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Associations of long-term fine particulate matter exposure with prevalent hypertension and increased blood pressure in older Americans

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Abstract

Background—Hypertension is a highly prevalent cardiovascular risk factor. It is possible that air pollution, also an established cardiovascular risk factor, may contribute to cardiovascular disease through increasing blood pressure. Previous studies evaluating associations between air pollution and blood pressure have had mixed results.

Methods—We examined the association between long-term (one-year moving average) air pollutant exposures, prevalent hypertension and blood pressure in 4,121 older Americans (57+ years) enrolled in the National Social Life, Health, and Aging Project. We estimated exposures to $PM_{2.5}$ using spatio-temporal models and used logistic regression accounting for repeated measures to evaluate the association between long-term average $PM_{2.5}$ and prevalence odds of hypertension. We additionally used linear regression to evaluate the associations between air pollutants and systolic, diastolic, mean arterial, and pulse pressures. Health effect models were adjusted for a number of demographic, health and socioeconomic covariates.

Results—An inter-quartile range (3.91 μg/m^3) increase in the one-year moving average of $PM_{2.5}$ was associated with increased: Odds of prevalent hypertension (POR 1.24, 95% CI: 1.11, 1.38), systolic blood pressure (0.93 mm Hg, 95% CI: 0.05, 1.80) and pulse pressure (0.89 mm Hg, 95% CI: 0.21, 1.58). Dose-response relationships were also observed.

Conclusions—$PM_{2.5}$ was associated with increased odds of prevalent hypertension, and increased systolic pressure and pulse pressure in a cohort of older Americans. These findings add to the growing evidence that air pollution may be an important risk factor for hypertension and perturbations in blood pressure.

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Keywords

Air pollution; Elderly; Hypertension; Blood pressure; Pulse pressure

1. INTRODUCTION

Approximately 75 million people, or 29% of all adults in the United States (US), have hypertension, and an additional third of Americans have pre-hypertension.[1] In 2013, 360,000 Americans died from hypertension-related diseases such as myocardial infarctions, cerebrovascular accidents, congestive heart failure and renal insufficiency.[1 2] It is estimated that treatment of hypertension and its sequelae in the U.S. costs over 46 billion dollars per year.[2] Since hypertension is both easily diagnosed and treatable, with successful treatment correlated with decreased subsequent morbidity and mortality,[3] control of hypertension has emerged as a cornerstone of preventative cardiovascular care.[4] While current clinical recommendations focus on individual, modifiable exposures to hypertension risk factors such as physical inactivity, poor diet, alcohol and tobacco use,[5] it is possible that environmental risk factors, such as air pollution, may also be important determinants of hypertension risk.

Air pollution, like hypertension, is a known, key risk factor for adverse cardiovascular health outcomes.[6 7] Evidence from animal [8 9] and epidemiologic [10] studies suggests that air pollution may impair cardiovascular function by inducing a chronic, systemic inflammatory response, increasing oxidative stress and plasma viscosity and altering autonomic nervous input to the heart and vasculature.[11] These pathophysiologic events can in turn lead to endothelial dysfunction, altered arterial diameter [12] or vascular tone and changes in heart rate, all of which ultimately can result in increased blood pressure and hypertension.[12 13]

While studies have provided key evidence linking air pollution exposures to adverse cardiovascular outcomes, epidemiological findings for hypertension are less consistent. Several studies have identified positive associations between air pollution and incident [14–17] and prevalent [18–20] hypertension; however, others have reported no association.[21–23] This inconsistency may reflect heterogeneity in a number of factors between studies, including differences in the studied populations, the exposure assessment methods, and the methods used to assess hypertension. Few studies, for example, have investigated the air pollution-hypertension association in large US populations, and none in nationally representative samples, significantly limiting inference.

A number of studies have examined associations between air pollution and blood pressure as a continuous measure, and those that have report heterogeneous results.[21 23 25–27] As cardiovascular risk increases at pressures lower than those used to diagnose hypertension (such as in pre-hypertension),[28 29] studies that evaluate hypertension as a dichotomous outcome might fail to adequately quantify the effects of air pollution on blood pressure-related cardiovascular outcomes. Furthermore, blood pressure has a number of component measurements, each a quantification of a different aspect of cardiovascular function. The systolic pressure (the maximum arterial pressure during the cardiac cycle) and the diastolic pressure (the minimum arterial pressure during a cardiac cycle) are perhaps the best studied,

and increases in either lead to a diagnosis of hypertension due to their established, adverse long-term cardiovascular effects. In addition to maximum and minimum measurements, blood pressure is further composed of steady components (quantified by mean arterial pressure) and pulsatile components (quantified by pulse pressure) which describe the cardiovascular function between blood pressure peaks and troughs.[30] Physiologically, mean arterial pressure is a proxy measure for tissue and organ perfusion pressure, while pulse pressure is proportional to cardiac stroke volume and a measure of conduit artery stiffness. Importantly, increases in all of these aspects of blood pressure have been previously associated with elevated cardiovascular risk,[30 31] while a number of recent studies have shown pulse pressure to have the greatest predictive ability for cardiovascular disease in older (>60 years) populations.[32] To date, few studies have investigated the effects of air pollution on mean arterial pressure and pulse pressure, but those that have observed significant, positive associations.[25 27] While elevations in these measures have both been associated with cardiovascular outcomes, they are not incorporated into the diagnosis of hypertension. So studies examining hypertension as an outcome may fail to completely capture the cardiovascular risk associated with changes in these blood pressure parameters.

To address these gaps in the current literature, we investigate whether fine particulate air pollution ($PM_{2.5}$) is associated with odds of prevalent hypertension in a nationally representative cohort of older Americans utilizing high quality exposure estimates of $PM_{2.5}$. We additionally investigate the association between particulate air pollutants and a number of continuous blood pressure measures, including systolic blood pressure, diastolic blood pressure, mean arterial pressure and pulse pressure.

2. METHODS

2.1 Population

We used demographic, health, and other data from a nationally representative probability sample of Americans participating in the National Social Life, Health, and Aging Project (NSHAP). NSHAP is a national area probability sample of 4,121 community residing, older (57+ year) adults, selected from households identified in the Health and Retirement Study (a national, multi-stage area probability sample with a target population of all U.S. adults) in 2004. [33 34] Participants included 4,121 older Americans. Wave 1 recruited 3,005 participants examined in 2005–2006 and Wave 2 included 3,377 participants in 2011–2012, with 2,261 individuals participating in both waves; 744 Wave 1 participants were either too sick to participate in Wave 2 or deceased. Additional Wave 2 participants were selected from eligible respondent (n=907) and non-respondent (n=209) households originally identified from the HRS probability sample. The survey over-sampled African-Americans, Latinos, men and individuals between 75–84 years.[33] Response rates for each wave were high, with 75% and 74% of individuals selected for Wave 1 and Wave 2 opting to participate, respectively. For each data collection wave, participants underwent interviews to obtain demographic, social (social networks, social support, marital history and intimate partnerships and sexuality), and health (self-reported health, physical function and morbidity) data, including medical history and a comprehensive list of current medications.

At the time of the interviews, biomeasure data on anthropometrics (height, weight, waist circumference) and cardiovascular health (blood pressure, pulse) were also collected.[33 34]

2.2 Outcome Assessment

During each data collection wave, two blood pressure measurements were collected for each participant from the left arm using a Lifesource digital blood pressure monitor (Model: UA-767PVL) according to manufacturer specifications.[35] If measured systolic blood pressure differed by >20 mm Hg or diastolic blood pressure differed by >14 mm Hg across the two measurements, then a third measurement was taken. Systolic blood pressure and diastolic blood pressure were calculated for inclusion in statistical models as the arithmetic mean of all measurements taken for an individual. Mean arterial pressure was calculated as [systolic blood pressure + (2*diastolic blood pressure)]/3; pulse pressure was calculated as the difference between systolic blood pressure and diastolic blood pressure.[36] In addition, hypertensive status was assessed using 1) Self-report of history of physician-diagnosed hypertension and 2) Self-report of current anti-hypertensive medication consumption. From these data, we defined prevalent hypertension as having a self-report of either hypertension history or medication use or having a measured systolic blood pressure or diastolic blood pressure greater than or equal to 140 mm Hg or 90 mm Hg, respectively.[37]

2.3 Covariates

We controlled for potential confounding using wave-specific covariates previously associated with hypertension or air pollution,[19 21 23 27] including body mass index (BMI), race/ethnicity, socio-economic status (SES), current smoking status, physical activity, alcohol consumption and residence location. Race/ethnicity was categorized as White, Black, Hispanic or other. Socioeconomic status (SES) was assessed on an individual level using self-reported educational attainment and on a neighborhood level using median household income and percent of households below the federal poverty line as reported in the 2000 US Census. Behavioral factors included smoking status (current, historical or none), self-reported frequency of physical activity, alcohol use (ever versus none), and estimated number of alcoholic drinks consumed per week. We additionally control for self-reported use of anti-hypertensive medications in any of the following classes: Diuretics, calcium channel blockers, beta blockers, alpha blockers, angiotensin converting enzyme (ACE) inhibitors, vasodilators and angiotensin receptor blockers (ARBs). Each class of anti-hypertensive was modeled with an individual indicator variable to reduce potential for residual confounding in those on more than one anti-hypertensive. Geographic covariates included region of residence (North Atlantic, South, Great Lakes region, Plains States, Pacific) as well as six categories of urbanicity (Categorized as: [1) 12 largest Standard Metropolitan Statistical Areas (SMSAs) 2) 13–100 largest SMSAs, 3) 12 largest suburbs, 4) 13–100 largest suburbs, 5) Other urban, 6) Other rural].

2.4 Exposure assessment

For each person, we estimated exposures to $PM_{2.5}$ using spatio-temporal models as described in Yanosky et al..[38] Briefly, $PM_{2.5}$ data were obtained from the US Environmental Protection Agency (EPA) Air Quality System database and Interagency Monitoring of Protected Visual Environments (IMPROVE) network.[39 40] These data,

along with meteorological (wind speed, temperature, total precipitation) and geospatial (county population density, line-source traffic density, point-source $PM_{2.5}$ emissions, elevation) covariates were used to fit a series of generalized additive mixed models. The daily spatio-temporal estimates derived from these models were used to calculate long-term moving average exposure estimates for each point on a 6 km grid of the conterminous US. NSHAP participants were assigned exposure estimates based on the grid point closest to their permanent addresses (mean distance, 2.23 km). The spatio-temporal estimates have been previously validated using cross-validation techniques, demonstrating high accuracy and low bias (cross-validation R^2 of 0.76).[38]

2.5 Statistical Analysis

Logistic regression models with a generalized estimating equation (GEE) approach to account for repeated measures were employed to investigate the associations between an interquartile range (IQR) increase in one-year $PM_{2.5}$ moving average and prevalence odds ratios (POR) of hypertension as a dichotomous outcome. Linear regression models using GEEs to account for repeated measures were used to examine associations between air pollution and various measures of blood pressure. All fully adjusted models included all participants (4,121 total participants, of which 2,261 had repeated measures) and controlled for BMI, age, sex, education, race/ethnicity, history of diabetes, behavioral variables (self-reported tobacco and alcohol use, physical activity) area-level socioeconomic indicators (including census-track data on median household income and percent of households living below the poverty line) and geographic covariates (region and urbanicity). Linear models of blood pressure additionally controlled for hypertensive medication use, while logistic models included did not, as hypertension medication use was a component of the outcome definition. All models were weighted to account for non-response and oversampling of certain populations..

Effect modification was investigated by education, physical activity, smoking status, age, sex, diabetes status, race and BMI. Last, dose-response relationships were modeled utilizing indicator variables for quartiles of exposure and non-parametric dose-response curves.

Data completeness for the blood pressure measurements was high, with only 84 (2.8%) Wave 1 and 120 (3.6%) Wave 2 participants missing blood pressure measurements. Data completeness for covariates was similarly high, with most covariates having less than 5% missing data, while BMI had a slightly higher (6.3%) missingness. Multiple imputation with chained equations was used to handle all covariate and outcome variable missingness by creating ten datasets with complete data and pooling estimates across datasets.[41]

A number of sensitivity analyses were conducted. First, mean arterial pressure was alternatively calculated as mean arterial pressure = diastolic blood pressure + 0.412 (systolic blood pressure – diastolic blood pressure) as a prior study identified this as producing a better approximation of actual mean arterial pressure.[42] Second, to minimize outcome misclassification for hypertension, we examined a more conservative definition of hypertension defined as: 1) Self report of current anti-hypertensive medication consumption, or 2) Systolic blood pressure ≥ 140 mm Hg, or diastolic blood pressure ≥ 90 mm Hg. Third, variables (i.e., BMI, history of diabetes) that are potentially on the causal pathway between

air pollution and hypertension were excluded from models. Last, complete case analyses were undertaken to examine the potential effects of the imputation methods. Analyses were completed using SAS Version 9.4 (SAS INC, Cary, NC).

3. RESULTS

3.1 Participant characteristics

Table 1 shows participant demographic, socioeconomic, health and average exposure characteristics. The average age of participants was approximately 70 years (SD 8.1) and 53.7% were female. Hypertension was highly prevalent, with 80.1% percent in Wave 1 hypertensive and 83.2% in Wave 2 meeting the study definition of hypertension and 58.6% of participants across both waves reporting anti-hypertension medication use.

Participants with hypertension tended to be older (P<0.001), male (47.3% versus 42.3%, p=0.010), have a higher BMI (29.8 kg/m^2 versus 26.8 kg/m^2, p<0.001), be more likely to have diabetes (26.1% versus 8.3%, p<0.001) and be less physically active than participants without hypertension. Hypertensive patients also had significantly higher one-year moving average $PM_{2.5}$ exposures as compared to non-hypertensive participants (10.5 $\mu g/m^3$ versus 10.1 $\mu g/m^3$, p=0.001).

3.2 Associations between $PM_{2.5}$ and hypertension

$PM_{2.5}$ exposures were positively associated with prevalent hypertension (Table 2). In base models, an IQR (3.91 $\mu g/m^3$) increase in $PM_{2.5}$ was associated with an 18% increased odds of prevalent hypertension (POR 1.18, 95% CI: 1.08, 1.29). Upon adjusting for geographic, behavioral, and area-level socioeconomic variables, the $PM_{2.5}$-associated POR increased 33% (POR 1.24, 95% CI: 1.11, 1.38). In models of quartiles of $PM_{2.5}$ exposure and prevalent hypertension, the PORs associated with the third and fourth quartiles of exposure were significantly higher than those in the lowest quartile of exposure, suggesting a dose-response relationship (Table 2; *p*- trend<0.001).

3.3 Associations between $PM_{2.5}$ and blood pressure measures

Table 3 summarizes the associations between $PM_{2.5}$ and the blood pressure measures. In base models, an IQR increase in one-year moving average $PM_{2.5}$ exposure was associated with an increase in systolic blood pressure of 0.73 mm Hg (95% CI: 0.00, 1.46); the effect estimate was 27.4% higher (0.93 mm Hg, 95% CI: 0.05, 1.80) in the fully adjusted models compared to that in base models. Similar magnitude and trend were also found for pulse pressure, with an IQR increase in $PM_{2.5}$ associated with an 0.89 mm Hg (95% CI: 0.21, 1.58) increase in pulse pressure in fully adjusted models. On the other hand, one-year $PM_{2.5}$ exposures were not associated with diastolic blood pressure or mean arterial pressure in either base or fully-adjusted models. Further analyses of systolic blood pressure and pulse pressure show the presence of dose-response relationships, with the third and fourth quartile effects higher than those in lower quartiles of exposure (Table 4, p_{trend}=0.056 for systolic blood pressure and p_{trend}=0.069 for pulse pressure). Interestingly, for both outcomes third quartile effect estimates were higher than those in the fourth quartile. This was particularly true for pulse pressure, suggesting a non-linear association. Consistent with these findings,

in dose-response curves fit with natural cubic splines, the associations between air pollution and systolic blood pressure (Figure 1) were largely linear, while a more pronounced deviation from linearity was observed for pulse pressure (Figure 2), consistent with a threshold effect.

3.4 Effect modification

No significant effect modifiers were identified for prevalent hypertension (data not shown). Participants with a history of type 2 diabetes mellitus, however, were found to have increased $PM_{2.5}$-associated impacts on pulse pressure (1.85 mm Hg versus 0.64 mm Hg), although the interaction term was borderline statistically significant ($p_{interact}$=0.071; Table 5).

3.5 Sensitivity Analysis

Models using an alternative calculation of mean arterial pressure did not significantly affect the null findings for this outcome (data not shown), nor did logistic regression models investigating a more conservative definition of HTN (Supplement Table 1). Models excluding covariates potentially on the causal pathway (BMI, diabetes) between air pollution and systolic blood pressure (0.89 mm hg, 95% CI: 0.02, 1.77), pulse pressure (0.93 mm hg, 95% CI: 0.25, 1.62) and hypertension (OR 1.22, 95% CI: 1.10, 1.35) and analyses restricted to complete cases (Supplement Table 2) did not differ appreciably from our fully adjusted, imputed models.

4. DISCUSSION

We observed that $PM_{2.5}$ was significantly associated with increased prevalence of hypertension and with increased measures of blood pressure, as assessed using systolic blood pressure and pulse pressure in a nationally representative cohort of older Americans. The impact of $PM_{2.5}$ on hypertension prevalence and systolic blood pressure was greatest in the third and fourth as compared to lowest quartile of exposure, consistent with a dose-response relationship, while a non-linear dose response was identified for pulse pressure. Diabetic participants had larger associations between pulse pressure and $PM_{2.5}$ exposures, although this was marginally significant ($p_{interact} < 0.10$).

Our finding of an association between $PM_{2.5}$ and hypertension prevalence is consistent with some, but not all, previous literature. Babisch et al. (2014) reported positive associations (OR 1.15, 95% CI: 1.02, 1.30) between increases in $PM_{2.5}$ (1 μg increment) and prevalent hypertension,[20] as did Pitchika et al. [43], who observed a 1 μg increment increase in $PM_{2.5}$ to be associated with a 15% increase in hypertension prevalence (95% CI: 2.5%, 28.0%) in a German population. This is consistent with findings by Liu et al. [44] who observed a 41.7 $\mu g/m^3$ increment in $PM_{2.5}$ to be significantly associated with increased odds of prevalent hypertension (POR 1.11, 95% CI: 1.05, 1.17) in a Chinese population. Dong et al. (2013) similarly observed increased odds of prevalent hypertension for long-term PM_{10} exposures (OR 1.12, 95% CI: 1.08, 1.16),[19] in a Chinese population. In contrast, Fuks et al. [24] examined the association between prevalent hypertension and residential distance to nearest major roadway in a population-based German cohort and observed no association, as

did Foraster et al. (2014) in a European cohort.[21] This heterogeneity in study findings may reflect design differences in terms of populations studied and differences in pollution constituents, as both studies which found null effects were in European populations.[21 22] Our study, in a nationally-representative cohort of older Americans using high-quality $PM_{2.5}$ exposure estimates, adds significantly to the extant literature on $PM_{2.5}$ and hypertension prevalence.

Most literature investigating the association of air pollution with blood pressure as a continuous measure has reported similar, positive associations to those we observe for $PM_{2.5}$.[21 23–27 45] Auchincloss et al. (2008) reported positive associations between $PM_{2.5}$, systolic blood pressure and pulse pressure in the Multi-Ethnic Study of Atherosclerosis (MESA) cohort [25] while in a Taiwanese cohort Chuang et al. (2010) observed particulate matter exposure to be associated with systolic blood pressure, but not diastolic blood pressure.[45] Similarly, Fuks et al. (2011) observed long-term $PM_{2.5}$ exposure to be positively associated with both systolic blood pressure and diastolic blood pressure in a German cohort,[46] while Chan et al. (2015) reported a 10 $\mu g/m^3$ increase in $PM_{2.5}$ to be associated with increases in systolic blood pressure (1.4 mm Hg, $p<0.001$), mean arterial pressure (0.8 mm Hg, $p=0.01$) and pulse pressure (1.0 mm Hg, $p=0.01$), but not diastolic blood pressure.[27] This study is similar to ours in that it used a national sample, high-quality exposure estimates and controlled for a number of likely confounders. In contrast, Pitchika et al. (2017) found 1 $\mu g/m^3$ increase in long-term $PM_{2.5}$ exposure to be associated with diastolic pressure (0.7 mmHg, 95% CI: 0.2; 1.2), but not systolic pressure in a German cohort, [43] while Zhang et al. (2018) observed a 10 $\mu g/m^3$ 2-year moving average increment in $PM_{2.5}$ concentration was associated with increases in SBP (0.45 mmHg, 95% CI: 0.40, 0.50), DBP (0.07 mmHg, 95% CI: 0.04, 0.11), and PP (0.38 mmHg, 95% CI: 0.33, 0.42) in a Taiwanese population.[47] The observed heterogeneity in the literature is likely influenced by differences in populations and exposure constituents and magnitudes across studies.

Importantly, our findings of significantly increased systolic blood pressure and pulse pressure are potentially of clinical relevance, as both have been associated with increased risk of cardiovascular events in older individuals.[30 32 48 49] In a 2001 study Franklin et al. found that the importance of each blood pressure fraction as a measure of risk differs by age. In individuals <50 years old, diastolic blood pressure was found to be the strongest predictor of coronary heart disease (CHD) development (HR per 10 mm Hg increment, 1.34; 95% CI, 1.18 to 1.51) while systolic blood pressure (HR 1.14, 95% CI: 1.06, 1.24) and pulse pressure (HR 1.02, 95% CI: 0.89, 1.17) were less strongly associated. In individuals 50–59 years, risks for all three measures were comparable in terms of CHD risk, while in individuals >60 years (the age group with by far the largest representation in our study) pulse pressure was the strongest predictor of CHD risk (HR 1.24, 95% CI: 1.16, 1.33) while diastolic blood pressure became a non-significant predictor (HR 1.12, 95% CI: 0.99, 1.27). [32] Similar results were reported in a 2003 analysis of the Framingham Heart Study in which systolic blood pressure (HR 1.56, 95% CI: 1.37, 1.77) and pulse pressure (HR 1.55, 95% CI: 1.37, 1.75) elevations conferred the greatest risk for incident congestive heart failure among adults 50–79 years.[50] As we observed air pollution exposures to be significantly associated with elevated systolic blood pressure and pulse pressure but not

diastolic blood pressure in a population >57 years, these previous studies suggest that air pollution may confer a blood pressure phenotype which is at particularly high risk for detrimental cardiovascular outcomes in this age group.

Interestingly, the observed pattern of results suggests physiologic mechanisms through which $PM_{2.5}$ increases blood pressure in older individuals. Blood pressure is a function of cardiac output (stroke volume and heart rate) as well as peripheral resistance.[51] Peripheral resistance (R) is described by:

$$R \propto \frac{\eta \cdot L}{r^4}$$

in which resistance is directly proportional to vessel length (L) and blood viscosity (η), but inversely proportional to the fourth power of vessel radius (r), making vessel radius the most important determinant of peripheral vascular resistance.[52] Current biological hypotheses involve air pollution affecting blood viscosity, autonomic nervous activity and endothelial function via direct irritation of pulmonary receptors, oxidative stress, and systemic inflammation.[12 13] Plasma viscosity increases due to pulmonary macrophages increasing cytokine production, leading to increases in hepatic soluble acute phase protein production (i.e. fibrinogen, CRP, complement, haptoglobin).[9] The resultant increase in plasma viscosity is directly proportional to increased blood pressure.[53] Increasing sympathetic autonomic activity (or conversely, withdrawing parasympathetic activity) increases heart rate and myocardial contractility (and thus cardiac output) as well as peripheral resistance due to constriction of vascular smooth muscle. Endothelial dysfunction, characterized by decreased production of vasodilators (i.e. nitric oxide) and increased production of vasoconstrictors (i.e. endothelin), also leads to net arterial vasoconstriction.[11] As both of these latter mechanisms serve to decrease vessel radius, the single most important determinant of peripheral resistance, even small changes in sympathetic tone and endothelial function can have dramatic effects on all blood pressure measures we investigate.

These mechanisms, however, do not completely explain the pattern of our findings, as we observed significant increases in pulse pressure and systolic pressure associated with increased air pollution, but not diastolic pressure or mean arterial pressure. Pulse pressure and systolic pressure are impacted by heart rate, left ventricular ejection volume, large artery elasticity and the magnitude and speed of the backward pressure wave arising in the peripheral circulation.[30] Elastin, a ubiquitous protein in the large conduit arteries which contributes to arterial wall elasticity, breaks down with increased age, leading to increased aortic stiffness, impedance and pulse wave velocity.[54 55] Pulse pressures and systolic pressures increase with increased pulse wave velocity, as the systolic pressure wave is reflected back into the large conduit arteries earlier than in normal physiology (in systole rather than diastole). These processes, taken together, serve to increase systolic blood pressure and pulse pressure, but not diastolic blood pressure or mean arterial pressure.[55] Thus, while all of the aforementioned mechanisms likely contribute to the observed increased hypertension risk, increased large arterial stiffness may explain the pattern of significant and non-significant associations seen in our study. Concordant with this, systemic

inflammation, a major biological pathway by which air pollution is hypothesized to exert cardiovascular health effects, has also been related to increased aortic arterial stiffness.[56]

We observed borderline significant effect modification by diabetes status. Interestingly, diabetes has also been shown to accelerate age-associated increases in conduit artery stiffness which may help mechanistically explain the observed effect modification.[57] Importantly, concomitant hypertension has been estimated to account for up to 75% of cardiovascular disease among diabetics.[58] As diabetics are already at increased risk of cardiovascular events as compared to non-diabetics,[59] the observed increased risk of blood pressure elevations among diabetics may thus be of significant clinical import. Specifically, it may demonstrate that diabetics are a particularly susceptible population to the increased cardiovascular risk consistently associated with air pollution.

Our study has a number of limitations. First, the observational nature of the study design limits our ability to draw causal inferences. Second, exposure error for $PM_{2.5}$ is probable. While our $PM_{2.5}$ estimates have been previously validated, the estimates are for outdoor pollution concentrations and do not estimate personal exposures *per se*. However, previous studies in elderly populations report high correlations between outdoor $PM_{2.5}$ concentrations and personal exposures.[60 61] Last, outcome misclassification is possible, as use of any medication in a pharmacologic class traditionally used to treat hypertension was incorporated into the dichotomous definition of the outcome. It is likely that at least some individuals were taking antihypertensive medications for treatment of non-hypertensive conditions.

Despite these limitations, our study is among very few to examine these associations in a nationally representative cohort of older Americans. We employed $PM_{2.5}$ estimates that have been shown to have high accuracy and precision in previous validation studies [38] and were able to control for an extensive list of likely confounders. Our reliance on three distinct measures of hypertension and consistency in results using both conservative and more liberal hypertension definitions limits the likelihood that outcome misclassification significantly affected our results.

5. CONCLUSIONS

Particulate matter was significantly associated with elevated odds of prevalent hypertension as well as increases in systolic and pulse pressures in a nationally representative cohort of older Americans. These findings add further to the growing body of evidence that air pollution may be an important risk factor for hypertension and perturbations in blood pressure.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Abbreviations

$PM_{2.5}$	Particulate matter with an aerodynamic diameter of ≤2.5μm
POR	Prevalence odds ratio

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Highlights

- Studies of air pollution and hypertension have reported mixed results.
- Few studies have examined systolic, diastolic, pulse pressure, and mean arterial blood pressure components.
- Increased long-term particulate matter exposure was found to be associated with increased odds of prevalent hypertension.
- Significant associations between air pollution and systolic and pulse pressures were also identified.

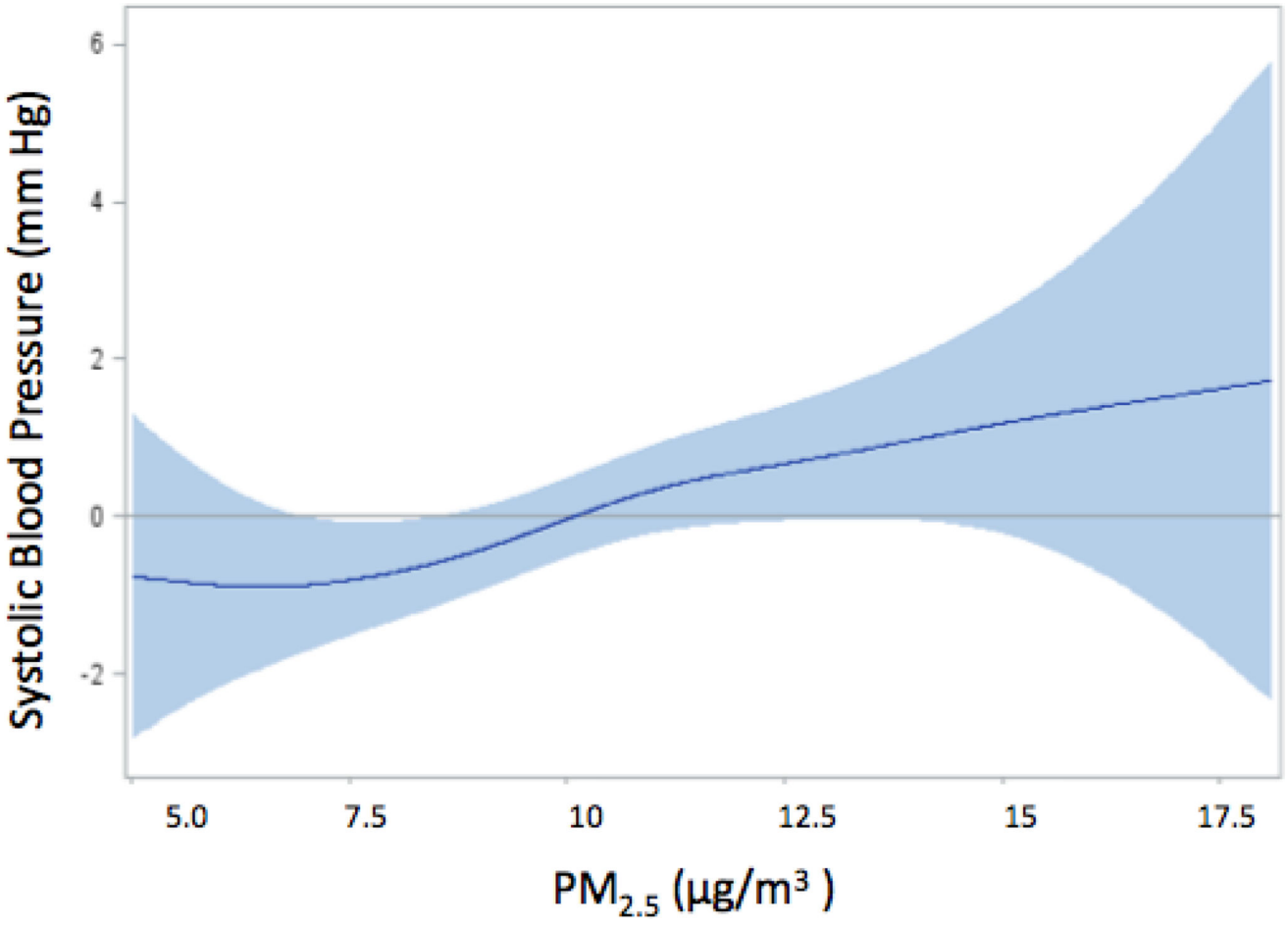

Figure 1. Spline representation of the association between an IQR increment in $PM_{2.5}$ and levels of systolic pressure[a]

[a]The shaded region indicates 95% confidence intervals

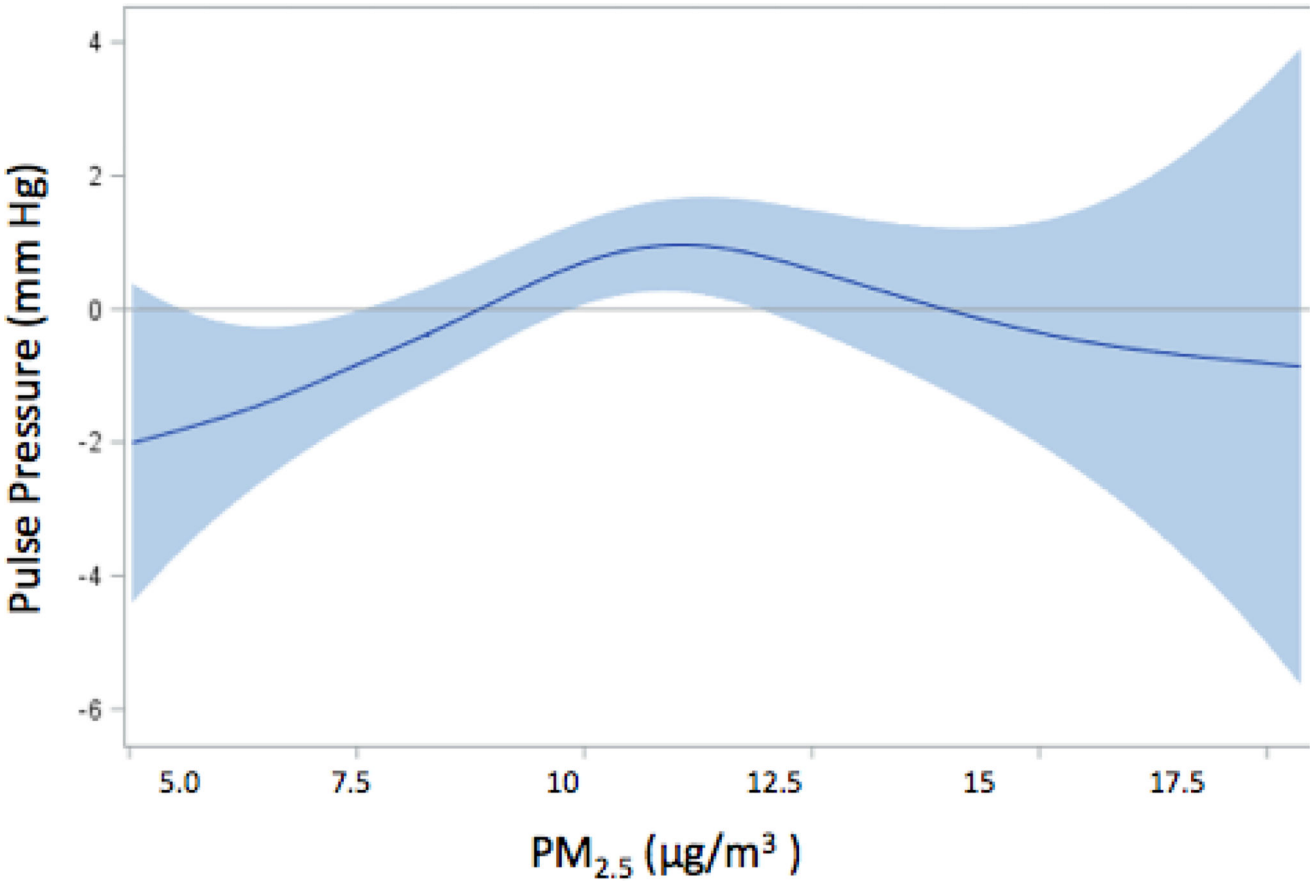

Figure 2. Spline representation of the association between an IQR increment in $PM_{2.5}$ and levels of pulse pressure[a]

[a]The shaded region indicates 95% confidence intervals

Table 1

Characteristics of participants at study entry

Covariates	Total Observations (N=4121)	With HTN (N=3250)	Without HTN (N=871)	p-value for difference
Age (mean, SD)	69.6 ± 8.1	70.2 ± 0.14	66.8 ± 8.1	<0.001
Male (n, %)	1909 (46.3%)	1568 (47.3%)	341 (42.3%)	0.010
HEALTH STATUS VARIABLES				
BMI kg/m2 (mean, SD)	29.2 ± 6.2	29.8 ± 6.4	26.8 ± 5.0	<0.001
-Obese (n, %)	1471 (35.7%)	1272 (42.3%)	199 (23.8%)	<0.001
-Overweight (n, %)	1430 (34.7%)	1109 (34.5%)	321 (35.3%)	0.660
Systolic pressure (mean, SD)	136.9 ± 20.4	140.5 ± 20.5	121.8 ± 10.6	<0.001
Diastolic pressure (mean, SD)	80.8 ± 11.9	82.0 ± 12.3	75.5 ± 7.8	<0.001
Mean arterial pressure (mean, SD)	99.5 ± 13.0	101.5 ± 13.2	90.9 ± 7.6	<0.001
Pulse pressure (mean, SD)	56.2 ± 16.9	58.5 ± 17.4	46.3 ± 45.6	<0.001
History of diabetes (n, %)	933 (22.6%)	866 (26.1%)	67 (8.3%)	<0.001
RACE/ETHNICITY				
-White (n, %)	2920 (70.0%)	2316 (70.1%)	604 (75.0%)	<0.001
-Black (n, %)	651 (15.8%)	574 (17.4%)	77 (9.6%)	
-Hispanic (n, %)	439 (10.7%)	332 (10.1%)	107 (13.3%)	
-Other (n, %)	97 (2.4%)	80 (2.4%)	17 (2.1%)	
BEHAVIORAL VARIABLES				
Current tobacco use				0.115
-Yes (n, %)	714 (17.3%)	559 (16.9%)	155 (19.2%)	
-No (n, %)	1719 (41.7%)	2755 (83.1%)	652 (80.8%)	
Physical Activity				<0.001
- <3 times per week (n, %)	1,172 (28.4%)	1017 (31.3%)	155 (17.8%)	
- ≥3 times per week (n, %)	2,949 (71.6%)	2292 (70.5%)	651 (74.7%)	
SOCIOECONOMIC VARIABLES				
Education level				<0.001
- <High school (n, %)	887 (21.5%)	758 (22.9%)	129 (16.0%)	
- High school equivalent (n, %)	1059 (25.7%)	879 (26.5%)	180 (22.3%)	
- Some college (n, %)	1239 (30.1%)	990 (29.9%)	249 (30.9%)	
- College degree or greater (n, %)	936 (22.7%)	687 (20.7%)	249 (30.9%)	
Median household income (mean, SD)	54,259 ± 26,039	53,152 ± 28,783	58,838 ± 28,783	<0.001
Percent census track below poverty line (mean, SD)	14.8 ± 12.2	15.2 ± 12.3	13.2 ± 11.3	<0.001
POLLUTANT (mean, SD)				
- $PM_{2.5}$ ($\mu g/m^3$)	10.4 ± 3.0	10.5 ± 3.0	10.1 ± 3.0	0.001

Table 2

Association between odds of prevalent hypertension and quartiles of one-year moving average of $PM_{2.5}$

$PM_{2.5}$ IQR increment[a]	POR (95% CI)	
	Base model[b]	Adjusted Model[c]
1 year	1.18 (1.08, 1.29)	1.24 (1.11, 1.38)
Trend Analysis		
Quartile1	--	--
Quartile 2	1.12 (0.94, 1.34)	1.15 (0.94, 1.39)
Quartile 3	1.39 (1.16, 1.68)	1.48 (1.19, 1.84)
Quartile 4	1.35 (1.12, 1.62)	1.46 (1.17, 1.81)
P trend	<0.001	<0.001

[a] IQR PM: 1 year: 3.91 μg/m^3

[b] Base model includes $PM_{2.5}$, BMI, age, sex, education, diabetes status, race

[c] Adjusted model additionally controls for tobacco and alcohol use, physical activity, region, urbanicity, census track data on median household income and percent of households living below the poverty line

Table 3

Association between blood pressure measures and one-year moving averages of $PM_{2.5}$

$PM_{2.5}$[a]	β (95% CI)	
	Base model[b]	Adjusted Model[c]
Systolic pressure (mm Hg)	0.73 (0.00, 1.46)	0.93 (0.05, 1.80)
Diastolic pressure (mm Hg)	−0.01 (−0.41, 0.40)	0.02 (−0.47, 0.50)
Mean arterial pressure (mm Hg)	0.24 (−0.23, 0.70)	0.32 (−0.24, 0.88)
Pulse pressure (mm Hg)	0.73 (0.16, 1.30)	0.89 (0.21, 1.58)

[a] IQR PM: 1 year: 3.91 μg/m3

[b] Base model includes $PM_{2.5}$, BMI, age, sex, , anti-hypertensive medication (loop diuretics, thiazide diuretics, potassium sparing diuretics, calcium channel blockers, beta blockers, alpha blockers, ACE inhibitors, vasodilators and ARBs), education, diabetes status, race,

[c] Adjusted model additionally controls for tobacco and alcohol use, physical activity, region, urbanicity, census track data on median household income and percent of households living below the poverty line

Table 4

Association between systolic and pulse pressures and quartiles of one-year moving averages of $PM_{2.5}$

$PM_{2.5}$ IQR increment[a]	**β (95% CI)**	
	Systolic pressure (mm Hg)[b]	**Pulse pressure (mm Hg)**[b]
1 year	0.93 (0.05, 1.80)	0.89 (0.21, 1.58)
Trend Analysis		
Quartile1	--	--
Quartile 2	0.47 (−1.13, 2.07)	1.20 (−0.06, 2.46)
Quartile 3	1.76 (0.00, 3.52)	2.51 (1.14, 3.87)
Quartile 4	1.47 (−0.30, 3.25)	1.24 (−0.15, 2.64)
P trend	0.056	0.069

[a] IQR PM: 1 year: 3.91 μg/m^3

[b] Model includes $PM_{2.5}$, BMI, age, sex, , anti-hypertensive medication (loop diuretics, thiazide diuretics, potassium sparing diuretics, calcium channel blockers, beta blockers, alpha blockers, ACE inhibitors, vasodilators and ARBs), education, diabetes status, race, tobacco and alcohol use, physical activity, region, urbanicity, census track data on median household income and percent of households living below the poverty line

Table 5

Association between systolic and pulse pressures and one-year moving averages of $PM_{2.5}$[a] stratified by participant characteristics

Characteristics	Systolic pressure (mm Hg)[b] β (95% CI)	Interaction p value	Pulse Pressure (mm Hg)[b] P (95% CI)	Interaction p value
Sex		0.999		0.648
-Male	0.93 (−0.24, 2.09)		0.75 (−0.15, 1.66)	
-Female	0.93 (−0.16, 2.01)		1.01 (0.16, 1.87)	
Age				0.770
-Below Median (70)	1.04 (0.11, 1.96)	0.466	0.93 (0.21, 1.65)	
-Above Median (70)	0.81 (−0.13, 1.75)		0.85 (0.12, 1.58)	
BMI		0.453		0.078
<30	0.83 (−0.67, 2.65)		1.08 (0.37, 1.80)	
≥30	1.06 (0.11, 2.01)		0.64 (−0.09, 1.38)	
Smoker		0.930		0.081
-Current tobacco	0.99 (−0.82, 2.83)		1.93 (0.61, 3.25)	
-No current	0.91 (−0.03, 1.86)		0.68 (−0.06, 1.41)	
Physical Activity		0.834		0.464
>3 times/week	0.99 (−0.07, 2.05)		1.07 (0.24, 1.90)	
≤3 times/week	0.85 (−0.30, 2.00)		0.68 (−0.22, 1.57)	
Education		0.166		0.218
>High school	0.49 (−0.55, 1.54)		0.59 (−0.25, 1.42)	
≤High school	1.53 (0.27, 2.78)		1.33 (0.35, 2.31)	
History of Diabetes		0.220		0.071
-Yes	1.75 (0.18, 3.31)		1.85 (0.62, 3.09)	
-No	0.71 (−0.24, 1.66)		0.64 (−0.10, 1.38)	
Race/Ethnicity		0.571		0.485
-White	1.07 (0.11, 2.02)		1.00 (0.26, 1.75)	
-Non-White	0.54 (−1.14, 2.22)		0.49 (−0.83, 1.81)	

[a] IQR PM: 1 year: 3.91 μg/m^3

[b] Model includes $PM_{2.5}$, BMI, age, sex, , anti-hypertensive medication (loop diuretics, thiazide diuretics, potassium sparing diuretics, calcium channel blockers, beta blockers, alpha blockers, ACE inhibitors, vasodilators and ARBs), education, diabetes status, race, tobacco and alcohol use, physical activity, region, urbanicity, census track data on median household income and percent of households living below the poverty line