-level exposure [23, 24].

The linear association between the quartile $PM_{2.5}$ and DM prevalence, which is also consistent with previous studies [23], might partly support our results for continuous $PM_{2.5}$ exposure. This would indicate that there is no detectable threshold above which $PM_{2.5}$ affects DM, since we found significant adverse effects even in the second quartile levels (*i.e.*, 13.0-14.1 μg/m^3).

The possible mechanism by which $PM_{2.5}$ affects DM

prevalence was examined by experimental and clinical studies [25]. Because air pollution exposure can alter inflammatory [26] and endothelial [27] functions, such impairment can reduce peripheral glucose uptake [28], affect insulin signaling in liver by endoplasmic reticulum stress [29], and alter mitochondrial function [30]. These mechanisms may explain the possible impact of air pollution on DM.

In the stratified analysis by sex, men had stronger effect estimates than women, which is discrepant with previous studies that reported women tend to suffer a greater severity of adverse effects due to air pollution for health outcomes in general and also in DM [10, 17, 31]. Although the reason for this discrepancy between the present and previous results is not clear, it may be explained by the fact that Japanese females, especially among older ages, were still more likely to be home managers (*i.e.*, they could be less exposed to the outside environment) compared to males. Otherwise, inadequate adjustment of socioeconomic status or other potential confounders might explain this discrepancy. Indeed, we only adjusted for the proportion of white-collar workers in each census area in the model, and this may not reflect the socioeconomic status of the female participants.

The present study has some strengths. First, we used three different types of definitions of DM status based on treatment status and blood testing, which enabled us to evaluate outcome misclassification. Second, we could adjust for potential confounders by using individual information obtained from the baseline questionnaire.

In contrast, several limitations should be noted. First, we used a cross-sectional study design, which may have limitations in capturing the insidious onset of DM since disease onset is difficult to identify. Second, we used a 2-year average (*i.e.*, 2006 and 2007) $PM_{2.5}$ exposure as a main exposure indicator and did not account for yearly changes in $PM_{2.5}$. However, Okayama City is well-developed and did not experience large demographic shifts during the study period, so our assumption would be reasonable. Third, because we could only assign $PM_{2.5}$ exposure to the census level, there is a possibility of exposure misclassification. Any such misclassification, however, would be nondifferential. Then estimated PRs would be changed toward the null, which may limit our ability to explain the observed results. Finally, residual confounding is still possible because we only adjusted for the proportion of white-collar workers as an area-level indicator of socioeconomic status (*i.e.*, we could not adjust for individual socioeconomic status). Moreover, our adjustment of the variables using categorical variables may not suffice to adjust for the confounding.

In conclusion. the present study indicates that long-term low-level exposure to $PM_{2.5}$ is associated with an increased prevalence of DM. This finding is consistent with previous studies and suggests that long-term exposure to $PM_{2.5}$ is associated with an increased prevalence of DM in Okayama City, Japan, where the $PM_{2.5}$ level is lower than in other cities in Asian countries.

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