Association of long-term exposure to PM$_{2.5}$ with hypertension prevalence and blood pressure in China: a cross-sectional study

Jiali Song, Yan Gao, Shuang Hu, Emanuela Medda, Guigang Tang, Di Zhang, Wenbo Zhang, Xi Li, Jing Li, Matteo Renzi, Maria Antonietta Stazi, Xin Zheng

**ABSTRACT**

**Objective** Evidence of the effects of long-term fine particulate matter (PM$_{2.5}$) exposure on hypertension and blood pressure is limited for populations exposed to high levels of PM$_{2.5}$. We aim to assess associations of long-term exposure to PM$_{2.5}$ with hypertension prevalence and blood pressure, and further explore the subpopulation differences and effect modification by participant characteristics in these associations in China.

**Methods** We analysed cross-sectional data from 883,827 participants aged 35–75 years in the China Patient-Centred Evaluative Assessment of Cardiac Events Million Persons Project. Data from the monitoring station were used to estimate the 1-year average concentration of PM$_{2.5}$. The associations of PM$_{2.5}$ exposure with hypertension prevalence and blood pressure were investigated by generalised linear models, with PM$_{2.5}$ included as either linear or spline functions.

**Results** The 1-year PM$_{2.5}$ exposure of the study population ranged from 8.8 to 93.8 μg/m$^2$ (mean 49.2 μg/m$^2$). The adjusted OR of hypertension prevalence related to a 10 μg/m$^2$ increase in 1-year PM$_{2.5}$ exposure was 1.04 (95% CI, 1.02 to 1.05). Each 10 μg/m$^2$ increment in PM$_{2.5}$ exposure was associated with increases of 0.19 mm Hg (95% CI, 0.10 to 0.28) and 0.13 mm Hg (95% CI, 0.08 to 0.19) in systolic blood pressure and diastolic blood pressure, respectively. The concentration–response curves for hypertension prevalence and systolic blood pressure showed steeper slopes at higher PM$_{2.5}$ levels; while the curve for diastolic blood pressure was U-shaped. The elderly, men, non-current smokers and obese participants were more susceptible to the exposure of PM$_{2.5}$.

**Conclusions** Long-term exposure to PM$_{2.5}$ is associated with higher blood pressure and increased risk of hypertension prevalence. The effects of PM$_{2.5}$ on hypertension prevalence become more pronounced at higher PM$_{2.5}$ levels. These findings emphasise the need to reduce air pollution, especially in areas with severe air pollution.

**INTRODUCTION**

Hypertension is the leading risk factor for death globally. Although hypertension is a worldwide public health concern, three-quarters of the world’s population with the condition are living in low-income and middle-income countries (LMICs). The causes of hypertension are complex. Apart from genetic predisposition, social determinants and lifestyle factors, air pollution, especially fine particulate matter with an aerodynamic diameter of 2.5 μm or less (PM$_{2.5}$), may also contribute to increased risk for hypertension.

Over the past few years, a growing body of epidemiological evidence indicated the association of long-term exposure to PM$_{2.5}$ with blood pressure and hypertension. However, most of the studies were undertaken in high-income countries. Compared with high-income countries, exposure to PM$_{2.5}$ is substantially higher in LMICs. Furthermore, prior studies have largely focused on specific populations or regions. Therefore, further studies are needed to assess the associations of PM$_{2.5}$ with blood pressure and prevalence of hypertension among a wider spectrum of populations with high PM$_{2.5}$ concentrations, especially in LMICs.
China is experiencing a growing epidemic of hypertension and is estimated to have 300 million individuals with hypertension by 2025.1,11 Meanwhile, outdoor PM$_{2.5}$ has become one of China’s most serious environmental problems with population-weighted annual means of PM$_{2.5}$ ranging from 19.1 to 79.3 µg/m$^3$ in 2015.12 A deeper understanding of the chronic health effects of PM$_{2.5}$ on hypertension prevalence and blood pressure in moderate to high PM$_{2.5}$ concentrations will help to develop policies to improve air quality and combat the hypertension epidemic in China.

Accordingly, incorporating PM$_{2.5}$ data with a large-scale population-based screening project in China, the China Patient-Centred Evaluative Assessment of Cardiac Events (PEACE) Million Persons Project, we aimed to: (1) explore the association of long-term PM$_{2.5}$ exposure with blood pressure level and hypertension prevalence, and evaluate subpopulation differences and effect modification by characteristics of participants in these associations; (2) assess the concentration–response relationships of long-term PM$_{2.5}$ exposure with hypertension prevalence and blood pressure.

METHODS

Study population

Our study population is derived from the China PEACE Million Persons Project, which has been described previously.13 In brief, we selected county-level regions using a convenience sampling strategy in all 31 provinces in mainland China from September 2014 to March 2019. These regions are designated as rural counties or urban districts according to urban–rural division codes of the National Bureau of Statistics of China.14 Local residents aged 35–75 years, who were currently registered in the selected region’s Hukou (a record officially identifying a person as a resident of an area) or had lived in the region for at least 6 of the previous 12 months, were enrolled in this project. After excluding participants with missing data on education level (n=13 714), body mass index (n=321) or blood pressure measurement (n=2), we further excluded participants with systolic blood pressure (SBP) ≥140 mm Hg or diastolic blood pressure (DBP) of 90 mm Hg or higher, BP difference of 10 mm Hg, a third measurement was obtained and the average of the last two readings was used. Hypertension was defined as SBP of 140 mm Hg or higher, DBP of 90 mm Hg or higher or use of antihypertensive medications, which is consistent with the US Joint National Committee and Chinese definitions.15 16 Body mass index (BMI) was defined as weight in kilograms divided by height in square metres. Obesity was defined as 28.0 kg/m$^2$ or higher, based on recommendations from the Working Group on Obesity in China.17

Exposure assessment

We geocoded each participant’s current address (either rural counties or urban districts) into latitude and longitude data and identified air monitors located within 10 km (online supplemental file 2). The average distance between the address of participants and assigned monitors was 2.7 (IQR 1.2–3.5) kilometres. The measurements from these monitors strictly followed the methodological standards set by the State Environmental Protection Administration of China. For each participant, daily average PM$_{2.5}$ concentrations measured at the nearest monitors to the residence were used to estimate PM$_{2.5}$ exposure. The 1-year average concentration before the medical examination was calculated and treated as an indicator of long-term exposure to PM$_{2.5}$. In the present study, we included participants with more than 330 valid PM$_{2.5}$ values for assessing long-term exposure, to ensure that, for each participant, the missing rate of PM$_{2.5}$ data in the preceding 1 year of medical examination is less than 10%.18

Statistical analysis

Continuous variables were reported as means with SD; categorical variables were presented as percentages. We developed mixed models with a logit link function to assess the effect of long-term exposure to PM$_{2.5}$ on hypertension prevalence. To assess the association of PM$_{2.5}$ and blood pressure, SBP and DBP were modelled using linear regressions with township-specific random intercepts. For each of these analyses, we started with a model (model 1) which only included age and sex. We then incrementally adjusted for additional covariates. The second model (model 2) included model 1 and socioeconomic factors (education level and urbanity). The third model (model 3) included model 2 and cardiovascular disease risk factors (BMI, smoking status, alcohol consumption and diabetes). Models of blood pressure were controlled for hypertensive medication use; while models of hypertension were not, as hypertension medication use was a

frequency of each drug. Those who did not remember the exact dose stated the number of pills or tablets taken (online supplemental file 1).

The blood pressure of each participant was measured twice on the right upper arm after 5 min of rest in a seated position with a standardised electronic blood pressure monitor (Omron HEM-7430). If the difference between the two SBP readings was greater than 10 mm Hg, a third measurement was obtained and the average of the last two readings was used. Hypertension was defined as SBP of 140 mm Hg or higher, DBP of 90 mm Hg or higher or use of antihypertensive medications, which is consistent with the US Joint National Committee and Chinese definitions.13 16 Body mass index (BMI) was defined as weight in kilograms divided by height in square metres. Obesity was defined as 28.0 kg/m$^2$ or higher, based on recommendations from the Working Group on Obesity in China.17

Statistical analysis

We geocoded each participant’s current address (either rural counties or urban districts) into latitude and longitude data and identified air monitors located within 10 km (online supplemental file 2). The average distance between the address of participants and assigned monitors was 2.7 (IQR 1.2–3.5) kilometres. The measurements from these monitors strictly followed the methodological standards set by the State Environmental Protection Administration of China. For each participant, daily average PM$_{2.5}$ concentrations measured at the nearest monitors to the residence were used to estimate PM$_{2.5}$ exposure. The 1-year average concentration before the medical examination was calculated and treated as an indicator of long-term exposure to PM$_{2.5}$. In the present study, we included participants with more than 330 valid PM$_{2.5}$ values for assessing long-term exposure, to ensure that, for each participant, the missing rate of PM$_{2.5}$ data in the preceding 1 year of medical examination is less than 10%.18

Statistical analysis

Continuous variables were reported as means with SD; categorical variables were presented as percentages. We developed mixed models with a logit link function to assess the effect of long-term exposure to PM$_{2.5}$ on hypertension prevalence. To assess the association of PM$_{2.5}$ and blood pressure, SBP and DBP were modelled using linear regressions with township-specific random intercepts. For each of these analyses, we started with a model (model 1) which only included age and sex. We then incrementally adjusted for additional covariates. The second model (model 2) included model 1 and socioeconomic factors (education level and urbanity). The third model (model 3) included model 2 and cardiovascular disease risk factors (BMI, smoking status, alcohol consumption and diabetes). Models of blood pressure were controlled for hypertensive medication use; while models of hypertension were not, as hypertension medication use was a
component of the outcome definition. To account for potential time-variant factors, we additionally adjusted for the day of week (one indicator variable per day) and season of measurement (summer: June–August; fall: September–November; winter: December–February; spring: March–May). We also used restricted cubic splines to characterise the concentration–response (C–R) relationships of PM$_{2.5}$ with hypertension prevalence and blood pressure.$^{19}$ In addition, to examine effect modification by age, gender, smoking status, alcohol consumption, diabetes and obesity, each potential modifier was tested by adding an interaction term in the regression model separately and testing its statistical significance as well as the association per categories of the tested variable through subgroup analyses.

Analyses were conducted with SAS V.9.4, 64-bit Windows (SAS Institute, Cary, North Carolina). All tests of significance were two-tailed, with a level of significance set at an alpha of 0.05.

Patient and public involvement

Patients and/or the public were not involved in the design, or conduct, or reporting, or dissemination plans of this research.

RESULT

Study population and baseline characteristics

We included a total of 883 827 participants distributed in 83 county-level regions of mainland China (online supplemental file 3). The mean age was 55.5 years, 60.4% were women, 43.1% had hypertension, mean SBP was 143.5 mm Hg, mean DBP was 83.1 mm Hg and 19.4% were taking antihypertensive medications. The 1-year PM$_{2.5}$ exposure of the total study population ranged from 8.8 to 93.8 µg/m$^3$ (mean 49.2 µg/m$^3$). There were 864 119 (97.8%), 842 356 (95.3%) and 706 415 (79.9%) participants with 1-year PM$_{2.5}$ exposure higher than 15 µg/m$^3$ (WHO Interim Target 3 (IT-1)), 25 µg/m$^3$ (WHO Interim Target 2 (IT-2)) and 35 µg/m$^3$ (WHO Interim Target 3 (IT-3)), respectively (online supplemental file 4).

Associations of long-term PM$_{2.5}$ exposure with hypertension prevalence

After adjusting for sociodemographic characteristics and cardiovascular risk factors, the OR of hypertension was 1.09 (95% CI: 1.08 to 1.10) for each 10 µg/m$^3$ increase in PM$_{2.5}$. The adjustment for day of week and season of blood pressure measurements resulted in a slight decrease in the effect estimate of the association of PM$_{2.5}$ exposure with hypertension prevalence (OR: 1.04, 95% CI: 1.02 to 1.05) (online supplemental file 5). For the C–R relationship, the curve showed steeper slopes at high PM$_{2.5}$ exposure levels (ie, higher than ~50 µg/m$^3$). Compared with individuals with PM$_{2.5}$ exposure of 38.5 µg/m$^3$, the adjusted ORs for hypertension of individuals with 15, 25 and 35 µg/m$^3$ of PM$_{2.5}$ exposure were 1.02 (95% CI: 1.01 to 1.04), 1.01 (95% CI: 1.00 to 1.02) and 1.00 (95% CI: 1.00 to 1.00), respectively (figure 1).

Associations of long-term PM$_{2.5}$ exposure with systolic and diastolic blood pressure

In the adjusted model 4, each 10 µg/m$^3$ increment was associated with increases of 0.50 mm Hg (95% CI: 0.41 to 0.59) in SBP and 0.23 mm Hg (95% CI: 0.18 to 0.28)
Open access

in DBP. After adjustment for the day of week and season, there was some reduction in the effect estimates of the associations between PM$_{2.5}$ exposure and blood pressure (0.19 mm Hg (95% CI: 0.10 to 0.28) in SBP; 0.13 mm Hg (95% CI: 0.08 to 0.18) in DBP) (online supplemental file 6). In addition, we found that the shapes of the C–R curves for SBP and DBP were different. The fitted C–R functions showed upward trends with greater effect estimates of PM$_{2.5}$ at higher concentrations for SBP but were generally U-shaped for DBP (figure 2).

Subpopulation difference and effect modification

The associations of PM$_{2.5}$ exposure with hypertension were stronger among men and non-current smokers, compared with their counterparts (figure 3). Gender significantly modified the effects of PM$_{2.5}$ exposure on all three outcomes (all p for interaction <0.05) with stronger associations among men (eg, OR for hypertension per 10 µg/m$^3$ increase in PM$_{2.5}$ of 1.01 (95% CI: 1.00 to 1.03) for women, and 1.06 (95% CI: 1.05 to 1.08) for men); while these associations were not modified by alcohol consumption and prevalence of diabetes (all p for interaction >0.05). Although smoking status was the effect modifiers in the associations of PM$_{2.5}$ exposure with SBP, DBP and hypertension, the impacts of smoking on these associations differed depending on the outcome. We observed greater effect estimates of PM$_{2.5}$ exposure for SBP (elevation in SBP per 10 µg/m$^3$ increase in PM$_{2.5}$ of 0.97 mm Hg (95% CI: 0.77 to 1.17) for the elderly, and −0.02 mm Hg (95% CI: −0.12 to 0.08) for their younger counterparts). Besides, obesity was also found to be an effect modifier in the associations of PM$_{2.5}$ exposure with SBP and DBP, with greater effect estimates in obese participants (figure 4).

**DISCUSSION**

In this study, we investigated the associations between long-term exposure to PM$_{2.5}$ and hypertension prevalence, SBP and DBP. We found that PM$_{2.5}$ was associated with increased risk of hypertension prevalence and elevation of blood pressure. Non-linearity in these associations was also observed. The C–R curves for hypertension and SBP showed steeper slopes for PM$_{2.5}$ concentration above 50 µg/m$^3$; while the C–R curve for PM$_{2.5}$–DBP was U-shaped, with the turning point around 50 µg/m$^3$. The elderly, men, non-current smokers and obese participants appeared to be more susceptible to the exposure of PM$_{2.5}$.

Our study contributes to the existing scientific literature in several ways. First, by including participants in a national cross-sectional survey with long-term exposure to PM$_{2.5}$ ranged from 8.8 to 93.8 µg/m$^3$, we are able to comprehensively assess the association of PM$_{2.5}$ exposure with hypertension prevalence and blood pressure among a more diverse spectrum of population with a wider range of PM$_{2.5}$ concentrations. We found exposure to PM$_{2.5}$ was positively associated with hypertension prevalence and blood pressure. This echoed the data showing that there was an absolute increase of 139 million individuals with hypertension in China during a decade from 2002 to 2013/2014,20 with the national annual mean PM$_{2.5}$ increasing from 39.5 to 47 µg/m$^3$ between 2000 and 2013.21 Specifically, in some high-polluted areas such as Beijing-Tianjin-Hebei region of 2013, the annual average concentrations of PM$_{2.5}$ had reached 98.9 µg/m$^3$, and daily average concentrations had exceeded 300 µg/m$^3$.22 23 Furthermore, the magnitude of the effects for each 10 µg/m$^3$ increment in PM$_{2.5}$ was also similar compared with other studies.3 17 24–26 For example, one study found that each 10 µg/m$^3$ in PM$_{2.5}$ was associated
with increases of 0.45 and 0.07 mm Hg in SBP and DBP, respectively.\textsuperscript{24} And ORs for hypertension prevalence related to a 10 µg/m\textsuperscript{3} increase in PM\textsubscript{2.5} were ranged from 1.01 to 1.14 in prior studies.\textsuperscript{5 25 26} It is also noteworthy that others have reported no or inconsistent associations.\textsuperscript{27 28} Adar et al found no associations between exposures to PM\textsubscript{2.5} and blood pressure based on a longitudinal cohort.\textsuperscript{27} However, this study only included a small fraction of Chinese populations (10\%), and was conducted in the USA with a mean annual average PM\textsubscript{2.5} of 17 µg/m\textsuperscript{3}, which was lower than the present study (49.2 µg/m\textsuperscript{3}).

Second, we provided new information on the C–R relationship between long-term PM\textsubscript{2.5} exposure and hypertension prevalence. Previous research reported a U-shaped relationship with a threshold PM\textsubscript{2.5} of 47.9 µg/m\textsuperscript{3}.\textsuperscript{3} However, in our study, the risk of hypertension associated with PM\textsubscript{2.5} became even more pronounced when the exposure was extended to higher levels. This finding was in line with a prior study based on prospective cohorts that showed there was a stepwise increase in the risk of developing hypertension with increasing quartiles of long-term PM\textsubscript{2.5} exposure.\textsuperscript{29} This result suggests that, for a given decrease in the concentration of PM\textsubscript{2.5}, a greater reduction in excess hypertension prevalence would be obtained in highly polluted regions compared with regions with low to moderate levels of PM\textsubscript{2.5} exposure. In this respect, the implication of air quality improvements in highly polluted regions of China in recent years would be more profound. It has shown that from 2013 to 2018, the annual average concentration of PM\textsubscript{2.5} in Beijing-Tianjin-Hebei region has declined by 49\%.\textsuperscript{30} Considering the population size and baseline PM\textsubscript{2.5} levels in these areas, the public health impact related to PM\textsubscript{2.5} reduction would be huge.

Third, we found evidence of non-linearity in the relationships of PM\textsubscript{2.5} exposure with SBP and DBP. Interestingly, the shapes of curves for SBP and DBP were different. Although the relevant mechanism remains unclear, pathophysiological changes, such as systemic inflammation, atherosclerosis, endothelial dysfunction and increased arterial stiffness,\textsuperscript{4} may have contributed to the observed patterns. The increased arterial stiffness induced by PM\textsubscript{2.5} would initially lead to elevation in SBP and decline in DBP, creating an increased pulse pressure.\textsuperscript{31} While with further increase of arterial stiffness, the heart rates got higher to maintain the stroke volume, which could result in the rise of DBP afterward. In addition, prior studies have reported the effects of PM\textsubscript{2.5} exposure on pulse pressures showing a tendency of rising first and then declining at higher PM\textsubscript{2.5} levels, which partially supports this hypothesis.\textsuperscript{24}

Fourth, we assessed the subpopulation differences through stratified analyses and identified the susceptible individuals to the exposure of PM\textsubscript{2.5}. We observed the large effect estimates of PM\textsubscript{2.5} on SBP among the elderly. Elderly subjects may commonly represent a higher prevalence of pre-existing cardiovascular and respiratory diseases, which may confer susceptibility to PM\textsubscript{2.5}. Also, the results showed that PM\textsubscript{2.5} exposure had larger effects on hypertension in men, and such increased susceptibility may be related to sex-related differences in the deposition localisation and rates of air pollutants; specifically, men have larger airways and slightly lower airway reactivity.\textsuperscript{32} Furthermore, we found stronger associations of PM\textsubscript{2.5} and blood pressure in obese participants. The greater response observed in obese individuals may be attributed to the higher deposition rate of PM\textsubscript{2.5} exposure. This has been demonstrated in overweight children, where there was an increase in tidal volume and resting minute ventilation.\textsuperscript{33} Additionally, smoking status was found to be an effect modifier with smaller effects on hypertension among current smokers. This finding is also supported by prior research.\textsuperscript{8 24} One possible explanation is that smoking and PM\textsubscript{2.5} exposure may share the same pathways in mediating cardiovascular effects and smoking may play a dominant role in smokers. Thus, exposure to PM\textsubscript{2.5} might not exert additional harmful effects via the same pathway.\textsuperscript{24} There is also some potential that the greater effect size in non-current smokers could also be connected to some of them being advised to quit smoking because of multiple comorbidities.

Our study should also be interpreted in the context of several limitations. First, given the nature of the cross-sectional study design, the causal relationship could not be established. Additional research is needed to examine these relationships in a prospective manner. Second, a selection bias is possible because our analysis was restricted to participants with available PM\textsubscript{2.5} data. Third, while we included a number of potential confounders in the analyses, there might be unmeasured confounders that affected the observed associations. In particular, we were unable to control for other confounders such as diet and physical activity, because these data were only available in a subset of the China PEACE Million Persons Project cohort. Fourth, we used the data from the fixed monitors to estimate the exposure of PM\textsubscript{2.5} and did not account for residential proximity to major roads, time-activity patterns and indoor-related characteristics, which would likely result in non-differential measurement errors. However, this approach is commonly used and previous research has indicated that PM\textsubscript{2.5} exposure estimated by the nearest monitor was highly correlated with other sophisticated approaches.\textsuperscript{34} Fifth, other gaseous pollutants, such as NOx and ozone, temperature and noise were not included in this study. As a result, we were not able to determine whether the observed effects were specifically attributable to PM\textsubscript{2.5} or the combined effects of these factors.

In conclusion, our study demonstrated that long-term exposure to PM\textsubscript{2.5} was positively associated with blood pressure and hypertension prevalence. The effect of PM\textsubscript{2.5} on hypertension prevalence was more pronounced at higher PM\textsubscript{2.5} concentrations. Our findings reinforce the need to develop comprehensive strategies for addressing air pollution problems, especially for areas with severe air pollution.
Acknowledgements
We thank all study individuals for their participation and appreciate the contributions made by project teams at the National Centre for Cardiovascular Diseases in the realms of project design, operations and data collection.

Contributors
JS and XZ conceived of this article. JS wrote the manuscript with further contributions from XZ, YG, XL, EM, MR, GT, DZ, WZ, JL and MAS. JS and SH completed all the statistical analyses. All authors interpreted data, contributed to critical revisions and approved the final version of the article. XZ accepts full responsibility for the finished work and/or the conduct of the study, had access to the data, and controlled the decision to publish.

Funding
This project was supported by the National Key Research and Development Program (2016YFE0103800) from the Ministry of Science and Technology of China, the CAMS Innovation Fund for Medical Science (2016-2M1-1006), the Major Public Health Service Project from the Ministry of Finance and National Health and Family Planning Commission of China and the 111 Project (B160055) from the Ministry of Education of China.

Competing interests
JL discloses that she is a recipient of research grants from the government of China, through Fuwai Hospital, for research to improve the management of hypertension and blood lipids, and to improve care quality and patient outcomes of cardiovascular disease; is a recipient of research agreement with Amezen, through National Centre for Cardiovascular Diseases (NCCD) and Fuwai Hospital, for a multicentre trial to assess the efficacy and safety of Omeacmt Mecarbil, and for dyslipidemic patient registration; is a recipient of a research agreement with Sanofi, through Fuwai Hospital, for a multicentre trial on the effects of saxagliptin; is a recipient of a research agreement with University of Oxford, through Fuwai Hospital, for a multicentre trial on empagliflozin and was a recipient of a research agreement, through NCCD, from AstraZeneca for clinical research methods training.

Patient consent for publication
Consent obtained directly from patient(s).

Ethics approval
The central ethics committee at the China National Centre for Cardiovascular Diseases approved this project (Ethical code 2014-574). All enrolled participants provided written informed consent.

Provenance and peer review
Not commissioned; externally peer reviewed.

Data availability statement
No data are available. The China PEACE Million Persons Project is a major national programme, and as the government policy stipulates, it is not permissible for the researchers to make the raw data publicly available at this time. All data generated during this study are included in this manuscript and its supplementary information files.

Supplemental material
This content has been supplied by the author(s). It has not been vetted by BMJ Publishing Group Limited (BMJ) and may not have been peer-reviewed. Any opinions or recommendations discussed are solely those of the author(s) and are not endorsed by BMJ. BMJ disclaims all liability and responsibility arising from any reliance placed on the content. Where the content includes any translated material, BMJ does not warrant the accuracy and reliability of the translations (including but not limited to local regulations, clinical guidelines, terminology, drug names and drug dosages), and is not responsible for any error and/or omissions arising from translation and adaptation or otherwise.

Open access
This is an open access article distributed in accordance with the Creative Commons Attribution Non Commercial (CC BY-NC 4.0) license, which permits others to distribute, remix, adapt, build upon this work non-commercially, and license their derivative works on different terms, provided the original work is properly cited, appropriate credit is given, any changes made indicated, and the use is non-commercial. See: http://creativecommons.org/licenses/by-nc/4.0/.

ORCID iDs
Xi Li http://orcid.org/0000-0003-2249-688X
Jing Li http://orcid.org/0000-0002-8840-8638
Xin Zhong http://orcid.org/0000-0001-5285-4989

REFERENCES
1. GBD 2017 Risk Factor Collaborators. Global, regional, and national comparative risk assessment of 84 behavioural, environmental and occupational, and metabolic risks or clusters of risks for 195 countries and territories, 1990-2017: a systematic analysis for the Global Burden of Disease study 2017. Lancet 2018;392:1923–94.
2. Mills KT, Bundy JD, Kelly TN, et al. Global disparities of hypertension prevalence and control. Circulation 2016;134:441–50.
3. Rajagopalan S, Al-Kindi SG, Brook RD. Air pollution and cardiovascular disease. JACC state-of-the-art review. J Am Coll Cardiol 2018;72:2054–70.
4. Carey RM, Munther P, Bosworth HB, et al. Prevention and control of hypertension: JACC health promotion series. J Am Coll Cardiol 2018;72:1278–93.
5. Xie W, Wang Y, Yang Y, et al. Long-term Effects of Ambient Particulate Matter (With an Aerodynamic Diameter ≤2.5 µm) on Hypertension and Blood Pressure and Attributable Risk Among Reproductive-Age Adults in China. J Am Heart Assoc 2018;7:e008553.
6. Dong G-H, Qian ZM, Xaverius PK, et al. Association between long-term air pollution and increased blood pressure and hypertension in China. Hypertension 2013;61:576–81.
7. Liu C, Chen R, Zhao Y, et al. Associations between ambient fine particulate air pollution and hypertension: a nationwide cross-sectional study in China. Sci Total Environ 2017;584–585:869–74.
8. Fuks KB, Weinmayr G, Foraster M, et al. Arterial blood pressure and long-term exposure to traffic-related air pollution: an analysis in the European study of cohorts for air pollution effects (escape). Environ Health Perspect 2014;122:896–905.
9. Schwartz J, Alexeeff SE, Mordukhovich I, et al. Association between long-term exposure to traffic particles and blood pressure in the Veterans administration normative aging study. Occup Environ Med 2012;69:422–7.
10. Chan SH, Van Hee VC, Bergen S, et al. Long-term air pollution exposure and blood pressure in the sister study. Environ Health Perspect 2015;123:951–8.
11. Kearney PM, Whelton M, Reynolds K, et al. Global burden of hypertension: analysis of worldwide data. Lancet 2005;365:217–23.
12. Cohen AJ, Brauer M, Burnett R, et al. Estimates and 25-year trends of the global burden of disease attributable to ambient air pollution: an analysis of data from the Global Burden of Disease study 2015. Lancet 2017:389:1907–18.
13. Lu J, Xuan S, Downing NS, et al. Protocol for the China peace (patient-centered Evaluative assessment of cardiac events) million persons project pilot. BMJ Open 2016;6:e010200.
14. Compilation rules of zoning codes for statistics and urban-rural division codes. Available: http://www.stats.gov.cn/tjsj/tjbz/20091/t20091125_8667.html [Accessed 01 Jul 2021].
15. James PA, Oparil S, Carter BL, et al. 2014 evidence-based guideline for the management of high blood pressure in adults: report of the panel members appointed to the eighth joint National Committee (JNC 8). JAMA 2014;311:507–20.
16. Liu L-S, Writing Group of 2010 Chinese Guidelines for the Management of Hypertension. [2010 Chinese guidelines for the management of hypertension]. Zhonghua Xin Xue Guan Bing Za Zhi 2011;39:579–615.
17. Zhou B-F, Cooperative Meta-Analysis Group of the Working Group on Obesity in China. Predictive values of body mass index and waist circumference for risk factors of certain related diseases in Chinese adults—study on optimal cut-off points of body mass index and waist circumference in Chinese adults. Biomed Environ Sci 2002;15:93–96.
18. Bennett DA. How can I deal with missing data in my study? Aust N Z J Public Health 2001;25:464–9.
19. Desquilbet L, Mariotti F. Dose-response analyses using restricted cubic spline functions in public health research. Stat Med 2010;29:1037–57.
20. Li Y, Yang L, Wang L, et al. Burden of hypertension in China: a nationally representative survey of 174,621 adults. Int J Cardiol 2017;227:516–23.
21. Liang F, Xiao Q, Huang K, et al. The 17-γ spatiotemporal trend of PM2.5 and its mortality in China. Proc Natl Acad Sci U S A 2020;117:25601–8.
Song J, et al. BMJ Open 2021;11:e050159. doi:10.1136/bmjopen-2021-050159

22. Wang L, Xiong Q, Wu G, et al. Spatio-Temporal Variation Characteristics of PM$_{2.5}$ in the Beijing-Tianjin-Hebei Region, China, from 2013 to 2018. *Int J Environ Res Public Health* 2019;16:4276.

23. Batterman S, Xu L, Chen F, et al. Characteristics of PM$_{2.5}$ Concentrations across Beijing during 2013-2015. *Atmos Environ* 2016;145:104–14.

24. Zhang Z, Guo C, Lau AKH, et al. Long-Term exposure to fine particulate matter, blood pressure, and incident hypertension in Taiwanese adults. *Environ Health Perspect* 2018;126:017008.

25. Cai Y, Zhang B, Ke W, et al. Associations of short-term and long-term exposure to ambient air pollutants with hypertension: a systematic review and meta-analysis. *Hypertension* 2016;68:62–70.

26. Lin H, Guo Y, Zheng Y, et al. Long-Term Effects of Ambient PM$_{2.5}$ on Hypertension and Blood Pressure and Attributable Risk Among Older Chinese Adults. *Hypertension* 2017;69:806–12.

27. Adar SD, Chen Y-H, D’Souza JC, et al. Longitudinal analysis of long-term air pollution levels and blood pressure: a cautionary tale from the multi-ethnic study of atherosclerosis. *Environ Health Perspect* 2018;126:107003.

28. Chen S-Y, Wu C-F, Lee J-H, et al. Associations between long-term air pollutant exposures and blood pressure in elderly residents of Taipei City: a cross-sectional study. *Environ Health Perspect* 2015;123:779–84.

29. Huang K, Yang X, Liang F, et al. Long-Term exposure to fine particulate matter and hypertension incidence in China. *Hypertension* 2019;73:1195–201.

30. Zhai S, Jacob DJ, Wang X, et al. Fine particulate matter (PM$_{2.5}$) trends in China, 2013–2018: separating contributions from anthropogenic emissions and meteorology. *Atmos Chem Phys* 2019;19:11031–41.

31. Bavishi C, Goel S, Messerli FH. Isolated systolic hypertension: an update after sprint. *Am J Med* 2016;129:1251–8.

32. Yunginger JW, Reed CE, O’Connell EJ, et al. A community-based study of the epidemiology of asthma. Incidence rates, 1964–1983. *Am Rev Respir Dis* 1992;146:888–94.

33. Bennett WD, Zeman KL. Effect of body size on breathing pattern and fine-particle deposition in children. *J Appl Physiol* 2004;97:821–6.

34. Miller KA, Spalt EW, Gassett AJ, et al. Estimating ambient-origin PM$_{2.5}$ exposure for epidemiology: observations, prediction, and validation using personal sampling in the Multi-Ethnic Study of Atherosclerosis. *J Expo Sci Environ Epidemiol* 2019;29:227–37.