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Abbreviations
ANOVA, analysis of variance; BMI, body mass
index; BP, blood pressure; CI, confidence

Association between ambient particulate matter levels and hypertension: results from the Korean Genome and Epidemiology Study

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ABSTRACT

Background: Recently, there has been increasing worldwide concern about outdoor air pollution, especially particulate matter (PM), which has been extensively researched for its harmful effects on the respiratory system. However, sufficient research on its effects on cardiovascular diseases, such as hypertension, remains lacking. In this study, we examine the associations between PM levels and hypertension and hypothesize that higher PM concentrations are associated with elevated blood pressure.

Methods: A total of 133,935 adults aged ≥ 40 years who participated in the Korean Genome and Epidemiology Study were analyzed. Multiple linear regression analyses were conducted to investigate the short- (1–14 days), medium- (1 and 3 months), and long-term (1 and 2 years) impacts of PM on blood pressure. Logistic regression analyses were conducted to evaluate the medium- and long-term effects of PM on blood pressure elevation after adjusting for sex, age, body mass index, health-related lifestyle behaviors, and geographic areas.

Results: Using multiple linear regression analyses, both crude and adjusted models generated positive estimates, indicating an association with increased blood pressure, with all results being statistically significant, with the exception of PM levels over the long-term period (1 and 2 years) in non-hypertensive participants. In the logistic regression analyses on non-hypertensive participants, moderate PM_{10} (particulate matter with diameters < 10 μm) and $PM_{2.5}$ (particulate matter with diameters < 2.5 μm) levels over the long-term period and all high PM_{10} and $PM_{2.5}$ levels were statistically significant after adjusting for various covariates. Notably, high $PM_{2.5}$ levels of the 1 year exhibited the highest odds ratio of 1.23 (95% confidence interval: 1.19–1.28) after adjustment.

Conclusions: These findings suggest that both short- and long-term exposure to PM is associated with blood pressure elevation.

Keywords: Blood pressure; Hypertension; Outdoor air pollution; Particulate matter

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BACKGROUND

In recent decades, outdoor air pollution has emerged as a pressing global health concern,[1] with particulate matter (PM) representing a primary contributor.[2] The adverse effects of PM on respiratory health, particularly its association with diseases such as asthma and chronic obstructive pulmonary disease (COPD), have been extensively studied and well documented.[3] However, there remains a need to investigate potential links between PM and cardiovascular diseases, such as hypertension, which present a substantial public health burden worldwide.[4,5] More than two hundred million disability-adjusted life years (DALYs) were lost and ten million people died due to hypertension worldwide in 2021.[6]

Hypertension, commonly known as high blood pressure (BP), is a complex and multifactorial cardiovascular disorder affecting millions of individuals globally.[7] Hypertension is a well-established risk factor for various cardiovascular diseases, including coronary artery disease,[8] stroke,[9] and heart failure.[10] Despite advances in medical science, the pathophysiology underlying essential hypertension remains incompletely understood. Apart from traditional risk factors, such as age, genetics, diet, and physical inactivity, emerging evidence suggests that environmental factors, including PM, may significantly contribute to the development and progression of hypertension.[1113]

PM, a diverse mixture of solid and liquid particles suspended in air, varies in size and composition based on its source, atmospheric conditions, and geographic location.[14] Fine particular matter ($PM_{2.5}$) and coarse particular matter (PM_{10}), which are less than 2.5 μm and 10 μm in diameter, respectively,[15] are of particular concern owing to their ability to deeply penetrate the respiratory system and possibly cross into the circulatory system, triggering a cascade of physiological responses.[16] $PM_{2.5}$ and PM_{10} particles originate from both natural sources, such as dust storms and wildfires, and anthropogenic sources, including industrial emissions, vehicle exhaust, and residential heating.[17] The ubiquity of these sources makes PM exposure almost inevitable, rendering understanding its potential health effects of paramount importance.[18]

While research on the effects of PM on respiratory health has flourished, the link between PM and hypertension remains an area that requires more thorough investigation, as it holds the potential to considerably enhance our understanding of the health risks associated with air pollution and its broader implications on public well-being.[19] Moreover, there have been limited studies on the health effects of PM on individuals who reside in Korea, underscoring the need for additional research.

As with previous studies,[20,21] we categorized the influence of PM into acute and chronic effects to better elucidate the potential relationships between ambient PM and hypertension. We hypothesized that exposure to PM is associated with blood pressure and that long-term exposure to high PM concentrations would increase the odds of hypertension among people not previously diagnosed as hypertension by a doctor.

When we explored the long-term effect of PM in particular, we performed a classification based on the on-site BP after excluding subjects diagnosed with hypertension because the time of diagnosis is unknown. Instead, we made the operational definition of hypertensive subjects as "someone whose on-site BP is over the reference value and never diagnosed with hypertension." In this study, we retrieved data from the Korean Genome and Epidemiology

Study (KoGES)[22] and analyzed both urban and rural residents in Korea to examine the association between PM levels and hypertension.

METHODS

Study population

The KoGES, which was initiated in 2001, is a multicenter cohort project conducted by the National Institutes of Health of the Korea Disease Control and Prevention Agency to identify risk factors for chronic diseases common to Koreans and to provide a scientific basis for implementing personalized preventive medicine.[23] The KoGES consists of six prospective cohort studies whose study designs and survey methods have been previously described elsewhere.[22] This cross-sectional study was based on data from the KoGES integrated with estimated air pollution data.[24]

The inclusion criteria for the survey included enrollment in the population-based cohort of the KoGES, age ≥ 40 years at baseline, and having visited health screening centers or hospitals participating in this study, which are located in metropolitan areas of large cities or rural areas where most people are engaged in agriculture. The participants underwent a series of anthropometric measurements and clinical examinations (e.g., blood and urine analyses) and completed a self-administered survey that collected sociodemographic information, lifestyle factors, and disease history.

Participant selection

In total, 211,562 people participated in the baseline KoGES. For air pollution data that have been available since 2005, we used baseline data from the Health Examinee cohort and Cardiovascular Disease Association cohort (2005–2013). The community-based cohort of the Ansung and Ansan areas in the Gyeonggi province (n = 10,030) was excluded from this study because its baseline recruitment was conducted beforehand.

Among 201,532 participants, those without on-site BP levels (n = 1,162) or average PM levels over the previous 2 years (n = 63,442) were excluded. After further exclusion of those with missing covariate values such as body mass index (BMI), health-related lifestyle behaviors, and self-reported physician-diagnosed diseases, the final study population included 133,935 participants (**Fig. 1**).

Primary variables

We used data on short- and long-term exposures to PM from the KoGES cohort, in which the daily average meteorological and air quality data for up to 3 years following examination were matched to the participant's residential address. However, due to the large number of missing data points from 3 years prior, only data up to 2 years were used. The levels of PM exposure were determined using the Community Multi-scale Air Quality (CMAQ) model, which relies on a chemical transport model. After quantifying the $PM_{2.5}$ and PM_{10} concentrations every 1 km^2 (1 km × 1 km) and meteorological data every 81 km^2 (9 km × 9 km), air quality data were generated using a geocoding method, where the local exposure data were tabulated by regressing the gridded data as weighted sums based on city, county, and district borders.[25] Previous studies have elaborately explained the procedures involved in air quality and meteorological data generation and correlation.[26]

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┌──────────────────────────┐
│   All KoGES participants │
│     (n = 211,562)        │
└──────────────────────────┘
              │
              │────────────────────────►┌──────────────────────────────────┐
              │                         │ Excluded:                          │
              │                         │ - Community-based cohort of        │
              │                         │   the Ansan and Anseong areas      │
              │                         │   (n = 10,030)                     │
              ▼                         └──────────────────────────────────┘
┌──────────────────────────┐
│ Participants who are from │
│ urban or rural parts of   │
│ Korea (n = 201,532)       │
└──────────────────────────┘
              │
              │────────────────────────►┌──────────────────────────────────┐
              │                         │ Excluded:                          │
              │                         │ - Without blood pressure data      │
              │                         │   (n = 1,162)                      │
              │                         │ - Those who have missing values    │
              │                         │   in any of the covariates         │
              │                         │   (n = 2,993)                      │
              │                         │ - Without particulate matter       │
              ▼                         │   levels data (n = 63,442)         │
┌──────────────────────────┐          └──────────────────────────────────┘
│     Study subjects       │
│     (n = 133,935)        │
└──────────────────────────┘
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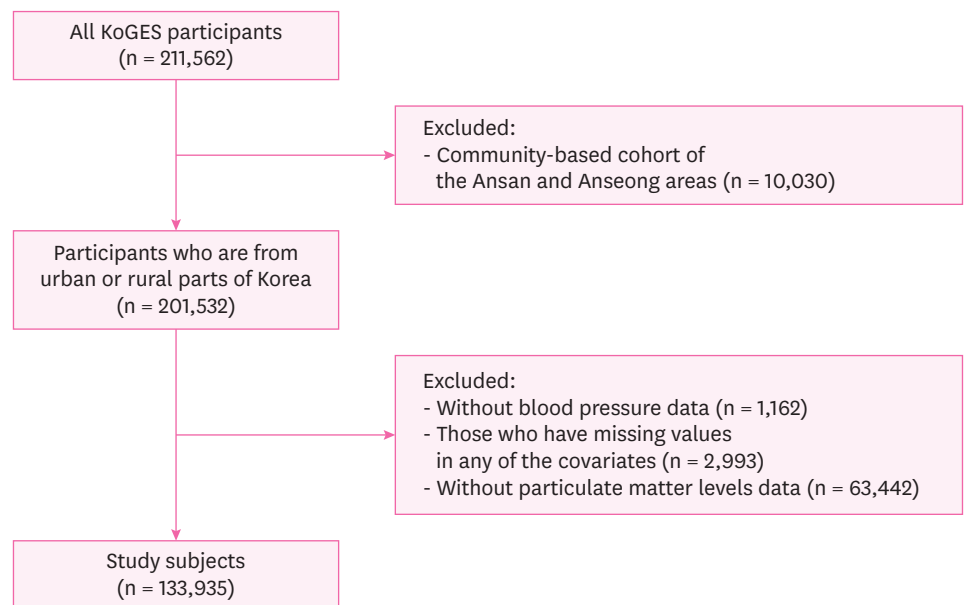
Fig. 1. Flow chart of the selection of study subjects.
KoGES: Korean Genome and Epidemiology Study.

Hypertensive patients were defined as follows: those who answered "Yes" to the question "Have you ever been diagnosed with high BP by a doctor?" and those whose on-site BP met the criteria for hypertension (systolic blood pressure [SBP] ≥ 140 mmHg or diastolic blood pressure [DBP] ≥ 90 mmHg). The protocol for measuring BP in the KoGES was explained as follows: After the participants had urinated and rested in a sitting position for at least 10 minutes, the BP was measured twice in both arms, with intervals ≥ 5 min, and the average BP was used for the analysis. Trained technicians measured the participant's BP in the brachial arteries using mercurial sphygmomanometers (Baumanometer-Standby; W.A. Baum Co., Inc., New York, NY, USA).[27]

Covariates

Potential covariates were identified by reviewing the relevant literature concerning the relationship between air pollution exposure and BP; these covariates included sex, age, BMI, family history of hypertension, smoking and drinking habits, physical activity level, and geographic location. If any parent or sibling had hypertension, the participant was classified as having a family history, and BMI was calculated using the participant's weight in kilogram and height in squared height in meter (kg/m^2).

Items such as smoking, drinking, and exercise regularity were primarily evaluated using a self-reported questionnaire format. If participants were unable to complete the self-report questionnaires, trained interviewers asked a series of questions to acquire information regarding demographic characteristics, socioeconomic status, medical and family history, and health-related lifestyle behaviors. Three response categories were provided to determine the smoking and alcohol consumption statuses of the participants: 1) those who had never smoked or drank alcohol, 2) those who had quit smoking or drinking, and 3) those who continue to smoke or drink today. The geographical classification was divided into 2 categories: urban and rural.

Statistical analysis

The study participants were separated into hypertensive and non-hypertensive groups, and the ambient PM_{10} and $PM_{2.5}$ levels were collected before the study visit. Depending on the duration of PM exposure, we categorized short-, medium-, and long-term exposures as follows: 1) short-term exposure, ≤ 2 weeks; 2) medium-term exposure, 1 and 3 months; and 3) long-term exposure, 1 and 2 years. Multiple linear regression analyses were conducted to evaluate the association between short-, medium-, and long-term exposures to PM and the on-site mean arterial pressure (MAP) of the participants. The MAP is the average BP in an individual during a single cardiac cycle calculated using the following formula: MAP = SBP × 2/3 + DBP × 1/3.

The study subjects were divided into three groups based on the tertial values of the PM concentrations, and logistic regression analyses were conducted to assess the association between medium- and long-term (≥ 1 month) exposures to PM and the elevation of BP in participants who had never been diagnosed with hypertension. The odds of hypertension among the group with moderate and high concentrations were compared to those of the low-concentration group.

Both regression models were adjusted for the covariates of sex, age, BMI, family history, smoking status, alcohol consumption, exercise regularity, and geographic area. A 2-tailed p-value of < 0.005 was considered statistically significant because of the large number of subjects.[28] All statistical analyses were performed using SAS statistical software version 9.4 (SAS Institute, Cary, NC, USA).

Ethics statement

The present study protocol was reviewed and approved by the Institutional Review Board (IRB) of Korea University Medical Center (IRB No. 2023AS0126).

RESULTS

The general characteristics of the study population are described in **Table 1**. Among the 133,935 study subjects, 47,237 were male (35.3%) and 86,698 were female (64.7%). The number of participants who had ever been diagnosed with hypertension or whose BP satisfied the criteria for hypertension was 44,936 (33.6%). By age group, 34,525 (34.1%) were in their 40s, 51,936 (38.8%) were in their 50s, and 36,373 (27.2%) were ≥ 60 years.

To compare the distributions of characteristics among different groups, the χ^2 test was used for categorical variables, including sex, family history, smoking and alcohol consumption statuses, exercise regularity, and geographic location. For ordinal variables, such as age groups and BMI, the Cochran-Armitage trend test was used to assess trends across the groups. Significant differences were observed in all characteristics among the groups.

Tables 2 and **3** show the geometric mean (GM) and 95% confidence interval (CI) of the PM concentrations for different categories of potentially confounding characteristics. To assess whether there were significant differences in PM concentrations between the subgroups, the concentrations within each characteristic were compared using Student's t-test or analysis of variance (ANOVA).

Table 1. General characteristics of the subjects

Characteristics	Category	All (n = 133,935)	With HTN (n = 44,936)	Without HTN (n = 88,999)	p-value
Sex	Male	47,237 (35.3)	17,728 (39.5)	29,509 (33.2)	< 0.001[a]***
	Female	86,698 (64.7)	27,208 (60.5)	59,490 (66.8)	
Age (years)	40–49	45,626 (34.1)	9,619 (21.4)	36,007 (40.5)	< 0.001[b]***
	50–59	51,936 (38.8)	17,405 (38.7)	34,531 (38.8)	
	≥ 60	36,373 (27.2)	17,912 (39.9)	18,461 (20.7)	
BMI (kg/m^2)	≤ 23	52,801 (39.4)	13,272 (29.5)	39,529 (44.4)	< 0.001[b]***
	23–25	36,760 (27.4)	11,907 (26.5)	24,853 (27.9)	
	≥ 25	44,374 (33.1)	19,757 (44.0)	24,617 (27.7)	
Family history	Yes	94,986 (70.9)	28,406 (63.2)	66,580 (74.8)	< 0.001[a]***
	No	38,949 (29.1)	16,530 (36.8)	22,419 (25.2)	
Smoking	Never	96,447 (72.0)	31,254 (69.6)	65,193 (73.3)	< 0.001[a]***
	Ex-smoker	20,367 (15.2)	8,178 (18.2)	12,189 (13.7)	
	Current	17,121 (12.8)	5,504 (12.2)	11,617 (13.1)	
Drinking	Never	68,157 (50.9)	22,734 (50.6)	45,423 (51.0)	< 0.001[a]***
	Ex-drinker	5,247 (3.9)	2,070 (4.6)	3,177 (3.6)	
	Current	60,531 (45.2)	20,132 (44.8)	40,399 (45.4)	
Exercise	Yes	65,677 (49.0)	21,544 (47.9)	44,133 (49.6)	< 0.001[a]***
	No	68,258 (51.0)	23,392 (52.1)	44,866 (50.4)	
Geographical area	Urban	123,827 (92.5)	40,355 (89.8)	83,472 (93.8)	< 0.001[a]***
	Rural	10,108 (7.5)	4,581 (10.2)	5,527 (6.2)	

Values are presented as number (%)
HTN: hypertension; BMI: body mass index.
[a]Analyzed by χ² test. [b]Analyzed by Cochran-Armitage trend test.
***$p < 0.001$.

Table 4 displays the outcomes of multiple linear regression analyses that assessed the relationship between PM concentration and MAP, classified by medical history of hypertension. In the hypertensive group, both crude and adjusted regressions revealed positive estimates toward increasing MAP, and all were statistically significant. In contrast, in the non-hypertension group, 1- and 2-year PMs were not statistically significant. The greatest increases in short-term periods in the hypertension group were observed in 2-week average PMs: 10 μg/m³ increases in PM_{10} and $PM_{2.5}$ were associated with 0.56 mmHg (95% CI: 0.46–0.67) and 0.97 mmHg (95% CI: 0.76–1.18) increases in MAP, respectively. These results are plotted in **Fig. 2** and the linear assumption was evaluated visually. Furthermore, additional parallel multiple linear regression analyses categorized by current smoking status, yielded similar outcomes and are detailed in **Supplementary Table 1**.

Table 5 shows the results of both crude and adjusted logistic regression analyses comparing the moderate and high PM concentration groups with the low PM concentration group in association with hypertension risk. These analyses examined how average PM concentrations over a longer period, such as a month or more, influenced BP elevations in participants who had not been previously diagnosed with hypertension. To be more explicit, outcome variables were binary: one group who were classified into hypertension as their on-site BP was over the reference value (SBP ≥ 140 mmHg or DBP ≥ 90 mmHg) and another group who were not classified into hypertension, among participants who reported that they were not hypertensive.

Moderate PM_{10} levels over 1 and 2 years showed the same adjusted odds ratio (OR) of 1.10 (95% CI: 1.05–1.14) after adjusting for sex, age, BMI, family history, smoking status, alcohol consumption, exercise regularity, and geographic area. Moderate $PM_{2.5}$ levels over 1 and 2 years showed adjusted ORs of 1.06 (95% CI: 1.02–1.11) and 1.08 (95% CI: 1.04–1.12), respectively. No other period in the moderate PM level group was statistically significant. In contrast, all associations between high PM levels and the risk of hypertension were

Table 2. Levels of PM_{10} regarding characteristics of the subjects

Characteristics	Category	Geometric mean ($\mu g/m^3$)			
		The day	1 week	3 months	2 years
All subjects		45.18 (45.08–45.29)	46.91 (46.83–46.99)	48.95 (48.90–49.00)	52.81 (52.77–52.84)
Sex	Male	45.29 (45.11–45.47)	47.09 (46.95–47.22)	48.88 (48.79–48.97)	52.91 (52.86–52.97)
	Female	45.13 (45.00–45.26)	46.81 (46.71–46.91)	48.99 (48.92–49.06)	52.75 (52.70–52.79)
	p-value[a]	< 0.001***	< 0.001***	< 0.001***	< 0.001***
Age (years)	40–49	45.36 (45.18–45.54)	47.17 (47.03–47.31)	48.72 (48.63–48.81)	53.19 (53.14–53.25)
	50–59	45.18 (45.01–45.36)	46.82 (46.69–46.95)	48.88 (48.80–48.97)	52.70 (52.64–52.75)
	≥ 60	44.97 (44.77–45.17)	46.71 (46.56–46.87)	49.34 (49.24–49.44)	52.48 (52.41–52.54)
	p-value[b]	0.001**	< 0.001***	< 0.001***	< 0.001***
BMI	≤ 23	44.84 (44.68–45.01)	46.50 (46.38–46.63)	48.62 (48.54–48.71)	52.63 (52.58–52.68)
	23–25	45.12 (44.92–45.32)	46.82 (46.66–46.97)	48.98 (48.87–49.08)	52.77 (52.71–52.83)
	≥ 25	45.65 (45.46–45.84)	47.47 (47.33–47.62)	49.32 (49.23–49.41)	53.05 (52.99–53.10)
	p-value[b]	< 0.001***	< 0.001***	< 0.001***	< 0.001***
Family history	Yes	45.42 (45.22–45.62)	47.09 (46.94–47.24)	48.86 (48.76–48.96)	52.88 (52.82–52.95)
	No	45.09 (44.96–45.21)	46.84 (46.74–46.93)	48.99 (48.92–49.05)	52.77 (52.73–52.81)
	p-value[b]	< 0.001***	< 0.001***	< 0.001***	< 0.001***
Smoking	Never	45.18 (45.06–45.31)	46.87 (46.77–46.97)	49.00 (48.94–49.06)	52.80 (52.76–52.84)
	Ex-smoker	45.31 (45.04–45.59)	46.98 (46.77–47.18)	48.83 (48.70–48.97)	52.67 (52.59–52.76)
	Current	45.06 (44.76–45.36)	47.05 (46.82–47.28)	48.82 (48.67–48.97)	53.00 (52.90–53.09)
	p-value[b]	0.604	0.194	0.006**	< 0.001***
Drinking	Never	45.09 (44.94–45.23)	46.78 (46.67–46.90)	48.96 (48.89–49.04)	52.71 (52.66–52.76)
	Ex-smoker	45.14 (44.61–45.67)	47.25 (46.86–47.65)	49.31 (49.05–49.57)	52.65 (52.48–52.82)
	Current	45.30 (45.14–45.46)	47.02 (46.90–47.14)	48.90 (48.82–48.98)	52.93 (52.88–52.98)
	p-value[b]	0.236	0.035*	0.016*	< 0.001***
Exercise	Yes	45.51 (45.36–45.66)	47.08 (46.96–47.19)	49.04 (48.97–49.12)	52.93 (52.88–52.98)
	No	44.85 (44.69–45.00)	46.74 (46.62–46.85)	48.85 (48.78–48.93)	52.68 (52.63–52.73)
	p-value[b]	< 0.001***	< 0.001***	< 0.001***	< 0.001***
Geographical area	Urban	45.36 (45.25–45.47)	47.04 (46.96–47.13)	48.75 (48.70–48.81)	52.70 (52.66–52.73)
	Rural	43.14 (42.75–43.53)	45.30 (44.99–45.62)	51.41 (51.23–51.59)	54.18 (54.05–54.30)
	p-value[b]	< 0.001***	< 0.001***	< 0.001***	< 0.001***

Values are geometric mean of average PM concentration of each duration before the test day with 95% confidence interval.
PM_{10}: particulate matter with diameters < 10 μm; PM: particulate matter; BMI: body mass index.
[a]Analyzed by Student's t-test. [b]Analyzed by analysis of variance test.
*p < 0.05, **p < 0.01, ***p < 0.001.

statistically significant. In particular, a high $PM_{2.5}$ level over 1 year had the greatest adjusted OR of 1.23 (95% CI: 1.19–1.28).

DISCUSSION

In this study, we investigated the associations between PM levels and BP using data from the KoGES cohort. In both crude and adjusted multiple linear regression analyses, positive estimates indicating an association with increased BP were observed in all PM levels except for 1- and 2-year average PMs in non-hypertensive participants. In logistic regression analyses, the association between long-term exposure to PM and hypertensive BP among participants not previously diagnosed as hypertension was statistically significant. Specifically, participants with high PM concentrations exhibited a significantly higher hypertension risk than those with low PM concentrations, but this relationship was inconsistent among participants with moderate PM levels. To the best of our knowledge, this is the first epidemiological study to explore short- and long-term associations between ambient $PM_{2.5}$ and PM_{10} exposure and BP in middle-aged to senior Korean adults using KoGES data encompassing a relatively large cohort of 133,935 participants. The results of this study could be used as the rationale behind government policies that attempt to reduce

Table 3. Levels of $PM_{2.5}$ regarding characteristics of the subjects

Characteristics	Category	Geometric mean (µg/m³)			
		The day	1 week	3 months	2 years
All subjects		22.43 (22.37–22.48)	23.56 (23.52–23.60)	24.41 (24.39–24.44)	25.95 (25.94–25.97)
Sex	Male	22.43 (22.33–22.53)	23.58 (23.51–23.65)	24.33 (24.28–24.37)	25.91 (25.88–25.94)
	Female	22.43 (22.35–22.50)	23.55 (23.50–23.60)	24.46 (24.43–24.49)	25.98 (25.96–26.00)
	p-value[a]	< 0.001***	< 0.001***	< 0.001***	< 0.001***
Age (years)	40–49	22.41 (22.31–22.51)	23.59 (23.52–23.66)	24.25 (24.21–24.30)	26.00 (25.97–26.02)
	50–59	22.45 (22.36–22.55)	23.58 (23.51–23.65)	24.43 (24.39–24.47)	25.94 (25.91–25.96)
	≥ 60	22.41 (22.30–22.52)	23.50 (23.42–23.58)	24.58 (24.54–24.63)	25.92 (25.89–25.95)
	p-value[b]	0.661	0.031*	< 0.001***	< 0.001***
BMI	≤ 23	22.28 (22.19–22.37)	23.38 (23.31–23.44)	24.27 (24.23–24.31)	25.89 (25.86–25.91)
	23–25	22.37 (22.26–22.48)	23.50 (23.42–23.58)	24.40 (24.36–24.45)	25.92 (25.89–25.95)
	≥ 25	22.65 (22.54–22.75)	23.83 (23.76–23.90)	24.59 (24.54–24.63)	26.06 (26.03–26.09)
	p-value[b]	< 0.001***	< 0.001***	< 0.001***	< 0.001***
Family history	Yes	22.60 (22.49–22.71)	23.73 (23.65–23.81)	24.47 (24.42–24.51)	25.99 (25.96–26.02)
	No	22.36 (22.29–22.43)	23.49 (23.44–23.54)	24.39 (24.36–24.42)	25.94 (25.92–25.96)
	p-value[b]	< 0.001***	< 0.001***	< 0.001***	< 0.001***
Smoking	Never	22.43 (22.36–22.50)	23.55 (23.50–23.60)	24.44 (24.41–24.47)	25.98 (25.96–26.00)
	Ex-smoker	22.48 (22.33–22.63)	23.59 (23.48–23.70)	24.32 (24.26–24.39)	25.82 (25.78–25.87)
	Current	22.33 (22.17–22.50)	23.59 (23.47–23.71)	24.35 (24.29–24.42)	25.94 (25.90–25.99)
	p-value[b]	0.798	0.711	0.001**	< 0.001***
Drinking	Never	22.42 (22.34–22.50)	23.55 (23.49–23.61)	24.47 (24.43–24.50)	25.99 (25.97–26.02)
	Ex-smoker	22.27 (21.99–22.56)	23.62 (23.41–23.83)	24.39 (24.27–24.52)	25.86 (25.78–25.94)
	Current	22.45 (22.36–22.53)	23.57 (23.51–23.63)	24.35 (24.31–24.39)	25.92 (25.89–25.94)
	p-value[b]	0.202	0.986	< 0.001***	< 0.001***
Exercise	Yes	22.58 (22.50–22.66)	23.64 (23.58–23.69)	24.41 (24.38–24.45)	25.97 (25.95–25.99)
	No	22.26 (22.18–22.35)	23.48 (23.42–23.54)	24.41 (24.38–24.45)	25.94 (25.91–25.96)
	p-value[b]	< 0.001***	< 0.001***	< 0.001***	< 0.001***
Geographical area	Urban	22.49 (22.43–22.55)	23.61 (23.57–23.65)	24.25 (24.22–24.27)	25.87 (25.85–25.88)
	Rural	21.64 (21.44–21.85)	22.97 (22.82–23.13)	26.53 (26.45–26.62)	27.05 (26.99–27.11)
	p-value[b]	< 0.001***	< 0.001***	< 0.001***	< 0.001***

Values are geometric mean of average PM concentration of each duration before the test day with 95% confidence interval.
$PM_{2.5}$: particulate matter with diameters < 2.5 µm; PM: particulate matter; BMI: body mass index.
[a]Analyzed by Student's t-test. [b]Analyzed by analysis of variance test.
*$p < 0.05$, **$p < 0.01$, ***$p < 0.001$.

ambient PM levels and bring clinically significant benefits to Korea, both in short- and long-term periods.

Past research on the associations between PM and BP has resulted in inconsistent findings. This inconsistency can be attributed to various factors, including the participants' general characteristics, ambient PM exposure durations and levels, PM assessment methods, and other variables.[29] As a result, caution should be used when comparing the present findings with those of previous studies because some studies reported a connection with lower BP,[30] others with higher BP,[31-33] and some found no associations at all.[34] According to a recent meta-analysis, the medium- and long-term (≥ 1 month) effects of PM on BP did not indicate a significant increase in SBP, but the short-term (< 1 month) exposure to $PM_{2.5}$ indicated a significant 0.53 mmHg (95% CI: 0.26–0.80) increase in SBP per 10 µg/m³ increase.[35] Another meta-analysis demonstrated that long-term exposure to ambient PM was associated with increased SBP (PM_1, $PM_{2.5}$), DBP (PM_1, $PM_{2.5}$, PM_{10}), and hypertension risk (PM_1, $PM_{2.5}$, PM_{10}), but PM_{10} was not associated with SBP.[36]

This research found the magnitude of association for $PM_{2.5}$ to be more significant than that for PM_{10}, suggesting that $PM_{2.5}$ poses a greater risk to cardiovascular health than PM_{10}. The reason for this increased risk is due to the size of these particles; while PM_{10} tends to deposit

Table 4. Crude and adjusted association between PM concentrations and MAP in the hypertensive and non-hypertensive subjects

PM	Period	Crude		Adjusted[a]	
		β (95% CI)	p-value	β (95% CI)	p-value
With HTN					
PM$_{10}$	0 day	0.020 (0.013–0.026)	< 0.001***	0.019 (0.013–0.025)	< 0.001***
	2 days	0.029 (0.022–0.036)	< 0.001***	0.028 (0.021–0.035)	< 0.001***
	1 week	0.044 (0.035–0.053)	< 0.001***	0.042 (0.033–0.051)	< 0.001***
	2 weeks	0.059 (0.049–0.069)	< 0.001***	0.056 (0.046–0.067)	< 0.001***
	1 month	0.067 (0.055–0.079)	< 0.001***	0.064 (0.052–0.076)	< 0.001***
	3 months	0.036 (0.022–0.050)	< 0.001***	0.033 (0.019–0.047)	< 0.001***
	1 year	0.093 (0.070–0.116)	< 0.001***	0.084 (0.061–0.107)	< 0.001***
	2 years	0.108 (0.085–0.131)	< 0.001***	0.099 (0.076–0.122)	< 0.001***
PM$_{2.5}$	0 day	0.029 (0.017–0.041)	< 0.001***	0.028 (0.017–0.040)	< 0.001***
	2 days	0.045 (0.031–0.059)	< 0.001***	0.044 (0.030–0.058)	< 0.001***
	1 week	0.069 (0.051–0.087)	< 0.001***	0.066 (0.049–0.084)	< 0.001***
	2 weeks	0.101 (0.080–0.122)	< 0.001***	0.097 (0.076–0.118)	< 0.001***
	1 month	0.121 (0.096–0.146)	< 0.001***	0.114 (0.089–0.139)	< 0.001***
	3 months	0.053 (0.023–0.084)	< 0.001***	0.047 (0.016–0.077)	< 0.001***
	1 year	0.117 (0.071–0.162)	< 0.001***	0.107 (0.061–0.152)	< 0.001***
	2 years	0.164 (0.116–0.212)	< 0.001***	0.152 (0.104–0.200)	< 0.001***
Without HTN					
PM$_{10}$	0 day	0.013 (0.010–0.016)	< 0.001***	0.012 (0.009–0.015)	< 0.001***
	2 days	0.017 (0.013–0.020)	< 0.001***	0.015 (0.011–0.018)	< 0.001***
	1 week	0.025 (0.021–0.030)	< 0.001***	0.023 (0.019–0.027)	< 0.001***
	2 weeks	0.037 (0.031–0.042)	< 0.001***	0.032 (0.027–0.037)	< 0.001***
	1 month	0.046 (0.040–0.052)	< 0.001***	0.039 (0.033–0.045)	< 0.001***
	3 months	0.021 (0.014–0.028)	< 0.001***	0.011 (0.005–0.018)	< 0.001***
	1 year	−0.004 (−0.016–0.008)	0.509	−0.006 (−0.017–0.005)	0.250
	2 years	0.009 (−0.003–0.021)	0.125	0.008 (−0.003–0.019)	0.172
PM$_{2.5}$	0 day	0.024 (0.018–0.030)	< 0.001***	0.022 (0.016–0.027)	< 0.001***
	2 days	0.030 (0.023–0.038)	< 0.001***	0.026 (0.020–0.033)	< 0.001***
	1 week	0.048 (0.039–0.057)	< 0.001***	0.043 (0.035–0.051)	< 0.001***
	2 weeks	0.070 (0.060–0.081)	< 0.001***	0.062 (0.052–0.072)	< 0.001***
	1 month	0.092 (0.079–0.104)	< 0.001***	0.079 (0.067–0.091)	< 0.001***
	3 months	0.038 (0.022–0.053)	< 0.001***	0.019 (0.005–0.034)	< 0.001***
	1 year	−0.003 (−0.026–0.021)	0.823	−0.009 (−0.031–0.013)	0.404
	2 years	0.034 (0.009–0.058)	0.007**	0.026 (0.003–0.049)	0.028*

Analyzed by multiple linear regression model.
PM: particular matter; MAP: mean arterial blood pressure; CI: confidence interval; HTN: hypertension; PM$_{10}$: particulate matter with diameters < 10 μm; PM$_{2.5}$: particulate matter with diameters < 2.5 μm.
[a]Model was adjusted for sex, age, body mass index, family history, smoking, drinking, exercise, and geographical area.
*p < 0.05, **p < 0.01, ***p < 0.001.

in the upper airways and cause allergenic and irritation responses, PM$_{2.5}$ is small enough to enter the terminal bronchioles, penetrate the alveolar–capillary barrier, and spread to other organ systems. The farther these particles travel through blood vessels, the greater the likelihood that they will cause systemic inflammation and increase an individual's BP. Similarly, other observational studies that have examined both PMs have reported greater effects of PM$_{2.5}$ than PM$_{10}$.[37-39]

The pathophysiological mechanism of the health effects of atmospheric PM on the cardiovascular system remains to be identified. Among previously published papers, several studies have suggested that certain factors, including oxidative stress,[40] systemic inflammatory response,[41] and autonomic nervous system disturbance,[42] could play significant roles. For instance, Jiang et al.,[41] conducted a study of 371 adults in metropolitan Shanghai, China, and found that a group of participants living within 50 m of a major road were exposed to levels of PM$_{2.5}$ that were 1.6-times higher than those of another group of participants who lived more than 200 m away from the major road; this resulted in an 8.4-fold increase in
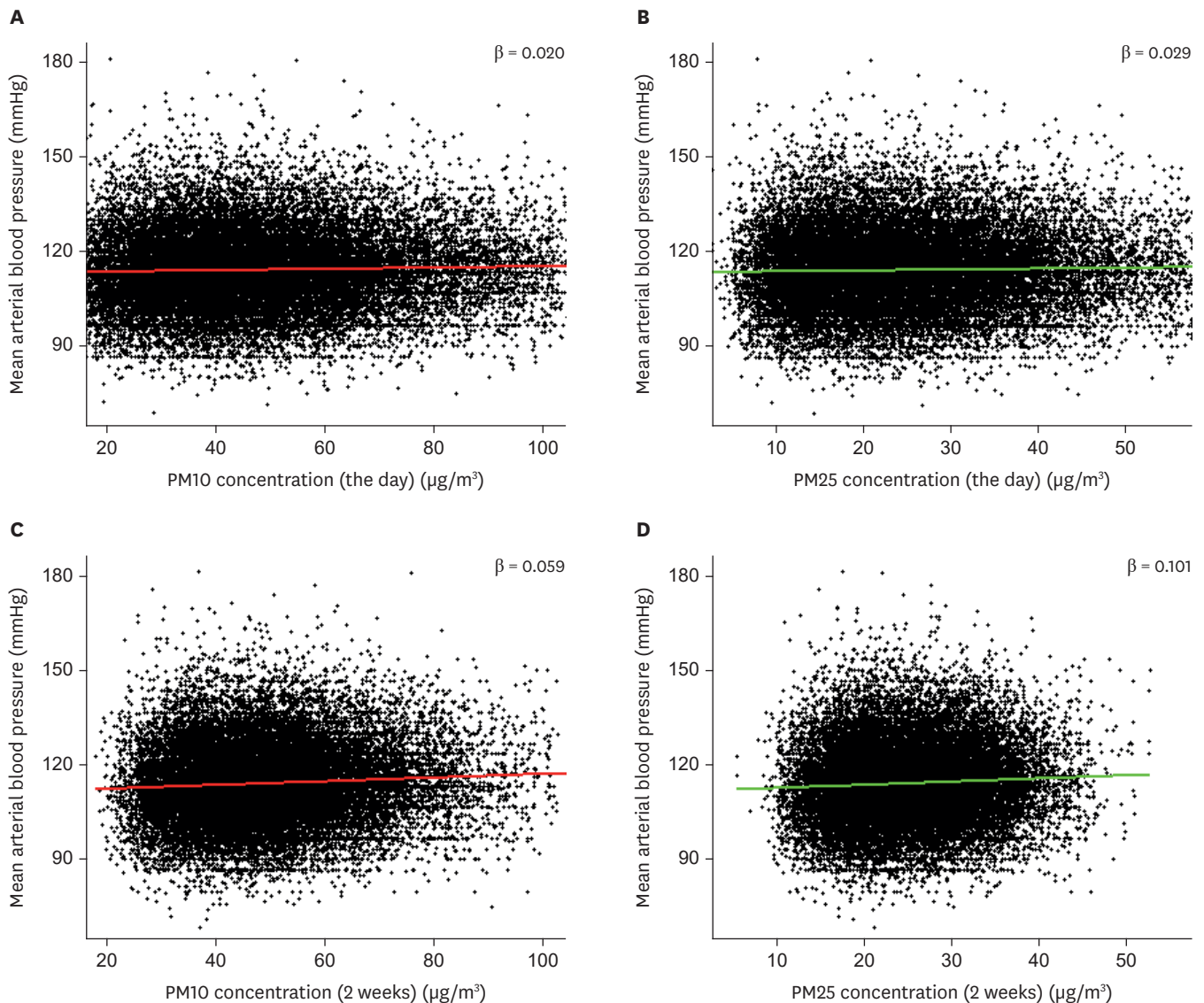
Fig. 2. Association between PM concentrations and MAP by scatter plot. Regression lines are displayed. (A, B) Average PM$_{10}$ and PM$_{2.5}$ levels of the health examination day and MAP. (C, D) Average PM$_{10}$ and PM$_{2.5}$ levels for 2 weeks and MAP.
PM: particulate matter; MAP: mean arterial pressure; M$_{10}$: particulate matter with diameters < 10 µm; PM$_{2.5}$: particulate matter with diameters < 2.5 µm.

the levels of interleukin-6, a 1.6-fold increase in SBP, and a 1.9-fold increase in DBP levels. Zhang et al.,[42] conducted a randomized trial of 56 adults who stayed at either a main road or a park and observed that the participants who were assigned to the main road were exposed to PM$_{2.5}$ concentrations that were 24% higher and showed increased BP and decreased heart rate variability, implying disturbances of the autonomic nervous system. All of these types of insidious damage can accumulate over time, increasing cardiovascular disease risk and resulting in premature death.

As a cross-sectional study using national cohort data, this research had certain limitations that should be acknowledged. First, while a statistically significant association between PM exposure and BP was observed, a definitive cause-and-effect relationship was not established, nor was the underlying pathophysiological mechanism responsible for the observed

Table 5. Crude and adjusted ORs for hypertension of subjects without prior diagnosis in moderate/high PM level groups

PM	Period	Crude		Adjusted[a]	
		OR (95% CI)	p-value	OR (95% CI)	p-value
Moderate					
PM$_{10}$	1 month	1.02 (0.98–1.06)	0.275	1.02 (0.98–1.06)	0.357
	3 months	1.03 (0.99–1.07)	0.212	0.99 (0.95–1.03)	0.483
	1 year	1.10 (1.06–1.14)	< 0.001***	1.10 (1.05–1.14)	< 0.001***
	2 years	1.09 (1.05–1.14)	< 0.001***	1.10 (1.05–1.14)	< 0.001***
PM$_{2.5}$	1 month	1.01 (0.98–1.06)	0.466	1.01 (0.97–1.05)	0.502
	3 months	1.02 (0.98–1.06)	0.464	0.99 (0.95–1.03)	0.488
	1 year	1.07 (1.03–1.12)	< 0.001***	1.06 (1.02–1.11)	0.004**
	2 years	1.08 (1.04–1.13)	< 0.001***	1.08 (1.04–1.12)	< 0.001***
High					
PM$_{10}$	1 month	1.07 (1.03–1.11)	0.001**	1.07 (1.03–1.11)	0.001**
	3 months	1.10 (1.06–1.15)	< 0.001***	1.07 (1.03–1.11)	0.001**
	1 year	1.21 (1.17–1.26)	< 0.001***	1.20 (1.16–1.25)	< 0.001***
	2 years	1.20 (1.15–1.25)	< 0.001***	1.20 (1.16–1.25)	< 0.001***
PM$_{2.5}$	1 month	1.08 (1.03–1.12)	< 0.001***	1.07 (1.03–1.11)	< 0.001***
	3 months	1.13 (1.08–1.17)	< 0.001***	1.08 (1.04–1.12)	< 0.001***
	1 year	1.28 (1.23–1.33)	< 0.001***	1.23 (1.19–1.28)	< 0.001***
	2 years	1.26 (1.21–1.31)	< 0.001***	1.22 (1.17–1.27)	< 0.001***

Analyzed by multiple logistic regression model. Groups were determined by tertile of each metabolite (low, moderate, and high); Group with the lowest tertile set as reference group.

PM: particular matter; OR: odds ratio CI: confidence interval; PM$_{10}$: particulate matter with diameters < 10 μm; PM$_{2.5}$: particulate matter with diameters < 2.5 μm.

[a]Model was adjusted for sex, age, body mass index, family history, smoking, drinking, exercise, and geographical area.

**p < 0.01, ***p < 0.001.

increase in BP identified. Therefore, further research is needed to elucidate the cause of this association. Second, in this study, one of the primary variables and several covariates were assessed through self-reported questionnaires. This introduces the possibility of recall bias, where participants may not accurately remember or report certain details, potentially impacting the accuracy of the findings. Third, an important aspect of the study was the lack of information on participant medication usage. This omission may have influenced the results, as certain medications can affect BP and may have confounded the observed association with PM exposure. Lastly, despite our efforts to account for various factors using comprehensive covariate adjustments in the statistical model, there remains a possibility of unidentified and unmeasured confounders that could have influenced the results.

Notably, the division of participants into three tertial groups by PM exposure created misclassification because the life patterns of individual participants were incompletely considered. Moreover, we used only ambient PM exposure levels based on the geographic location of participants' addresses; therefore, indoor PM levels, sources of indoor PM emissions (e.g., heating and cooking), and occupational factors were not considered. According to previous studies, indoor PM levels are affected by outdoor PM levels due to the exchange of air between the 2 environments. However, indoor PM levels can exceed outdoor PM levels, and individual PM exposure is highly dependent on the indoor air environment if people spend most of their time indoors.[25] Therefore, in future research, individual particle exposure monitors should be used to assess both outdoor and indoor PM levels.

Despite its limitations, this study also has several strengths. First, it is based on a large population study from the KoGES cohort dataset, which is representative of the entire Korean population. The participants demonstrated spatial diversity, as evidenced by their residency throughout the country. Second, by integrating air quality data with individual participant addresses, the ambient air quality prediction model generated more precise estimates of

individual exposure to ambient air pollution. This advancement enables the extension of air pollution research beyond individuals living in cities or metropolitan areas to those living in rural areas that have limited air quality monitoring stations. Third, diverse individual-level information, such as lifestyle factors, was available, which enabled adjustment for an extensive list of covariates during the statistical analysis. Moreover, despite the inherent limitations owing to the cross-sectional design, it is worth noting that the assessment of PM exposure for participants occurred before the health examination date, implying a potential temporal causality in this research. Last but not least, only a limited number of studies have attempted to investigate the link between PM and BP, which have shown inconsistent results. In our research, we further explored the conflicting relationships between PM and BP to improve understanding of this subject.

In this study, $PM_{2.5}$ and PM_{10} were used as indicators of ambient air pollution exposure because they are regularly monitored and aligned with international standards. Nevertheless, the latest research on the effects of air pollution is focused on even smaller particles, such as PM_1 or $PM_{0.1}$, given that they are considered to have even more detrimental effects than $PM_{2.5}$ and PM_{10} due to their size. As some of the results obtained in this study may have resulted from the effects of PM_1 or $PM_{0.1}$, it is essential to conduct future investigations that consider smaller PM to examine their potential association with BP. Prospective cohort studies are also required to investigate the incidence rates of various diseases induced by PM.

CONCLUSIONS

This study was the first to analyze data from the KoGES integrated with Korea-wide ambient air monitoring data and to examine the association between PM exposure and elevated BP. The results indicate notable linear associations between PM levels and BP, suggesting that high PM levels may be associated with hypertension risk. This finding concurs with existing research showing that fine PM has a detrimental effect on the cardiovascular system. More extensive samples are necessary for future investigations, and longitudinal studies should be conducted to more accurately evaluate the effects of PM on health.

ACKNOWLEDGEMENTS

SUPPLEMENTARY MATERIAL

Supplementary Table 1
Crude and adjusted association between PM concentrations and MAP in the smoking and non-smoking subjects

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