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ORIGINAL RESEARCH

Long-Term Particulate Matter Exposure and Incidence of Arrhythmias: A Cohort Study

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BACKGROUND: Studies have shown that short-term exposure to air pollution is associated with cardiac arrhythmia hospitalization and mortality. However, the relationship between long-term particulate matter air pollution and arrhythmias is still unclear. We evaluate the prospective association between particulate matter (PM) air pollution and the risk of incident arrhythmia and its subtypes.

METHODS AND RESULTS: Participants were drawn from a prospective cohort study of 178 780 men and women who attended regular health screening exams in Seoul and Suwon, South Korea, from 2002 to 2016. Exposure to PM with an aerodynamic diameter of ≤10 and ≤2.5 μm (PM_{10} and $PM_{2.5}$, respectively) was estimated using a land-use regression model. The associations between long-term PM air pollution and arrhythmia were examined using pooled logistic regression models with time-varying exposure and covariables. In the fully adjusted model, the odds ratios (ORs) for any arrhythmia associated with a 10 μg/m^3 increase in 12-, 36-, and 60-month PM_{10} exposure were 1.15 (1.09, 1.21), 1.12 (1.06, 1.18), and 1.14 (1.08, 1.20), respectively. The ORs with a 10 μg/m^3 increase in 12- and 36-month $PM_{2.5}$ exposure were 1.27 (1.15, 1.40) and 1.10 (0.99, 1.23). PM_{10} was associated with increased risk of incident bradycardia and premature atrial contraction. $PM_{2.5}$ was associated with increased risk of incident bradycardia and right bundle-branch block.

CONCLUSIONS: In this large cohort study, long-term exposure to outdoor PM air pollution was associated with increased risk of arrhythmia. Our findings indicate that PM air pollution may be a contributor to cardiac arrhythmia in the general population.

Key Words: air pollution ■ arrhythmias ■ particulate matter

Cardiac arrhythmias, a group of conditions defined by an abnormal electrical activity of the heart, are associated with increased risk of cardiovascular disease (CVD) and mortality.[1,2] The prevalence of certain types of arrhythmia, such as atrial fibrillation (AF), is projected to increase exponentially in the next decade,[3] partly as a reflection of the rising prevalence of increased CVD risk factors, such as hypertension, diabetes mellitus, and obesity.[4] Nevertheless, environmental biomarkers (such as air pollution, noise, and natural environment) other than traditional CVD risk factors are implicated in the development of arrhythmia and could be potential targets for its prevention and management.[5]

While a meta-analysis showed that short-term exposure to air pollution was associated with cardiac arrhythmia hospitalization and mortality,[6] the relationship between long-term particulate matter (PM) air pollution and arrhythmia is still unclear. Some studies have found a positive association between long-term PM air pollution exposure and ventricular arrhythmia in patients in high risk.[7,8] More recently, 2 general population-based cohort studies from Canada and South Korea found that air pollution was associated with increased onset

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CLINICAL PERSPECTIVE

What Is New?

- Long-term particulate matter air pollution was associated with increased incidence of bradycardia, right bundle-branch block, and premature atrial contraction arrythmias in the general population.

What Are the Clinical Implications?

- Measures to control the level of particulate matterair pollution are required and may reduce the burden of arrythmias, as exposure to particulate matter air pollution is chronic.
- Prevention and treatment of cardiac arrythmias might benefit from identification of relevant pathophysiological pathways associated with particulate matter air pollutions.

Nonstandard Abbreviations and Acronyms

CVD	cardiovascular disease
NCDSS	the Korean National Climate Data Service System
PM_{10}	particulate matter with an aerodynamic diameter of ≤10
$PM_{2.5}$	particulate matter with an aerodynamic diameter of ≤2.5
WPW	Wolff-Parkinson-White

of atrial fibrillation.[9,10] However, no previous study has evaluated the association between long-term PM air pollution and other types of cardiac arrhythmias in a general population setting with relatively high air pollution levels.

We therefore evaluated the association between PM air pollution and risk of incident arrhythmia, including AF, bradycardia, premature cardiac contractions, right bundle-branch block (RBBB), tachycardia, and other types of arrhythmias, in a large cohort of middle-aged Korean men and women who attended repeated health screening exams between 2002 and 2016. We hypothesized that participants exposed to a higher concentration of PM air pollution would be at higher risk of arrhythmias compared with those exposed to a lower concentration of PM air pollution.

METHODS

Study Population

We obtained data from the KSHS (Kangbuk Samsung Health Study), an ongoing cohort study of South Korean men and women 18 years of age or older who underwent comprehensive annual or biennial health examinations at the clinics of the Kangbuk Samsung Hospital Total Healthcare Center in Seoul and Suwon, South Korea.[11] In our study, over 80% of the participants were employees from various companies or local government organizations and their spouses. The remaining participants voluntarily purchased screening exams at the health exam center.

We included participants who lived in the Greater Seoul metropolitan areas (605.3 km^2 in Seoul, 1063 km^2 in Incheon, and 10 184 km^2 in Gyeonggi-do Province [Suwon center]) and who attended at least 2 health screening exams from January 1, 2002 through December 31, 2016 (n=196 330; Figure 1). We excluded participants with any arrhythmia (n=6404) or history of heart diseases (n=4132) at baseline. We further excluded participants with missing data on potential risk covariates (n=3306). The final sample included 182 488 participants (102 698 men and 79 790 women). The study was approved by the institutional review board of the Kangbuk Samsung Hospital, and the requirement of informed consent was waived because we only used deidentified data routinely collected during health screening visits.

Assessment of PM Air Pollution

We used a land use regression model to estimate monthly PM_{10} and $PM_{2.5}$ concentration at a 1-km resolution for each participant based on their address postal codes in Seoul or Suwon city.[12] Briefly, PM concentrations were collected from the Seoul Metropolitan Government Atmospheric Environment, the Gyeonggi-do Institute of Health & Environment, as well as the Korean National Climate Data Service System. For PM_{10}, data were collected since 2002 for Seoul and Suwon. We used mixed generalized additive regression models to estimate individual monthly mean PM_{10} concentrations from January 1, 2002 through December 31, 2016. Covariates included smoothed county population density, seasonal terms (year and month), meteorological variables (temperature, humidity, wind speed, pressure, etc), digital altitude elevation, distance to the nearest power plants (industrial pollution), percentage of land coverage within 300, 500, 1000, 3000, and 5000-m buffers, and distance to the nearest road (traffic pollution). For $PM_{2.5}$, data were collected since 2008 for Seoul and since 2014 for Suwon, and were modeled in a way analogous to PM_{10}, as described above. For Suwon, we imputed $PM_{2.5}$ levels between 2008 and 2014 using the product of the $PM_{2.5}/PM_{10}$ ratio calculated using 2008 to 2014 data from Seoul and concurrent PM_{10} levels in Suwon.

In a cross-validation study, we built the models in a training data set including 70% of the measurements,

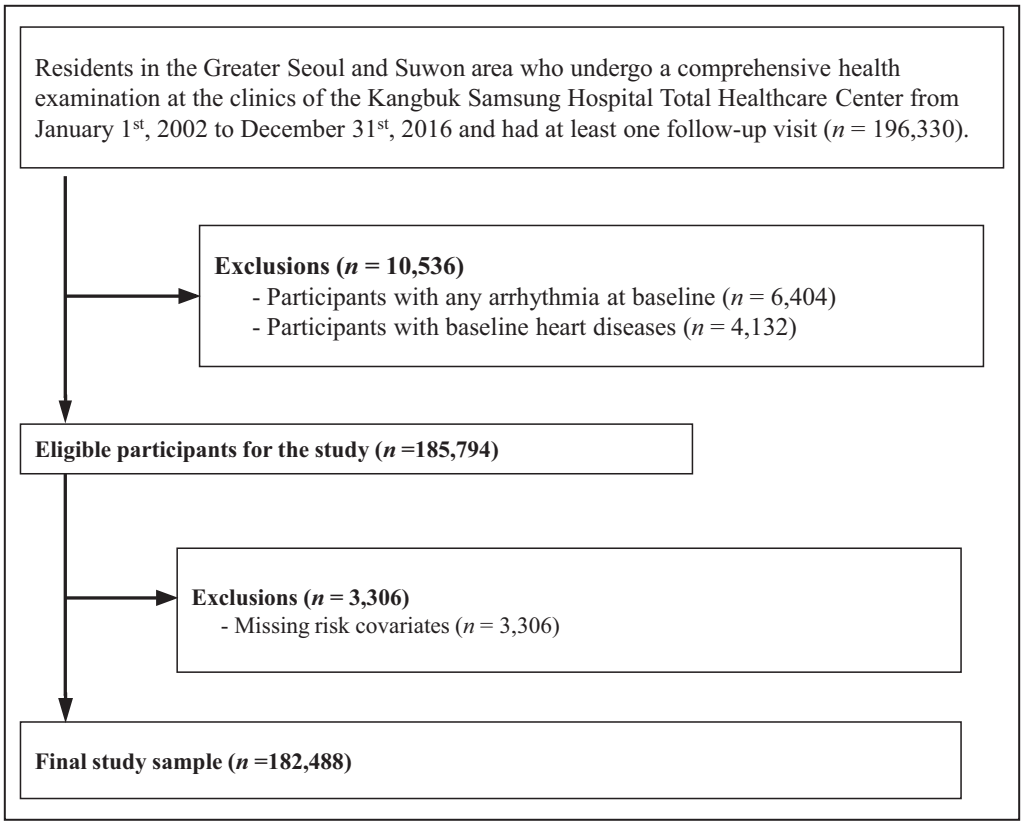

Figure 1. Flowchart of study participants.

used the models to predict PM_{10} and $PM_{2.5}$ values in a test data set including the rest 30% of the measurements, and compared the predicted values with the observed measurements. The models had a high predictive accuracy (cross-validation R^2 values of 0.80 and 0.77 for PM_{10} and $PM_{2.5}$, respectively).

For each participant, we calculated the 12-, 36-, and 60- month mean PM_{10} concentration and 12- and 36-month mean $PM_{2.5}$ concentration before each visit (60-month $PM_{2.5}$ analyses were unavailable because of the shorter time-frame of measurements). The follow-up for 12-month PM_{10} analysis was from January 1, 2003 through December 31, 2016; the follow-up for 12-month $PM_{2.5}$ was from January 1, 2009 through December 31, 2016.

Assessment of Cardiac Arrhythmias

In each health screening exam, standard 12-lead ECGs were digitally acquired using an ECG recorder (CARDIMAX FX-7542; Fukuda Denishi Co., Ltd., Tokyo, Japan) at 1 mV/cm calibration and 25 mm/s speed. All ECGs were initially inspected visually for technical errors and inadequate quality. Arrhythmias were automatically documented from the ECGs analysis program (PI-19E, Fukuda Denshi, Tokyo, Japan) and interpreted by a cardiologist.

We defined 6 types of arrhythmia as end points for our analyses: AF or flutter, bradycardia (resting heart rate <50 beats/min), premature atrial contractions, premature ventricular contractions, right bundle branch blocks (RBBB, QRS ≥120 ms, RSR' pattern in V1–3, and slurred S wave in the lateral leads [I, aVL and V5–V6]), tachycardia (resting heart rate ≥100 beats/min), and other arrhythmias (Wolff-Parkinson-White [WPW] syndrome, sinus arrhythmia, etc).[13]

Health Screening Exams

At each screening exam, standardized questionnaires were used to collect information on sociodemographic factors, lifestyle characteristics, disease history, and medication use. Alcohol consumption was categorized as none, moderate (men: >0 to ≤30 g/day, women: >0 to ≤20 g/day), or excessive (men >30 g/day, women >20 g/day). Physical activity was classified into 3 categories: none, <3 times per week, ≥3 times per week. Diabetes mellitus was defined as fasting serum glucose ≥126 mg/dL, a self-reported history of diabetes mellitus, or self-reported use of antidiabetic medications. Hypertension was defined as blood pressure ≥140/90 mm Hg, a self-reported history of hypertension, or current use of antihypertensive medications.

Patient and Public Involvement

Patients were not directly involved in the design of the study, the writing or editing of this document, or the interpretation or dissemination of the results.

Statistical Analysis

We used pooled logistic regression models with time-varying exposures and covariates to evaluate the association of long-term PM_{10} and $PM_{2.5}$ exposure and the development of arrhythmia. For time-dependent analyses, the pooled logistic regression model closely approximates a Cox model when the risk of outcome between intervals is low.[14] The primary outcome of the study was the development of any arrhythmia. The secondary outcomes were the development of each individual type of arrhythmia. Participants who developed arrhythmia contributed person-time from the baseline visit until the visit in which they developed the arrhythmia. The rest of participants contributed person-time until their last available visit.

Odds ratios (OR) and 95% CIs were calculated per 10 μg/m^3 increment in PM concentration. We used 2 models with increasing degrees of adjustment: model 1 adjusted for age, sex, study center, year of visit, and education level; model 2 additionally adjusted for systolic blood pressure, smoking status, height, weight, alcohol consumption, physical activity, diabetes mellitus, history of diabetes mellitus, and history of hypertension.

As for sensitivity analyses for PM_{10} air pollution, we conducted analyses for 12- and 36-month PM_{10} exposure among the same participants as the 60-month PM_{10} exposure. For $PM_{2.5}$ air pollution, we conducted analyses for 12-month $PM_{2.5}$ exposure among the same participants as the 36-month $PM_{2.5}$ exposure.

To evaluate the nonlinear dose-response relationships between PM air pollution and risk of arrhythmia, we modeled PM_{10} and $PM_{2.5}$ air pollution using restricted cubic splines with knots at the 5th, 25th, 75th, and 95th percentiles of the distribution of PM concentrations. We also assessed potential effect modification by age group, sex, alcohol intake, physical exercise, and smoking status, using likelihood ratio tests comparing models with an interaction (product) term between air pollution exposure and the effect modifier versus models without the interaction term.

This study was done without adjustment for multiplicity (multiple exposures, multiple time points, and multiple end points) and, thus, the Type I error was not controlled to 0.05. All analyses were performed with Stata (version 15.0; Stata Corporation) and R (Version 3.4.1; R Development Core Team).

RESULTS

The mean (SD) age at inclusion was 36.5 (7.0) years, and 56.3% of study participants were male. The monthly mean (SD) concentrations of PM_{10} during the 12-, 36-, and 60-month periods before the baseline visit were 56.6 (7.5), 55.6 (6.2), and 56.1 (5.3) μg/m^3, respectively; the mean (SD) concentration of $PM_{2.5}$ during the 12- and 36-month periods before the baseline visit were 26.6 (2.3) and 26.3 (2.3) μg/m^3, respectively (Table 1).

The median (range) follow-up time was 4.8 (0.5–13.9) years for PM_{10} and 3.8 (0.5–7.9) years for $PM_{2.5}$ analyses. For PM_{10} analyses, there were 16 149 incident cases of any arrhythmia during follow-up (6373 bradycardias, 180 AF, 1705 premature ventricular contraction, 670 premature atrial contraction, 669 tachycardia, 4760 RBBB, and 1792 other arrythmias). For $PM_{2.5}$ analyses, there were 12 769 incident cases of any arrhythmia during follow-up (5499 bradycardias, 116 AF, 1186 premature ventricular contraction, 488 premature atrial contraction, 450 tachycardia, 3732 RBBB, and 1298 other arrythmias) (Table S1).

In the fully adjusted models, the ORs (95% CI) for any arrhythmia associated with a 10 μg/m^3 increase in 12-, 36-, and 60-month PM_{10} exposure were 1.14 (1.09–1.20), 1.11 (1.05–1.17), and 1.13 (1.07–1.19), respectively (Table 2). The ORs for a 10 μg/m^3 increase in 12- and 36-month $PM_{2.5}$ exposure were 1.27 (1.15–1.40) and 1.11 (1.00–1.23). Spline regression analyses confirmed that increasing 12-month mean PM_{10} and $PM_{2.5}$ concentrations were associated with an increased risk of any arrhythmias, although the associations were non-linear (*P* for non-linear spline terms <0.001; Figure 2).

Among individual arrhythmia types, an increase in PM_{10} or in $PM_{2.5}$ air pollution exposure was associated with a statistically significant increase in the risk of bradycardia, irrespective of the duration of the exposure period (Table 3). In addition, PM_{10} exposure during the 12- and 36-months before baseline was associated with a statistically significant increased risk of premature atrial contraction, and $PM_{2.5}$ exposure 12- and 36-months before baseline was associated with RBBB disorders. In spline regression analyses, the risk of bradycardia and premature contraction increased with the increasing 12-month mean PM_{10} throughout most of its distribution (Figure 3).

In sensitivity analyses, the associations between PM air pollution and general arrhythmias were consistent when we restricted the analyses for 12- and 36-month PM_{10} exposure using the same participants as the 60-month PM_{10} exposure, and for 12-month $PM_{2.5}$ exposure using the same participants as the 36-month $PM_{2.5}$ exposure (Tables S2 and S3).

Table 1. Baseline Characteristics and Medical History of KSHS Cohort Participants

Characteristic	Overall	No Arrhythmia During Follow-Up	Any Arrhythmia During Follow-Up	*P* Value
N	182 488	167 763	14 725	
Age, y	36.5 (7.0)	36.6 (7.0)	36.3 (6.5)	<0.001
Male, %	102 698 (56.3)	92 371 (55.1)	10 327 (70.1)	<0.001
12-mo $PM_{2.5}$ mean, μg/m^3	26.6 (2.3)	26.2 (2.3)	26.4 (2.2)	<0.001
36-mo $PM_{2.5}$ mean, μg/m^3	26.3 (2.3)	26.3 (2.3)	26.5 (2.2)	<0.001
12-mo PM_{10} mean, μg/m^3	56.6 (7.5)	56.4 (7.5)	58.8 (7.0)	<0.001
36-mo PM_{10} mean, μg/m^3	55.6 (6.2)	55.5 (6.2)	57.6 (5.9)	<0.001
60-mo PM_{10} mean, μg/m^3	56.1 (5.3)	56.0 (5.3)	57.8 (5.2)	<0.001
Height	167.7 (8.3)	167.5 (8.3)	169.7 (7.9)	<0.001
Weight	65.6 (12.7)	65.5 (12.7)	66.5 (11.9)	<0.001
Smoking				<0.001
Never	96 912 (53.1)	90 894 (54.2)	6018 (40.9)	
Former	41 784 (22.9)	37 396 (22.3)	4388 (29.8)	
Current	43 792 (24.0)	39 473 (23.5)	4319 (29.3)	
Alcohol intake				<0.001
None	50 091 (27.4)	46 351 (27.6)	3740 (25.4)	
Moderate	115 219 (63.1)	105 594 (62.9)	9625 (65.4)	
High	17 178 (9.4)	15 818 (9.4)	1360 (9.2)	
Daily physical activity				<0.001
None	110 357 (60.5)	102 242 (60.9)	8115 (55.1)	
<3 times	54 608 (29.9)	49 844 (29.7)	4764 (32.4)	
3 times or more	17 523 (9.6)	15 677 (9.3)	1846 (12.5)	
Diabetes mellitus, %	2254 (1.2)	2090 (1.2)	164 (1.1)	0.176
Hypertension, %	19 006 (10.4)	17 528 (10.4)	1478 (10.0)	0.121
Systolic BP, mm Hg	110.9 (13.5)	110.8 (13.5)	112.1 (13.1)	<0.001
Creatinine, mg/dL	0.9 (0.2)	0.9 (0.2)	1.0 (0.2)	<0.001

Numbers in the table are mean (SD) or count (%). BP indicates blood pressure; KSHS, Kangbuk Samsung Health Study; PM_{10}, particulate matter of <10 μm in aerodynamic diameter; and $PM_{2.5}$, particulate matter of <2.5 μm in aerodynamic diameter.

In subgroup analyses, the association between exposure to PM_{10} and risk of any arrhythmia was stronger in participants ≥40 years of age compared with those <40 years (*P* for interaction=0.001), in heavy alcohol drinkers compared with moderate drinkers or non-drinkers (*P* for interaction=0.001), and in physically inactive compared with active participants (*P* for interaction=0.001, Figure 4).

DISCUSSION

In this large prospective study of middle-aged Korean men and women, long-term exposure to $PM_{2.5}$ and PM_{10} air pollution was associated with increased risk of arrhythmia, more specifically bradycardia, premature atrial contractions, and RBBB. The associations were

Table 2. ORs (95% CI) for Each 10 μg/m^3 Increase in Particulate Matter Exposure Associated with Risk of Any Arrhythmias in KSHS Cohort From 2002 to 2016

Exposures	Models	12-mo	36-mo	60-mo
PM_{10}	Model 1	1.13 (1.08, 1.19)	1.10 (1.05, 1.16)	1.12 (1.06, 1.18)
	Model 2	1.14 (1.09, 1.20)	1.11 (1.05, 1.17)	1.13 (1.07, 1.19)
$PM_{2.5}$	Model 1	1.29 (1.17, 1.42)	1.12 (1.02, 1.25)	
	Model 2	1.27 (1.15, 1.40)	1.11 (1.00, 1.23)	

Model 1. Adjusted for age (continuous), sex (men or women), study center (Seoul or Suwon), year of visit (continuous), and education level (no education, elementary school, middle school, high school, technical college, university, or more). Model 2. Additionally, systolic blood pressure (continuous), smoking status (never, current, former), height (continuous), weight (continuous), alcohol consumption (none, moderate, or excessive), physical activity (none, <3 times per week, or ≥3 times per week), and history of diabetes mellitus (yes or no) and history of hypertension (yes or no). KSHS indicates Kangbuk Samsung Health Study; PM_{10}, particulate matter of <10 μm in aerodynamic diameter; and $PM_{2.5}$, particulate matter of <2.5 μm in aerodynamic diameter.

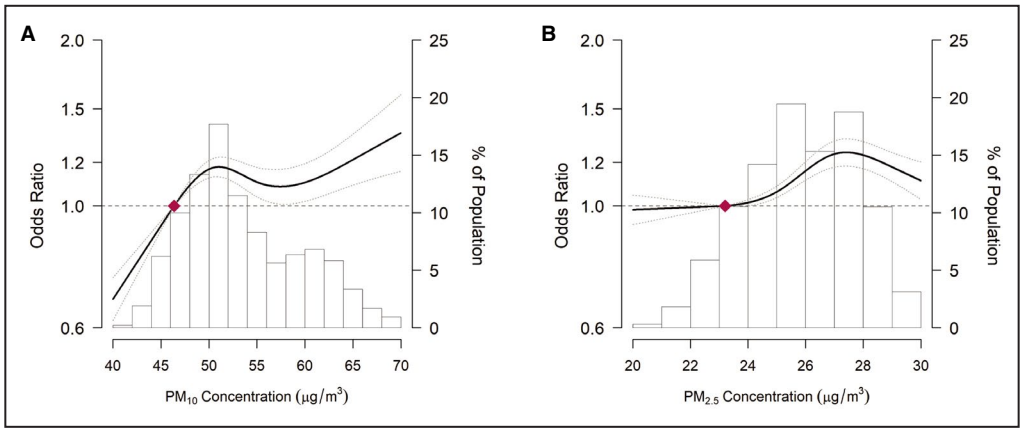

Figure 2. ORs for risks of arrhythmias by the level of exposure to 12-month PM_{10} and $PM_{2.5}$ concentration.
(A) OR for PM_{10} exposure; (**B**) OR for $PM_{2.5}$ exposure. $PM_{2.5}$ (particulate matter with an aerodynamic diameter of ≤2.5), PM_{10} (particulate matter with an aerodynamic diameter of ≤10). The dose-response curve was calculated using restricted cubic splines with knots at the 5th, 27.5th, 50th, 72.5th, and 95th percentiles of the distribution of 60-month PM_{10} concentrations. The reference exposure level was set at the 10th percentile of the distribution of 12-month PM_{10} concentrations (47.7 μg/m^3) and 12-month $PM_{2.5}$ concentrations (23.2 μg/m^3). ORs were adjusted for age (continuous), sex (men or women), study center (Seoul or Suwon), year of visit (continuous), education level (no education, elementary school, middle school, high school, technical college, university, or more), systolic blood pressure (continuous), smoking status (never, current, former), height (continuous), weight (continuous), alcohol consumption (none, moderate, or excessive), physical activity (none, <3 times per week, or ≥3 times per week), and history of diabetes mellitus (yes or no) and history of hypertension (yes or no).

stronger in older participants, heavy alcohol drinkers, and in those who were physically inactive.

The mechanisms underlying the association between PM air pollution and arrhythmias are unclear. Inhalation of PM and absorption of its constituents into the bloodstream could result in high levels of reactive oxygen species, systemic inflammation, and prothrombotic factors. Additionally, PM could alter endothelial structure and function and affect the autonomic tone, leading to acute or chronic vasoconstriction and cardiac arrhythmias.[15] In rats, ECG morphology changed and atrioventricular block arrhythmias increased in response to traffic derived $PM_{2.5}$.[16] This finding is consistent with our observation of increased risk of RBBB associated with ambient $PM_{2.5}$. In experimental studies on healthy adults, controlled short-term exposure of concentrated ambient particles was also associated with significant changes in indices of inflammation, hemostasis, autonomic heart rate control, and cardiac repolarization.[17] Moreover, in cross-sectional studies of subjects at high risk of CVD, short-term and long-term exposure to PM air pollution were associated with increased risk of arrhythmia.[7,18]

Bradycardia

Bradycardia is a common, usually asymptomatic condition in the general population, but severe bradycardia could predict cardiac arrest and other CVD outcomes.[19] Few studies have investigated the effect of PM air pollution on the risk of bradycardia. Higher air pollution concentrations may increase the occurrence of bradycardia in infants in high-risk.[20] In a study of 10 healthy participants over 60 years of age, short-term exposure to concentrated ambient air pollution particles immediately decreased their heart rate variability and was associated with a 5-fold increase in bradycardia.[21] The physiological importance of the observed association of long-term PM air pollution exposure with bradycardia is unclear, and more research is warranted.

Premature Atrial Contraction

Premature atrial contraction is a common, benign cardiac dysrhythmia with no pathological significance; however, growing evidence has suggested that premature atrial contraction may be associated with the development of atrial fibrillation and stroke.[22,23] In a study of 105 community-dwelling healthy nonsmokers, exposure to $PM_{2.5}$ was not associated with increased premature atrial contraction frequency.[24] For long-term PM exposure, the Reasons for Geographic and Racial Differences in Stroke (REGARDS) study showed that increased long-term, but not short-term $PM_{2.5}$ exposure, was associated with higher prevalence of premature atrial contraction.[25] Possible explanations for the discrepancies between the REGARDS study and our study include: the study

Table 3. ORs (95% CI) for Secondary Outcomes Associated with Particulate Matter Exposures

Arrhythmias	Exposure	Models	Exposure Duration		
			12-mo	36-mo	60-mo
Atrial Fibrillation	PM_{10}	Model 1	1.04 (0.63, 1.74)	0.98 (0.54, 1.79)	1.33 (0.66, 2.67)
		Model 2	1.04 (0.62, 1.74)	0.99 (0.54, 1.80)	1.34 (0.67, 2.70)
	$PM_{2.5}$	Model 1	1.50 (0.64, 3.52)	0.93 (0.47, 1.84)	
		Model 2	1.51 (0.66, 3.49)	0.94 (0.48, 1.84)	
Bradycardia	PM_{10}	Model 1	1.21 (1.12, 1.31)	1.22 (1.13, 1.32)	1.19 (1.10, 1.29)
		Model 2	1.23 (1.14, 1.33)	1.23 (1.14, 1.33)	1.21 (1.12, 1.31)
	$PM_{2.5}$	Model 1	1.22 (1.06, 1.40)	1.22 (1.05, 1.41)	
		Model 2	1.20 (1.04, 1.38)	1.19 (1.03, 1.38)	
Premature Ventricular Contraction	PM_{10}	Model 1	1.11 (0.96, 1.29)	1.08 (0.92, 1.27)	1.04 (0.86, 1.25)
		Model 2	1.11 (0.96, 1.29)	1.08 (0.92, 1.27)	1.04 (0.86, 1.25)
	$PM_{2.5}$	Model 1	0.99 (0.72, 1.35)	0.83 (0.59, 1.16)	
		Model 2	0.99 (0.72, 1.36)	0.84 (0.60, 1.16)	
Premature Atrial Contraction	PM_{10}	Model 1	1.31 (1.03, 1.65)	1.33 (1.05, 1.69)	1.38 (1.06, 1.79)
		Model 2	1.31 (1.03, 1.66)	1.34 (1.05, 1.70)	1.39 (1.07, 1.80)
	$PM_{2.5}$	Model 1	1.09 (0.63, 1.88)	0.96 (0.53, 1.74)	
		Model 2	1.09 (0.63, 1.87)	0.96 (0.53, 1.74)	
RBBB	PM_{10}	Model 1	1.06 (0.97, 1.16)	0.97 (0.88, 1.06)	1.02 (0.92, 1.12)
		Model 2	1.08 (0.98, 1.18)	0.98 (0.89, 1.08)	1.03 (0.94, 1.14)
	$PM_{2.5}$	Model 1	1.74 (1.46, 2.08)	1.19 (0.99, 1.44)	
		Model 2	1.70 (1.42, 2.04)	1.17 (0.97, 1.41)	
Tachycardia	PM_{10}	Model 1	0.95 (0.74, 1.22)	1.09 (0.84, 1.41)	1.06 (0.78, 1.43)
		Model 2	0.92 (0.71, 1.18)	1.05 (0.81, 1.36)	1.03 (0.76, 1.39)
	$PM_{2.5}$	Model 1	0.90 (0.60, 1.34)	0.97 (0.63, 1.50)	
		Model 2	0.90 (0.60, 1.34)	1.00 (0.65, 1.55)	
Other	PM_{10}	Model 1	1.15 (0.99, 1.33)	1.13 (0.97, 1.32)	1.22 (1.04, 1.42)
		Model 2	1.15 (0.99, 1.33)	1.14 (0.97, 1.32)	1.22 (1.05, 1.43)
	$PM_{2.5}$	Model 1	0.88 (0.69, 1.11)	0.87 (0.64, 1.18)	
		Model 2	0.87 (0.69, 1.10)	0.86 (0.64, 1.16)	

Model 1. Adjusted for age (continuous), sex (men or women), study center (Seoul or Suwon), year of visit (continuous), and education level (no education, elementary school, middle school, high school, technical college, university, or more). Model 2. Additionally, systolic blood pressure (continuous), smoking status (never, current, former), height (continuous), weight (continuous), alcohol consumption (none, moderate, or excessive), physical activity (none, <3 times per week, or ≥3 times per week), and history of diabetes mellitus (yes or no) and history of hypertension (yes or no). PM_{10} indicates particulate matter of <10 μm in aerodynamic diameter; $PM_{2.5}$, particulate matter of <2.5 μm in aerodynamic diameter; and RBBB, right bundle branch block.

design (cross-sectional versus longitudinal), population (Black and White versus Asian), and exposure levels (1-year $PM_{2.5}$ mean concentration of 13.5 μg/m^3 versus 26.6 μg/m^3 in our study). The association between PM_{10} and premature cardiac contraction, however, has not been previously examined, and the positive association between long-term PM_{10} and the risk of premature atrial contraction in our study needs to be confirmed in future studies.

Right Bundle-Branch Block

The relationship between air pollution and the development of RBBB has not been extensively studied. In the Multi-Ethnic Study of Atherosclerosis (MESA) study, long-term exposure to $PM_{2.5}$ was associated with an increased prevalence of ventricular conduction abnormalities in adults without clinical cardiovascular disease.[26] In our study, $PM_{2.5}$, but not PM_{10}, was associated with a higher risk of incident RBBB. It is known that $PM_{2.5}$ penetrates deeper into the lungs and reaches the alveolar region because of its smaller size compared with PM_{10}. Also, $PM_{2.5}$ may be more toxic, as it includes nitrates, sulfates, metals, and particles with various chemicals, and the higher surface area per unit mass for $PM_{2.5}$ could increase the adsorption and condensation toxicants.[27] In this study, we did not test the associations between long-term PM exposure and other types of conduction disorders, including AV block and left bundle branch block, because of the limited number of cases during follow-up visits.

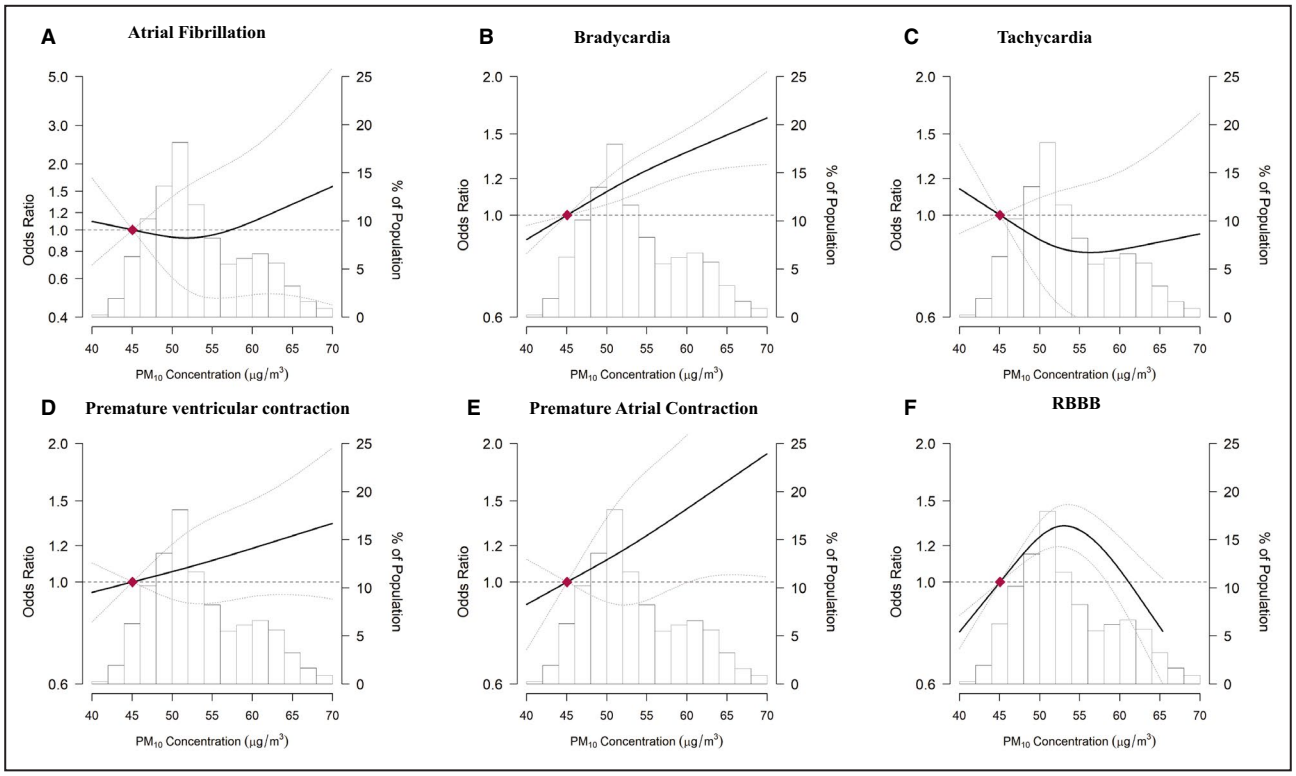

Figure 3. ORs for risks of specific arrhythmia diseases by the level of exposure to 12-month PM_{10} concentration.
A, OR for atrial fibrillation; (**B**) OR for bradycardia; (**C**) OR for tachycardia; (**D**) OR for premature ventricular contraction; (**E**) OR for premature atrial contraction; (**F**) OR for RBBB. $PM_{2.5}$ (particulate matter with an aerodynamic diameter of ≤2.5), PM_{10} (particulate matter with an aerodynamic diameter of ≤10), RBBB (right bundle branch block). The dose-response curve was calculated using restricted cubic splines with knots at the 5th, 27.5th, 50th, 72.5th, and 95th percentiles of the distribution of 12-month PM_{10} concentrations. The reference exposure level was set at the 10th percentile of the distribution of 12-month PM_{10} concentrations (47.7 μg/m^3). ORs were adjusted for age (continuous), sex (men or women), study center (Seoul or Suwon), year of visit (continuous), education level (no education, elementary school, middle school, high school, technical college, university, or more), systolic blood pressure (continuous), smoking status (never, current, former), height (continuous), weight (continuous), alcohol consumption (none, moderate, or excessive), physical activity (none, <3 times per week, or ≥3 times per week), and history of diabetes mellitus (yes or no) and history of hypertension (yes or no). OR indicates odds ratio; and RBBB, right bundle branch block.

Atrial Fibrillation and Tachycardia

Short- and long-term exposure to PM or gaseous air pollution, including $PM_{2.5}$, PM_{10}, nitrogen dioxide (NO_2), sulfur dioxide (SO_2), carbon dioxide (CO), ozone, and black carbon (BC) have been positively associated with AF and tachycardia.[18,28] In our study, long-term PM air pollution exposure was associated with an increased risk of AF and tachycardia, but the associations were not statistically significant. Possible reasons for the inconsistent associations across studies include: differences in sample size, study settings, air pollution levels, and pre-existing conditions of the study populations.

In our study, 12-month $PM_{2.5}$ had a stronger association than 36- or 60-month $PM_{2.5}$ with atrial fibrillation and RBBB. This could attributable to a more robust association with a longer follow-up time for 12-month $PM_{2.5}$ analysis.

Interaction Effects

The positive association between air pollution and the risk of arrhythmias was more pronounced in participants ≥40 years of age, and in participants with higher levels of alcohol intake and lower levels of physical exercise. The mechanisms underlying these differences are unclear. Vulnerable populations, including the elderly, or participants with underlying conditions, tend to be more susceptible to the effects of PM air pollution exposure, but these differences need to be confirmed in future studies.[10,28,29]

Strengths and Limitations

The strengths of our study included the large sample size, the long follow-up period, and the availability of detailed information on various potential lifestyle and socioeconomic confounders. Our findings, however, should be interpreted with the consideration of some limitations. First, model-estimated exposures are surrogates for personal exposure, which depends on the time spent indoors, daily activity patterns, and workplace exposure. Exposure measurement error may underestimate the underlying associations between air pollution and arrhythmias.

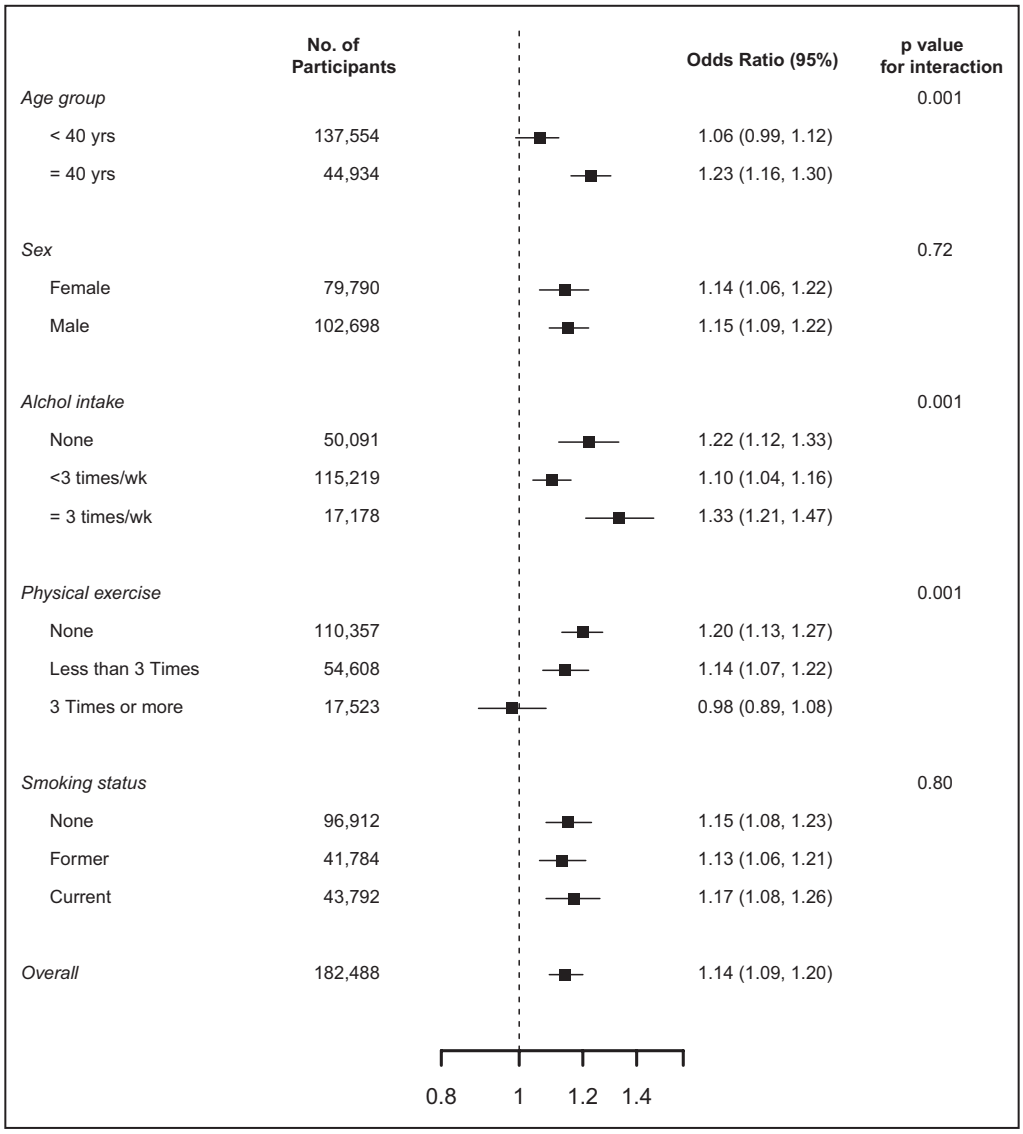

Figure 4. ORs (95% CI) for risks of arrhythmias associated with a 10 μg/m^3 increase in 12-month PM_{10} concentrations, by baseline participant characteristics.
ORs were adjusted for age (continuous), sex (men or women), study center (Seoul or Suwon), year of visit (continuous), education level (no education, elementary school, middle school, high school, technical college, university, or more), systolic blood pressure (continuous), smoking status (never, current, former), height (continuous), weight (continuous), alcohol consumption (none, moderate, or excessive), physical activity (none, <3 times per week, or ≥3 times per week), and history of diabetes mellitus (yes or no) and history of hypertension (yes or no). OR indicates odds ratio.

Second, arrhythmias were detected from a single 10-second electrocardiogram recording. Consequently, we were more likely to detect arrhythmias that were persistent or that occurred frequently, and may have missed episodes of paroxysmal arrhythmias, including paroxysmal atrial fibrillation. We were also unable to differentiate between long-term disease and observation of acute events because of the limited duration of ECG. Furthermore, we do not have data on PR, QRS, and corrected QT intervals, which could better quantify the underlying arrhythmic abnormalities.

Third, our study population was relatively young, healthy, and highly educated, which reduces the risk of selection bias because of competing risks and the risk of confounding becasue of the presence of comorbidities or medication use; thus, our results may not be generalized to other populations.

Our study suggests that PM air pollution is associated with increased incidence of bradycardia, RBBB, and premature atrial contraction arrythmias in the general population. The dose-response trend highlights the importance and potential opportunities for

reducing the burden of arrythmias, as exposure to PM air pollution is chronic. Prevention and treatment of cardiac arrythmias might benefit from the identification of relevant pathophysiological pathways associated with PM air pollution.

CONCLUSIONS

In this large cohort of middle-aged men and women, exposure to long-term PM_{10} air pollution was independently associated with an increased risk of bradycardia and premature ventricular contractions, and $PM_{2.5}$ with an increased risk of bradycardia and RBBB. Future studies are needed to understand the underlying mechanistic pathways, the interactions between PM exposure and other traditional CVD risk factors, and the benefits of PM reduction on cardiac rhythmicity.

ARTICLE INFORMATION

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Zhang contributed to data analysis, reporting and drafting the work; Zhao and Shin contributed to planning, data analysis, and revising the work; Kang, Hong, Chang, Ryu, Park, Cho, Guallar, Shin, and Zhao contributed to revising and final approval of the work.

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Disclosures

None.

Supplementary Material

Tables S1–S3

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Supplemental Material

Table S1. **Cases and person-year for arrhythmias during follow-up visits.**

	Outcomes								
	Bradycardia	Atrial fibrillation	Premature Ventricular Contraction	Premature Atrial Contraction	Tachycardia	RBBB	LBBB	Atrioventricular block	Others
PM_{10}	6,373/1,031,827	180/1,049,008	1,705/1,042,506	670/1,047,251	669/1,046,556	4,760 /1,032,103	42/1,043,399	26/1,043,417	1,792 /1,042,900
$PM_{2.5}$	5,499/590,311	116/602,578	1,186/600,144	488/601,674	450/601,803	3,732/585,312	22/598,223	5/598,251	1,298/599,501

Table S2. ORs (95% CI) for each 10 μg/m^3 increase in particulate matter exposure associated with risk of any arrhythmias in Kangbuk Samsung Health Study (KSHS) cohort from 2002 to 2016. (use longer exposure datasets)

Exposures	Models	12-month	36-month	60-month
PM_{10}	model 1	1.10 (1.04, 1.16)	1.08 (1.03, 1.14)	1.12 (1.06, 1.18)
	model 2	1.11 (1.05, 1.18)	1.09 (1.03, 1.15)	1.12 (1.06, 1.19)
$PM_{2.5}$	model 1	1.20 (1.08, 1.33)	1.13 (1.01, 1.25)	
	model 2	1.19 (1.07, 1.32)	1.11 (1.00, 1.23)	

Model 1. Adjusted for age (continuous), sex (men or women), study center (Seoul or Suwon), year of visit (continuous), and education level (no education, elementary school, middle school, high school, technical college, and university or more). Model 2. Additionally, systolic blood pressure (continuous), smoking status (never, current, former), height (continuous), weight (continuous), alcohol consumption (none, moderate, or excessive), physical activity (none, <3 times per week, or ⩾3 times per week), and history of diabetes (yes or no) and history of hypertension (yes or no). PM_{10}, particulate matter of less than 10 μm in aerodynamic diameter; $PM_{2.5}$, particulate matter of less than 2.5 μm in aerodynamic diameter.

Table S3. ORs (95% CI) for secondary outcomes associated with particulate matter exposures (use longer exposure datasets)

Arrhythmias	Exposure	Models	Exposure Duration		
			12-month	36-month	60-month
Atrial Fibrillation	PM_{10}	Model 1	1.24 (0.61, 2.55)	1.28 (0.62, 2.66)	1.33 (0.66, 2.67)
		Model 2	1.25 (0.61, 2.56)	1.29 (0.62, 2.69)	1.34 (0.67, 2.70)
	$PM_{2.5}$	Model 1	1.02 (0.50, 2.07)	0.93 (0.48, 1.84)	
		Model 2	0.93 (0.47, 1.84)	0.94 (0.48, 1.84)	
Bradycardia	PM_{10}	Model 1	1.19 (1.10, 1.28)	1.19 (1.10, 1.29)	1.19 (1.10, 1.29)
		Model 2	1.21 (1.12, 1.31)	1.21 (1.11, 1.31)	1.21 (1.12, 1.31)
	$PM_{2.5}$	Model 1	1.14 (0.99, 1.31)	1.22 (1.05, 1.41)	
		Model 2	1.12 (0.97, 1.29)	1.19 (1.03, 1.38)	
Premature Ventricular Contraction	PM_{10}	Model 1	1.07 (0.90, 1.28)	1.03 (0.85, 1.24)	1.04 (0.86, 1.25)
		Model 2	1.07 (0.90, 1.28)	1.03 (0.85, 1.24)	1.04 (0.86, 1.25)
	$PM_{2.5}$	Model 1	0.85 (0.61, 1.20)	0.83 (0.59, 1.16)	
		Model 2	0.86 (0.61, 1.21)	0.84 (0.60, 1.16)	
Premature Atrial Contraction	PM_{10}	Model 1	1.28 (0.98, 1.68)	1.35 (1.03, 1.75)	1.38 (1.06, 1.79)
		Model 2	1.28 (0.98, 1.68)	1.35 (1.04, 1.76)	1.39 (1.07, 1.80)
	$PM_{2.5}$	Model 1	0.99 (0.55, 1.80)	0.96 (0.53, 1.74)	
		Model 2	0.99 (0.55, 1.80)	0.96 (0.53, 1.74)	
Conduction Disorder	PM_{10}	Model 1	0.99 (0.90, 1.09)	0.94 (0.85, 1.04)	1.02 (0.93, 1.13)
		Model 2	1.01 (0.92, 1.11)	0.96 (0.86, 1.06)	1.04 (0.94, 1.15)
	$PM_{2.5}$	Model 1	1.55 (1.27, 1.89)	1.19 (0.99, 1.44)	
		Model 2	1.52 (1.24, 1.85)	1.17 (0.97, 1.41)	
Tachycardia	PM_{10}	Model 1	1.03 (0.75, 1.41)	1.08 (0.79, 1.47)	1.06 (0.78, 1.43)
		Model 2	0.98 (0.72, 1.34)	1.04 (0.77, 1.42)	1.03 (0.76, 1.39)
	$PM_{2.5}$	Model 1	0.92 (0.60, 1.41)	0.97 (0.63, 1.50)	
		Model 2	0.93 (0.61, 1.43)	1.00 (0.65, 1.55)	
Other	PM_{10}	Model 1	1.11 (0.95, 1.30)	1.10 (0.94, 1.30)	1.22 (1.04, 1.42)
		Model 2	1.11 (0.95, 1.31)	1.11 (0.94, 1.31)	1.22 (1.05, 1.43)
	$PM_{2.5}$	Model 1	0.97 (0.70, 1.34)	0.87 (0.64, 1.18)	
		Model 2	0.96 (0.69, 1.33)	0.86 (0.64, 1.16)	

Model 1. Adjusted for age (continuous), sex (men or women), study center (Seoul or Suwon), year of visit (continuous), and education level (no education, elementary school, middle school, high school, technical college, and university or more). Model 2. Additionally, systolic blood pressure (continuous), smoking status (never, current, former), height (continuous), weight (continuous), alcohol consumption (none, moderate, or excessive), physical activity (none, <3 times per week, or ⩾3 times per week), and history of diabetes (yes or no) and history of hypertension (yes or no). PM_{10}, particulate matter of less than 10 μm in aerodynamic diameter; $PM_{2.5}$, particulate matter of less than 2.5 μm in aerodynamic diameter.