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Components of particulate matter as potential risk factors for acute myocardial infarction



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Abstract

Background Particulate matter with an aerodynamic diameter of $\leq 2.5 \mu\text{m}$ ($\text{PM}_{2.5}$) is a heterogeneous mixture, and specific substances that affect cardiovascular events remain unknown. We aimed to examine the association of short-term exposure to $\text{PM}_{2.5}$ and its components with hospital admissions for acute myocardial infarction (AMI).

Methods The concentrations of total $\text{PM}_{2.5}$ and its individual components were continuously measured using Aerosol Chemical Speciation Analysers. From a national-scale administrative database collected from 828 facilities in 47 prefectures across Japan from April 2017 to December 2019, we extracted AMI data for seven prefectures where these aerosol analysers were installed. The primary outcome was the relationship of $\text{PM}_{2.5}$ and its components with AMI hospitalisation. A time-stratified case-crossover analysis was conducted, and the approximate risk of AMI by pollutant concentrations was estimated using a conditional logistic regression model. In total, 44,232 patients with AMI aged 40–104 years (74.9% male) were examined.

Results The estimated effect of an increase in the total $\text{PM}_{2.5}$ concentration is significantly associated with AMI-related hospitalisation. Upon further examination of the components of $\text{PM}_{2.5}$, black carbon has a more substantial influence on AMI development than water-soluble organic compounds, nitrate, and sulphate ions.

Conclusions Short-term exposure to $\text{PM}_{2.5}$ is associated with an increased incidence of AMI. Future research prioritises the components of $\text{PM}_{2.5}$, with particular focus on whether a decrease in black carbon concentrations can mitigate the risk of future air quality-related AMI.

Plain language summary

This study examined whether short-term exposure to fine air particles, known as $\text{PM}_{2.5}$, increases the risk of heart attacks. $\text{PM}_{2.5}$ consists of tiny substances that can enter the lungs and bloodstream. Researchers analysed over 44,000 heart attack cases in seven regions of Japan between 2017 and 2019. They found that higher $\text{PM}_{2.5}$ levels shortly before each case were linked to more hospital visits for heart problems. Among the components of $\text{PM}_{2.5}$, black carbon—produced by car exhaust, factories, and smoke—showed the strongest link to heart attacks. The findings suggest that reducing black carbon in the air may help lower the risk of heart attacks. Cleaner air could lead to better heart health and benefit public well-being.

The World Health Organization (WHO) data indicate that almost the entire global population (99%) is exposed to air containing high levels of pollutants, exceeding the limits set in the WHO guidelines. This widespread exposure significantly contributes to the global disease burden, posing substantial health challenges worldwide¹. Exposure to air pollution is estimated to result in millions of deaths and lead to significant years of healthy life lost annually, with air pollution ranking as the fourth leading risk factor for death in both men and women².

Air pollution increases the incidence of cardiovascular and respiratory diseases, as well as neurological diseases such as stroke, dementia, Alzheimer's disease, and Parkinson's disease. Air pollution is now recognised as the greatest environmental threat to human health^{3,4}. The concentration of particulate matter (PM) with an aerodynamic diameter of $\leq 2.5 \mu\text{m}$ ($\text{PM}_{2.5}$) has been associated with the risk of developing myocardial infarction^{5,6}. $\text{PM}_{2.5}$ exposure has been hypothesised to lead to the development of cardiovascular disease through various potential mechanisms, including the

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generation of inflammation and oxidative stress within the lungs, influx of particulate matter into the circulation, alterations in the composition of the gut microbiota resulting from oral intake, and activation of neurohormonal pathways, which may lead to adrenal and autonomic imbalances⁷. However, the underlying pathogenesis of cardiovascular disease remains unknown due to the diversity and complexity of PM_{2.5} components, making it challenging to determine their exact effects and mechanisms. It is probable that not all components of PM_{2.5} are equally toxic to humans^{5,6}. Black carbon, which is one of the components of PM_{2.5}, represents a significant risk factor for cardiovascular disease and is strongly associated with mortality⁸. Therefore, we hypothesised that the identification of black carbon from PM_{2.5} as a risk factor for cardiovascular disease can play a crucial role in the development of future regulations for black carbon.

This study confirms the association between short-term exposure to PM_{2.5} and its components and hospitalisation due to acute myocardial infarction (AMI). Furthermore, we explore the individual components of PM_{2.5} and to demonstrate that black carbon can serve as an additional indicator of air quality management for AMI risk.

Methods

This retrospective observational study with a time-stratified case-cross-over design included multiple centres in Japan and was conducted using the Japanese Registry of all Cardiac and Vascular Diseases (JROAD) –Diagnostic Procedure Combination (DPC) database. As of 2019, the JROAD-DPC is a national survey of cardiovascular care in which a total of 828 Japanese Cardiovascular Society (JCS)-accredited hospitals participated. This database is a comprehensive resource for improving the quality of cardiovascular care⁹. The DPC system calculates the cost of inpatient care for acute care hospitals in Japan according to the International Classification of Diseases, 10th Revision diagnoses and medical treatment. The validity of the primary diagnosis, procedure records, and surgical records in the DPC database has been confirmed^{10,11}. This study was conducted in accordance with the Declaration of Helsinki and its subsequent amendments. This study was approved by the Ethics Committees at the National Institute for Environmental Studies (No. 2022-003), Toho University (No. A22035), and Sakurajyuji Hospital (No. 2022-01). As all data were anonymised by the participating institutions before collection by the data holder, i.e., the JCS, the requirement for individual informed consent was waived.

Air pollutants and environmental data

In April 2017, the Ministry of the Environment, Japan, initiated the deployment of the Aerosol Chemical Speciation Analyser (ACSA-14, Kimoto Electric Co., Ltd., Osaka, Japan) at 10 monitoring sites across Japan, aiming to enhance the PM_{2.5} monitoring system¹². This device performs continuous automatic measurement of the chemical and mass components of atmospheric aerosols separately for fine and coarse particles, enabling more stable measurements¹³. The concentrations of PM with an aerodynamic diameter of >2.5 and ≤10 µm (PM_{10–2.5}), along with PM_{2.5} concentrations and PM_{2.5} composition, were continuously measured at 1-h intervals. The PM_{2.5} components included optically measured black carbon (OBC), water-soluble organic compounds (WSOC), nitrate ion (NO₃[−]), and sulphate ion (SO₄^{2−}), all of which can contribute to PM_{2.5} countermeasures by enabling a detailed and rapid understanding of domestic sources and the adverse effects of transboundary pollution from the continent¹². The mass concentration of PM was determined using the beta-ray absorption method. The OBC mass concentration was measured optically using the near-infrared spectroscopy method. The mass concentrations of WSOC and NO₃[−] were determined using the ultraviolet photometric absorption method. The mass concentration of SO₄^{2−} was measured using the BaSO₄-based turbidimetric method with the addition of BaCl₂ dissolved in polyvinylpyrrolidone solution¹³. For the days that had ≥20 h or more measurements, we calculated daily mean concentrations. From the JROAD-DPC database collected from 828 facilities in 47 prefectures across Japan from April 2017 to December 2019, we extracted AMI data for seven

prefectures (Hokkaido, Niigata, Tokyo, Aichi, Osaka, Hyogo, and Fukuoka) where ACSA was installed. These sites were used to measure the concentrations of urban background total PM_{10–2.5}, total PM_{2.5}, and individual PM_{2.5} components. The remaining three sites situated in three distinct prefectures (Miyagi, Shimane, and Nagasaki) were not included in the present analysis. This was because each ACSA in these three sites is situated in a rural area, the mountains, or an island where people do not reside (ACSA was initially installed with the primary objective of monitoring transboundary pollution) (Supplementary Fig. 1).

We assigned prefecture-based concentrations to each patient because there was one monitoring site in each prefecture. Although exposure misclassification was inevitable, we used concentrations measured in populated areas where many patients with AMI resided. In our previous study, the daily mean concentrations of total PM_{2.5} measured at individual sites were highly correlated with those measured at other sites within the same prefecture¹⁴. For co-pollutants, including nitrogen dioxide (NO₂), sulphur dioxide (SO₂), and ozone (O₃), we obtained data on hourly concentrations that were measured at ambient air pollution monitoring stations located in each prefectural capital, separately from the ACSA measurements, from the atmospheric environment database of the National Institute for Environmental Studies in Japan. From hourly measurements, we estimated the daily mean concentrations of NO₂ and SO₂ and the daily maximum 8 h mean concentrations of O₃. Data published by the Japan Meteorological Agency were used to evaluate the daily mean temperature and relative humidity levels. Influenza incidence was referenced from the National Institute of Infectious Diseases database in Japan. An influenza epidemic period was defined as a week in which the number of recorded cases exceeded the 90th percentile of the distribution during the study period.

Study participants and participants' selection

Data on patients admitted with AMI at 828 JCS-accredited hospitals between April 1, 2017, and December 31, 2019 (prior to the onset of the coronavirus pandemic), were extracted from the JROAD-DPC database⁹. During the study period between April 2017 and December 2019, we extracted the data of 102,233 patients with AMI along with the International Classification of Disease, Tenth Revision (ICD-10) codes for AMI (I21-0, I21-1, I21-2, I21-3, I21-4, and I21-9) in the fields for the main diagnosis, admission-precipitating diagnosis, and/or most resource-consuming diagnosis from the JROAD-DPC database. We selected 101,124 patients aged ≥40 years and identified 52,157 patients admitted to hospitals located in seven prefectures (Hokkaido, Niigata, Tokyo, Aichi, Osaka, Hyogo, and Fukuoka) where Aerosol Chemical Speciation Analysers were installed. After excluding 3,166 patients without emergency hospitalisation, 4488 patients who had not undergone coronary angiography (procedure code [claim code]; D206-2 [170020710]) and/or re-vascularisation (procedure code [claim code]; K5461 [150374910], K5462 [150375010], K5463 [150375110], K547 [150260350], K5481 [150284310], K5482 [150359310], K5491 [150375210], K5492 [150375310], K5493 [150375410], K550 [160107550], K550-2 [150318310]), and 271 patients who were not assigned concentration of PM_{2.5} total mass and/or its components, a total of 44,232 patients with AMI (33,143 men and 11,089 women) were included in the present analysis (Supplementary Fig. 2).

Statistics and Reproducibility

Patient characteristics and variables are summarised using descriptive statistics, such as mean (standard deviation [SD]), median, quartiles, and interquartile range (IQR) for continuous variables and as frequencies and percentages for categorical variables. A time-stratified case-cross-over design was employed to estimate the association between short-term exposure to PM_{2.5} and admission of patients with AMI. The odds ratio (OR) and 95% confidence interval (CI) for IQR increase in PM_{2.5} concentrations were calculated using a conditional logistic regression model. The results were presented as %increase in AMI incidence, calculated as follows: [(OR − 1) × 100]. The ‘case’

Table 1 | Overall characteristics of patients with AMI between 1 April 2017 and 31 December 2019

Variable	n	%
Age, years	40–64	14,251
	65–74	12,989
	≥75	16,992
Male sex	33,143	74.9
BMI ≥ 25 kg/m ²	14,232	34.8
Smoking history	21,508	55.6
Hypertension	28,565	64.6
Diabetes	13,633	30.8
Hyperlipidaemia	29,111	65.8
Season	Warm (May to October)	23,972
	Cool (November to April)	20,260

AMI, acute myocardial infarction, BMI body mass index.

day was defined as the day of admission, and three or four ‘control’ days were selected from the same days of the week within the same year and month as the ‘case’ day. Time-variant factors, including ambient temperature at lag0-20, relative humidity at lag0-1, and influenza epidemics, were included in the model. For temperature, we modelled using a cross-basis function with a quadratic b-spline with internal knots at the 10, 75, and 90th percentiles of temperature distributions for the exposure–response relationship between temperature and AMI, and with a natural cubic spline for lag with 3 degrees of freedom for its lag–response relationship over the lag of 21 days^{15–17}. For humidity, we used a cubic spline function with 3 degrees of freedom. A single-component model was used to evaluate the association between individual PM_{2.5} components and hospital admission of patients with AMI, which was adjusted for PM_{10–2.5} concentrations. A multi-pollutant model was constructed, adjusted for PM_{10–2.5} and/or each co-pollutant one by one (NO₂, SO₂, and O₃). Furthermore, a multi-component model was constructed, mutually adjusted for concentrations of four PM_{2.5} components, to examine the potential confounding effects of different pollutants and PM_{2.5} components. For example, when calculating the point estimate for OBC, adjustments were made for WSOC, NO₃[−], and SO₄^{2−}.

From the JROAD-DPC database, we extracted information on the date of admission, the prefecture code of residence, age, sex, height, weight, Brinkman index (number of cigarettes per day multiplied by smoking year), and comorbidities (including hypertension, diabetes, and hyperlipidaemia). We categorised the hospitalisation season into warm (May to October) and cold (November to April). Analyses were stratified by age (40–64, 65–74, or ≥75 years), sex, the season of onset (warm [May to October] or cold [November to April]), body mass index (BMI) (<25 or ≥25 kg/m²), smoking history, hypertension, diabetes mellitus, and hyperlipidaemia. Smokers were defined as patients who had a Brinkman index >0. A likelihood ratio test was employed to assess the statistical associations between age, sex, season of onset, BMI, smoking history, hypertension, diabetes mellitus, and hyperlipidaemia with AMI. Pearson’s correlation coefficients were calculated between the daily mean concentrations of total PM_{2.5} and individual PM_{2.5} components. All analyses were conducted using STATA version 16.1 (Stata Corporation, College Station, TX, USA), and R version 4.0.0 (R Foundation for Statistical Computing, Vienna, Austria) with the *dlnm* package¹⁵. All tests were two-sided and performed at a 5% level of statistical significance.

Reporting summary

Further information on research design is available in the Nature Portfolio Reporting Summary linked to this article.

Table 2 | Summary statistics for environmental factors at lag0 from 1 April 2017 to 31 December 2019

Ambient pollutants	Percentile					
	Mean	SD	25	50	75	IQR
Total PM _{2.5} (µg/m ³)	11.5	6.7	6.8	10.0	14.7	7.88
PM _{2.5} components (µg/m ³)						
OBC	0.2	0.3	0.0	0.2	0.4	0.33
WSOC	0.7	0.7	0.3	0.6	0.9	0.60
NO ₃ [−]	1.0	1.2	0.4	0.6	1.1	0.70
SO ₄ ^{2−}	2.9	2.5	1.3	2.2	3.7	2.41
Total PM _{10–2.5} (µg/m ³)	12.7	9.4	6.8	10.5	15.8	8.95
SO ₂ (ppb)	1.5	1.4	0.6	1.1	2.0	1.46
NO ₂ (ppb)	11.4	8.2	5.1	9.3	15.8	10.70
O ₃ (ppb) ^a	42.0	14.1	32.9	40.7	50.1	17.25
Ambient temperature (°C)	16.6	8.6	9.6	17.4	23.3	13.70
Relative humidity (%)	70	13	61	71	79	18.00

IQR interquartile range NO₂ nitrogen dioxide, OBC optically measured black carbon, O₃ ozone, PM_{2.5} particulate matter with an aerodynamic diameter ≤2.5 µm, PM_{10–2.5} particulate matter concentrations with an aerodynamic diameter of >2.5 µm and ≤10 µm, SD standard deviation, SO₂ sulphur dioxide, WSOC water-soluble organic compounds.

^aDaily maximum 8-h mean concentrations.

Results

Patient characteristics at baseline

Of the 44,232 patients with AMI (mean [SD]: 69.6 [12.5] years), 14,251 were aged ≥75 years (38.4%). The study population comprised 74.9% male patients. The prevalence of hypertension, diabetes, and hyperlipidaemia was 64.6, 30.8, and 65.8%, respectively. Furthermore, 54.2% of the patients experienced AMI during the warm season, defined as the period between May and October (Table 1).

Total PM_{2.5} and PM_{2.5} components

The environmental factors during the study period are summarised in Table 2. The mean (SD) daily total PM_{2.5} and total PM_{10–2.5} concentrations were 11.5 (6.7) and 12.7 (9.4) µg/m³, respectively. The mean concentrations (SD) of PM_{2.5} components, including OBC, WSOC, NO₃[−], and SO₄^{2−}, were 0.2 (0.3), 0.7 (0.7), 1.0 (1.2), and 2.9 (2.5) µg/m³, respectively. The proportion of daily mean OBC concentrations in the total mass of PM_{2.5} concentrations was 3.1% (2.4%), with a minimum of 0.0% and a maximum of 27.7%. The remaining PM_{2.5} components, including WSOC, NO₃[−], and SO₄^{2−}, accounted for 6.6% (3.5%) (minimum 0.0%, maximum 35.7%), 8.9% (5.4%) (minimum 0.1%, maximum 49.4%), and 23.7% (10.9%) (minimum 0.1%, maximum 89.9%), respectively.

IQR increase in total PM_{2.5} and AMI hospitalisation

A clear health effect was observed for various lags in this study (Fig. 1). On examining the relationship between total PM_{2.5} and AMI hospitalisation, we observed that the estimated effects of %increase for an IQR increase in PM_{2.5} at lag0-1 were associated with a significantly higher incidence of AMI (% increase: 2.44, 95% CI: 0.85–4.07) following adjustment for ambient temperature, relative humidity, and influenza epidemics. The observed significance remained even after additional adjustment for PM_{10–2.5}, with a % increase of 2.22 (95% CI: 0.30–4.19).

The association between total PM_{2.5} exposure and AMI hospitalisation was sustained after adjustment for co-pollutants in addition to PM_{10–2.5} (Supplementary Table 1). Exposure to PM_{10–2.5} at lag0-1 was not associated with AMI hospitalisation (PM_{2.5} adjusted %increase for an IQR increase = 0.34; 95% CI: −1.37–2.08).

IQR increase in PM_{2.5} components and AMI hospitalisation

We examined the relationship between each PM_{2.5} component and AMI hospitalisation for various lags. Compared with WSOC, NO₃⁻, and SO₄²⁻, OBC at lag 0-1 was strongly associated with AMI hospitalisation, even after adjustment for ambient temperature, relative humidity, and influenza epidemics (%increase: 2.75, 95% CI: 0.96–4.57) (Fig. 2). This significant relationship remained unchanged in a multi-component model, with a % increase of 2.54 (95% CI: 0.00–5.14) (Supplementary Table 2).

Stratified analysis for the relationship between total PM_{2.5} and AMI hospitalisation

Supplementary Fig. 3 presents the stratified analysis data on the relationship between total PM_{2.5} and AMI hospitalisation at lag0-1. There were percentage increases associated with an IQR increase in PM_{2.5} levels for age ≥75 years (%increase: 3.45%; 95% CI: 0.30–6.70%), male sex (%increase: 2.59%; 95% CI: 0.36–4.87%), diabetes mellitus (%increase: 4.43%; 95% CI:

0.89–8.10%), smoking history (%increase: 2.82%; 95% CI: 0.07–5.65%), and warm season (%increase: 2.81%; 95% CI: 0.05–5.65%). Nevertheless, although all factors underwent effect modification, the results did not reach statistical significance.

Stratified analysis for the relationship between OBC and AMI hospitalisation

Regarding the relationship between OBC concentration and AMI hospitalisation at lag 0-1, similar results were obtained in stratified analysis as those obtained for PM_{2.5}. In particular, male sex (%increase: 3.27%; 95% CI: 0.31–6.31%), diabetes mellitus (%increase: 4.70%; 95% CI: 0.10–9.51%), smoking status (%increase: 3.76%; 95% CI: 0.10–7.56%), and warm season (%increase: 5.33%; 95% CI: 1.24–9.58%) were associated with an increased risk of AMI hospitalisation. However, no significant effect modification was observed (Supplementary Fig. 4).

Correlations between daily total PM_{2.5} and PM_{2.5} components

The correlations between the daily total PM_{2.5} mass and each component were examined. Positive correlations were evident; however, OBC exhibited the weakest correlation ($r = 0.58$) compared with WSOC ($r = 0.72$), SO₄²⁻ ($r = 0.71$), and NO₃⁻ ($r = 0.65$) (Supplementary Fig. 5).

Discussion

This study used a time-stratified case-crossover design to assess the association between short-term PM_{2.5} exposure and AMI admissions. The main findings highlighted a significant association between increased PM_{2.5} concentrations and increased incidence of AMI, with black carbon identified as a critical component of PM_{2.5} in assessing air quality-related cardiovascular risks. The present study makes a novel contribution to the field by focusing on the PM_{2.5} components and their relationship to AMI hospitalisation.

In seven of the 47 prefectures with continuous automatic analysers for measuring the air aerosol chemical composition in Japan, elevated PM_{2.5} concentrations were significantly associated with an increased risk of AMI hospitalisation. Furthermore, even after adjustment for PM_{2.5} components, OBC was still associated with AMI hospitalisation.

PM components are unlikely to all be equally involved in disease development or equally important in causing health effects¹⁸. Given the variability in the components of PM_{2.5} across seasons and regions¹⁹, these

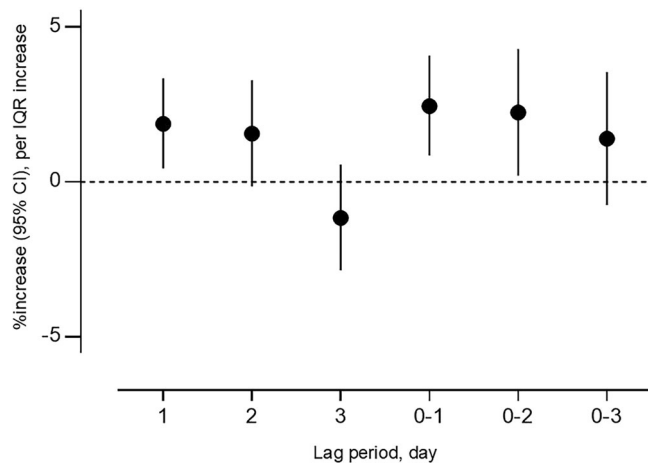
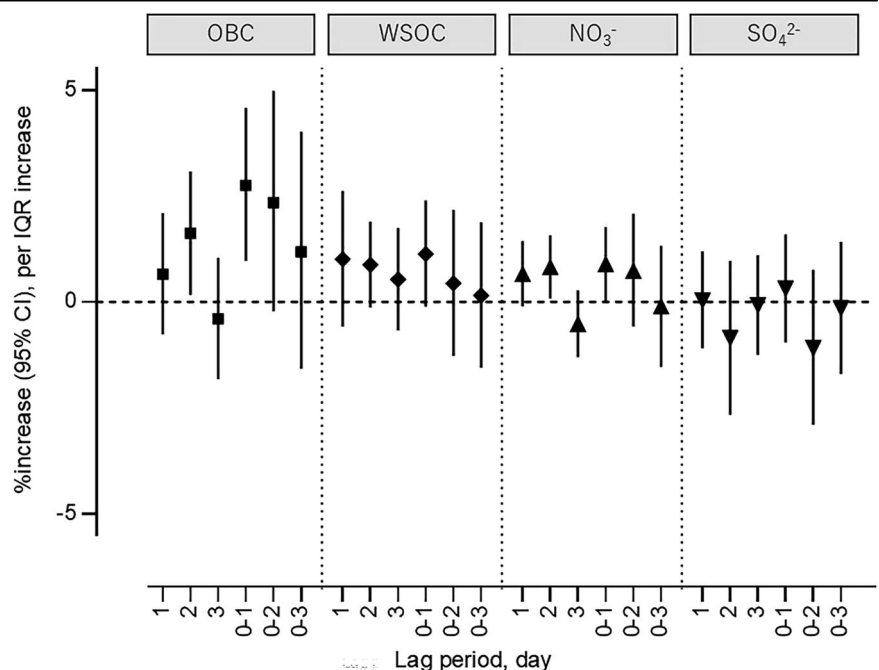


Fig. 1 | Estimated effect of %increase for AMI incidence in total PM_{2.5} for an IQR increase. Estimated effects of %increase for an IQR increase in PM_{2.5} at lag0-1 were associated with a significantly higher incidence of AMI following adjustment for ambient temperature, relative humidity, and influenza epidemics. The error bars in the forest plot show the 95% confidence intervals for $n = 44,232$.

Fig. 2 | Estimated effect of %increase for AMI incidence in individual PM_{2.5} components for an IQR increase. The relationship between each PM_{2.5} component (OBC, WSOC, NO₃⁻ and SO₄²⁻) and AMI hospitalisation for lag 1, lag 2, lag 3, lag 0-1, lag 0-2, and lag 0-3 after adjustment for ambient temperature, relative humidity, and incidence of the influenza epidemic was examined. Compared with WSOC, NO₃⁻, and SO₄²⁻, OBC at lag 0-1 was strongly associated with AMI hospitalisation, even after adjustment for ambient temperature, relative humidity, and influenza epidemics. The error bars in the forest plot show the 95% confidence intervals for $n = 44,232$.



components should be considered when investigating the health effects due to exposure of PM_{2.5}. This study suggests that black carbon, a component of PM_{2.5}, is associated with the risk of developing AMI. Black carbon is generated by incomplete combustion of fossil fuels and other materials and is a measure of sooty carbon in the air that is quantified optically^{4,20}. Combustion-related particles, such as black carbon, are more harmful to human health than PM components that are not produced by combustion²¹. Previous studies have demonstrated a significant association of elevated concentrations of black carbon with acute coronary syndrome development and cardiovascular mortality, given that black carbon was previously regarded as an independent pollutant, rather than as a component of PM_{2.5}^{22,23}. The present study raises the possibility that black carbon is an important factor in the development of AMI due to PM_{2.5}. To date, only few studies have examined the potential future consequences of exposure to black carbon in PM_{2.5}²⁴.

This epidemiological study revealed that identification of the causal relationship of specific PM with increased risk of developing AMI is challenging. Nevertheless, the results demonstrated a significant association of PM_{2.5} and black carbon emissions with the incidence of AMI. Consequently, reducing these emissions may help in decreasing the future incidence of AMI. Given that black carbon is also related to global warming^{25,26}, regulating emissions may also help in mitigating climate change.

This study has some limitations. First, similar to past epidemiologic environmental studies on the health effects of air pollution, the occurrence of exposure misclassification was inevitable, given that the results were based on pollutant data measured at a single monitoring station in each prefecture. It was difficult to estimate the bias effect of a combination of the Berkson error and classical error on our estimates of the association between PM_{2.5} and AMI risk²⁷. Second, as in all epidemiologic studies, the data integrity, validity, and ascertainment bias in this study were potential limitations. However, the uniform data collection, large sample size, and implementation of a population-based design that covered all known AMI in Japan may have minimised the effects of these potential sources of bias^{28,29}. Third, our previous investigation revealed a strong correlation between the daily average concentrations of total PM_{2.5} measured at monitoring stations in the same prefecture¹⁴. However, this assertion may not be applicable to PM_{2.5} components. Fourth, there are considerable interrelationships among atmospheric particle composition, especially among composition originating from the same source⁸. In addition to black carbon, PM_{2.5} contains various other known components, including aluminium, iron, potassium, sodium, Ni, sulphur, silicon, vanadium, and zinc³⁰. It is also possible that PM_{2.5} contains many unknown chemical compositions. However, the WHO regional office for Europe has indicated that black carbon represents a significant risk factor for cardiovascular disease and is strongly associated with mortality⁸. Although black carbon is not typically considered a direct cause of AMI, it is often regarded as an indicator of a wide variety of combustion-derived chemical components.

The study utilised a nationwide Japanese cardiovascular disease database to ascertain that black carbon, a component of PM_{2.5}, is a significant factor in AMI hospitalisation and may serve as a valuable indicator for future regulations and measures to reduce cardiovascular risk. Further detailed regional analyses, including differences between urban and rural areas and seasonal effects, could lead to more effective countermeasures in the future. Furthermore, a more comprehensive health risk assessment is warranted to evaluate not only the immediate but also the long-term health effects of exposure. Concrete data on the health effects of air pollution to policymakers can aid in the development of effective environmental policies. For the general public, the dissemination of such data can raise awareness about the health effects of air pollution, thereby increasing individual participation in preventive actions and environmental improvements. This study represents an important step toward understanding the link between air pollution and cardiovascular disease and could have a significant impact on future health measures and environmental policies. In addition to reducing the burning of fossil fuels and biomass, it may be important to implement measures to safeguard public health, such

as encouraging the use of masks and discouraging outdoor activities when PM_{2.5} concentrations are elevated.

In conclusion, short-term exposure to PM_{2.5} and its component black carbon particles is associated with an increased incidence of AMI. Although other components may be involved in the development of AMI, black carbon is a valuable indicator for assessing the AMI risk associated with air quality.

Data availability

The data that support the findings of this study were used under licence from the Japanese Circulation Society for the current study and are not publicly available. However, the data are available from the authors upon reasonable request and with permission from the Centre for the Japanese Registry of all Cardiac and Vascular Diseases (JROAD) at the Japanese Circulation Society (https://www.j-circ.or.jp/jittai_chosa/). The air pollution data utilised for this study are publicly available from the following websites: https://www.env.go.jp/air/osen/pm_resultmonitoring/post_25.html (the Ministry of the Environment) and <https://tenbou.nies.go.jp/download/> (National Institute for Environmental Studies). The air pollution dataset used in the present study can be available upon reasonable request for research purposes to Dr Takehiro Michikawa (takehiro.michikawa@med.toho-u.ac.jp). The numerical data (source data) for Fig. 1 are in Supplementary Data 1. The numerical data (source data) for Fig. 2 are in Supplementary Data 2. The numerical data (source data) for Supplementary Fig. 2 are in Supplementary Data 3. The numerical data (source data) for Supplementary Fig. 3 are in Supplementary Data 4. The numerical data (source data) for Supplementary Fig. 4 are in Supplementary Data 5. The numerical data (source data) for Supplementary Fig. 5 are in Supplementary Data 6.

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S.K.: study conceptualisation, methodology, formal analysis, investigation, data curation, writing - original draft, writing - review & editing, visualisation, supervision, project administration, and funding acquisition; T.M.: study conceptualisation, methodology, software, formal analysis, investigation, data curation, writing - review & editing, visualisation, project administration, and funding acquisition; A.Y.: study investigation, resources, and writing - review & editing; K.T.: study resources and writing - review & editing; T.I. contributed to resources and writing - review & editing; Y.N.: study investigation and writing - review & editing; A.T.: study conceptualisation, methodology, investigation, resources, and writing - review & editing.

Competing interests

The authors declare no competing interests.

Additional information

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