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Exposure to fine particulate matter and acute effects on blood pressure: effect modification by measures of obesity and location

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Abstract

Background: Observational studies and controlled experiments have provided evidence that airborne particulate matter (PM) is capable of acutely increasing blood pressure (BP) in certain scenarios. The goal of this study was to evaluate whether and to what extent obesity and community location affect relationships between fine particulate matter ($PM_{2.5}$) and blood pressure (BP) measures.

Methods: Using data from a stratified random sample survey of adults conducted in 2002–3 in Detroit, Michigan, we tested body mass index (BMI) and waist circumference (WCIR) in separate models as effect modifiers of the relationship between $PM_{2.5}$ exposure and BP. We also tested interactions with community location. Models were adjusted for covariates with established prohypertensive effects.

Results: $PM_{2.5}$ exposure was positively associated with increased pulse pressure (PP) for those categorised as obese (BMI≥30) across lags 2 (β 4.16, $p<0.05$) and 3 days (μ 2.55, $p<0.05$) prior to BP measure. WCIR similarly modified the effect of exposure to $PM_{2.5}$ on PP (β 4.34, $p<0.003$). The observed effects were enhanced in the community with closer proximity to local emissions of $PM_{2.5}$, and for residents classified as obese (BMI≥30) or with WCIR above high-risk cuts points.

Conclusions: This community-based study suggests that positive associations between $PM_{2.5}$ exposure and PP and systolic BP are enhanced in areas proximate to sources of $PM_{2.5}$ emissions.

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These patterns were observed for all residents, but were more visible and consistent among those who were obese. Research is needed to examine the mechanistic pathways by which air particles interact with obesity and location to affect BP, and inform community interventions to reduce the population burden of hypertension and related co-morbidities.

Researchers have analysed multiple risk factors for cardiovascular mortality and morbidity in association with air pollution.[1] One potential mechanism explaining this association is that acute exposure to particulate matter (PM) at high concentrations is capable of raising blood pressure (BP) within hours to days.[2-5] Population studies of air pollution effects found an increase in systolic blood pressure (SBP) with elevated concentrations of particulates.[3] Controlled exposure to concentrated ambient $PM_{2.5}$+ozone resulted in a significant acute increase in diastolic BP.[6] However, not all studies have shown positive effects. For example, Ibald-Mulli and colleagues[7] found a small significant decrease in SBP and diastolic blood pressure (DBP) in association with $PM_{2.5}$, in patients with coronary heart disease (CHD).

The search for possible elevated risk conditions has drawn recent attention.[8-12] The most recent reports by the National Research Council[13] and the American Heart Association writing group[14] have emphasised the need to better characterise vulnerable subgroups at high risk for negative effects of air particles. Given that hypertension affects approximately one-third of the US adult population and is a leading risk factor for cardiovascular morbidity and mortality,[15] it is critical to understand those groups at highest risk of increases in BP due to short-term increases in PM exposure.

Observational studies and controlled experiments have provided evidence that PM is capable of acutely increasing BP in certain scenarios.[2-5] Increases in mortality rates,[16-18] hospital admissions and emergency department visits[12-19] and symptom exacerbations in patients with CHD[2021] have been demonstrated in response to PM exposure. Cardiovascular disease (CVD) mortality rates are associated with environmental exposure to background and traffic air pollutants.[22] Short-term increases in exposure to ambient $PM_{2.5}$ have been associated with acute increases in SBP and pulse pressure (PP) with enhanced effects observed in neighbourhoods most proximate to emissions sources.[23] To our knowledge, the modification effects on PP, a well-established risk factor for cardiovascular mortality and morbidity, have not yet been documented for air pollution exposures in multiethnic populations. PP reflects stiffness of the large arteries,[24] and increased PP becomes a stronger predictor of CVD in the presence of additional cardiovascular risk factors.[25]

Obesity may increase susceptibility to the adverse effects of PM exposure.[82627] Obesity enhances effects of $PM_{2.5}$ on CVD[27] and biomarkers of vascular inflammation.[26] Although body mass index (BMI) has become the gold standard for identifying those at increased risk for obesity-related adverse health outcomes, waist circumference (WCIR) is an emerging measure that can identify those at greatest risk for cardiometabolic diseases (eg, hypertension, dyslipidaemia, CHD and diabetes) more effectively than BMI alone.[28] Furthermore, there is evidence that BMI and WCIR are independently associated with BP.[29]

Building on the extant literature, in the analysis presented here we postulated that individuals with higher BMI and WCIR would have a greater acute response to the effects of $PM_{2.5}$ exposure on baseline BP measures than those with lower BMI and WCIR. Furthermore, we hypothesised that community location may amplify the effect modification by obesity. The effect modification may depend on proximity to sources of pollutants, and the full scope of effect modification by obesity for air pollution may not be visible until community location and level of exposure are taken into account. Recent findings document that long-term differences in $PM_{2.5}$ exposure within cities are associated with the risk of cardiovascular events.[27]

METHODS

Study sample

Study design and methods have been described extensively by Schulz and colleagues.[30] Briefly, data for this investigation were collected as part of the Healthy Environments Partnership (HEP) study,[30] a community-based participatory research (CBPR) partnership initiated in October 2000 with funding from National Institute for Environmental Health Sciences (NIEHS) and affiliated with the Detroit Community–Academic Urban Research Center (URC).[31] The URC Board, which consists of representatives from community-based organisations, health service and public health institutions, and academic institutions, identified health disparities, with a particular focus on the contributions of the environment, as a priority area to address. HEP contributes to this goal by investigating the prevalence of more proximate social, psychological, behavioural and biological indicators of CVD risk, and the contributions of social and physical environments to those risk factors.[28] The HEP study was approved in January 2001 by the University of Michigan Institutional Review Board for Protection of Human Subjects. All participants provided written informed consent as part of the study protocol.

The HEP survey sample is a stratified, two-stage probability random sampling of occupied housing units (or households) in the three areas of Detroit in which air quality was monitored (n = 919). The communities vary in their socioeconomic and racial–ethnic composition. Eastside (ES) and Northwest (NW) Detroit are predominantly African American and Southwest (SW) Detroit is predominantly Latino. Of the 919 survey participants who were 25 or more years of age living in the three study areas, 57% were non-Hispanic black/African American, 20% Latino and 22% non-Hispanic white. Of the HEP survey participants, 53% reported household income <$20 000/year.[30] Survey participants were invited to participate in an additional biomarker portion of the study, and 348 elected to do so. Participants scheduled a follow-up appointment at a community site, where fasting blood samples were taken and BP was measured at a second point in time.

Blood pressure measurement

There was a mean of 4 weeks between measurement points for BP. Biomarker site staff were trained and required to demonstrate competency and were certified in measuring BP. Biomarker data collection began in May 2002 and ended in April 2003.

The BP measures included SBP and DBP collected using a portable cuff device (Omron model HEM 711AC; Omron Healthcare, Inc., Bannockburn, Illinois, USA) that passed Association for the Advancement of Medical Instrumentation (AAMI) standards.[32] BP was measured following the National Health and Nutrition Examination Survey methodology,[33] in a seated position using the right arm. Three consecutive measures of SBP and DBP were taken at each of the two time points. There were 60 min time intervals each between the three BP measurements. The mean of the second and third measures were used for all data analyses, except for 11 respondents for whom only the first two measurements were available and the mean of these measures was used. In addition, BP assessment included calculated components—PP calculated as the difference between SBP and DBP, and one-third of the SBP plus two-thirds of the DBP (mean arterial pressure; MAP).

PM exposure variables and lag time periods

Levels of ambient $PM_{2.5}$ were characterised in NW, SW and ES Detroit during the years 2000–3. These measures were made continuously (30 min intervals) at each community monitoring site using tapered element oscillating microbalances (TEOM Model 1400a,

Rupprecht and Patashnick, Inc., Albany, New York, USA), as previously described. Study participants lived within a 5 km radius of the air quality monitoring sites.

PM exposures were characterised in two different ways: (1) individual 24 h spans: exposure measured 1 day (lag 1), 2 days (lag 2) and up to 5 days before (lags 3–5) the day BP was measured; and (2) large spans: 48 h average before (2 days average), 72 h average before (3 days average) up to 120 h average before (5 days average). Standard meteorological variables including temperature, atmospheric pressure, relative humidity, wind speed and wind direction were also recorded in 30 min intervals at each site.

Potential effect modifiers and covariates

Survey data were collected during the period May 2002 to April 2003. Survey interviewers measured body weights (kg), standing heights and WCIR (measured at the high point of the iliac crest at minimal respiration to the nearest 1 mm) using standard anthropometric procedures. Obesity measures included dichotomous indicators of BMI calculated as [(weight in pounds)/(height in inches)^2×703] and BMI≥30 = obese, and WCIR in centimetres using gender-specific cut points (88 cm for females, 102 cm for males) for high and low risk.[35]

Covariates included demonstrated risk factors for hypertension.[102736-44] Covariates derived from the survey included: race–ethnicity (non-Hispanic black, non-Hispanic white, Hispanic/Latino), household income (≤$10 K, $10–19.9 K, $20–34.9 K, ≥$35 K), educational level (<12 years, 12 years, >12 years), age (25–44, 45–64, >65 years), gender (0, male; 1, female), smoking history (never, current, past), dietary sodium intake (<2300 mg per day, >2300 mg per day), physical activity (PA) (0, never; 1, light activity; 2, moderate activity; 3, regular activity; and 4, vigorous activity) and preexisting health conditions (physician diagnosis of diabetes, and medication use for hypertension). Temperature, atmospheric pressure and relative humidity derived from the monitoring sites described above were also included as covariates.

Data analysis

All analyses were conducted using the Statistical Analysis System (SAS V.9.1, SAS Institute Inc., Cary, North Carolina, USA). We used the Student t test (for continuous variables) and χ^2 (for categorical) to compare the demographic data and health characteristics for survey respondents who participated in the biomarker component and those who did not. A p value <0.05 was considered significant. Multivariate associations between ambient $PM_{2.5}$ and BP outcomes were assessed using SAS PROC SURVEYREG, which allowed the use of complex sample designs (weight, strata and PSUs).

To address effect modification, we used models that include the interactive effect of obesity and community-level PM exposure at baseline at varying lag levels (1–5 days). Model 1 assessed the modification of the association between exposure to $PM_{2.5}$ and SBP, DBP, PP and MAP by obesity (BMI and WCIR) while accounting for survey time point exposure relative to biomarker time exposure, and other relevant baseline covariates. Model 2 incorporated an interaction term for community location interaction with exposure. These variables were not significant, and patterns were similar to results from models that did not include these variables.

In order to maximise statistical power, models presented are those that do not include smoking or physician diagnosis of diabetes. Similarly, we ran models incorporating the covariates sodium intake and PA. We also tested the models controlling for the interaction between medication use and $PM_{2.5}$. In all cases, the patterns were similar to the tables reported. In order to maximise statistical power and precision of the point estimates, models

presented do not include these additional variables. Finally, we tested models that controlled for meteorological variables (temperature, atmospheric pressure and relative humidity). However, owing to multicollinearity with $PM_{2.5}$, these meteorological variables were not included in the final models.

RESULTS

Table 1 presents the sociodemographic data and health characteristics for the study participants. The mean (SD) age of the 348 biomarker participants was 46.3 years (SD 1.1), with 55.6% female and 58.9% African Americans. Fifty-one per cent were obese (BMI>30) and 57.4% were above the high-risk cut point for WCIR. Sixty-eight per cent were prehypertensive or hypertensive and 36.0% had elevated total blood cholesterol (>200 mg/dl). The characteristics of the biomarker subsample were not statistically different from those who did not participate in the biomarker data collection, with the following exceptions: biomarker participants were more likely to report that they currently smoked ($p = 0.019$), had lower income ($p = 0.013$) and were less likely to fall into the prehypertensive BP category ($p = 0.033$) (data not shown).

The mean level of $PM_{2.5}$ measured across all three Detroit community-level monitoring sites for the period 2000–3 was 15.1 mg/m^3 (SD 8.2). Measures made at the SW Detroit site were approximately 20% ($p<0.05$) higher than those measured at the NW and ES monitoring locations. These levels are above the United States Environmental Protection Agency (USEPA) National Ambient Air Quality Standard (NAAQS) of 15 mg/m^3 for annual $PM_{2.5}$.

Table 2 presents results for the modification of individual day lag effects by obesity status, after adjustment for the covariates. $PM_{2.5}$ was associated with increased risk for elevated PP among those categorised as obese based on their BMI measures (BMI>30 kg/m^2), for $PM_{2.5}$ exposure lags 2 and 3. For example, in obese individuals, a 10 mg/m^3 increase in daily $PM_{2.5}$ was associated with a 4.2 mm Hg increase in PP for lag 2 ($p<0.003$) compared with a 3.3 mm Hg increase in PP among non-obese participants.

WCIR similarly modified the day lag effects for lag 2 for the association between exposure to $PM_{2.5}$ and increased PP, adjusted for the covariates (table 3).

Tables 4 and 5 present results for the introduction of community location and exposure ($PM_{2.5}$) interaction terms in the models.

The observed effect of $PM_{2.5}$ on multiple haemodynamic indicators is enhanced by community proximity to point sources of PM, and this effect is observed for both BMI (tables 2 and 4) and WCIR (tables 3 and 5). After accounting for the interaction between location and exposure, the effect of $PM_{2.5}$ on SBP and pulse pressure is heightened. These effects are slightly larger, and more likely to be statistically significant among those who are obese, as measured by either BMI or WCIR. Furthermore, after accounting for the interaction between location and exposure, a significant effect of $PM_{2.5}$ on systolic blood pressure is also observed among those who are not obese (lag 4 for BMI and lags 4 and 5 for WCIR), and on pulse pressure (lags 2 and 4 for BMI and lag 4 for WCIR).

Models also assessed effect modification of multiday averaged exposure to $PM_{2.5}$ on BP outcomes (data not shown). In contrast to the observed effect modification for individual day lags described above, analysis of multiday averaged exposures found significant effects only on PP (3 days, 4 days and 5 days). However, adding location enhanced these effects on PP in SW Detroit relative to the other two communities. In addition, significant effects on SBP were also observed with location incorporated (4 days, 5 days), similar to those observed for the individual day lags.

DISCUSSION

Results of this community-based study with a multiethnic sample of adults suggest that communities most proximate to point sources of $PM_{2.5}$ experience heightened PP and SBP responses. In other words, the effect modification of the positive associations between $PM_{2.5}$ exposure and PP and SBP by BMI and WCIR is enhanced in areas proximate to sources of $PM_{2.5}$ emissions. This effect is most visible among those who are obese, as measured by BMI or WCIR, although similar trends are also apparent among those who are not obese. This increase manifested predominantly within 48–96 h after exposure.

These findings are partially consistent with our hypothesis that anthropometric measures may confer susceptibility to acute cardiovascular effects of fine particles. They add to a growing body of work,[82627] suggesting that a priori states of obesity and central adiposity may enhance susceptibility to changes in haemodynamic effects associated with ambient air pollution. The results reported here suggest that, after accounting for proximity to point sources of PM, individuals with central adiposity are more sensitive to increased BP in response to PM exposure. However, we also found evidence for a similar trend among the non-obese. Thus $PM_{2.5}$ exposure increases risk among all segments of the population, with exacerbated risk among those with central adiposity.

Previous research suggests that obesity- and central adiposity-dependent mechanisms may contribute to vascular and perhaps cardiovascular effects of exposure to fine particles.[8142644-50] Possible pathways include the association of obesity with established CVD risk factors, such as metabolic syndrome, enhanced insulin resistance and hyperglycaemia that are cumulatively damaging to the endothelium,[51] and risk factors as potential modulators of the endothelial phenotype in obesity, including oxidative stress[52] and chronic inflammation.[5354] Community residents with diabetes, hypertension and respiratory conditions may be at increased risk of cardiovascular morbidity and mortality associated with ambient air pollution.[1244]

Findings for elevated PP are particularly of interest, and suggest that PP may be considered a BP response indicator in PM toxicology. Further, we found that individuals residing in SW Detroit, the area most proximate to local emissions sources of $PM_{2.5}$, were more likely than those residing in other areas of the city to experience elevated effects of $PM_{2.5}$ exposure on SBP and PP. These effects were more likely to be statistically significant among those who were obese. Potential mechanisms for these differences include differences across locations in the PM source contributions, and PM chemical composition, including metal constituents,[8] such as from diesel truck traffic.[55] For example, researchers have shown that diesel-derived air pollution may have adverse vascular effects particularly in the presence of mild systemic inflammation.[49]

Southwest Detroit contains a high density of industrial facilities relative to other areas of the city.[5657] Industries include iron/steel manufacturing, coke ovens, chemical plants, refineries, sewage sludge incineration and coal-fired utilities. SW Detroit also experiences heavy car and truck traffic along two major interstate roads and the entrance/exit of the Ambassador Bridge, the most travelled border crossing between Canada and the USA. Previous analyses found that 35% of the $PM_{2.5}$ measured at the SW Detroit monitoring site was due to locally emitted particles from motor vehicles.[57] Further study to examine the extent to which these differences in composition of $PM_{2.5}$ contribute to the excess risk of increased BP experienced by residents of SW Detroit are needed.

Our study was subject to limitations. Significant relationships were observed controlling for a number of potential confounders: age, BMI, cigarette smoking and preexisting co-morbidities (diabetes and hypercholesterolaemia); however, residual confounding remains

possible, and other important variables may not have been considered. Because PM exposure, obesity and hypertension are associated with socioeconomic status, the finding of significant differences within this sample with limited income may be conservative. Moreover, we modelled the data for participants who had biomarker measurements available (37% of all HEP survey participants). More detailed studies of community-level biomedical status are warranted. The sociodemographic differences among the three community sites may have influenced the interpretation of the findings. It should be further emphasised that all exposure estimates are of an ecological nature, resulting in a high probability of misclassifying individual exposures. As a result, a high level of uncertainty is expected with effects presented here likely to be conservative estimates. Future studies should therefore consider taking truly individual exposure measurements.

Despite these limitations, our study with a multiethnic community sample confirms and extends previous epidemiological studies in a large and diverse population of adults.[3-582644] Our results find effects of $PM_{2.5}$ on BP for all residents, with enhanced effects visible for those who are obese, defined by either BMI or WCIR. Future research should investigate the mechanism(s) responsible for the effects of excess abdominal fat distribution on the relationship between $PM_{2.5}$ and BP measures.

Our findings add to a small body of research that documents differential effects of air pollution levels on health across neighbourhoods within the same city.[2758] The effects of $PM_{2.5}$ on PP and SBP are noted in all HEP community locations, and are most visible among those who are obese. The mechanism by which obesity and location jointly contribute to increased BP requires further explication. Other studies have shown that community-level characteristics (eg, higher unemployment) modify relationships between ozone and mortality.[59] Future air pollution studies should focus on identifying biological, anthropometric and community susceptibility characteristics in addition to exploring the potential for interplay with socio-demographic influences. Our findings of differential effects by location suggest the need to characterise the contribution of major source categories to observed $PM_{2.5}$ concentrations in communities and the role of specific particle physicochemical and toxicological properties. The extent to which particles associated with specific emission sources (eg, motor vehicles, coal-burning power plants) confer additional risks for all residents, and are exacerbated for those with additional metabolic risk factors, remains a critical public health issue.

In conclusion, given study findings suggesting that rates of obesity are higher in communities with reduced economic resources, and that poor communities and communities of colour also experience increased exposure to PM, our findings add weight to a growing literature that suggests that residents of older urban neighbourhoods encounter multiple conditions that exacerbate risk of CVD, and that those risks may have not additive, but multiplicative effects on health. Continued vigilance regarding regulation of emissions sources in communities of colour and low-income communities is critical, combined with efforts to address obesity, in order to reduce well-established disparities in cardiovascular health.

What is already known on this subject

Research studies point to the possibility that obesity may impart greater susceptibility to the adverse cardiovascular effects of airborne particulate matter (PM) exposure. Although body mass index (BMI) has become the gold standard for identifying those at increased risk for obesity-related adverse health outcomes, waist circumference is an emerging measure of central adiposity that can identify greatest risk for cardiometabolic

diseases more effectively than BMI alone. Studies provide evidence that PM is capable of acutely increasing blood pressure in certain scenarios (eg, BMI status, neighbourhoods).

What this study adds

We document, here, that the effect modification of the positive associations between particulate matter ($PM_{2.5}$) exposure and pulse pressure and systolic blood pressure (BP) by body mass index (BMI) and waist circumference (WCIR) is enhanced in areas proximate to sources of $PM_{2.5}$ emissions. Our results support the need to identify those at increased risk of cardiovascular disease (CVD) due to clustering of environment, location and individual-level risk factors. As public health researchers and practitioners develop evidence-based community interventions for reducing the risk for CVD in multiethnic communities, the cumulative and interactive contributions of environmental exposures with biological and anthropometric variables to unequal risk must be considered. If confirmed by longitudinal studies and larger sample sizes, these results provide insight into the plausible mechanistic pathways by which air particles evoke specific BP responses. Furthermore, community health interventions to reduce the population burden of CVD risk need to focus on regulating $PM_{2.5}$ exposure in concert with interventions to reduce BMI and WCIR and related co-morbidities.

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Table 1

Weighted descriptive characteristics for Healthy Environments Partnership (HEP) biomarker participants

	HEP biomarker participants			
Descriptive characteristics	**N**	**Percentage**	**Mean**	**SE**
Age	348		46.27	1.11
Gender				
Male	99	44.43		
Female	249	55.57		
Race–ethnicity				
Hispanic	55	18.12		
Non-Hispanic white	70	20.24		
Non-Hispanic black	213	58.85		
Other	10	2.79		
Education				
<12 years	115	34.32		
12 years	97	30.14		
>12 years	130	35.54		
Household income				
<$10 000	107	31.95		
$10 000–20 000	93	29.21		
$20 000–35 000	82	23.36		
$35 000+	49	15.48		
Body mass index (BMI)	348		30.93	0.46
Percentage obese (BIVII>30)				
No	167	49.86		
Yes	181	50.14		
Waist circumference (WCIR)	344		99.13	1.05
High-risk WCIR				
No	128	42.58		
Yes	216	57.43		
Diabetes				
No	303	65.24		
Yes	45	10.67		
Blood pressure				
Normal	114	32.02		
Prehypertensive	74	24.19		
Hypertensive	149	43.79		
High cholesterol >200 mg/dl				
No	225	64.05		
Yes	123	35.95		
Systolic blood pressure	344		128.84	1.30
Diastolic blood pressure	344		80.08	0.72

	HEP biomarker participants			
Descriptive characteristics	**N**	**Percentage**	**Mean**	**SE**
Pulse pressure	344		48.77	0.91
Arterial blood pressure	344		96.33	0.85
Smoking behaviour				
Never	118	33.99		
Current	152	43.33		
Former	78	22.68		

Table 2

Body mass index as effect modifier of the association between PM2.5 and hemodynamic measures in the Healthy Environments Partnership sample (n = 348)

	Lag 1		Lag 2		Lag 3		Lag 4		Lag 5	
	Δmm Hg	p value	Δmm Hg	p value	Δmm Hg	p value	Δmm Hg	p value	Δmm Hg	p value
Systolic BP										
PerObese	0.49	0.819	3.75	0.056	2.42	0.150	3.80	0.162	0.54	0.601
No PerObese	−1.99	0.251	1.25	0.515	−0.85	0.547	2.39	0.270	1.40	0.249
Diastolic BP										
PerObese	−1.96	0.136	−0.45	0.695	−0.10	0.933	1.41	0.437	0.36	0.657
No PerObese	−1.19	0.287	−2.06	0.126	−0.72	0.536	1.10	0.499	0.83	0.320
Pulse										
PerObese	2.50	0.118	**4.16**	**0.005**	**2.55**	**0.018**	2.58	0.113	0.27	0.712
No PerObese	−0.79	0.527	3.32	0.057	−0.09	0.931	1.51	0.323	0.60	0.367
Arterial BP										
PerObese	−1.17	0.417	0.93	0.474	0.74	0.552	2.22	0.276	0.41	0.622
No PerObese	−1.47	0.226	−0.99	0.452	−0.79	0.494	1.46	0.392	0.99	0.286

PerObese, BMI≥30; No PerObese, BMI<30; BP, blood pressure; PM, particulate matter.

Covariates included race–ethnicity, household income, educational level, age, gender, medication use for hypertension and location.

Table 3

Waist circumference as effect modifier of the association between PM2.5 and haemodynamic measures in the Healthy Environments Partnership sample (n = 348)

	Lag 1		Lag 2		Lag 3		Lag 4		Lag 5	
	Δmm Hg	p value	Δmm Hg	p value	Δmm Hg	p value	Δmm Hg	p value	Δmm Hg	p value
Systolic BP										
WCIR	−0.54	0.756	3.22	0.054	1.95	0.237	3.77	0.181	0.03	0.785
No WCIR	−2.57	0.199	0.81	0.731	−1.31	0.399	2.14	0.357	0.22	0.052
Diastolic BP										
WCIR	−1.87	0.107	−1.12	0.276	−0.23	0.847	1.46	0.418	0.01	0.862
No WCIR	−0.93	0.439	−1.82	0.224	−0.80	0.477	0.96	0.575	0.13	0.106
Pulse										
WCIR	2.35	0.070	**4.34**	**0.003**	2.22	0.055	2.45	0.160	0.02	0.752
No WCIR	−1.61	0.241	2.67	0.190	−0.41	0.694	1.44	0.345	0.09	0.164
Arterial										
WCIR	−1.11	0.359	0.30	0.781	0.50	0.690	2.24	0.277	0.02	0.828
No WCIR	−1.48	0.277	−0.97	0.527	−1.00	0.393	1.28	0.478	0.16	0.072

WCIR, waist circumference ≥88 cm for females, 102 cm for males. No WCIR, waist circumference <88 cm for females, 102 cm for males.

BP, blood pressure; PM, particulate matter.

Covariates included race–ethnicity, household income, educational level, age, gender, medication use for hypertension and location.

Table 4

Body mass index as effect modifier of the association between PM2.5 and haemodynamic measures in the Healthy Environments Partnership sample (n = 348), including location interaction with exposure

	Lag 1		Lag 2		Lag 3		Lag 4		Lag 5	
	Δmm Hg	**p value**	**Δmm Hg**	**p value**	**Δmm Hg**	**p value**	**Δmm Hg**	**p value**	**Δmm Hg**	**p value**
Systolic BP										
PerObese	−1.81	0.462	**4.82**	**0.013**	**4.68**	**0.011**	**8.99**	**0.012**	0.90	0.448
No PerObese	−4.22	0.069	2.52	0.161	1.27	0.394	**7.22**	**0.024**	1.70	0.131
Diastolic BP										
PerObese	−2.80	0.080	−0.72	0.631	0.99	0.564	3.02	0.345	0.29	0.834
No PerObese	−2.03	0.106	−2.33	0.161	−0.01	0.996	2.34	0.402	0.77	0.388
Pulse										
PerObese	1.10	0.659	**5.60**	**0.005**	**3.87**	**0.006**	**6.30**	**0.003**	0.68	0.547
No PerObese	−2.12	0.343	**4.97**	**0.044**	1.42	0.278	**5.15**	**0.011**	0.97	0.315
Arterial BP										
PerObese	−2.50	0.107	1.11	0.406	2.23	0.177	4.98	0.117	0.47	0.702
No PerObese	**−2.78**	**0.038**	−0.73	0.542	0.39	0.794	3.86	0.166	1.03	0.233

PerObese, BMI≥30; No PerObese, BMI<30; BP, blood pressure; PM, particulate matter.

Covariates included race–ethnicity, household income, educational level, age, gender, medication use for hypertension and location.

Table 5

Waist circumference as effect modifier of the association between PM2.5 and haemodynamic measures interaction with exposure in the Healthy Environments Partnership sample (n = 348), including location interaction with exposure

	Lag 1		Lag 2		Lag 3		Lag 4		Lag 5	
	Δmm Hg	**p value**	**Δmm Hg**	**p value**	**Δmm Hg**	**p value**	**Δmm Hg**	**p value**	**Δmm Hg**	**p value**
Systolic BP										
WCIR	−2.18	0.343	**4.22**	**0.023**	**3.87**	**0.029**	**8.36**	**0.026**	0.69	0.516
No WCIR	**−5.02**	**0.038**	1.51	0.537	0.59	0.715	**6.78**	**0.043**	**2.38**	**0.028**
Diastolic BP										
WCIR	−2.73	0.073	−1.20	0.424	0.76	0.654	2.89	0.343	0.01	0.995
No WCIR	−1.90	0.130	−2.24	0.203	−0.09	0.956	2.20	0.442	1.20	0.179
Pulse										
WCIR	0.64	0.781	**5.50**	**0.009**	**3.26**	**0.030**	**5.76**	**0.009**	0.75	0.501
No WCIR	−3.03	0.197	3.90	0.199	0.84	0.556	**4.93**	**0.020**	1.26	0.181
Arterial										
WCIR	−2.56	0.082	0.59	0.636	1.80	0.258	4.68	0.138	0.21	0.837
No WCIR	**−2.96**	**0.029**	−1.01	0.458	0.10	0.946	3.61	0.209	1.54	0.069

WCIR, waist circumference ≥ 88 cm for females, 102 cm for males. No WCIR, waist circumference <88 cm for females, 102 cm for males.

BP, blood pressure; PM< particulate matter.

Covariates included race–ethnicity, household income, educational level, age, gender, medication use for hypertension and location.