




Effects of Short-Term Exposure to Air Pollutants on Real-Time Blood Pressure: A Wearable Device-Based Study in China

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Abstract Air pollutants have been known as the most persistent environmental risk factors of elevated blood pressure (BP). However, most of the existing studies measured resting BP only, which is not accurate as ambulatory BP. This study investigated the effects of short-term exposure to air pollutants on ambulatory BP. Wearable devices were used to measure personal ambulatory systolic BP (ASBP) and diastolic BP (ADBP) and movement trajectories of 172 participants for one week, with a 1-min interval. Daily concentrations of the six major air pollutants were estimated at a spatial resolution of 1 km. Linear mixed-effect models and distributed lag non-linear models estimated the associations between air

pollutant exposure and ambulatory BP. ASBP was positively associated with PM_{2.5} ($\beta=0.010$ [95% CI: 0.005, 0.015]), PM₁₀ ($\beta=0.006$ [0.005, 0.008]), and SO₂ ($\beta=0.046$ [0.021, 0.071]), and negatively with NO₂ ($\beta=-0.009$ [-0.017, -0.001]); ADBP was positively associated with PM_{2.5} ($\beta=0.008$ [0.005, 0.010]) and PM₁₀ ($\beta=0.003$ [0.002, 0.004]), and negatively with NO₂ ($\beta=-0.008$ [-0.012, -0.004]), O₃ ($\beta=-0.002$ [-0.004, -0.001]), and CO ($\beta=-0.366$ [-0.652, -0.081]). At 14-h lag, ASBP and ADBP were positively associated with PM₁₀ and O₃, and negatively with NO₂. The cumulated exposure to PM₁₀ and CO was associated with ambulatory BP at all levels of concentration, while SO₂ was associated with ambulatory BP only when the concentration was over 15 $\mu\text{g}/\text{m}^3$. The findings have important implications for BP management and hypertension prevention, by providing solid evidence for developing cost-effective strategies of minimizing adverse environmental exposure and improving health equity.

Chen Li, Xiu Yang and Kun Qin contributed equally to this work.

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Introduction

High blood pressure (BP), also known as hypertension, is characterized by elevated arterial BP in the systemic circulation, which has been considered a primary risk factor of cardiovascular diseases and mortality [1]. According to the latest Global Burden of Disease Study conducted in 2017, hypertension has caused 10.4 million deaths and 218 million disability-adjusted life years, making it a large global health burden, particularly in Asian countries such as China [2–4]. The prevalence of hypertension in China has increased from 7.7% to 23.3% over the past three decades[5]. To control and prevent hypertension, a wide array of modifiable risk factors of hypertension have been identified, including lifestyle behaviors and environmental factors[6, 7]. Among them, numerous epidemiological studies have examined the associations of elevated BP and air pollutants, which have been regarded as the predominant, also most persistent, risk factors of elevated BP[7, 8]. Air pollutants that are harmful to human health mainly include particulate matter with aerodynamic diameter $< 2.5\mu\text{m}$ ($\text{PM}_{2.5}$) and $< 10\mu\text{m}$ (PM_{10}), nitrogen dioxide (NO_2), sulfur dioxide (SO_2), ozone (O_3), and carbon monoxide (CO)[9]. Exposure to these air pollutants may be related to cardiovascular autonomic nervous system imbalance, inflammation, and oxidative stress, which could lead to endothelial dysfunction, acceleration of the development of atherosclerosis, vasoconstriction and further elevated BP[10, 11].

Several studies have reported positive associations between air pollutants and BP[12]. For example, a study of 39,259 adults in China found that exposure to higher concentrations of $\text{PM}_{2.5}$, PM_{10} , and NO_2 over the past three years was associated with the elevated BP[13]; another study of 9,354 children in China also found that exposure to higher concentrations of PM_{10} and O_3 over the past five days was associated with the elevated BP[14]. However, in those studies, the resting BP was measured only in a clinic environment for a limited number of times (e.g., annual physical examinations, cohort follow-ups every 2–3 years), which was only a snapshot and may not reflect one's

BP status in normal times (also referred to as a white-coat effect), making their findings less stable and reliable. Ambulatory blood pressure (ABP) monitoring can capture dynamic BP over time, which could more accurately describe one's BP profile and be more reliable than conventional, clinic-based BP measurements[15]. Limited studies have examined the effects of air pollutants on ABP, and their conclusions have been inconsistent. For example, a multi-city study of 7,108 adults in China found that very short-term (e.g., within 12 h) exposure to $\text{PM}_{2.5}$ was associated with the participants' elevated BP, measured by a portable blood-pressure meter every 30 or 60 min for 24 consecutive hours[16]. However, the exposure to air pollutants in all these existing studies has been calculated as the $\text{PM}_{2.5}$ concentrations over the participants' residence, which were either measured at the nearest fixed-site monitoring stations to the participants' residence, or averaged from satellite-derived estimates over one's residence during a given period of time (ranging from three months to three years)[17, 18]. These methods, without considering individuals' movement trajectory, cannot accurately estimate the real-world exposure to $\text{PM}_{2.5}$ due to a spatiotemporal mismatch between $\text{PM}_{2.5}$ and BP measurements[19]. Moreover, the impacts of other air pollutants than $\text{PM}_{2.5}$ remain unknown. Therefore, accurate measurements of multiple air pollutants and BP at a high spatiotemporal resolution are necessary for a comprehensive understanding of their associations.

To fill the aforementioned gaps, this study aimed to examine the effects of the short-term exposure to common air pollutants on ABP, on the basis of their high-resolution dynamic measures and spatiotemporal match. The daily concentrations of air pollutants were estimated at a spatial resolution of 1 km by a newly developed multi-output LightGBM model, which has demonstrated better accuracy than all existing products of air pollutant concentrations. Wearable device developed by the International Institute of Spatial Lifecourse Health (ISLE) were used to measure individuals' real-time BP and movement trajectory, which allows more accurate estimation of individuals' exposure to air pollutants. The findings are expected to help multiple stakeholders, including people with hypertension, physicians, and policy-makers, better understand the effects of air pollutants on the risk for hypertension, and have important public health implications for BP prediction and management at large

scales. They would also provide solid evidence for future intervention design and policy making aimed at alleviating air pollution and improving health equity.

Materials and Methods

Study Participants

The 172 participants of this study were recruited from Pujiang County of Chengdu, the capital city of Sichuan Province in West China, via a multi-stage stratified cluster random sampling method. Individuals were invited to participate in the survey if meeting all the following criteria: 1) living at the current residence over the past three years or longer; 2) having no pulmonary, cardiovascular, and other chronic diseases; and 3) having no severe mental or terminal illnesses. The survey consists of a face-to-face questionnaire-based interview collecting individuals' sociodemographic and lifestyle information at enrollment, and a 1-week real-time monitoring of physiological indicators (e.g., BP) and movement trajectories by a wearable device in July 2023. After excluding the ones that had missing information on basic sociodemographic and lifestyle characteristics, or did not have time to return the wearable device, 140 participants were finally included in this study. Only anonymous data were used for this study for confidentiality protection. The study was approved by the Ethics Committee of Wuhan University (WHU-LFMD-IRB2023018), and written informed consent was obtained from all participants prior to the survey.

ABP Measurements

The ambulatory systolic blood pressure (ASBP, mmHg) and ambulatory diastolic blood pressure (ADBP, mmHg) of each participant were measured, with an interval of 1 min during 8 am and 10 pm (devices were being charged during 10 pm and 8 am) in 7 consecutive days, by a wearable device using photoplethysmography with the accuracy of up to 97% [20]. The wearable device was bound to the left wrist under the instruction of the trained staffs at the beginning of the 1-week monitoring, which can automatically record the time (hour and minute in the 24-h system) and location (latitude

and longitude coordinates in the World Geodetic System 1984) of each measurement. Participants were instructed to carry out their usual daily activities throughout the monitoring period, ensuring that the data reflected routine behaviors and exposure. To make data collection smooth and easy, the device was programmed to automatically upload measurements to the backstage server throughout the study period, without any actions required from the participants. In cases of failed readings, it could continue attempting measurements until a valid result was obtained. Furthermore, at 8 am every morning during the study period, the trained staffs checked the backstage server and confirmed all devices were functioning properly, to minimize missing values and enhance data integrity.

Exposure Measurements

The exposure to air pollutants was calculated as the mean daily concentrations of six air pollutants (i.e., $PM_{2.5}$, PM_{10} , NO_2 , SO_2 , O_3 , and CO) over one's movement trajectory during the 1-week study period, on the basis of the daily products of these pollutants. The daily concentrations of air pollutants were modeled at a spatial resolution of 1 km by a two-stage machine learning model, coupled with a synthetic minority oversampling technique and a tree-based gap-filling method, on the basis of multiple data sources including ground observations, satellite aerosol optical depth, operational chemical transport model simulations, and other ancillary data (e.g., meteorological fields, land use data, population, and elevation) [21]. Also, the concentrations over one's movement trajectory during a day were matched to ABP records for all participants by time (hour and minute) and location information.

Covariates

The covariates adjusted in the models included age ($<45, \geq 45$ years), sex (male, female), occupation (employed, unemployed), marital status (married, unmarried), and annual household income ($\leq 10,000$, 10,000–50,000, 50,000–100,000, and $> 100,000$ Chinese yuan).

Statistical Analysis

The characteristics of the participants were presented as the number and percentage for categorical variables, and as the mean and standard deviation (SD) for continuous variables. Linear mixed-effect models were fit to estimate associations between individuals' exposure to air pollutants and BP changes, expressed as coefficient (β) and 95% confidence interval (CI), with random intercepts accounting for correlations of repeated BP measurements at the individual level. Subgroup analysis was conducted to examine variations of those associations across age, sex, occupation, and marital status, with the significance of differences between the subgroups examined by Z-tests. Distributed lag non-linear mixed-effect models (DLNM) were used to estimate the lag effects and cumulative effects of the air pollutants on BP. As BP was measured over the 14 consecutive hours (8 am to 10 pm) during the waking time every day, 14 h were used as the maximum lag with natural cubic splines with 3 degrees of freedom for air pollutants and 3 degrees of freedom for lag space to establish the cross-basis function in DLNM. Moreover, all covariates were used as fixed-effect terms and a natural cubic spline with 7 degrees of freedom was used to control for time trends in DLNM. The 14 h were also used as the maximum lag for plotting exposure–response curves for the overall cumulative effects of air pollutant exposure on ASBP and ADBP. Sensitivity analysis was conducted by varying the degree of freedom of time trends from 6 to 8. All statistical analyses were performed in R (version 4.3.0) with the “lme4” package for fitting linear mixed-effect models and the “dlnm” package for fitting DLNM. All tests were 2-sided, with the level of significance set as 0.05.

Results

Characteristics of the Study Subjects

The 140 participants had a mean age of 46.6 ± 15.7 , with 58.6% being female (Table 1). Among them, 83.6% were employed and 82.9% were married; more than

Table 1 Characteristics of the study participants

Variables	Number (%) or Mean \pm SD All ($n = 140$)
Sex	
Male	58 (41.4)
Female	82 (58.6)
Age (year)	46.6 ± 15.7
< 45	55 (39.3)
≥ 45	85 (60.7)
Occupation status	
Employed	117 (83.6)
Unemployed	23 (16.4)
Marital status	
Married	116 (82.9)
Unmarried	24 (17.1)
Annual household income (Chinese yuan)	
$\leq 10,000$	15 (10.7)
$> 10,000$ but $\leq 50,000$	90 (64.3)
$> 50,000$ but $\leq 100,000$	26 (18.6)
$> 100,000$	9 (6.4)
Daily mean concentration of air pollutants	
PM _{2.5} ($\mu\text{g}/\text{m}^3$)	32.6 ± 6.8
PM ₁₀ ($\mu\text{g}/\text{m}^3$)	153.6 ± 22.4
NO ₂ ($\mu\text{g}/\text{m}^3$)	16.2 ± 4.2
SO ₂ ($\mu\text{g}/\text{m}^3$)	11.2 ± 1.5
O ₃ ($\mu\text{g}/\text{m}^3$)	88.8 ± 14.0
CO (mg/m^3)	0.6 ± 0.1
Daily mean level of ABP	
ASBP (mmHg)	120.7 ± 8.4
ADBP (mmHg)	76.0 ± 4.3

ABP ambulatory blood pressure, ADBP ambulatory diastolic blood pressure, ASBP ambulatory systolic blood pressure

half had a household annual income of 10,000–50,000 Chinese *yuan*. The average ASBP and ADBP of the participants were 120.7 ± 8.4 mmHg and 76.0 ± 4.3 mmHg, respectively. The individuals' average ASBP and ADBP ranged from 102.0–140.0 mmHg and from 69.0–112.0 mmHg, respectively. In general, the average concentrations of PM_{2.5}, PM₁₀, NO₂, SO₂, O₃, and CO over one's moving trajectory were 32.6 ± 6.8 $\mu\text{g}/\text{m}^3$, 153.6 ± 22.4 $\mu\text{g}/\text{m}^3$, 16.2 ± 4.2 $\mu\text{g}/\text{m}^3$, 11.2 ± 1.5 $\mu\text{g}/\text{m}^3$, 88.8 ± 13.4 $\mu\text{g}/\text{m}^3$, and 0.6 ± 0.1 mg/m^3 , respectively (Fig. 1).

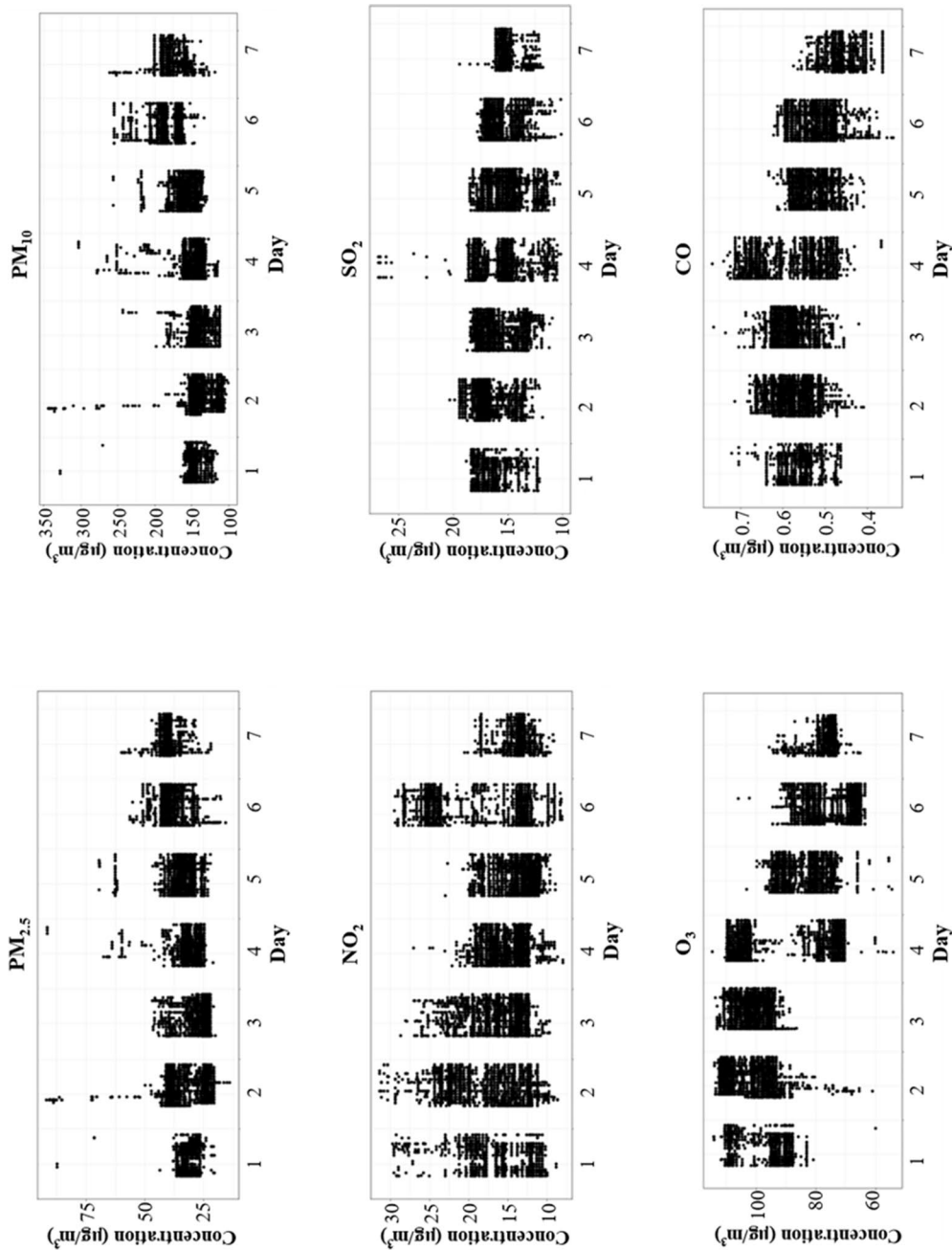


Fig. 1 Scatter plots of the air pollutant concentrations the participants were dynamically exposed to in seven days of the week. Each point denotes a record of air pollutant concentration one was exposed to at a given time and location

Associations between Air Pollutant Exposure and ABP

In crude models, the increased ASBP was associated with each unit increase in the concentration of PM_{2.5} ($\beta=0.010$ [95% CI: 0.005, 0.015]), PM₁₀ ($\beta=0.006$ [0.005, 0.008]), and SO₂ ($\beta=0.046$ [0.021, 0.071]), and decrease in NO₂ ($\beta=-0.009$ [-0.017, -0.001]) (Table 2). After adjusting for covariates, the associations remained significant ($\beta=0.010$ [0.005, 0.015] for PM_{2.5}, $\beta=0.006$ [0.005, 0.008] for PM₁₀, $\beta=0.046$ [0.021, 0.071] for SO₂, and $\beta=-0.009$ [-0.017, -0.001] for NO₂). The increased ADBP in crude models was associated with each unit increase in the concentration of PM_{2.5} ($\beta=0.008$ [95% CI: 0.005, 0.010]) and PM₁₀ ($\beta=0.003$ [0.002, 0.004]), each unit decrease in NO₂ ($\beta=-0.008$ [-0.012, -0.004]) and O₃ ($\beta=-0.002$ [-0.004, -0.001]), and CO ($\beta=-0.373$ [-0.658, -0.087]). Similarly, the associations remained significant after adjusting for covariates ($\beta=0.008$ [0.005, 0.010] for PM_{2.5}, $\beta=0.003$ [0.002, 0.004] for PM₁₀, $\beta=-0.008$ [-0.012, -0.004] for NO₂, $\beta=-0.002$ [-0.004, -0.001] for O₃, $\beta=-0.366$ [-0.652, -0.081] for CO).

Variations of the Associations between Air Pollutants and ABP

The ABP changes corresponding to each unit increase in the concentrations of air pollutants varied across subgroups (Fig. 2). PM_{2.5} was positively associated with ASBP in most subgroups, which were stronger in females ($\beta=0.022$ [0.016, 0.028]) than males ($\beta=-0.006$ [-0.013, 0.001]), in those aged < 45 ($\beta=0.023$ [0.016, 0.031]) than ≥ 45 years ($\beta=0.004$ [-0.002, 0.009]), in the unemployed ($\beta=0.030$ [0.018, 0.042]) than employed ($\beta=0.007$ [0.002, 0.012]), and in the unmarried ($\beta=0.044$ [0.033, 0.056]) than married ($\beta=0.005$ [-0.001, 0.010]). PM_{2.5} was also positively associated with ADBP in all subgroups, stronger in those aged < 45 ($\beta=0.14$ [0.010, 0.018]), the unemployed ($\beta=0.014$ [0.008, 0.020]), and the unmarried ($\beta=0.021$ [0.015, 0.027]). Similarly, PM₁₀ was positively associated with ASBP in most subgroups, which were stronger in females ($\beta=0.011$ [0.010, 0.013]), those aged < 45 years ($\beta=0.012$ [0.010, 0.015]), the unemployed ($\beta=0.014$ [0.011, 0.018]), and the unmarried ($\beta=0.017$ [0.013, 0.020]). PM₁₀ was also positively associated with ADBP in all subgroups, stronger in females ($\beta=0.004$

Table 2 Associations between each unit increase in the concentration of air pollutants and changes in ambulatory blood pressure

Variables	β (95% CI)			
	Crude	Model 1	Model 2	Model 3
Ambulatory systolic blood pressure (ASBP)				
PM _{2.5} ($\mu\text{g}/\text{m}^3$)	0.010*** (0.005, 0.015)	0.010*** (0.006, 0.015)	0.010*** (0.006, 0.015)	0.010*** (0.005, 0.015)
PM ₁₀ ($\mu\text{g}/\text{m}^3$)	0.006*** (0.005, 0.008)	0.006*** (0.005, 0.008)	0.006*** (0.005, 0.008)	0.006*** (0.005, 0.008)
NO ₂ ($\mu\text{g}/\text{m}^3$)	-0.009* (-0.017, -0.001)	-0.009* (-0.017, -0.001)	-0.009* (-0.017, -0.001)	-0.009* (-0.017, -0.001)
SO ₂ ($\mu\text{g}/\text{m}^3$)	0.046*** (0.021, 0.071)	0.046*** (0.021, 0.071)	0.045*** (0.020, 0.070)	0.046*** (0.021, 0.071)
O ₃ ($\mu\text{g}/\text{m}^3$)	-0.001 (-0.004, 0.001)	-0.001 (-0.004, 0.001)	-0.001 (-0.004, 0.001)	-0.001 (-0.004, 0.001)
CO (mg/m ³)	0.065 (-0.493, 0.622)	0.069 (-0.488, 0.627)	0.071 (-0.486, 0.629)	0.070 (-0.488, 0.627)
Ambulatory diastolic blood pressure (ADBP)				
PM _{2.5} ($\mu\text{g}/\text{m}^3$)	0.008*** (0.005, 0.010)	0.008*** (0.005, 0.010)	0.008*** (0.005, 0.010)	0.008*** (0.005, 0.010)
PM ₁₀ ($\mu\text{g}/\text{m}^3$)	0.003*** (0.002, 0.004)	0.003*** (0.002, 0.004)	0.003*** (0.002, 0.004)	0.003*** (0.002, 0.004)
NO ₂ ($\mu\text{g}/\text{m}^3$)	-0.008*** (-0.012, -0.004)	-0.008*** (-0.012, -0.004)	-0.008*** (-0.012, -0.004)	-0.008*** (-0.012, -0.004)
SO ₂ ($\mu\text{g}/\text{m}^3$)	-0.008 (-0.021, 0.005)	-0.008 (-0.021, 0.005)	-0.008 (-0.021, 0.005)	-0.008 (-0.021, 0.005)
O ₃ ($\mu\text{g}/\text{m}^3$)	-0.002*** (-0.004, -0.001)	-0.002*** (-0.004, -0.001)	-0.002*** (-0.004, -0.001)	-0.002*** (-0.004, -0.001)
CO (mg/m ³)	-0.373* (-0.658, -0.087)	-0.370* (-0.655, -0.084)	-0.368* (-0.653, -0.082)	-0.366* (-0.652, -0.081)

Model 1: Adjusted for sex and age; Model 2: Additionally adjusted for occupation and marital status upon Model 1; Model 3: Additionally adjusted for annual household income upon Model 2

* $p < 0.05$; ** $p < 0.005$; *** $p < 0.001$

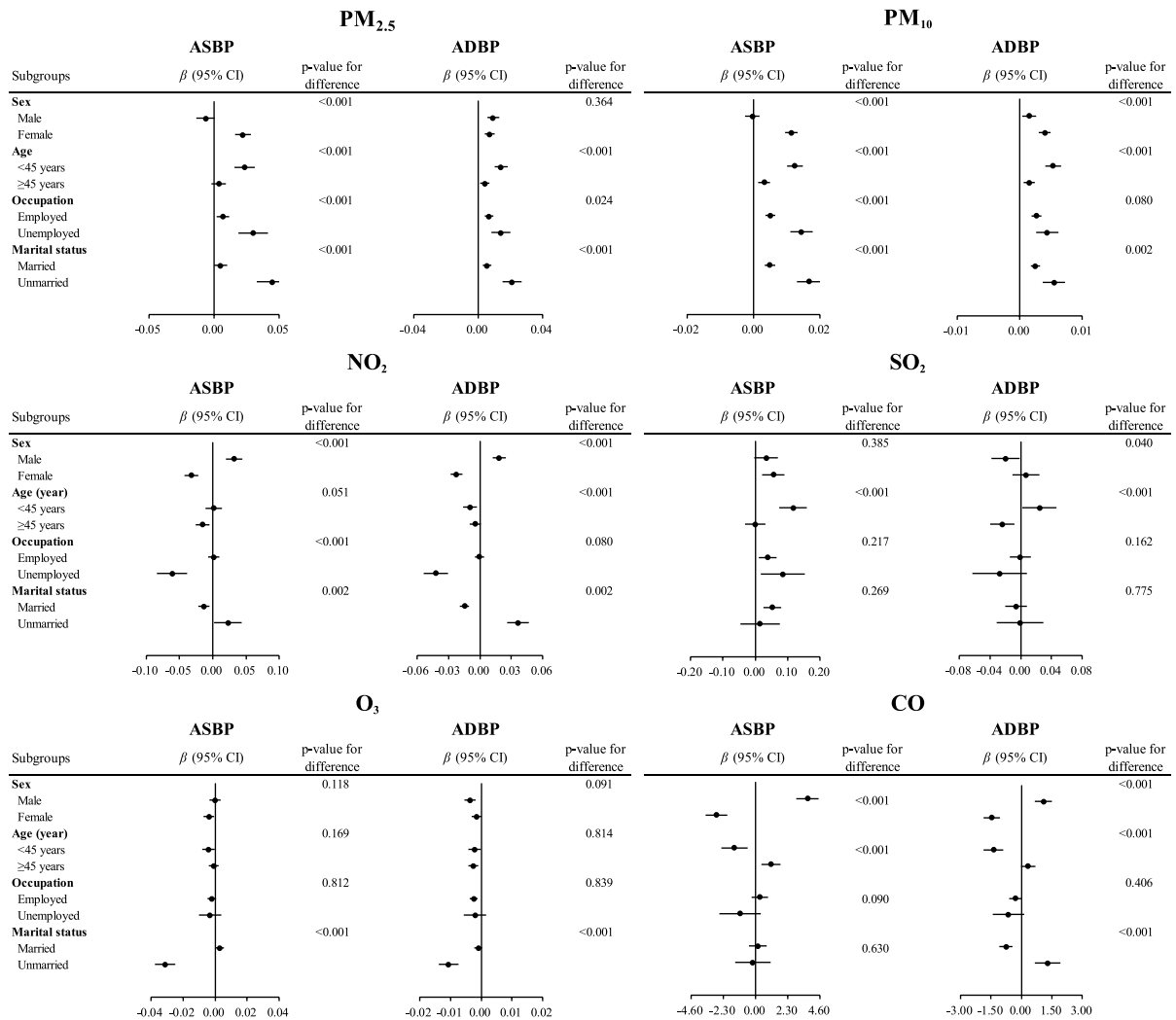


Fig. 2 Associations between air pollutant exposure and ambulatory blood pressure in subgroups. All covariates (age, sex, occupation, and marital status) except for the one used for stratification were adjusted. Z-tests were used to test for sig-

nificance of differences in the association across subgroups. ADBP, ambulatory diastolic blood pressure; ASBP, ambulatory systolic blood pressure

[0.003, 0.005]), those aged <45 years ($\beta=0.005$ [0.004, 0.007]), the unemployed ($\beta=0.004$ [0.003, 0.006]), and the unmarried ($\beta=0.006$ [0.004, 0.007]).

NO₂ and ASBP/ADBP were positively associated in males ($\beta=0.032$ [0.020, 0.044] for ASBP, $\beta=0.018$ [0.012, 0.025] for ADBP) and the unmarried ($\beta=0.023$ [0.002, 0.044] for ASBP, $\beta=0.036$ [0.026, 0.047] for ADBP) while negatively associated in females ($\beta=-0.032$ [-0.043, -0.022] for ASBP, $\beta=-0.022$ [-0.028, -0.017] for ADBP) and the married ($\beta=-0.013$ [-0.022, -0.005] for ASBP, $\beta=-0.015$ [-0.019, -0.010] for ADBP). SO₂ was

positively associated with ASBP in most subgroups, with the stronger association observed in those aged <45 years ($\beta=0.117$ [0.075, 0.159]) relative to those aged ≥45 years. Also, SO₂ was negatively associated with ADBP in males ($\beta=-0.020$ [-0.038, -0.001]) and in those aged ≥45 years ($\beta=-0.024$ [-0.040, -0.008]), while positively associated with ADBP in those aged <45 ($\beta=0.025$ [0.003, 0.047]). O₃ was positively associated with ASBP in the married ($\beta=0.003$ [0.001, 0.005]), while negatively associated with ASBP ($\beta=-0.031$ [-0.037, -0.025]) and ADBP in the unmarried ($\beta=-0.011$

$[-0.014, -0.007]$). CO and ASBP were positively associated in males ($\beta=3.696$ [2.897, 4.494]) and those aged ≥ 45 years ($\beta=1.107$ [0.417, 1.797]), while negatively associated in females ($\beta=-2.780$ $[-3.553, -2.007]$) and those aged <45 years ($\beta=-1.501$ $[-2.440, -0.562]$). Similarly, CO and ADBP were positively associated in males ($\beta=1.082$ [0.677, 1.487]) and those aged ≥ 45 ($\beta=0.334$ $[-0.013, 0.682]$), while negatively associated in females ($\beta=-1.457$ $[-1.856, -1.059]$) and those aged <45 years ($\beta=-1.374$ $[-1.866, -0.883]$).

Lag Effects of Air Pollutants on ABP

The 0–14 h lag effects of air pollutant exposure on ABP changes were estimated. At lag 0, ASBP was positively associated with exposure to PM_{10} ($\beta=1.133$ [0.162, 2.104] mmHg), and negatively associated with O_3 ($\beta=-3.069$ $[-0.771, -5.368]$ mmHg) and CO ($\beta=-1.553$ $[-0.151, -2.954]$ mmHg); ADBP was negatively associated with exposure to CO ($\beta=-1.172$

$[-0.453, -1.898]$ mmHg) (Fig. 3). These lag effects occurred immediately at the time of exposure, and attenuated thereafter and became not significant approximately at lags 0–2 h. At lags 2–4 h, exposure to SO_2 was positively associated with both ASBP and ADBP, which became negative at lags 8–12 h. At lags 12–14 h, exposure to $PM_{2.5}$ and O_3 was positively associated with ASBP, while NO_2 exposure was negatively associated with ASBP. Moreover, at 14-h lag, ASBP was positively associated with exposure to PM_{10} ($\beta=0.453$ [0.011, 0.884] mmHg), O_3 ($\beta=4.548$ [2.271, 6.828] mmHg) and negatively associated with NO_2 (-1.210 $[-1.981, -0.420]$ mmHg). ADBP was positively associated with exposure to PM_{10} ($\beta=1.811$ [0.790, 2.899] mmHg), O_3 ($\beta=1.273$ [0.211, 2.334] mmHg), and CO ($\beta=0.898$ [0.201, 1.396] mmHg) and negatively associated with NO_2 ($\beta=-1.66$ $[-0.162, -3.198]$ mmHg). The results remained robust after varying the time trends from 6 to 8 degrees of freedom (Figure S1).

The cumulative effects of air pollutant exposure for 14 h on ABP were also estimated. The cumulated

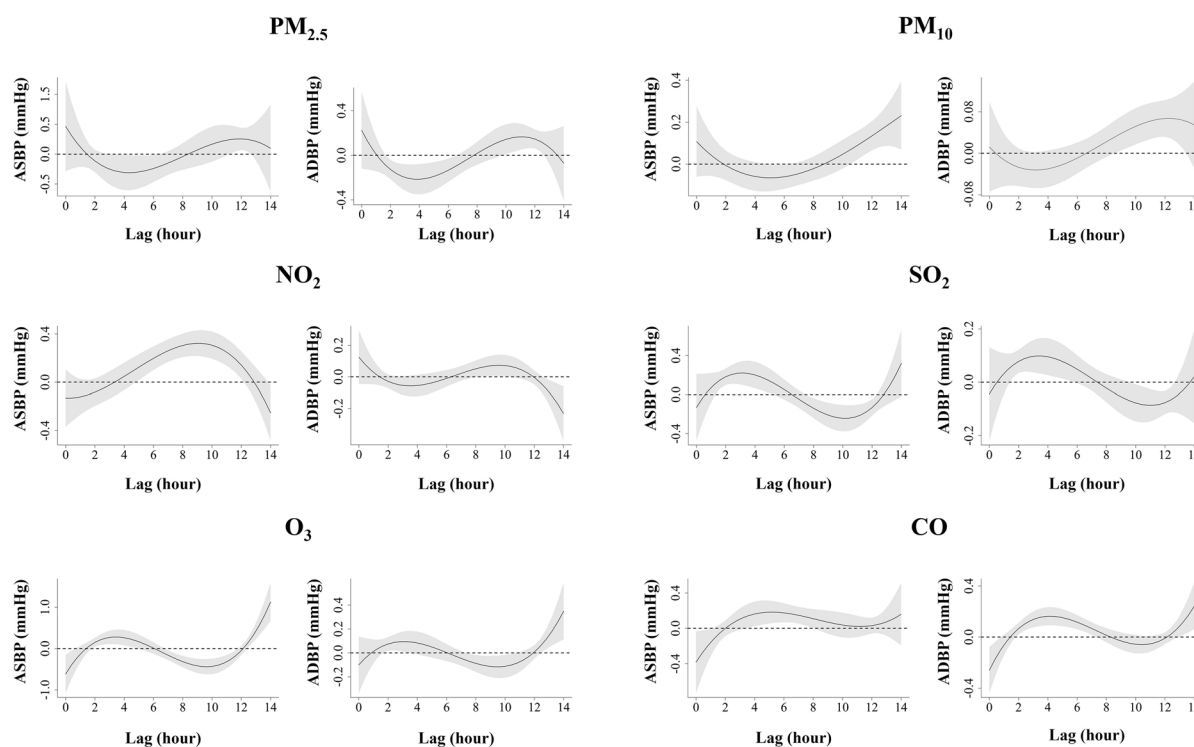


Fig. 3 Associations between air pollutant exposure and ambulatory blood pressure with 0–14 h lag. The solid curves denote the estimated effects, with the gray areas representing the 95%

confidence intervals. ADBP, ambulatory diastolic blood pressure; ASBP, ambulatory systolic blood pressure

exposure to PM_{10} was associated with ASBP and ADBP, particularly at the lower levels of concentration (Fig. 4). The cumulated exposure to CO was also associated with ASBP and ADBP, while SO_2 exposure was associated with ASBP and ADBP when its concentration was over $15 \mu\text{g}/\text{m}^3$. Exposure to NO_2 was associated with only ASBP, while exposure to O_3 was associated with only ADBP at all levels of concentration.

Discussion

To the knowledge of the authors, this is the first study examining the effects of the real-world exposure to the six major air pollutants on ABP. It revealed that short-term exposure to higher concentrations of particulate matter (PM_x) was significantly associated with the increased ABP, while exposure to higher concentrations of some gaseous pollutants (e.g., NO_2 and O_3) was significantly associated with the

decreased ABP. After 14-h lag, exposure to PM_{10} , O_3 , and CO was associated with the increased ABP, while exposure to NO_2 was associated with the decreased ABP. The cumulative effects of air pollutant exposure for 14 h on ABP were generally adverse. The cumulated exposure to PM_{10} and CO was associated with ABP at most levels of concentration, while SO_2 exposure was associated with ABP only when its concentration was over $15 \mu\text{g}/\text{m}^3$. In addition, stronger associations were mainly observed in males, the unemployed, and the unmarried.

The transient exposure to particulate matter ($\text{PM}_{2.5}$ and PM_{10}) exposure was associated with elevated ABP in this study, which was consistent with some previous findings. For example, a multi-city study of 7,108 adults in China found that transient $\text{PM}_{2.5}$ exposure was associated with elevated ABP [16]. A possible biological mechanism is that $\text{PM}_{2.5}$ inhaled into the lung could stimulate many subtypes of nervous system receptors in the airways, leading to cardiovascular autonomic nervous system imbalance and larger

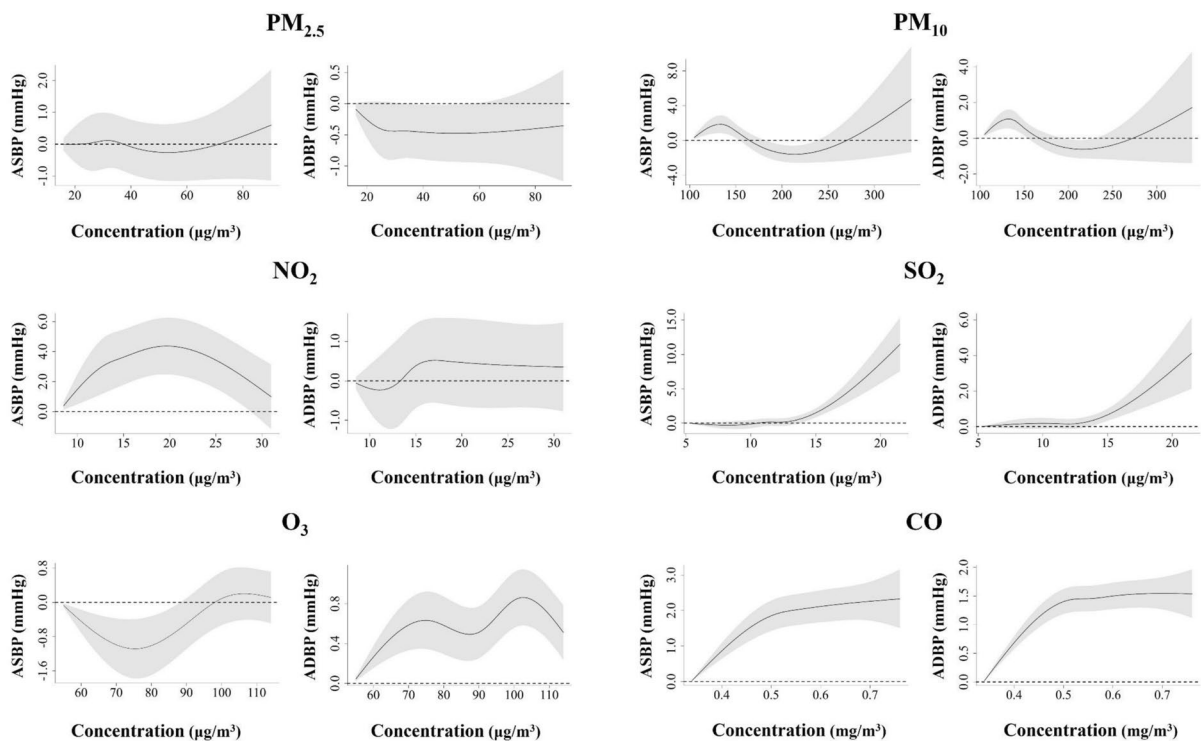


Fig. 4 Exposure–response curves for cumulative changes in ABP over 14 h (compared to the minimum observed concentration of each air pollutant). The solid curves denote the aver-

age effect estimates, with the gray areas representing the 95% confidence intervals

systemic effects relevant to higher BP, such as inflammation, oxidative stress, and vasoconstriction[11, 22]. However, that study matched the concentrations of air pollutants measured at the nearest fixed-site monitoring stations to the study subjects as surrogates for personal exposure, without considering individuals' spatial movement (e.g., outdoor activities), which may introduce inevitable exposure measurement errors. Another Chinese study of 37 college students measured personal exposure to PM_{2.5} by personal aerosol monitors for three consecutive days, and found that short-term exposure to PM_{2.5} was associated with decreased ABP[18]. The inconsistent results with the ones from this study may be due to the limited sample size and lack of generalizability of the findings from college students who usually shared similar daily activities (e.g., taking classes in the classrooms, dining in the canteens on campus, resting in the dormitories). In our study, wearable device was used to monitor individual real-time BP and movement trajectories among the subjects at all ages during the 1-week study period, which have reduced exposure and outcome misclassification, and also improve the generalizability and hence reliability of the findings.

In previous studies, the associations between exposure to gaseous pollutants and ABP were also inconsistent (sometimes contradictory)[23–25]. In this study, we observed that most gaseous pollutants (e.g., NO₂, O₃, and CO) involved in this study were negatively associated with ABP, which was inconsistent with previous findings. For example, a study of 48 male adults in Brazil found that the increases in daily concentrations of SO₂ and CO were associated with the increased BP, while NO₂ had no statistically significant effects on BP[23]. Unlike particulate matter that could directly stimulate many nervous system receptors in the airways after inhalation and hence lead to BP imbalance[18, 24], inhaling gaseous pollutants may cause more complex chemical reactions, and the mechanisms on human health effects remain not fully understood[25]. Although most gaseous pollutants are toxic to human body, some studies have found that inhaling a certain dose of NO, O₃, and CO may also lead to vasodilation and lower BP for a short period[26–28]. For example, although long-term exposure to O₃ could cause oxidative stress, inflammation of the airways, and even premature deaths in general population[29], some studies also showed that short-term exposure to a higher concentration

of O₃ may not only have no acute or chronic health effects on the human body, but also trigger several beneficial biochemical mechanisms and reactivate the antioxidant system, which has been widely used in the treatment of cardiovascular diseases including hypertension[28].

Elucidating the lag and cumulative effects of exposure to air pollutants on BP changes have great implications for BP management[30]. This study suggested that ABP was positively associated with exposure to PM₁₀, O₃, and CO, and negatively associated with NO₂ after 14-h lag. Previous studies have explored potential lag patterns between PM_{2.5} with ABP. For example, a multi-city study in China found that the association between PM_{2.5} and ASBP was strongest at the concurrent hour of exposure, and gradually weakened about 12 h later[16]. Another study of 132 adults in China and the US observed that the increased exposure to PM_{2.5} was associated with a significant increase in DBP with a 1-day lag, and then gradually weakened[31]. Gaseous pollutants (e.g., O₃) have showed strong oxidation after inhalation, which may cause cardiac physiological impairments and adversely affect the cardiovascular system[32]. However, the potential lag patterns of gaseous pollutants remain unclear. Moreover, we observed the thresholds of exposure to SO₂ for the adverse cumulative effects on ASBP and ADBP, which has not been reported in previous studies. One possible explanation is that SO₂ in blood vessel may be first converted to hydrogen sulfide in neutrophil that has physiological functions (e.g., vasorelaxation)[33], and, with the concentration of ambient SO₂ increases, the higher concentration of hydrogen sulfide than a certain threshold may result in the increased BP[33]. Hence, understanding the lag and the cumulative effects of the major air pollutants on ABP is crucial to reduce the incidence of hypertension and disease burden attributable to air pollutants. By using wearable devices to measure ABP frequently, this study provided convincing evidence and contributed to new knowledge on the lag and cumulative effects of air pollutants on BP changes.

The stronger associations between air pollutants and elevated BP were observed in male, those aged <45 years, and the unemployed and unmarried. Some existing studies suggested that males and younger people may generally participate in more outdoor activities and work, which may increase

their exposure to air pollutants[8, 18]. Similarly, the unemployed and unmarried people may tend to have a larger range of activities than the employed and married people who usually spend a considerable part of time in the workplace and at home[8]. In addition, we also observed that CO was positively associated with BP changes in males and those aged ≥ 45 years, while negatively associated in females and those aged < 45 years. Although CO has been recognized as a cardiovascular toxin, some animals studies found that it could change the respiratory rhythm driven by the central nervous system, which may decreased BP[34, 35]. However, some possible biological mechanisms, including systemic inflammation and endothelial dysfunction, may contribute to the positive association between CO and BP in older people[35]. Also, the cardiac function of the young people may be better adapted to the external environment, while the old people with body organs deteriorating with age may be rather susceptible to air pollutants and hence more sensitive to the impact of CO[36].

Our study has several strengths. First, we used wearable devices to collect individual ABP, which could minimize the influence of “white coat phenomenon”. Wearable devices have been considered the most accurate tool, so far, for short-term individual exposure assessment[37]. Compared to other large automated devices, wearable devices are more convenient to use, making much less interference in participants’ daily activities. Second, the high-resolution concentrations of air pollutants, modeled by advanced methods, and the individual movement trajectories, collected by wearable devices over the day, allow us to better estimate the real-world exposure to the six major air pollutants, which further enables a better examination of the effects of short-term exposure to air pollutants on ABP changes, as well as the lag patterns over hours. Compared to using the concentration of air pollutants from the nearest fixed-site monitoring stations to calculate the exposure of the participants, our approach could refine the assessment of air pollutant exposure and reduce measurement errors, leading to more reliable results, which is of great significance to the comprehensive understanding of health effects of air pollutants.

Some limitations in this study need to be acknowledged. First, although improved upon the previous

efforts by including the participants with a range of sociodemographic characteristics, the sample size and spatial distribution of the participants are still limited. Specifically, all participants were healthy adults without the known chronic diseases diagnosed, and the levels of air pollutants were lower than the average levels in China, which may affect (possibly underestimate) the results of this study to be generalized to other populations and regions[38]. Future efforts would expand the study population and region to further validate and strengthen the generalizability of these findings. Second, we did not mandate the participants to refrain from hypertension-related behaviors during the study period, such as caffeine and alcohol drinking. Moreover, all participants were instructed to maintain their usual daily activities throughout the study period, occasional increases in short-term exposure to higher concentrations of air pollutants may have occurred (e.g., when participants spent time near high-traffic roads or industrial zones), which may to some extent affect the findings of this study. Third, although using the best products of air pollutants with the highest spatiotemporal resolution to date, the accuracy of such products still has space to improve, e.g., by including some local measurements of air pollutant concentrations whenever available. Also, similar as most, if not all, of the existing studies in this field, outdoor and indoor exposures to air pollutants were not differentiated. However, the outdoor and indoor concentrations of air pollutants were found highly correlated, which, plus the fine-scale spatiotemporal match between air pollutant concentrations and movement trajectories, may still make our estimations reasonable and reliable. Fourth, the maximum lag was selected to be 14 h due to the limited maintenance of the battery in the wearable device (the device was being charged during 10 pm and 8 am, and cannot measure ABP). That is to say, the effects of air pollutants at, for example, 9 pm, can only have 1 h of lagged response (up to 10 pm), which is a compromise between the duration of the observations and the temporal resolution of the measurements. This limitation could be partially overcome in future research if the interval between two measurements (1 min in this study) was increased to make the battery stay longer, at the expense of the temporal resolution of ABP measurements.

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