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Dezhong Chen, Yiyue Yin, Weiyi Chen, Ting Xiao, Kai Su, Yiqian Zeng, Xuan Wang, G. Neil Thomas and Xiangqian Lao*

*E-mail: xq.lao@cityu.edu.hk

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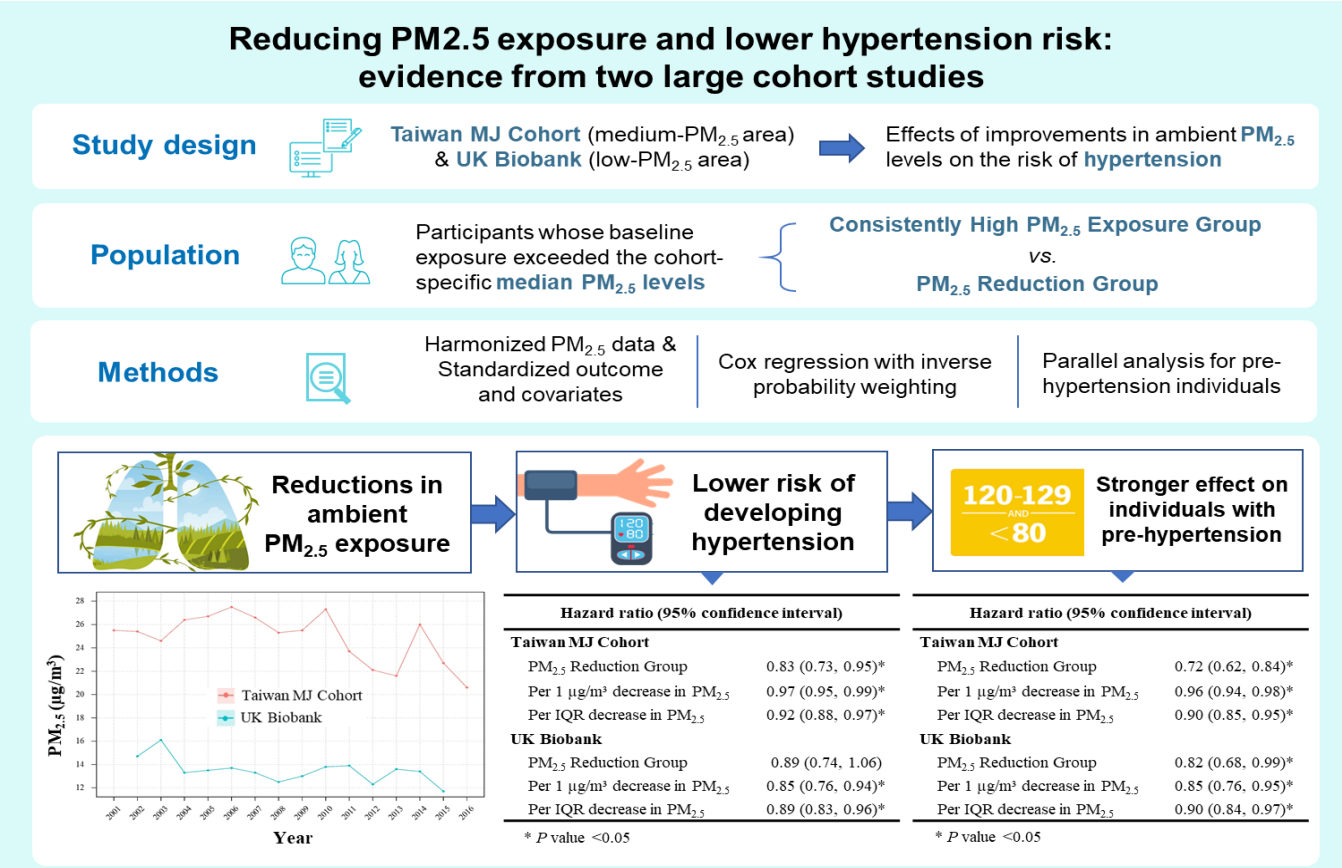
Graphical Abstract



Highlights:

- Reduced $PM_{2.5}$ exposure was associated with a lower risk of developing hypertension.
- This benefit was stronger among individuals with pre-hypertension in both cohorts.
- Findings are consistent in two cohorts representing varying air pollution levels.

Reducing $PM_{2.5}$ exposure and lower hypertension risk: evidence from two large cohort studies

Dezhong Chen[1], Yiyue Yin[1], Weiyi Chen[1], Ting Xiao[1], Kai Su[1], Yiqian Zeng[1], Xuan Wang[2], G. Neil Thomas[3] and Xiangqian Lao[1,*]

[1] Department of Biomedical Sciences, College of Biomedicine, City University of Hong Kong, Hong Kong, China

[2] China-UK Low Carbon College, Shanghai Jiao Tong University, Shanghai, China

[3] Department of Applied Health Sciences, University of Birmingham, Birmingham, UK

*E-mail: xq.lao@cityu.edu.hk.

Abstract: **Objective:** Although the adoption of air pollution mitigation strategies has greatly improved air quality over recent decades, their beneficial effects on health remain underexplored. We therefore investigated the association between reductions in $PM_{2.5}$ exposure and hypertension risk. **Methods:** This prospective cohort study utilized data from the Taiwan MJ Cohort (2005–2016) and the UK Biobank (UKB, 2006–2015), representing medium- and low-$PM_{2.5}$ areas, respectively. Ground-level $PM_{2.5}$ data were derived from the Atmospheric Composition Analysis Group and matched to each participant's residential address. Participants whose baseline exposure exceeded the cohort-specific median $PM_{2.5}$ level were included (MJ Cohort: n = 38,397; UKB: n = 66,837). Covariates and outcome definitions were standardized across cohorts, and Cox regression models with inverse probability weighting (IPW) were used to analyze the associations between $PM_{2.5}$ reduction and the 5-year risk of hypertension. A parallel analysis was performed among individuals with pre-hypertension. **Results:** In the MJ Cohort, participants who experienced a reduction in $PM_{2.5}$ exposure had a significantly lower risk of developing hypertension compared with those who continuously lived in high-$PM_{2.5}$ areas (hazard ratio [HR] = 0.83, 95% confidence interval [CI]: 0.73–0.95). Each 1 μg/m³ decrease in $PM_{2.5}$ exposure was also associated with a reduced risk of hypertension (HR = 0.97, 95% CI: 0.95–0.99). In the UKB, a significant association was observed only when $PM_{2.5}$ exposure was analyzed as a continuous variable (per 1 μg/m³ decrease: HR = 0.85; 95% CI: 0.76–0.94). Notably, the benefits of reduced $PM_{2.5}$ exposure were even more pronounced among individuals with pre-hypertension in both cohorts. **Conclusion:** Analysis of two large cohorts from regions with varying air pollution levels demonstrates that reductions in long-term $PM_{2.5}$ exposure are consistently associated with a lower risk of developing hypertension, providing direct evidence of the substantial health benefits of improved ambient $PM_{2.5}$ levels.

Keywords: hypertension; $PM_{2.5}$; air quality improvement; risk factor

1. Introduction

Hypertension is a major risk factor for cardiovascular disease and premature mortality worldwide. In 2019, an estimated 1.28 billion adults aged 30–79 years were affected, with the greatest burden observed in low- and middle-income countries [1]. Beyond established behavioral and metabolic determinants, growing evidence has identified ambient air pollution, particularly fine particulate matter with an aerodynamic diameter ≤ 2.5 μm ($PM_{2.5}$), as an important environmental contributor to elevated blood pressure and hypertension incidence [2–4]. A recent meta-analysis of 28 observational studies reported that each 10 μg/m³ higher long-term $PM_{2.5}$ exposure was associated with a 21% greater risk of incident hypertension and a 6% higher prevalence [3]. Additionally, $PM_{2.5}$ exposure has also been linked to dyslipidemia, diabetes, and broader cardiometabolic dysfunction, further amplifying cardiovascular risk [5–8].

In response to the substantial health burden attributable to air pollution, many governments have implemented aggressive air quality control policies over recent decades, resulting in marked declines in ambient $PM_{2.5}$ concentrations in certain countries and regions [9]. Although these improvements represent major public health achievements, evidence regarding whether reductions in $PM_{2.5}$ exposure translate into measurable cardiovascular benefits, particularly for hypertension, remains limited and inconsistent. Several studies have documented health benefits from declining air pollution, including reduced dyslipidemia risk and lower cardiometabolic mortality, following improvements in $PM_{2.5}$ across varying concentration levels [10–13]. By contrast, analyses based on the UK Biobank (UKB), conducted in a relatively low-exposure setting, have reported largely null associations between improved air quality and circulatory diseases [14]. These mixed findings underscore a critical gap in the literature: whether, and to what extent, lowering

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$PM_{2.5}$ exposure after prolonged exposure confers protection against hypertension. Moreover, few studies have directly compared the benefits of $PM_{2.5}$ reduction across populations with different concentration levels, and evidence among individuals at elevated risk, such as those with pre-hypertension, is particularly scarce.

To address these gaps, we investigated the association between reductions in ambient $PM_{2.5}$ exposure and the incidence of hypertension using data from two large prospective cohorts: the Taiwan Mei Jau (MJ) Cohort and the UKB. According to the WHO Global Air Quality Guidelines, annual $PM_{2.5}$ exposure in the MJ Cohort fell between Interim Targets 2 and 3, indicating a medium-exposure region, whereas exposure in the UKB fell between Interim Targets 3 and 4, indicating a lower-exposure region [15]. These cohorts therefore provide a unique opportunity to evaluate the health benefits of air quality improvement across distinct exposure environments and ethnic populations. By harmonizing exposure assessment, covariates, and analytic strategies across cohorts, and by explicitly contrasting individuals, including those with pre-hypertension, who experienced sustained high exposure with those whose $PM_{2.5}$ exposure declined over time, this study aims to directly quantify the cardiovascular benefits associated with cleaner air. These findings offer policy-relevant evidence supporting the importance of air pollution mitigation strategies for hypertension prevention.

2. Methods

2.1. Study design and participants

The study population was drawn from two large prospective cohorts: the Taiwan MJ Cohort and the UKB. The MJ Cohort has been recruiting Taiwanese residents since 1994 through a paid medical screening program operated by the MJ Health Management Institution. Its database was established in 1996 and contains detailed information on sociodemographic characteristics, lifestyle behaviors, physical measurements, and laboratory biomarkers collected during routine health examinations. By 2017, more than 615,000 participants had been enrolled [16]. For the present analysis, we used data from 345,719 adults who participated beginning in 2005, as this wave included more specific information on individual income.

The UKB is a large-scale population-based cohort in the United Kingdom. Between 2006 and 2010, over 500,000 adults aged 40–69 years attended baseline assessments at 22 centers across England, Scotland, and Wales. Participants completed touchscreen questionnaires on sociodemographic characteristics, medical history, and lifestyle factors, and underwent physical and biological measurements. Additional diagnostic information was obtained through linkage with national hospital records.

Detailed descriptions of study design and data collection procedures for both cohorts have been published previously [16,17]. All participants provided written informed consent. This study was conducted under UKB application 75,249 and approved by the Human and Artefacts Ethics Sub-Committee at the City University of Hong Kong (HU-STA-00001476).

From the initial populations of the MJ Cohort (n = 345,719) and the UKB (n = 501,928), we first excluded individuals with missing information on hypertension or key covariates, as well as those with hypertension at or before the baseline visit. Among the remaining participants, we further excluded those without residential address information or whose addresses could not be matched to $PM_{2.5}$ data for the entire study period. Since the MJ Cohort includes repeated health examinations, participants with only a single visit were additionally excluded.

To evaluate the impact of improved ambient $PM_{2.5}$ levels, we restricted the analytic sample to participants residing in areas where baseline $PM_{2.5}$ concentrations exceeded the cohort-specific median (MJ Cohort: n = 38,397; UKB: n = 66,837). We first examined the association between reduced $PM_{2.5}$ exposure and incident hypertension within this high-exposure baseline population. We then conducted a parallel analysis restricted to individuals with pre-hypertension drawn from the same high-exposure group (MJ Cohort: n = 12,547; UKB: n = 45,495). The complete participant selection process is illustrated in Supplementary Figure S1.

2.2. Outcome ascertainment

The health outcome in this study was incident hypertension. In the MJ Cohort, seated systolic blood pressure (SBP) and diastolic blood pressure (DBP) were measured in the morning using a computerized automated mercury sphygmomanometer (CH-5000, Citizen, Tokyo, Japan). If either SBP or DBP was ≥ 140 or ≥ 90 mmHg, respectively, a second measurement was obtained after 10 minutes of rest and used for diagnosis. Both baseline and incident hypertension were defined based on the following criteria: (1) SBP ≥ 140 mmHg and/or DBP ≥ 90 mmHg, (2) self-report of physician-diagnosed hypertension, or (3) use of blood pressure-lowering medications [4,11].

In the UKB, SBP and DBP were measured twice after a 5-minute rest using an Omron 705 IT electronic blood pressure monitor. The average of these two measurements was used to diagnose hypertension [18]. Date on hospital admissions and visits were obtained through linkage with national healthcare databases: Hospital Episode Statistics (HES) for England, the Scottish Morbidity Record (SMR) for Scotland, and the Patient Episode Database for Wales (PEDW). Incident hypertension during follow-up was defined as: (1) SBP ≥ 140 mmHg and/or DBP ≥ 90 mmHg, (2) self-reported

diagnosis of hypertension, or (3) hospital diagnosis of hypertension [International Classification of Diseases, Tenth Revision (ICD-10) codes: I10–I15]. An additional criterion of receiving anti-hypertensive treatment was used to identify individuals with hypertension at baseline.

For the pre-hypertension analysis, individuals without hypertension at baseline were classified as having pre-hypertension if their baseline SBP was 120–139 mmHg or DBP was 80–89 mmHg in either cohort.

To ensure comparability between cohorts, follow-up time was standardized to a fixed 5-year period. The endpoint was defined as the earliest occurrence of hypertension within five years after the baseline visit. Participants who did not develop hypertension during this interval were censored exactly five years after baseline. Time-to-event analyses were conducted using days as the time scale.

2.3. Exposure assessment

Ground-level $PM_{2.5}$ concentrations data were derived from the publicly available dataset of the Atmospheric Composition Analysis Group at Washington University in St. Louis [19]. Briefly, annual and monthly ground-level $PM_{2.5}$ concentration between 1998 and 2022 were estimated by integrating Aerosol Optical Depth (AOD) data retrievals from National Aeronautics and Space Administration's (NASA) Twin MODerate Resolution Imaging Spectroradiometer (MODIS), Multiangle Imaging Spectro Radiometer (MISR), Sea-Viewing Wide Field-of-View Sensor (SeaWIFS), and Visible Infrared Imaging Radiometer Suite (VIIRS) with the GEOS-Chem chemical transport model. A residual Convolutional Neural Network (CNN) was then applied to calibrate these geophysical $PM_{2.5}$ estimates using ground monitoring data across the entire time series. Model validation demonstrated acceptable performance ($R^2 = 0.73$) [20].

Participants' addresses were collected during each medical visit in the MJ Cohort and obtained through the Administrative Data Liaison Service for England and Wales, the Information Services Division for Scotland, or self-reports in the UKB [21]. These addresses were geocoded, and annual $PM_{2.5}$ concentrations with a spatial resolution of 0.01° × 0.01° (approximately 1 × 1 km²) were assigned to each location. Address changes were incorporated to ensure accurate exposure assessment over time: the number of months each participant resided at their respective addresses during each calendar year was calculated, and annual $PM_{2.5}$ concentrations were estimated by weighting exposure according to residential duration.

Baseline $PM_{2.5}$ exposure was defined as the average annual concentration during the four years preceding the baseline visit plus the baseline year. Follow-up $PM_{2.5}$ exposure was defined as the average annual concentration from the year after baseline until the study endpoint (*i.e.*, hypertension diagnosis or the end of the 5-year follow-up). These multi-year averages were used to enhance the stability of long-term exposure estimates and ensure comparability across participants. Based on these definitions, the periods of $PM_{2.5}$ exposure assessment were 2001–2016 for the MJ Cohort and 2002–2015 for the UKB.

Using these exposure metrics, we identified the cohort-specific median baseline $PM_{2.5}$ concentration (21.2 μg/m³ for the MJ Cohort and 12.8 μg/m³ for the UKB). Participants whose baseline exposure exceeded these medians were included in the analytic sample. Within this high-exposure baseline population, we defined two exposure groups:

(1) Consistently High $PM_{2.5}$ Exposure Group: Participants whose baseline and follow-up $PM_{2.5}$ exposure both remained above the cohort-specific median.

(2) $PM_{2.5}$ Reduction Group: Participants whose baseline exposure exceeded the median but whose follow-up exposure fell to or below the median.

These categories enabled evaluation of whether reductions in $PM_{2.5}$ exposure were associated with a lower risk of developing hypertension.

2.4. Covariates

Based on previous literature, we selected sociodemographic, lifestyle, health, and environmental factors associated with hypertension as covariates [8,11,22]. To ensure comparability between the two cohorts, we standardized and categorized these variables into consistent dichotomous or multi-level groups. Definitions of each covariate for both cohorts, along with the UKB data fields used in this study, are provided in Supplementary Tables S1 and S2.

Sociodemographic characteristics included sex (male or female), age (years), educational background (low, medium, or high), and average yearly household income (low, medium, or high). Ethnicity (White or non-White) was included only for the UKB. To account for area level socioeconomic conditions, we used the Area Deprivation Index (ADI) for the MJ Cohort and the Townsend Deprivation Index (TDI) for the UKB [23]. Details on the construction of the ADI are provided in the Supplementary Note 1.

Lifestyle variables included smoking status (never, former, or current), alcohol use (seldom, occasional, or regular), vegetable and fruit intake (seldom, moderate, or frequent), and exercise intensity (inactive, light, moderate, or high). Body mass index (BMI) was calculated as weight in kilograms divided by height in meters squared. Diabetes and dyslipidemia at baseline were categorized as either "yes" or "no."

Since baseline $PM_{2.5}$ exposure may influence hypertension risk and could be affected by residential relocation, we additionally included baseline $PM_{2.5}$ concentration as a covariate in all models.

2.5. Statistical analysis

For continuous variables, descriptive statistics were presented as mean ± standard deviation (SD) if normally distributed and as median (interquartile range) if skewed. Categorical data were expressed as a number (percentage).

Cox regression models with inverse probability weighting (IPW) were used to estimate the association between $PM_{2.5}$ reduction and hypertension risk across separate cohort. Stabilized IPWs were calculated using a logistic model, regressing exposure groups (*i.e.*, consistently high $PM_{2.5}$ exposure and $PM_{2.5}$ reduction group) on all covariates. To minimize bias from extreme weight values, we replaced observations below the 1st percentile with the 1st percentile value and those above the 99th percentile with the 99th percentile value. Covariate balance before and after weighting is shown in Supplementary Figure S2. Given the repeated measurement design of the MJ Cohort, a marginal structural model (MSM) was applied to account for time-varying covariates at each follow-up visit [24], including educational background, average yearly household income, ADI score, smoking status, alcohol use, vegetable and fruit intake, exercise intensity, BMI, diabetes, and dyslipidemia. These time-varying covariates were also adjusted in Cox regression models for the MJ Cohort. Additionally, person-level random intercepts were included to control for clustering within repeated measurements for MJ Cohort. Three hierarchical models were developed to evaluate the associations: Model 1 included exposure groups and baseline $PM_{2.5}$ exposure. Model 2 further adjusted for sociodemographic covariates, including sex, age, ethnicity (UKB only), educational background, average yearly household income, and area level socioeconomic conditions. Model 3 included all covariates from Model 2 and additionally adjusted for smoking status, alcohol use, vegetable and fruit intake, exercise intensity, diabetes, and dyslipidemia. All association estimates were presented as hazard ratios (HR) with corresponding 95% confidence intervals (CI). To visualise differences in hypertension risk between exposure groups, we plotted cumulative hazard curves for each cohort based on Model 3.

Quantitative associations were further explored by treating exposure as a continuous variable. The difference between baseline and follow-up $PM_{2.5}$ concentrations was calculated, and per 1 μg/m³ and interquartile range (IQR) decrease were used to evaluate risk reduction across cohorts. Additionally, restricted cubic spline (RCS) analyses were conducted to model concentration–response relationships between changes in $PM_{2.5}$ and hypertension risk, based on Model 3. The number of RCS knots was determined using model fit criteria (AIC and BIC) with a range of 3 to 5 knots. Statistical significance of the overall curve and non-linearity of the cubic spline terms was tested.

For the pre-hypertension analysis, the same modeling strategy was applied among participants with pre-hypertension at baseline. Additional subgroup analyses were performed based on predefined covariates: age (< 45 years *vs.* ≥ 45 years), sex (male *vs.* female), educational background (low/medium *vs.* high), household income (low/medium *vs.* high), area level socioeconomic score (low *vs.* high), smoking status (never *vs.* former/current), alcohol use (seldom *vs.* occasional/regular), vegetable intake (seldom/moderate *vs.* frequent), fruit intake (seldom/moderate *vs.* frequent), exercise intensity (inactive/light *vs.* moderate/high), and BMI (normal weight *vs.* overweight/obesity). To examine effect modification, we incorporated multiplicative interaction terms between each stratified factor and exposure group (stratified factor × exposure group) in Model 3. Statistical significance of interactions was determined using the Benjamini–Hochberg false discovery rate (FDR) procedure, with FDR-adjusted $P < 0.05$ considered significant [25].

Several sensitivity analyses were performed to assess the robustness of the findings: (1) repeating analyses including participants with baseline exposure below the cohort-specific median; (2) restricting baseline $PM_{2.5}$ exposure to a three-year window (average of the two years before baseline plus the baseline year); (3) treating $PM_{2.5}$ exposure as time-varying, with the baseline assessment period attributed to the Consistently High $PM_{2.5}$ Exposure Group; (4) excluding participants with baseline cardiovascular disease, stroke, kidney disease, or cancer (definitions in the Supplementary Note 2); (5) excluding participants who developed hypertension within the first year of follow-up; (6) applying multivariate imputation by chained equation to address missing covariate data across five imputed datasets; and (7) excluding participants who changed residential address during follow-up.

Statistical analyses were conducted using RStudio (version 2024.04.1; R Core Team, Vienna, Austria) with a series of packages, including 'survival', 'ggsurvfit', 'rms', and 'ggplot2'. Two-tailed *P* values less than 0.05 were considered statistically significant.

3. Results

Table 1 presents the population characteristics of both cohorts. The mean age of participants in the MJ Cohort was lower than that in the UKB, and in both cohorts, more than half of participants were women. In terms of education levels, participants in the MJ Cohort had a lower proportion of low educational attainment compared to the UKB. Participants in the MJ Cohort were less likely to smoke or consume alcohol, whereas those in the UKB reported higher levels of vegetable and fruit intake. Regarding health status, participants in the UKB had a higher mean BMI, SBP and DBP, and a greater prevalence of baseline dyslipidemia. Among participants in the two exposure groups, the median baseline $PM_{2.5}$ exposure was 25.9 μg/m³ in the

MJ Cohort and 13.2 μg/m³ in the UKB. After a median follow-up of 3.8 years in the MJ Cohort and 5 years in the UKB, median $PM_{2.5}$ exposure was reduced by 1.5 μg/m³ and 0.1 μg/m³, respectively. Detailed baseline characteristics by $PM_{2.5}$ exposure groups are provided in Supplementary Table S3. Temporal trends in average $PM_{2.5}$ exposure are presented in Figure 1. A significant declining trend was observed in the UKB (Mann-Kendall test: P = 0.007), whereas no significant trend was detected in the MJ Cohort (P = 0.117).

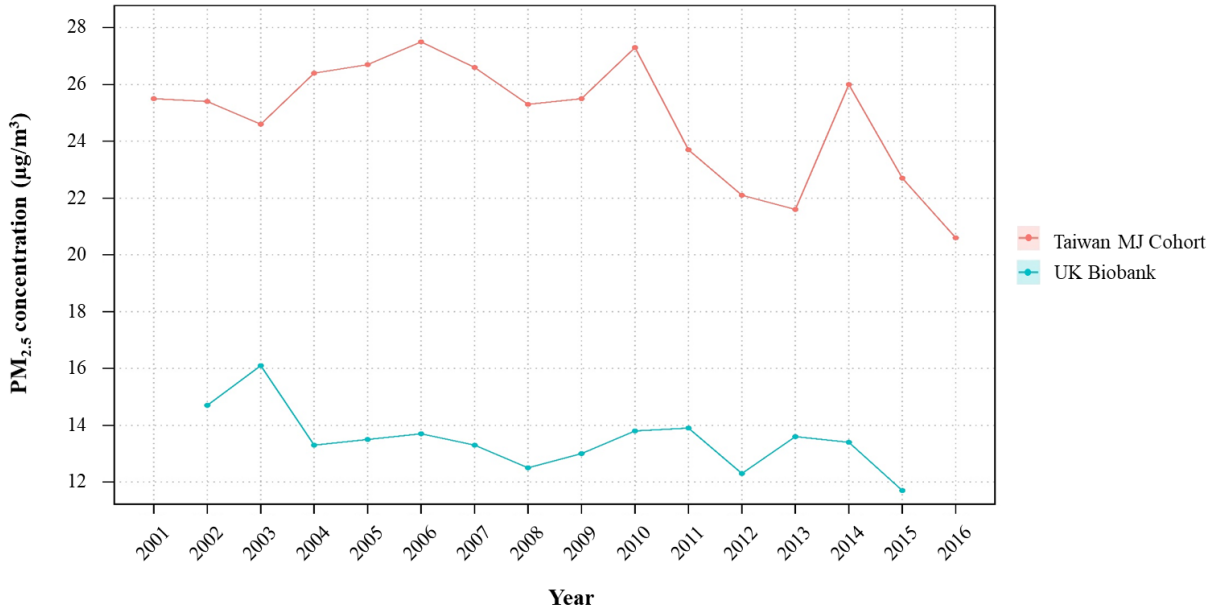

Figure 1. Trends in the $PM_{2.5}$ exposure for the Taiwan MJ Cohort (n = 38,397, 2001–2016) and UK Biobank (n = 66,837, 2002–2015). The dots represent the median annual $PM_{2.5}$ exposure for each year.

Table 1. Baseline characteristics of participants in the Taiwan MJ Cohort and the UK Biobank.

	Taiwan MJ Cohort (n = 38,397)	UK Biobank (n = 66,837)
Age (years), mean ± SD	38.5 ± 10.5	52.7 ± 7.9
Male, n (%)	18,072 (47.1%)	26,626 (39.8%)
Ethnicity, n (%)		
White	NA	59,446 (88.9%)
Non-White	NA	7391 (11.1%)
Educational background, n (%)		
Low	3606 (9.4%)	19,140 (28.6%)
Medium	8149 (21.2%)	13,168 (19.7%)
High	26,642 (69.4%)	34,529 (51.7%)
Average yearly household income, n (%)		
Low	7161 (18.6%)	9132 (13.7%)
Medium	22,534 (58.7%)	33,896 (50.7%)
High	8702 (22.7%)	23,809 (35.6%)
Area level socioeconomic score, mean ± SD†	0.4 ± 1.1	−1.2 ± 3.1
Smoking status, n (%)		
Never	29,759 (77.5%)	38,299 (57.3%)
Former	1922 (5.0%)	21,252 (31.8%)
Current	6716 (17.5%)	7286 (10.9%)
Alcohol use, n (%)		
Seldom	33,085 (86.2%)	18,552 (27.8%)
Occasional	3717 (9.7%)	17,503 (26.2%)
Regular	1595 (4.2%)	30,782 (46.1%)
Vegetable intake, n (%)		

Table 1. *Cont.*

	Taiwan MJ Cohort (n = 38,397)	UK Biobank (n = 66,837)
Seldom	4446 (11.6%)	1201 (1.8%)
Moderate	22,527 (58.7%)	34,683 (51.9%)
Frequent	11,424 (29.8%)	30,953 (46.3%)
Fruit intake, n (%)		
Seldom	11,823 (30.8%)	4543 (6.8%)
Moderate	21,294 (55.5%)	40,567 (60.7%)
Frequent	5280 (13.8%)	21,727 (32.5%)
Exercise intensity, n (%)		
Inactive	14,677 (38.2%)	16,711 (25.0%)
Light	8213 (21.4%)	16,714 (25.0%)
Moderate	6587 (17.2%)	16,709 (25.0%)
High	8920 (23.2%)	16,703 (25.0%)
BMI (kg/m²), mean ± SD	22.7 ± 3.4	25.8 ± 4.0
SBP (mmHg), mean ± SD	112.3 ± 12.5	123.4 ± 9.9
DBP (mmHg), mean ± SD	68.5 ± 8.9	76.2 ± 7.0
Diabetes, n (%)		
Yes	831 (2.2%)	1253 (1.9%)
No	37,566 (97.8%)	65,584 (98.1%)
Dyslipidemia, n (%)		
Yes	7959 (20.7%)	25,983 (38.9%)
No	30,438 (79.3%)	40,854 (61.1%)
Baseline $PM_{2.5}$ exposure (μg/m³), median [IQR]	25.9 [22.4, 31.4]	13.2 [13.0, 14.2]

† Different indicators were used in the two cohorts (Area Deprivation Index for the MJ Cohort and the Townsend Deprivation Index for the UK Biobank). Abbreviations: SD, standard deviation; BMI, body mass index; SBP, systolic blood pressure; DBP, diastolic blood pressure; IQR, interquartile range; NA, not applicable.

Associations between $PM_{2.5}$ reduction and hypertension risk are presented in Table 2. In the MJ Cohort, participants experiencing reductions in $PM_{2.5}$ exposure had a significantly lower risk of hypertension compared with those in the consistently high exposure group (Model 3: HR = 0.83, 95% CI: 0.73, 0.95; P = 0.006). In contrast, the association was not statistically significant in the UKB (Model 3: HR = 0.89, 95% CI: 0.74, 1.06; P = 0.179), although the point estimate suggested a protective direction. When $PM_{2.5}$ exposure was treated as a continuous variable, each 1 μg/m³ decrease was significantly associated with a 3% (95% CI: 1%, 5%) reduction in hypertension risk in the MJ Cohort and a 15% (95% CI: 6%, 24%) reduction in the UKB. Cumulative hazard graph (Figure 2) indicates that, in the MJ Cohort, participants in the $PM_{2.5}$ Reduction Group consistently exhibited lower hypertension risk throughout follow-up compared with those in the Consistently High Exposure Group. In the UKB, risks were similar between groups until approximately three years of follow-up. Figure 3 illustrates the concentration-response curves, with three knots applied for the MJ Cohort and four knots for the UKB. The curves revealed a near-linear relationship in the MJ Cohort (P for non-linearity = 0.380) and a significant non-linear association in the UKB (P for non-linearity < 0.001).

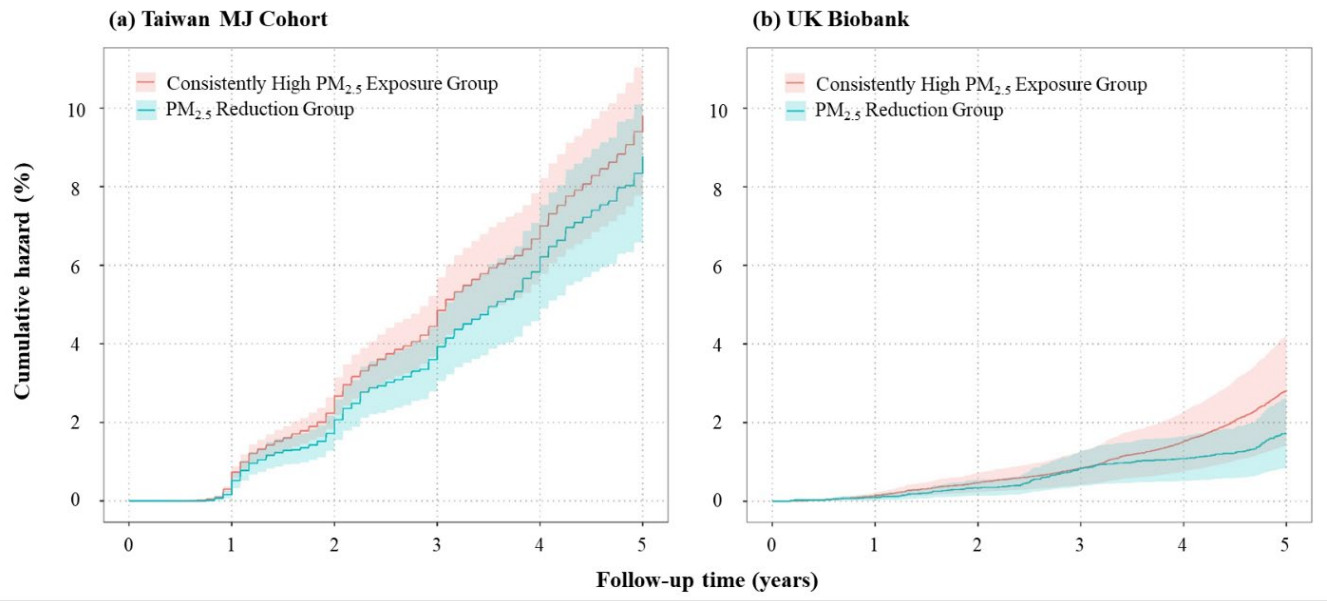

Figure 2. Cumulative hazard graph comparing the risk of hypertension between the two $PM_{2.5}$ exposure groups across cohorts. **(a)** Taiwan MJ Cohort; **(b)** UK Biobank.

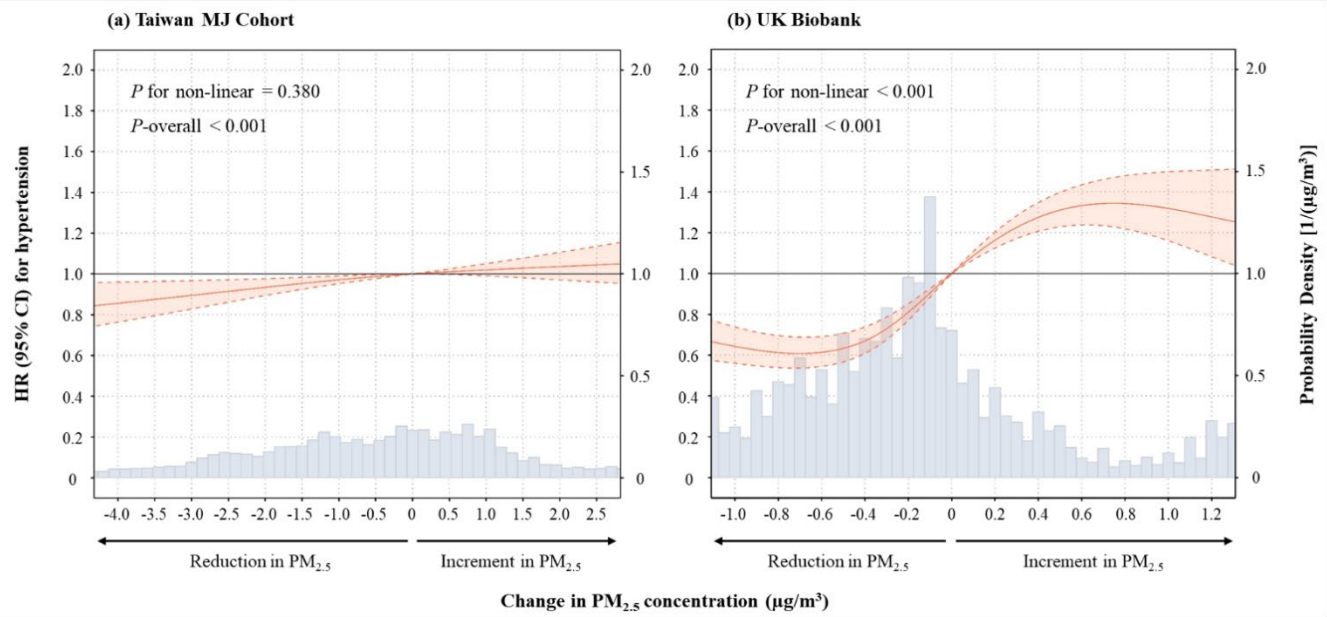

Figure 3. Concentration-response relationship between changes in $PM_{2.5}$ and hypertension risk across cohorts. **(a)** Taiwan MJ Cohort; **(b)** UK Biobank. Analyses were conducted among participants in both $PM_{2.5}$ exposure group. The X-axis displays the range of changes in $PM_{2.5}$ concentration from the 5th to the 95th percentile (Taiwan MJ Cohort: −4.3 to 2.5 μg/m³; UK Biobank: −1.1 to 1.3 μg/m³). Orange curve and its 95% CI correspond to the left y-axis (HR for hypertension), while the histogram bars correspond to the right y-axis (probability density).

Table 2. Association between reduced exposure to ambient $PM_{2.5}$ and the risk of incident hypertension.

	N of participants	N (%) of incident hypertension	Model 1		Model 2		Model 3	
			HR (95% CI)	*P* value	HR (95% CI)	*P* value	HR (95% CI)	*P* value
Taiwan MJ Cohort								
Consistently High $PM_{2.5}$ Exposure Group	32,221	2278 (7.1%)	Reference	-	Reference	-	Reference	-
$PM_{2.5}$ Reduction Group	6176	334 (5.4%)	0.85 (0.75, 0.97)	0.015	0.84 (0.73, 0.95)	0.008	0.83 (0.73, 0.95)	0.006
Per 1 μg/m³ decrease in $PM_{2.5}$	38,397	2612 (6.8%)	0.96 (0.95, 0.98)	< 0.001	0.98 (0.96, 0.99)	0.015	0.97 (0.95, 0.99)	0.002
Per IQR decrease in $PM_{2.5}$[†]	38,397	2612 (6.8%)	0.91 (0.87, 0.95)	< 0.001	0.94 (0.89, 0.99)	0.018	0.92 (0.88, 0.97)	0.002
UK Biobank								
Consistently High $PM_{2.5}$ Exposure Group	54,712	1144 (2.1%)	Reference	-	Reference	-	Reference	-
$PM_{2.5}$ Reduction Group	12,125	233 (1.9%)	0.91 (0.76, 1.09)	0.307	0.88 (0.74, 1.06)	0.178	0.89 (0.74, 1.06)	0.179
Per 1 μg/m³ decrease in $PM_{2.5}$	66,837	1377 (2.1%)	0.97 (0.89, 1.05)	0.449	0.87 (0.79, 0.97)	0.011	0.85 (0.76, 0.94)	0.003
Per IQR decrease in $PM_{2.5}$[†]	66,837	1377 (2.1%)	0.98 (0.92, 1.04)	0.449	0.91 (0.85, 0.98)	0.011	0.89 (0.83, 0.96)	0.003

[†] Analyses were conducted among participants in both $PM_{2.5}$ exposure group. The IQR for the change in $PM_{2.5}$ concentration was 2.6 μg/m³ for the Taiwan MJ Cohort and 0.7 μg/m³ for the UK Biobank. HR, hazard ratio; CI, confidence interval. Model 1 included exposure groups and baseline $PM_{2.5}$ exposure. Model 2 further adjusted for sex, age, ethnicity (only for the UK Biobank), educational background, average yearly household income, and area level socioeconomic conditions. Model 3 included all covariates from Model 2 and additionally adjusted for smoking status, alcohol use, vegetable and fruit intake, exercise intensity, diabetes, and dyslipidemia.

Analyses restricted to participants with pre-hypertension at baseline (Table 3) showed significant associations in both cohorts, whether $PM_{2.5}$ reduction was modeled as a dichotomous exposure group or as a continuous change in concentration. The magnitude of the association was stronger among individuals with pre-hypertension, indicating greater protective effect of $PM_{2.5}$ reduction in pre-hypertension participants compared with the overall population.

Table 3. Association between reduced exposure to ambient $PM_{2.5}$ and the risk of incident hypertension among pre-hypertension individuals.

	N of participants	N (%) of incident hypertension	Model 1		Model 2		Model 3	
			HR (95% CI)	*P* value	HR (95% CI)	*P* value	HR (95% CI)	*P* value
Taiwan MJ Cohort								
Consistently High $PM_{2.5}$ Exposure Group	10,532	1794 (17.0%)	Reference	-	Reference	-	Reference	-
$PM_{2.5}$ Reduction Group	2015	269 (13.3%)	0.73 (0.64, 0.84)	< 0.001	0.72 (0.63, 0.84)	< 0.001	0.72 (0.62, 0.84)	< 0.001
Per 1 μg/m³ decrease in $PM_{2.5}$	12,547	2063 (16.4%)	0.95 (0.94, 0.97)	< 0.001	0.96 (0.94, 0.98)	< 0.001	0.96 (0.94, 0.98)	< 0.001
Per IQR decrease in $PM_{2.5}$[†]	12,547	2063 (16.4%)	0.88 (0.84, 0.93)	< 0.001	0.91 (0.86, 0.96)	< 0.001	0.90 (0.85, 0.95)	< 0.001
UK Biobank								
Consistently High $PM_{2.5}$ Exposure Group	37,071	998 (2.7%)	Reference	-	Reference	-	Reference	-
$PM_{2.5}$ Reduction Group	8424	196 (2.3%)	0.83 (0.69, 1.01)	0.068	0.82 (0.68, 0.99)	0.043	0.82 (0.68, 0.99)	0.044
Per 1 μg/m³ decrease in $PM_{2.5}$	45,495	1194 (2.6%)	0.94 (0.86, 1.03)	0.207	0.87 (0.78, 0.97)	0.013	0.85 (0.76, 0.95)	0.005
Per IQR decrease in $PM_{2.5}$[†]	45,495	1194 (2.6%)	0.96 (0.90, 1.02)	0.207	0.91 (0.85, 0.98)	0.013	0.90 (0.84, 0.97)	0.005

[†] Analyses were conducted among participants in both $PM_{2.5}$ exposure group. The IQR for the change in $PM_{2.5}$ concentration was 2.6 μg/m³ for the Taiwan MJ Cohort and 0.7 μg/m³ for the UK Biobank. HR, hazard ratio; CI, confidence interval. Model 1 included exposure groups and baseline $PM_{2.5}$ exposure. Model 2 further adjusted for sex, age, ethnicity (only for the UK Biobank), educational background, average yearly household income, and area level socioeconomic conditions. Model 3 included all covariates from Model 2 and additionally adjusted for smoking status, alcohol use, vegetable and fruit intake, exercise intensity, diabetes, and dyslipidemia.

Figure 4 illustrates the subgroup-specific associations between hypertension and $PM_{2.5}$ reduction. In the MJ Cohort, the associations were more pronounced in participants with low and medium household income (HR = 0.80, 95% CI: 0.69, 0.93, adjusted *P* for interaction = 0.013) and those with dyslipidemia at baseline (HR = 0.71, 95% CI: 0.57, 0.90, adjusted *P* for interaction = 0.039). No significant subgroup differences were observed in the UKB.

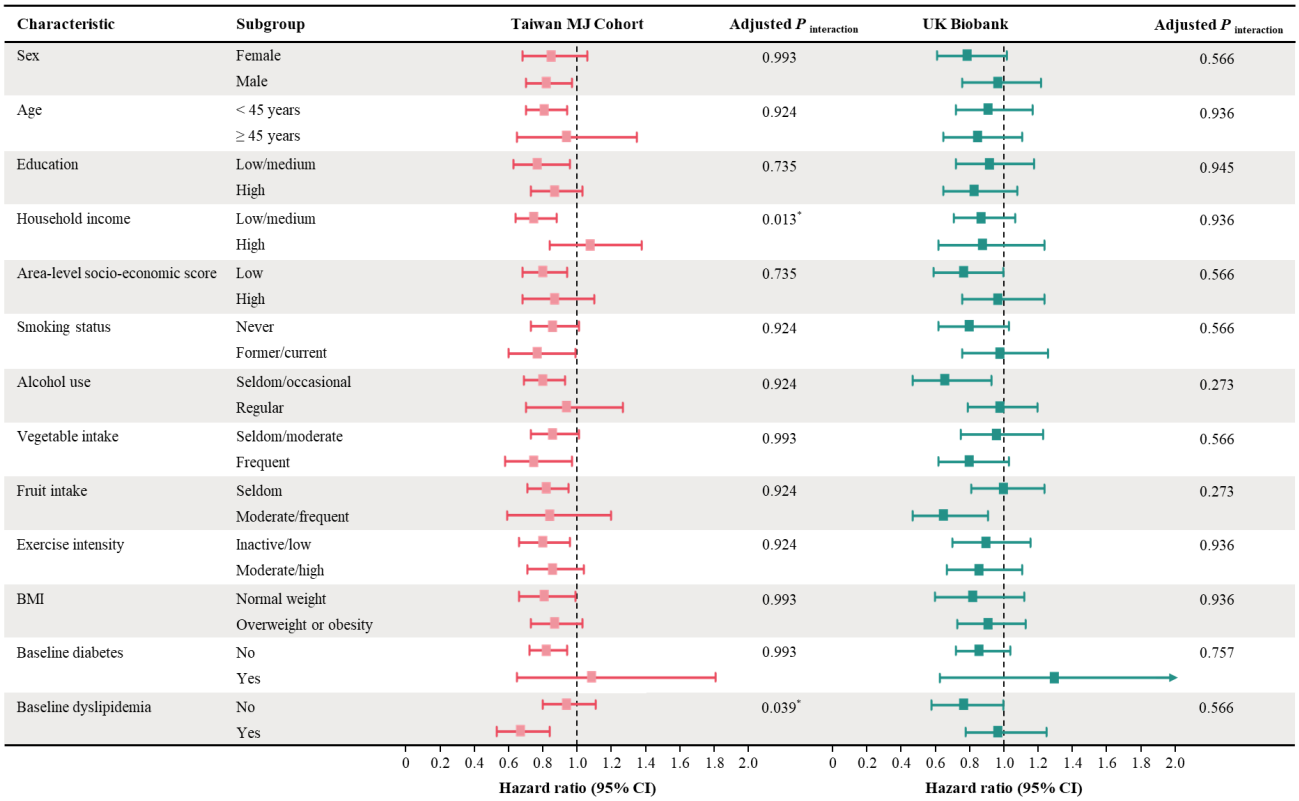

Figure 4. Stratified analysis on the association between reduced exposure to ambient $PM_{2.5}$ and risk of incident hypertension. * *P* value < 0.05.

Sensitivity analyses demonstrated consistent protective associations of $PM_{2.5}$ reduction with hypertension risk. Results remained robust when including participants with below-median baseline exposure (Supplementary Figure S3), restricting baseline exposure to a three-years window, treating exposure as time-varying, excluding participants with baseline cardiovascular disease (CVD) and stroke, kidney diseases, or cancer, excluding cases of incident hypertension in the first year, and employing the multiple imputation for missing covariates (Supplementary Tables S4–S8). Excluding participants with address changes during follow-up attenuated the continuous exposure association in the UKB to non-significance (Supplementary Table S9).

4. Discussion

In this large bi-cohort analysis spanning two distinct exposure settings, reductions in long-term ambient $PM_{2.5}$ were consistently associated with a lower risk of incident hypertension. Among all participants, individuals in the $PM_{2.5}$ Reduction Group experienced a 17% lower hypertension risk in the Taiwan MJ Cohort (HR = 0.83, 95% CI: 0.73, 0.95), with a similar but non-significant protective direction observed in the UKB (HR = 0.89, 95% CI: 0.74, 1.06). When modeled continuously, each 1 μg/m³ decrease in $PM_{2.5}$ was associated with a 3% (8% for IQR decrease) reduction in hypertension risk in the MJ Cohort and a 15% (11% for IQR decrease) reduction in the UKB, underscoring the cardiovascular benefits of improved air quality. Notably, protective associations were stronger among individuals with pre-hypertension, with $PM_{2.5}$ reduction linked to a 28% lower risk in the MJ Cohort (HR = 0.72, 95% CI: 0.62, 0.84) and an 18% lower risk in the UKB (HR = 0.82, 95% CI: 0.68, 0.99). These findings provide direct evidence that lowering $PM_{2.5}$ exposure after prolonged high exposure confers measurable cardiovascular benefits, particularly for populations already at elevated risk. By harmonizing analytic strategies across two large cohorts with different baseline pollution levels, this study strengthens the inference that air quality improvements can meaningfully reduce hypertension burden, offering timely and policy-relevant support for continued air pollution mitigation worldwide.

Although many cross-sectional and longitudinal studies have established that higher long-term $PM_{2.5}$ exposure increases the risk of hypertension [2–4], direct evidence demonstrating that improved air quality reduces hypertension risk remains limited. A recent UKB analysis examined changes in air pollution across 14 major disease categories and reported no significant associations between $PM_{2.5}$ reduction and circulatory diseases among individuals who relocated to cleaner areas [14]. However, that study did not specify the number of participants relocating from high- to low-exposure environments, limiting assessment of

statistical power. In our UKB analysis, the number of incident hypertension cases was relatively small (n = 1377, 2.1% of all participants; n = 1194, 2.6% among those with pre-hypertension), which likely constrained statistical power and may explain the lack of a significant association observed in the prior study. Our study addressed this by standardizing a fixed 5-year follow-up window, ensuring sufficient time to observe health effects following e6xposure reduction—an aspect not explicitly considered in the prior UKB relocation study. Unlike the earlier study, which only used categorized exposure, our analysis modeled $PM_{2.5}$ as a continuous variable, thereby increasing statistical power to detect significant associations (per 0.7 μg/m³ decrease: HR = 0.89; 95% CI: 0.83, 0.96). Furthermore, we included all eligible UKB participants in our analysis, regardless of relocation history, allowing assessment of improved ambient $PM_{2.5}$ air quality from a broader range of sources and providing a more accurate reflection of real-world conditions.

Evidence from other settings supports the cardiovascular benefits of air quality improvement. A Canadian study found that individuals relocating from high- to intermediate- or low-$PM_{2.5}$ regions experienced lower cardiometabolic mortality, indirectly reinforcing the notion that reducing pollution exposure yields cardiometabolic benefits [13]. Similarly, our previous work in the MJ Cohort showed that each 5 μg/m³ decrease in $PM_{2.5}$ during follow-up was associated with a 16% reduction in hypertension risk (HR = 0.84; 95% CI: 0.82, 0.86) [11]. The present study extends this evidence by applying a harmonized analytic framework across two large cohorts and by comparing 5-year $PM_{2.5}$ averages between baseline and follow-up periods. Despite differences in baseline exposure levels and exposure contrasts, both cohorts demonstrated consistent protective associations, reinforcing the conclusion that lowering $PM_{2.5}$ exposure can meaningfully reduce hypertension risk.

The stronger protective associations observed among individuals with pre-hypertension suggest that populations with elevated baseline blood pressure may be particularly sensitive to improvements in air quality. This pattern is biologically plausible: pre-hypertensive individuals already exhibit early vascular dysfunction, heightened sympathetic activity, and impaired endothelial regulation, all of which may amplify susceptibility to the inflammatory and oxidative effects of $PM_{2.5}$ [26]. Reducing $PM_{2.5}$ exposure may therefore interrupt these pathophysiological processes at a stage when blood pressure remains modifiable, resulting in a larger relative risk reduction compared with the general population. The pronounced benefit observed among participants with baseline dyslipidemia in the MJ Cohort further supports this interpretation [12]. Dyslipidemia and hypertension share mechanistic pathways—including endothelial injury, impaired nitric oxide bioavailability, and systemic inflammation—that are also exacerbated by $PM_{2.5}$ exposure [27,28]. Individuals with dyslipidemia may thus experience a "double burden" of vascular stress, making them more responsive to reductions in pollution-related insults. Taken together, these findings indicate that air quality improvements may yield disproportionate cardiovascular benefits for individuals with existing metabolic or vascular vulnerabilities. This highlights the potential value of targeting pollution reduction strategies toward communities with a high prevalence of pre-hypertension or cardiometabolic risk factors, where health gains may be especially substantial.

Several secondary findings contextualize differences observed between cohorts. The categorical comparison of exposure groups showed a significant association in the MJ Cohort but not in the UKB, likely reflecting the much narrower range of $PM_{2.5}$ reductions in the UKB (5th–95th percentile: −1.1 to 1.3 μg/m³), which limited the exposure contrast needed to detect effects. Dichotomizing continuous exposure may also reduce statistical power and obscure underlying associations [29]. Consistent with this, treating $PM_{2.5}$ change as a continuous variable yielded significant associations in both cohorts. Concentration–response curves further highlighted cohort differences: the MJ Cohort exhibited an approximately linear decline in hypertension risk with decreasing $PM_{2.5}$, whereas the UKB showed a modest but non-linear pattern, suggesting that even small improvements in already low-pollution settings may confer measurable benefits. Differences in baseline characteristics—including age, BMI, and blood pressure, all established hypertension risk factors [30]—likely contributed to cohort-specific shapes and effect sizes. Subgroup analyses also revealed heterogeneous associations, such as stronger effects by household income in the MJ Cohort; however, these findings were inconsistent across cohorts and should be interpreted cautiously. Sensitivity analyses generally yielded similar results, reinforcing the robustness of our findings. Notably, the UKB association did not reach statistical significance among participants who did not relocate during the follow-up period, likely due to exclusion of a relatively large sample (13.1%), which reduced statistical power. Nevertheless, the positive direction suggests that reduction in $PM_{2.5}$ tended to lower hypertension risk in the UKB ($PM_{2.5}$ Reduction Group: HR = 0.98; per 1 μg/m³ decrease: HR = 0.91). Overall, these secondary results underscore the influence of exposure contrast, baseline risk profiles, and population characteristics on effect estimation, without altering the central conclusion that reductions in $PM_{2.5}$ are associated with lower hypertension risk.

This study has several notable strengths. The use of two large independent cohorts enabled replication of findings across distinct exposure settings. By applying IPW and MSM, we approximated a pseudo-population less affected by time-varying confounding, strengthening

causal interpretation of the association between $PM_{2.5}$ reduction and hypertension risk [31]. The harmonized exposure assessment, drawing from the same high-resolution $PM_{2.5}$ dataset and applying consistent spatial–temporal assignment procedures, further enhanced comparability between cohorts and improved the robustness of the findings.

Several limitations should also be acknowledged. First, both cohorts primarily included middle-aged and older adults, many of whom already had elevated cardiometabolic risk. The generalizability of these findings to younger populations or regions with different demographic structures should be cautious. Second, defining follow-up exposure based on event timing may introduce potential bias (e.g., immortal time bias), potentially leading to overestimation of effect size. Although results remain consistent across sensitivity analyses, the possibility of bias influencing quantitative outcomes cannot be excluded. Third, although smoking status was included as a covariate, we lacked information on other indoor and outdoor pollutants, such as nitrogen dioxide (NO_2), ozone (O_3), sulfur dioxide (SO_2), household fuel use, and ventilation, which may contribute to residual confounding. Fourth, despite efforts to standardize covariates and outcome definitions, differences in data collection methods remain. For example, disease diagnoses in the UKB were obtained through linkage to national health records, whereas some conditions in the MJ Cohort relied on self-report, which may introduce misclassification and affect cross-cohort comparability. Finally, although extensive covariate adjustment was performed, unmeasured or residual confounding cannot be fully excluded.

5. Conclusion

In two large population-based cohorts representing distinct air pollution contexts, reductions in long-term $PM_{2.5}$ exposure were consistently associated with a lower risk of incident hypertension, with particularly pronounced benefits among individuals with pre-hypertension. By harmonizing exposure assessment and analytic strategies across cohorts, this study provides direct evidence that improvements in ambient $PM_{2.5}$ levels can yield measurable cardiovascular gains even after prolonged high exposure. These findings underscore the importance of sustained air pollution mitigation efforts and suggest that populations with elevated baseline hypertension risk may derive especially meaningful health benefits. Continued reductions in $PM_{2.5}$ concentrations therefore represent a critical public-health strategy for preventing hypertension and potentially alleviating the broader burden of cardiovascular disease.

Supplementary data

Supplementary material includes detailed definitions of variables (Supplementary notes 1–2 and Tables S1–S2), as well as supporting tables and figures (Tables S3–S9 and Figures S1–S3). The supplementary material is provided in a separate file.

Data availability statement

The data or datasets generated or analyzed in this study are available in Zenodo at https://doi.org/10.5281/zenodo.22160780. All datasets are fully de-identified and contain no potentially identifiable health information, ensuring that participants' privacy is protected.

Declaration of generative AI and AI-assisted technologies

During the preparation of this manuscript, the authors used generative AI tools only to improve language and readability. Specifically, the authors used ChatGPT and DeepSeek for language polishing in entire manuscript. The authors take full responsibility for the content of the manuscript.

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Authors' contribution

Conceptualization: D.C. and X.L.; methodology: D.C.; software: D.C.; validation: D.C. and Y.Y.; formal analysis: D.C.; investigation: X.W. and G.N.T.; resources: X.L.; data curation: W.C., Y.Z. and T.X.; writing—original draft preparation: D.C.; writing—review and editing: Y.Y., W.C., T.X., K.S., Y.Z., X.W., G.N.T. and X.L.; visualization: D.C.; supervision: X.W. and X.L.; project administration: X.L.; funding acquisition: X.L. All authors have read and agreed to the published version of the manuscript.

Conflicts of interest

Xiangqian Lao holds the position of Associate Editor for *International Journal of Environmental Epidemiology* and has not peer reviewed or made any editorial decisions for this paper. The authors declare no other conflicts of interest associated with this manuscript.

Ethical statement

The study was performed in accordance with the Declaration of Helsinki and approved by the Human and Artefacts Ethics Sub-Committee at the City University of Hong Kong (HU-STA-00001476).

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