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Ambient particulate air pollution and blood pressure in peri-urban India

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Abstract

Background—Evidence linking long-term exposure to particulate air pollution to blood pressure (BP) in high-income countries may not be transportable to low- and middle-income countries. We examined cross-sectional associations between ambient fine particulate matter ($PM_{2.5}$) and black carbon (BC) with BP (systolic [SBP] and diastolic [DBP]) and prevalent hypertension in adults from 28 peri-urban villages near Hyderabad, India.

Methods—We studied 5531 participants from the Andhra Pradesh Children and Parents Study (18-84 years, 54% men). We measured BP (2010-2012) in the right arm and defined hypertension as SBP ≥130 mm Hg and/or DBP ≥80 mm Hg. We used land-use regression models to estimate annual average $PM_{2.5}$ and BC at participant's residence. We applied linear and logistic nested mixed-effect models stratified by sex and adjusted by cooking fuel type to estimate associations between within-village $PM_{2.5}$ or BC and health.

Results—Mean (SD) $PM_{2.5}$ was 33 $\mu g/m^3$ (2.7) and BC was 2.5 $\mu g/m^3$ (0.23). In women, a 1 $\mu g/m^3$ increase in $PM_{2.5}$ was associated with 1.4 mm Hg higher SBP (95%CI: 0.12, 2.7), 0.87 mm Hg higher DBP (95%CI: -0.18, 1.9) and 4% higher odds of hypertension (95%CI: 0%, 9%). In men, associations with SBP (0.52 mm Hg; 95%CI: -0.82, 1.8), DBP (0.41 mm Hg; 95%CI: -0.69, 1.5), and hypertension (2% higher odds; 95%CI:-2%, 6%) were weaker. No associations were observed with BC.

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Data and code availability
Data is available through a formal collaborator request to APCAPS (see: http://apcaps.lshtm.ac.uk/apply-to-collaborate/). The computing code required to replicate the results reported is by contacting the corresponding author.

Conflicts of interest
None declared

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Conclusions—We observed a positive association between ambient $PM_{2.5}$ and BP and hypertension in women. Longitudinal studies in this region are needed to corroborate our findings.

Keywords

blood pressure; hypertension; ambient air pollution; particulate matter; black carbon; cardiovascular health; lower-middle income country; India

Introduction

High blood pressure (BP) is the leading risk factor for all-cause mortality and morbidity globally.1 The prevalence of high BP has increased over the past decades 2 and is projected to increase by 60% by 2025.3 Although high BP is a worldwide public health concern, 80% of the burden is in low- and middle-income countries.4 Of all adults with high BP in 2015 (1.1 billion), an estimated 44% lived in South and East Asia and 18% in India.2

Besides genetic and lifestyle factors, environmental factors such as air pollution can affect BP.5 A number of studies have reported an association between short-term changes (i.e., hours to days) in ambient levels of fine particulate matter ($PM_{2.5}$) and BP.6–9 Relatively fewer studies have assessed the association between long-term (i.e., months to years) exposure to $PM_{2.5}$ and BP, with most 6–12 (but not all 13–17) studies reporting a positive association. Identifying the key sources of $PM_{2.5}$ responsible for the observed associations remains an area of intense interest, with some evidence that combustion-related particles, often assessed as black carbon (BC), may be particularly relevant for cardiovascular health. 8,18,19

Air pollution levels in low- and middle-income countries are typically higher than in high-income countries, with 59% of air-pollution associated deaths occurring in Asia. 20 Despite the combined burden from high BP and air pollution in low- and middle-income countries, to date, most studies evaluating the association between long-term exposure to $PM_{2.5}$ and BP have been conducted in high-income countries.6–9,18,21,22 Findings from these studies may have limited transportability to populations in low- and middle-income countries because of a confluence of genetic, lifestyle, and environmental differences. 9,23,21,24 Epidemiologic studies in low- and middle-income countries can therefore shed light on the exposure-response relationship in populations exposed to higher ambient concentrations. Moreover, in low- and middle-income countries, the sources of ambient PM are potentially different than those found in high-income countries,23,24 implying differences in particle composition and toxicity.

There are various calls for greater understanding of the etiologic role of ambient air pollution in cardiovascular health in low- and middle-income countries,7,21,22,24–26 especially in India.22,24 In response, we examined associations between long-term exposure to ambient particulate air pollution, systolic (SBP) and diastolic blood pressure (DBP), and prevalent hypertension in adults from peri-urban India.

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Methods

Study population and ethics

We used data from the third follow-up of the Andhra Pradesh Children and Parents Study (APCAPS) intergenerational cohort. 27 This cohort includes individuals enrolled in the first follow-up (2003-2005) who were born during 1987-1990 (i.e. index children). The cohort was expanded in the third follow-up (2010-2012) to include their parents and siblings (eFigure 1). Questionnaire and vascular health data were collected from 6944 participants between 2010 and 2012, at one time point per participant. We included adults (≥18 years) and non-pregnant women (n=6227; 1315 index children and 4912 family members).

This study was approved by the ethics committees of the London School of Hygiene & Tropical Medicine (London, UK), the National Institute of Nutrition (Hyderabad, India), the Indian Institute of Public Health (Hyderabad, India), and Parc de Salut MAR (Barcelona, Spain). Signed informed consent forms were obtained from all participants.

Study area

Participants resided in 28 villages in a peri-urban area 28 (of 770 km^2) southeast of Hyderabad (Figure 1). Villages differed regarding their degree of urbanization, population size (from 546 to 21 262 people in 2013), proximity to Hyderabad (29 to 66 km), socioeconomic status, and primary cooking fuel.

Blood pressure measurements

We measured SBP and DBP in the right arm in a sitting position after 5 min of rest using an oscillometric device (Omron HEM 7300; Omron, Matsusaka Co., Japan) and an appropriate-sized cuff. Measurements were made in clinics established in study villages as part of APCAPS. Participants were asked to refrain from performing vigorous exercise, eating or drinking anything other than water, smoking or taking drugs 30 min prior to the measurement. Three consecutive BP readings were obtained, leaving 1 minute between successive readings. We used the average of the three readings as the estimate of BP in the main analyses, and the average of the last two of the three BP readings in sensitivity analyses. Research staff recorded the room temperature. We defined hypertension as SBP ≥130 mm Hg and/or DBP ≥80 mm Hg.29

Air pollution exposure

Within the framework of the CHAI project (Cardiovascular Health effects of Air pollution in Andhra Pradesh, India),30 we estimated annual average ambient concentrations of $PM_{2.5}$ and BC at participants' residential address using land-use regression models developed for the study area.31 Briefly, two monitoring sessions were performed in two seasons between 2015 and 2016 in 23 sites of the study area. Adjusted R^2 was 58% for $PM_{2.5}$ model and 79% for BC model. 31

Covariates

We collected data on socio-demographic, health, lifestyle, and household characteristics via questionnaire administered by a trained interviewer. The questionnaire (available at: http://

apcaps.lshtm.ac.uk/questionnaires/) also included questions related to dietary intake over the past year (evaluated through a semi-quantitative food frequency questionnaire) and physical activity over the preceding week. Development and validation of the APCAPS questionnaire sections is described elsewhere.32,33 We assessed socio-economic status using the Standard of Living Index (SLI), a household level asset-based scale based on principal component analysis and designed for the Indian population.27 Tertiles were derived to identify low, middle, and high SLI. We measured height (in m) and weight (in kg) during the clinic visit. We calculated body mass index (BMI) accordingly (weight divided by squared height).

Data analysis

We identified potential confounders using prior evidence and bivariate associations with the outcome and/or the exposure, as illustrated using DAGitty 2.3 34 in a directed acyclic graph (eFigure 2). Given the importance of sex as a determinant of baseline health status, socio-economic and lifestyle factors, and time-activity patterns influencing residential exposure,35 we decided *a priori* to stratify all analysis by sex, but we also report results for the whole study population. We excluded participants with missing data on sex (n=5), household ID (n=82), BP (n=3), and land-use regression-predicted estimates (n=580). We also excluded participants with SBP - DBP < 15 mm Hg (n=5) and those in whom BP was measured in the left arm (n=21); leaving 5531 participants for analysis (1165 index children and 4366 family members). Missingness of some covariates varied by village; we therefore multiply imputed missing data in our covariates using the method of chained equations.36 We created m=20 imputed datasets 37 using the same covariates included in the model 4 dataset (see below) as input and pooled each m estimate using Rubin's rules.38

Participants lived in 2296 households (on average two participants per household) within 28 villages. To estimate within-village associations between $PM_{2.5}$ or BC and health, we applied nested (linear for BP and logistic for hypertension) mixed-effects models in which both the within and between village exposure–outcome relationships were modeled explicitly, an approach referred to as within–between model specification.39,40 Compared to random-effects estimation, within–between specification is better suited to model scenarios in which the exposure may be correlated with the random effects (thereby being subject to bias), sample size is large, and within-group variability of the exposure is limited. 41 Although conceptually analogous to fixed-effects estimation, within–between specification has the advantage of adjusting for the between-group unobserved effects using fewer degrees of freedom.40 We used the following regression equation (all components expressed in scalar form):

$$y_{vhi} = \beta_0 + \beta_w(x_{vhi} - \bar{x}_v) + \beta_B\bar{x}_v + (u_v + u_{vh} + e_{vhi}) + covariates$$

where y_{vhi} represents the outcome in village v, household h and individual i; β_0 represents a constant; β_w represents the within-village effect estimated as the effect of the difference between the individual exposure (x_{vhi}) and the village mean ($\bar{x}_v$) on the outcome; β_B represents the village mean exposure (between effect); u represent the random intercepts for the nested household (u_{vh}) within village (u_v); and e_{vhi} the error term.

Household air pollution is an additional important source of personal and ambient air pollution in this region. We therefore explored the role of type of primary cooking fuel (biomass vs. clean) as potential confounder through adjustment and as a potential effect measure modifier through stratified analyses in women. For each air pollution metric and continuous outcome, we fitted the following regression models:

- **model 1** (basic): adjusted for age, antihypertensive medication, and mean village concentration
- **model 2** (cooking fuel adjusted): model 1 + cooking fuel
- **model 3** (main): model 2 + education attainment, SLI, physical activity, environmental tobacco smoke, active smoking (only in men), alcohol, room temperature, and salt intake
- **model 4** (including potential mediators): model 3 + BMI and diabetes

Results are expressed as change in BP outcome (in mm Hg) per 1 $\mu g/m^3$ increase in within-village $PM_{2.5}$ and per inter-quartile range width (IQRW) increase in within-village BC. For prevalent hypertension as a dichotomous outcome, we only fit model 3. We explored potential non-linearity for all continuous covariates (age, physical activity, temperature, salt intake, and BMI) by adding a natural spline with 3 degrees of freedom. The full model allowing for non-linearity in age is shown in eTable 1. For categorical covariates, we used the same categories shown in Table 1.

To assess the robustness of our findings, we conducted multiple sensitivity analyses using model 3: i) defining the outcome as the average of the last two BP readings, since the first BP reading can be higher than subsequent ones; ii) excluding participants taking antihypertensive medication (n=195); iii) conducting a leave-one-village-out analysis (i.e. removing each of the villages one at a time); and iv) including village as a fixed effect with only a random intercept for household. As secondary analysis, we refit models 3 and 4 stratified by age (≤40 years vs. > 40 years) while adjusting for sex and age. Analyses were conducted with R (version 3.5.0) using packages "mice" 36 and "lme4".42

Results

Participants' characteristics and blood pressure levels

The 5531 participants included were 54% male, had a mean age of 38 years, and had a mean BMI of 21 kg/m^2 (Table 1). Compared to men, women tended to be older, more physically active, had less formal education, higher BMI, lower household SLI, and consumed less tobacco and alcohol. Few participants (6%) reported previous diagnosis of hypertension, although we identified 46% of participants as hypertensive based on measured BP. On average, men had higher SBP (124 mm Hg vs. 118 mm Hg), DBP (81 mm Hg vs. 78 mm Hg), and prevalent hypertension (52% vs. 39%) than women.

Air pollution levels

Ambient annual averages were 33 $\mu g/m^3$ (range: 24 to 38) for $PM_{2.5}$ and 2.5 $\mu g/m^3$ (range: 1.6 to 3.1) for BC (Table 1). The IQRWs of within-village levels were 0.34 $\mu g/m^3$ for $PM_{2.5}$

and 0.13 μg/m^3 for BC. BC had more within-village variability than $PM_{2.5}$ (Figure 2). All participants were exposed to higher annual average $PM_{2.5}$ than the World Health Organization guideline (10 μg/m^3) and the US Environmental Protection Agency (12 μg/m^3) air quality standard. Almost all participants (96%) had exposures above the European Union Air Quality Standards (25 μg/m^3).

Associations between air pollution and blood pressure and hypertension

Crude models and models 1 and 2 are presented in eTable 2. Models 3 and 4 are presented in Table 2. A 1 μg/m^3 increase in within-village $PM_{2.5}$ was associated with 1.4 mm Hg (95% Confidence Interval (CI): 0.12, 2.7) higher SBP among women (Table 2; Model 3). The association for DBP was also positive but smaller in magnitude (0.87 mm Hg; 95% CI: -0.18, 1.9). In men, associations between $PM_{2.5}$ and SBP (0.52 mm Hg; 95% CI: -0.82, 1.8) and DBP (0.41 mm Hg; 95% CI: -0.69, 1.5) were smaller compared to women. BC was not associated with either SBP or DBP in either men or women. When further adjusting for BMI and diabetes – which may be considered either confounders or potential causal intermediates between air pollution and hypertension – associations were generally similar, but slightly weaker for $PM_{2.5}$ in men and for BC in women (Table 2; Model 4). In the whole study population (men and women), a 1 μg/m^3 increase in within-village $PM_{2.5}$ was associated with 0.98 mm Hg (95% CI: -0.02, 2.0) higher SBP and 0.64 mm Hg (-0.18, 1.5) higher DBP. BC was not associated with either SBP (-0.03 mm Hg; - 0.53, 0.48) or DBP (0.002 mm Hg; -0.41, 0.41). Stratified analyses by age are presented in eTable 3. There was slight indication of stronger $PM_{2.5}$-SBP and weaker $PM_{2.5}$-DBP associations in the older (vs. younger) group. However, differences in point estimates were small. Stratified analyses by cooking fuel for women are presented in eTable 4. Although the point estimate between $PM_{2.5}$ and SBP was larger in women using biomass; there were mainly no differences between the two groups.

A 1 μg/m^3 increase in within-village $PM_{2.5}$ was associated with an adjusted odds ratio of hypertension of 1.04 (95% CI: 1.00, 1.09) in women, 1.02 (0.98, 1.07) in men, and 1.03 (1.00, 1.07) across both sexes (data only shown in the main text). For each 0.13 μg/m^3 increase in within-village BC, the adjusted odds ratio of hypertension was 1.01 (0.99, 1.03) in women, 0.99 (0.97, 1.02) in men, and 1.00 (0.99, 1.02) when including both men and women.

Sensitivity analyses

Results were similar in sensitivity analyses (eTable 1). When excluding participants taking antihypertensive medication (model S2), the effect of $PM_{2.5}$ on DBP in women was slightly stronger (0.97; 95% CI: -0.09, 2.0) per 1 μg/m^3 increase in $PM_{2.5}$. When using fixed rather than random effects for village (model S3) which more stringently controls for differences between villages, we observed a very similar point estimate for the association between $PM_{2.5}$ and SBP in women. Also in women, results were fairly robust to the exclusion of specific villages, with exception of villages 1 and 14 (Figure 3), possibly because of the high number of participants in these villages. The pattern was similar for men (eFigure 3).

Discussion

In this cross-sectional study, we observed positive associations between long-term exposure to ambient $PM_{2.5}$ and BP and prevalent hypertension among women. Stronger associations were found for SBP than DBP. Associations in men were weaker and included the null. Long-term exposure to BC was not associated with BP or hypertension either in women or men. Results were robust in sensitivity analyses. Models adjusting for primary cooking fuel (biomass vs. clean) suggests that $PM_{2.5}$–SBP association in women was independent of type of fuel used for cooking.

Previous studies have reported sex-adjusted estimates or have focused only on one sex,9 making comparison of our sex-specific results difficult. Sex-specific (or gender-specific) effects of air pollution are often determined by differences in time–activity patterns. In the study population, women spend the majority of their time near home (83% of the daytime vs. 57% for men).35 This suggests that residence-based exposure estimates may be more relevant for women than for men in this setting, and may explain why we observed stronger associations between $PM_{2.5}$ and BP in women. Women cooking with solid fuels have generally higher SBP and DBP than clean fuel users.43 In a study by Liu *et al* 10 in China, higher levels of ambient $PM_{2.5}$ were associated with higher SBP in individuals using solid fuels for cooking. However, our stratified analysis in women was not sufficiently powered to assess if the association observed between ambient $PM_{2.5}$ and SBP may be modified by the cooking fuel used.

Most studies investigating long-term ambient PM in relation to BP have been conducted either in urban areas where air pollution is typically dominated by traffic sources or in high-income countries, where $PM_{2.5}$ concentrations are considerably lower (<20 $\mu g/m^3$) than in our study (33 $\mu g/m^3$). Our study is likely more comparable to two nationwide studies conducted in China, which include rural areas and with similar ambient $PM_{2.5}$ levels (≥30 $\mu g/m^3$).10,11 Both studies found stronger $PM_{2.5}$–SBP associations than $PM_{2.5}$–DBP, which is consistent with our results. Liu *et al* found a 0.60-mm Hg (95% CI: 0.05, 1.1) increase in SBP and 0.02-mm Hg increase in DBP (95% CI: -0.30, 0.34) per 42 $\mu g/m^3$ increase in $PM_{2.5}$ in adults ≥35 years old.10 Lin *et al* found an increase in both SBP (1.3 mm Hg; 95% CI: 0.04, 3.6) and DBP (1.0 mm Hg; 95% CI: 0.31, 1.8) per 10 $\mu g/m^3$ increase in $PM_{2.5}$ in middle-aged (≥50 years) adults.11 The magnitude of our $PM_{2.5}$–SBP association in women was ~10 times greater than these Chinese studies (after rescaling all estimates to 1 $\mu g/m^3$ increase). A range of factors may explain the higher magnitude of association observed in our study vs. some prior studies. First, our study had a high prevalence of undiagnosed (87%) and untreated (93%) hypertension, which may make this population more comparable to high risk subgroups elsewhere. Second, many prior studies have focused on differences in exposures between-cluster (e.g., between-city) rather than within-cluster. Estimates of association between- vs. within-cluster may be susceptible to different biases and thus provide different insights into the true effect of $PM_{2.5}$ exposure on BP. Third, published studies have used a range of approaches to estimate air pollution exposures, including satellite-based methods with relatively course spatial resolution (10 × 10 km) 10,11. Satellite-based methods may have limited ability to estimate small-area variations in air pollution exposures and may have larger exposure measurement error than the land-use

regression models, leading to smaller health effects estimates.44 Fourth, differences in particle composition and toxicity may contribute to apparent heterogeneity across studies.

Few studies have investigated the relationship between middle- or long-term exposure to BC (or $PM_{2.5}$ absorbance, comparable to BC) and BP.16,17,44–47 All were conducted either exclusively in urban areas 16,17,47 or in the USA and in older (mostly men) adults (~70 to 80 years).44–46 Although results were heterogeneous, they indicated positive associations between ambient BC and BP. Our lack of association is surprising, particularly because the BC land-use regression model had better performance and captured more local spatial variability compared to the $PM_{2.5}$ land-use regression model.31 A possible explanation is that ambient BC in this setting has a different toxicologic profile compared to settings where it is dominated by traffic.16,17,45–48 Further studies are needed to explore the role of ambient BC in cardiovascular health in low- to middle-income countries, perhaps with greater emphasis on source apportionment and composition or toxicity of particles.

The biologic mechanisms linking BP and air pollution likely differ according to $PM_{2.5}$ constituents, timing and duration of exposure, and underlying susceptibility of individuals. 6,49 Current knowledge indicates that inhaled particles can acutely induce pulmonary oxidative stress and inflammation, and also provoke an initial imbalance in the autonomic nervous system, stimulating the sympathetic response, and subsequently elevating BP due to an increase in arterial vasoconstriction.6,8 Long-term PM exposures can also trigger endothelial injury or dysfunction, perhaps driven by an increase in reactive oxygen species, and thus adversely alter systemic hemodynamics and increase risk of hypertension.6,8,49

Our study overcomes several limitations of previous studies. We collected demographic data for ~100% of all living residents of the study villages (eFigure 1). Adults (≥18 years) surveyed (n=63128) are similar to our adult study participants in terms of age (mean age of 38 years in both the general population and participants), sex (51% vs. 54% men), and education (47% vs. 53% without education; with slightly more women without education in our study sample) (eTable 5). Participants are therefore considered to be representative of the general population of this peri-urban area in South India. Regarding exposure assessment, land-use regression models provide finer spatial resolution and likely lower exposure measurement error compared with exposure estimates derived solely from satellite imagery or proximity of residence to fixed-site monitoring stations. Limitations of our study, however, should be considered while interpreting the results. Because of the cross-sectional design, we could not ensure that exposure preceded the outcome or investigate the influence of timing of exposure on BP. There were a few years between the BP measurement (2010-2012) and the air pollution monitoring campaign (2015-2016), although geographic predictors used in land-use regression models were from 2012-2013. We assume that the spatial pattern of sources in the study area remained constant between 2010 and 2015. Previous research supports this assumption in settings dominated by traffic sources;50 but no comparable evidence is available for peri-urban or rural areas. Although we considered a wide range of potential individual and household confounding factors, we cannot rule out the possibility of unmeasured confounding in the observed associations. Nonetheless, our model formulation allowed separating between and within village effects, thus accounting for factors that may vary across villages (e.g., exposure to other co-pollutants linked to high

BP). The fairly wide CIs likely reflect the limited variability of the within-village exposure and/or the random measurement error in the outcome by measuring BP in a single occasion. 29

In conclusion, our study suggests that long-term exposure to ambient fine particulate matter is positively associated with blood pressure in women, independently of the type of fuel used for cooking. Additional epidemiologic evidence is needed to corroborate our findings, ideally from studies using longitudinal data, to better inform the potential cardiovascular health benefits of air pollution control policies.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgments

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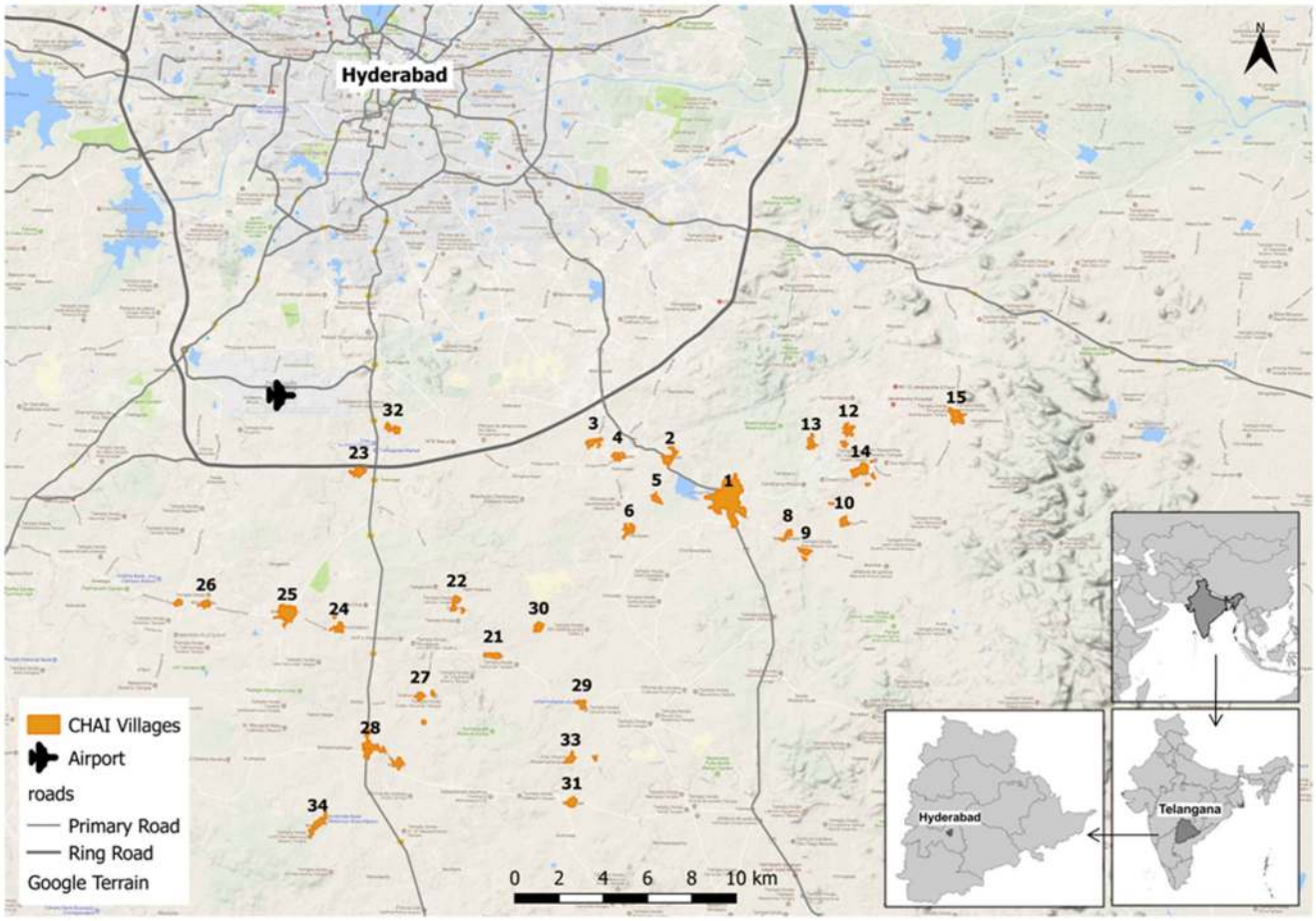

Figure 1. Map of the study area.

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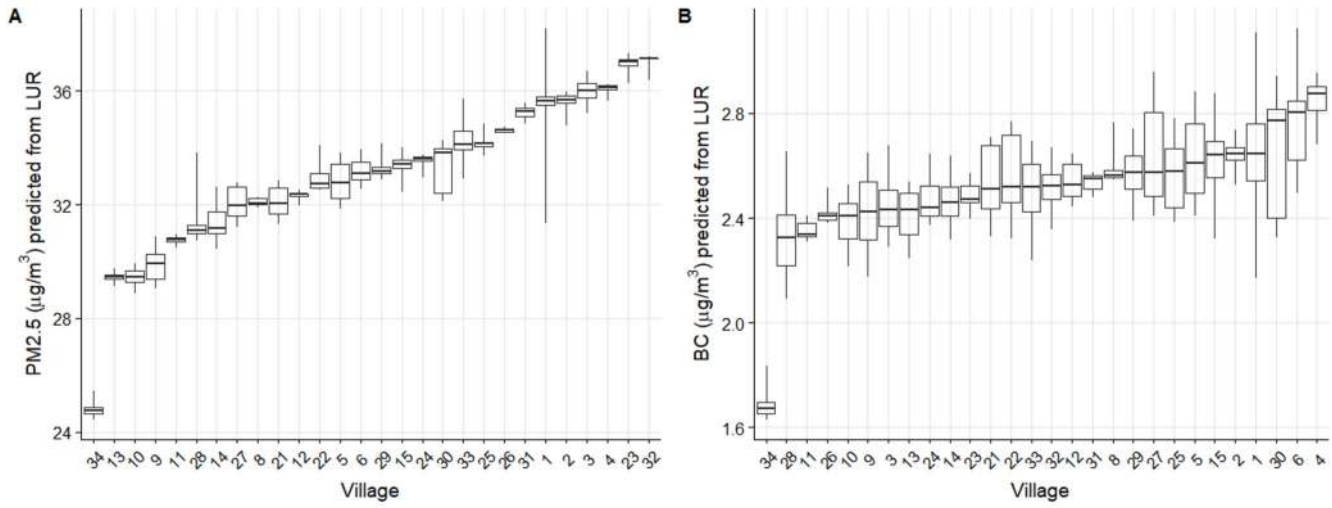

Figure 2. Box plots of estimated $PM_{2.5}$ (panel A) and BC (panel B) at residence according to village.

$PM_{2.5}$: particles less than 2.5 μm in diameter; BC: black carbon; LUR: Land-Use Regression model.

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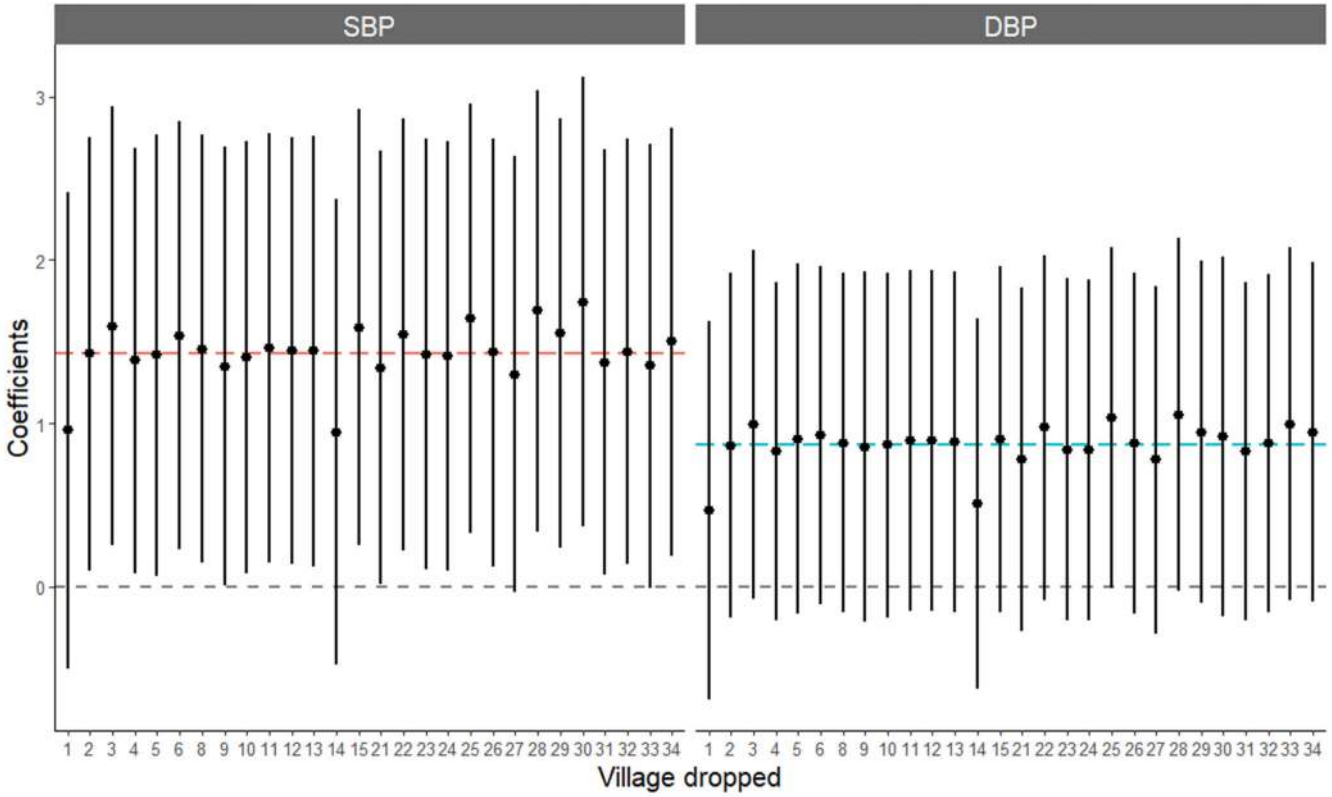

Figure 3. Regression coefficients for the association between fine particulate matter ($PM_{2.5}$) and blood pressure in women after the leave-one-village-out approach.

Error bars represent 95% Confidence Interval. Dashed black line corresponds to the zero level. Red dashed line corresponds to the systolic blood pressure (SBP) coefficient from the model considering all villages (showed for reference), whereas blue dashed line corresponds to diastolic blood pressure (DBP) coefficient.

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Table 1

Participants' characteristics, exposure levels, and blood pressure.

	n (% missing)[a]	ALL	MEN	WOMEN
CATEGORICAL VARIABLES				
Men; %	5531 (0)	54	-	-
Formal education; %	5530 (0.02)			
Without (either illiterate or literate)		53	38	70
With any kind		47	62	30
Standard of living index; %	5171 (6.5)			
Low		33	34	38
Medium		33	36	35
High		33	30	27
Smoking status; %	5530 (0.02)			
Never		83	68	99
Former (stopped 6 months ago)		1	2	0
Current (within last 6 months)		16	30	0.2
Exposure to ETS at home; %	5530 (0.02)	31	74	63
Alcohol intake frequency; %	5529 (0.04)			
Never		32	20	45
Occasional (monthly or special occasions)		36	35	37
Regular (daily or weekly)		32	44	17
Primary cooking fuel; %	5184 (6.3)			
Clean (gas or electricity)		42	43	40
Biomass		58	57	60
Self-reported hypertension; %	5375 (2.8)	6	6	6
Antihypertensive medication; %	5373 (2.9)	3	3	3
Measured hypertension; %	5531 (0)	46	52	39
Self-reported diabetes; %	5530 (0.02)	2	3	2
CONTINUOUS VARIABLES				
Age (years); mean ± SD	5531 (0)	37.7 ± 13.3	37.3 ± 14.9	38.1 ± 11.3
Physical activity (METs-week); mean ± SD	5235 (5.4)	1.6 ± 0.21	1.6 ± 0.21	1.7 ± 0.21
BMI (kg/m^2); mean ± SD	5519 (0.2)	21.1 ± 3.8	20.9 ± 3.6	21.4 ± 4.1
Temperature of the room (°C); mean ± SD	5531 (0)	26.4 ± 2.8	26.3 ± 2.8	26.5 ± 2.8

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	n (% missing)[a]	ALL	MEN	WOMEN
CATEGORICAL VARIABLES				
Salt intake (grams/day); mean ± SD	5523 (0.1)	6.4 ± 3.4	6.9 ± 3.7	5.8 ± 2.9
Ambient $PM_{2.5}$ ($\mu g/m^3$); mean ± SD	5531 (0)	32.8 ± 2.7	32.8 ± 2.7	32.9 ± 2.7
Ambient BC ($\mu g/m^3$); mean ± SD	5531 (0)	2.5 ± 0.23	2.5 ± 0.23	2.5 ± 0.23
SBP (mm Hg); mean ± SD	5531 (0)	120.9 ± 15.9	124.0 ± 16.2	117.8 ± 14.9
DBP (mm Hg); mean ± SD	5531 (0)	79.4 ± 12.5	81.3 ± 12.9	77.7 ±11.6

MET: metabolic equivalent task; BMI: body mass index; ETS: environmental tobacco smoke; SBP: systolic blood pressure; DBP: diastolic blood pressure; $PM_{2.5}$: particles less than 2.5 μm in diameter; BC: black carbon; SD: standard deviation

[a]% missing based on 5531 sample size; % distributions for a given covariate are based on complete cases. Values correspond to data prior to multiple imputation

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Table 2

Associations between residential exposure to particles and blood pressure according to sex. Changes in SBP and DBP are expressed as unit increase in mm Hg per 1 μg/m^3 increase in $PM_{2.5}$ and per IQRW increase in black carbon (=0.13 μg/m^3).

	MEN (n=2979)				WOMEN (n=2552)			
	SBP		DBP		SBP		DBP	
	β	*95% CI*	*β*	*95% CI*	*β*	*95% CI*	*β*	*95% CI*
$PM_{2.5}$								
Model 3	0.52	-0.82 to 1.8	0.41	-0.69 to 1.5	1.4	0.12 to 2.7	0.87	-0.18 to 1.9
Model 4	0.42	-0.86 to 1.7	0.31	-0.74 to 1.4	1.5	0.19 to 2.7	0.91	-0.08 to 1.9
Black carbon								
Model 3	-0.20	-0.86 to 0.47	-0.04	-0.59 to 0.51	0.15	-0.52 to 0.81	0.11	-0.41 to 0.64
Model 4	-0.27	-0.91 to 0.37	-0.12	-0.64 to 0.41	0.01	-0.64 to 0.65	-0.03	-0.53 to 0.47

$PM_{2.5}$: particles less than 2.5 μm in diameter; SBP: systolic blood pressure; DBP: diastolic blood pressure; IQRW: inter-quartile range width; CI: confidence interval.

Model 3 (main): adjusted for age, antihypertensive medication, mean village concentration, cooking fuel, education attainment, standard of living index, physical activity, environmental tobacco smoke, active smoking (only in men), alcohol, room temperature, and salt intake

Model 4 (including potential mediators): model 3 + body mass index and diabetes