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Acute Effects of Ambient Particulate Matter on Blood Pressure: Differential Effects across Urban Communities

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Abstract

Recent studies have suggested a link between exposure to ambient particulate matter <2.5μm in diameter ($PM_{2.5}$) and adverse cardiovascular outcomes. The objective of this study was to examine the effects of differing community-level exposure to $PM_{2.5}$ on daily measures of blood pressure (BP) among an adult population. During the period May 2002 through April 2003, BP was examined at two time points for 347 adults residing in three distinct communities of Detroit, MI. Exposure to $PM_{2.5}$ was assessed in each community during this period, along with multivariate associations between $PM_{2.5}$ and BP. In models combining all three communities, $PM_{2.5}$ was significantly associated with systolic pressure (SP); a 10 $\mu g/m^3$ increase in daily $PM_{2.5}$

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was associated with a 3.2 mm Hg increase in SP (p=0.05). However, in models that added a location interaction, larger effects were observed for SP within the community with highest $PM_{2.5}$ levels; a 10 $\mu g/m^3$ increase in daily $PM_{2.5}$ was associated with a 8.6 mm Hg increase in SP (p=0.01). We also found young age (<55 years) and not taking BP medications to be significant predictors of increased BP effects. Among those taking BP medications, the $PM_{2.5}$ effect on BP appeared to be mitigated, partially explaining the age effect, as those participants less than 55 years were less likely to take BP medications. Short-term increases in exposure to ambient $PM_{2.5}$ are associated with acute increases in BP in adults, especially within communities with elevated levels of exposure.

Keywords

air pollution; particulate matter; blood pressure; urban; cardiovascular outcomes

Introduction

Several observational studies have demonstrated that short-term exposure to fine particulate matter < 2.5 μm in diameter ($PM_{2.5}$) can acutely raise blood pressure (BP).[1-5] However, not all studies have been positive.[6-9] Discrepancies between previous studies may result from variations in: characteristics or susceptibility of study participants, PM exposure mischaracterizations, varying chemical composition of the PM, protective medication effects taken by some participants, possible lack of adjustments for other confounders, and inaccurate determinations of BP.[10] Importantly, no previous study that has linked $PM_{2.5}$ exposure and BP has reported the effect of varying pollutant exposure types within a metropolitan area in order to identify potentially sensitive sub-populations and/or particularly toxic local PM environments. This is important because the pro-hypertensive actions of $PM_{2.5}$ may be limited to a specific subset of at-risk individuals and/or may be mediated only by PM of certain chemical composition.

Thus, in the current study, we examined the effect of daily exposure to $PM_{2.5}$ on BP among an adult population characteristic of the general population across three distinct Detroit, Michigan communities with differing levels of exposure to ambient $PM_{2.5}$. Since the communities vary in their socioeconomic and racial-ethnic compositions, with high concentrations of socioeconomically and racially-ethnically disadvantaged persons, the study also contributes to understanding the potential role of differential exposure to air pollution in health disparities of socioeconomic and racial-ethnic classes.

Methods

Data for this study was collected as part of the Detroit Healthy Environments Partnership (HEP),[11] an affiliated project of the Detroit Community-Academic Urban Research Center (DCA-URC).[12] The goals of HEP include gathering and analyzing biological indicators of cardiovascular disease risk, and the contributions of social and physical environments to those risk factors, in eastside, northwest, and southwest Detroit. These three communities differ in racial, ethnic, and socioeconomic composition.[11] As a community-based participatory research effort,[13] HEP engages researchers based in academic institutions, and representatives from health service organizations and community-based organizations in a collaborative effort to address these questions. Representatives of partner organizations comprise the HEP Steering Committee, which is involved in all aspects of the research process. The HEP study was approved in January 2001 by the University of Michigan (UM) Institutional Review Board for Protection of Human Subjects.

Blood Pressure Measures and Covariates

A stratified probability sample of 919 residents of the 3 Detroit study communities (northwest, southwest, and eastside) participated in the HEP study, with 347 of those participants completing both a stratified face-to-face survey and a biomarker component of the study.[11] All BP measures and other relevant covariates were collected during the period May 2002 through April 2003 (see Table 1). These measures were made at 2 different time points for each study participant (mean of four weeks between each measurement time point). The measures included systolic and diastolic BP collected using a portable cuff device (Omron model #HEM 711AC) that passed Association for the Advancement of Medical Instrumentation (AAMI) standards.[14] Self-reports of age, sex, race-ethnicity, household income, education, body mass index, smoking behavior, doctor diagnosed diabetes, and medication use for hypertension, along with measures of total cholesterol. In brief, of the variables listed in Table 1, only 2 were found to be significantly different between biomarker participants and non-participants. A slightly higher percentage of biomarker participants had an annual household income of less than $10,000 (32% vs. 26%, p=0.01), and fewer biomarker participants were characterized as “never smoked” (34% vs. 45%, p=0.02).

BP was measured following the methodology utilized by the NHANES study,[15] in a seated position using the right arm, with a large cuff used in instances where arm circumference was greater than 15 inches. Three consecutive measures of systolic and diastolic pressure, separated by about one minute, were taken at each of the two time points, with the mean of the 2nd and 3rd measures used for all data analysis. Pulse pressure was calculated as systolic minus diastolic BP.

Community-Level Characterization of PM2.5

Levels of ambient $PM_{2.5}$ were characterized in the 3 Detroit communities during the years 2000 to 2003 using tapered element oscillating microbalances (TEOM Model 1400a, Rupprecht and Patashnick, Inc).[11,16] Two of the three monitoring sites were established for the sole purpose of conducting this study, the northwest site was previously established by the state of Michigan. Each monitoring site was located within a 5 km radius of all study participants in each respective community, allowing for a considerable increase in the geographic representativeness of community-level assessment of exposure to ambient $PM_{2.5}$ over many previous studies. For days in which $PM_{2.5}$ was not available from the northwest site, data was interpolated using regression with data from the eastside site, with justification for this being that daily comparative exposure data for both sites was available for 79% of the study days. Three full years of data collection found levels of $PM_{2.5}$ at these two sites to be nearly identical (Figure 1), allowing the eastside site to serve as a reliable surrogate estimator of exposure for the northwest site on days when northwest data was missing. Standard meteorological variables including temperature, atmospheric pressure, relative humidity, wind speed, and wind direction were also recorded at each site.

Statistical Analysis

Multivariate associations between ambient $PM_{2.5}$ and BP outcomes were assessed using the PROC SURVEYREG procedure of SAS for WINDOWS 9.13. These procedures are specially designed for the analysis of complex sample survey data. PROC SURVEYREG incorporates the complex sample weights (final weights, strata, and psu) for the standard error estimates, and was determined most appropriate for complex sampling designs like that of our study. Models investigated lagged exposure in two ways: 1) Individual 24-hour spans: exposure measured 1 day prior to health outcome (Lag1), 2 days prior (Lag2), up to 4 days prior (Lag4), and 2) Large spans: 48-hour average prior (2 days average), 72-hour average prior (3 days average) up to 120-hour average prior (5 days average). Covariates adjusted for

in all models included: age, sex, race-ethnicity, household income, education, body mass index, smoking behavior, doctor diagnosed diabetes, total cholesterol, and medication use for hypertension. We also estimated models that controlled for meteorological variables. However, due to the previously known high level of covariance between ambient $PM_{2.5}$ and temperature (correlation coefficients as high as 0.7 for our study), we were not able to include temperature in the final models since this resulted in non-convergence of the model.

Results

The mean (SD) level of $PM_{2.5}$ measured across all three community-level monitoring sites for the period 2000-2003 was 15.0 (8.2) $\mu g/m^3$ (mean levels at each individual site are shown in Figure 1). Concentrations observed at the southwest Detroit site were significantly elevated (by roughly 20%) over those measured at the northwest and eastside monitoring locations. These levels are above the USEPA-National Ambient Air Quality Standard (NAAQS) of 15 $\mu g/m^3$ for annual $PM_{2.5}$.

Multivariate associations between BP and community-level exposure to $PM_{2.5}$ were examined at varying lag levels (1-5 days), and included analyses to assess the modification of the relationship by community location, age, baseline BP, and medication use. Overall, regression equations demonstrated positive associations between exposure to $PM_{2.5}$ and increased systolic pressure and pulse pressure. In particular, significant effect modification of these associations were observed for community location, age, and medication use (data presented below), while no significant effects were found for baseline BP (data not presented).

Effects of Community Location

Table 2a presents analysis results for individual day lag effects. As is shown, $PM_{2.5}$ was significantly associated with systolic pressure (as well as pulse pressure) for Lag2 ($p=0.05$), as a 10 $\mu g/m^3$ increase in daily $PM_{2.5}$ was associated with a 3.2 mm Hg increase in systolic pressure. However, the inclusion of a community location interaction term in the model found the observed effects to be greatly enhanced in the southwest Detroit community relative to the other two communities. For example, as is seen in Table 2b, a significant increase in systolic pressure (as well as pulse pressure) was observed for Lags 2, 3, & 4. The effects of $PM_{2.5}$ were not only more consistent across lags for the location interaction model, but the magnitude of the effect was also greater [ex: a 10 $\mu g/m^3$ increase in daily $PM_{2.5}$ was associated with a 8.6 mm Hg increase in systolic pressure for Lag4 ($p=0.01$)]. Models were also assessed for effects of multi-day averaged exposure to $PM_{2.5}$ on BP outcomes. Similar to the analysis of individual day lag effects, analysis of multi-day averaged exposures found significant effects on systolic pressure (5 days) without a location interaction included in the model (Table 3a). However, inclusion of the location interaction found the observed effects on systolic pressure (as well as pulse pressure) to be enhanced in the southwest Detroit community relative to the other two communities (Table 3b).

Effects of Age and Medication Use

Table 4 presents analysis results for effect of age on individual day lag relationships. Contrary to expected outcomes based on previous literature, we found young age (those <55 years) to be a significant predictor of increased BP effects (both systolic and pulse pressure for Lag2 and Lag4). Since our data showed increased medication use among older study participants, we then analyzed for effect modification by prevalence of BP medication use. These results (Table 5a) clearly showed that not taking BP medication was a strong predictor of increased BP effects for both systolic and pulse pressure. When we then added the community location interaction to the model, we saw further increases in BP specific to

residing in the SW Detroit community (Table 5b). For example, a 10 $\mu g/m^3$ increase in daily $PM_{2.5}$ was associated with a 10.3 mm Hg increase in systolic pressure for Lag4 (p=0.01). Among those taking BP medications, the $PM_{2.5}$ effect on BP appeared to be mitigated, partially explaining the age effect, as those participants less than 55 years were less likely to use BP medications.

Discussion

In this study of 347 adults in three Detroit communities, short-term increases in exposure to $PM_{2.5}$ levels less than the current daily USEPA NAAQS (65 $\mu g/m^3$) were significantly associated with an increase in systolic and pulse pressure. These results confirm and extend previous epidemiological studies to a broad population of adults by demonstrating these effects in a multi-ethnic community sample. Moreover, not only was $PM_{2.5}$ related to alterations in BP, but the effect of air pollution varied by community location, age, and BP medication use. This provides critically important insight of the cardiovascular risk conveyed by air pollutants by strongly supporting that $PM_{2.5}$ from differing sources and/or chemical composition have a differential impact on BP, and therefore likely cardiovascular risk as well.

Even relatively small increases in systolic and/or pulse pressure of similar magnitudes found in this study are well-established to substantially increase the long-term risk for both coronary and cerebrovascular events.[17,18] However, these associations are presumably related to sustained BP elevations. It is not clear whether the differences in BP due to PM exposures found in this study are maintained in a chronic fashion and thereby contribute to a long-term elevated cardiovascular risk. This is hypothetically possible and requires further investigation. Nonetheless, this hemodynamic pro-hypertensive change has been consistently implicated as one of the major triggers of cardiovascular events in vulnerable individuals.[19] It is conceivable that in susceptible people, a rapid pro-hypertensive response (or the underlying mediating hemodynamics responsible such as arterial vasoconstriction and increased vascular resistance) over a few days could trigger atherosclerotic plaque disruption and thus promote an acute myocardial infarction or stroke. In vulnerable coronary heart disease patients, the BP increase could also instigate myocardial ischemia due to increases in cardiac afterload and oxygen demand. Moreover, the relation between BP increase and $PM_{2.5}$ was shown to be linear. The actual increase in BP therefore could be substantially larger on days with extreme elevations in air pollution. For example, the 5^{th} and 95^{th} percentile $PM_{2.5}$ pollution days for the SW Detroit community for our study period were 4.9 and 35.1 $\mu g/m^3$, respectively. Based on results in Table 5a, an individual residing in SW Detroit and not taking BP medications would have a theoretical increase in systolic pressure of 31 mm Hg (based on the 10.3 mm Hg increase in systolic pressure per 10 $\mu g/m^3$ increase in daily $PM_{2.5}$, Lag4) from $PM_{2.5}$ exposure on a 5^{th} percentile pollution day to a 95^{th} percentile pollution day. Finally, there is a wide range in the magnitude of BP elevation within subjects, and certain susceptible individuals may actually respond with much larger degrees of BP increase than the population mean. Therefore, our findings may provide an important explanation of a key mechanism whereby air pollutants are capable of increasing the risk both for acute coronary and cerebrovascular events over a few-day period.

Community Location Effect

Elevated levels of $PM_{2.5}$ have been reported for southwest Detroit[16] and attributed to the density of traffic and industrial facilities present in this community relative to other areas of the city.[20] Results of the community location analysis in this study suggest that increased levels of $PM_{2.5}$, and possibly differences in chemical composition of the PM emitted from nearby emission sources may be responsible for the adverse effect observed on BP outcomes. Two specific studies of PM using animal models have previously been conducted

in southwest Detroit and have observed impacts of nearby emission sources. One study assessed levels of plasma asymmetric dimethyl arginine (ADMA), an endogenous inhibitor of nitric oxide synthase, in rats following three days of exposure to concentrated ambient $PM_{2.5}$,[21] and found a significant increase of ADMA in rats exposed to PM compared to a control group exposed to filtered air. The measured meteorological conditions and the elemental tracers observed in the $PM_{2.5}$ suggested that emissions from a nearby industrial complex (including coal combustion, oil refineries and coke ovens) may have considerably contributed to the overall mass of $PM_{2.5}$ in this study. Another animal-based study conducted in southwest Detroit found that the chemical composition of PM, rather than the $PM_{2.5}$ mass concentration, was most indicative of adverse effects.[22] These analyses determined that increased pulmonary retention of specific chemical components of $PM_{2.5}$ were associated with the enhancement of airway inflammation, specifically in rodents with increased eosinophilic infiltrates in lungs of allergic rats. Further, the analysis determined the likely source of the retained chemical components in the lung tissue to be from the nearby industrial source complex located within southwest Detroit and upwind of the study site during the exposure period.

Most research to date has focused on ambient $PM_{2.5}$ mass and has not involved extensive exposure characterization; therefore, little is known regarding the effects of specific $PM_{2.5}$ sources and components on human health. Our findings provide evidence that exposure to $PM_{2.5}$ from different communities within the same city (differing sources and chemical composition) can have a differential impact on human health outcomes, in this case BP. This corroborates two recent studies, where long-term exposure to $PM_{2.5}$ was associated with widely different cardiovascular outcomes across different communities within the same urban area.[23,24] However, further studies are required in order to help determine the most toxic and responsible PM constituents.

Effects of Age and BP Medication Use

Contrary to what might have been expected based on previous literature on susceptibility to PM, we found that young age (those <55 years) modified the relationship between BP and individual day lag exposures to $PM_{2.5}$. Since there was higher medication use among older study participants, we then analyzed for effect modification by prevalence of medication use for hypertension. These results clearly showed that not taking medication was a strong predictor of increased BP effects (both systolic and pulse pressure). Among those taking BP medications, the $PM_{2.5}$ effect on BP appeared to be mitigated, partially explaining the age effect, as participants <55 years were less likely to take BP medications.

Blood pressure medications appeared to be protective in our study against the effects of PM exposure. While we were not able to assess if different classes of BP medications were more or less protective, it is likely that there would be differences, and further investigation of this finding is needed in future studies. Beta blockers may be most protective by blocking SNS responses, or perhaps that ACEI and ARBs may be most protective due to their anti-oxidant and anti-inflammatory responses. Controlled studies with hypertensive versus normotensive participants not on BP meds (looking at beta blockers vs. ace inhibitors or angiotensin receptor blockers vs. calcium blockers vs. diuretics in responses - each separately) could assess if there are differences in responses following PM exposure.

Potential Mechanisms

Several biological mechanisms could be responsible for affecting cardiovascular hemodynamics in response to $PM_{2.5}$.[25] While the actual etiology must remain speculative, plausible pathways have been described in human and animal studies, and theories to explain these findings include the release of pro-inflammatory/oxidative mediators from

pulmonary cells and/or trans-located PM constituents effecting the function of the system arterial circulation.[25] A third hypothesis is that PM within the lung may promote arterial vasoconstriction via altering CV autonomic nervous system balance. The inhalation of PM has been shown to induce changes in autonomic balance favoring sympathetic activity, mediate systemic oxidative stress and inflammation, and promote vascular dysfunction leading to arterial vasoconstriction.[25-28] The pulmonary tree is widely innervated by vagal afferents.[29] Stimulation of many of the nervous receptor subtypes can instigate reflex autonomic responses and alter CV sympathetic/parasympathetic balance.[29] Several studies have shown that PM rapidly effects CV autonomic tone.[30-34] Overlapping and different mechanisms may be responsible for alterations in BP at varying time points. Nevertheless, these pathways are each individually, or in sum, hypothetically capable of promoting physiological BP elevations.[35]

Limitations

Significant relationships were observed after controlling for several potential confounders; however, residual confounding remains possible and other important variables may not have been considered. Furthermore, this study was conducted over a relatively short time duration, and in a limited adult sample with a low median income. Because PM exposure and hypertension are associated with socioeconomic status, the finding of significant effects within this sample with limited income may be conservative. The results and conclusions reported here need to be confirmed with larger samples with a broader range of socioeconomic characteristics. The lack of detailed medication information was also a limitation, and this study did not determine PM chemical components and source impacts on a daily basis. Future studies will be required to clarify the relevant biologic mechanisms and to identify the specific PM constituents responsible for mediating the observed adverse BP effects.

Perspectives

Despite these limitations, we found that exposure to levels of $PM_{2.5}$ that do not exceed the current daily USEPA-NAAQS was associated with potentially clinically meaningful increases in systolic and pulse pressure. We found young age (<55 years) to be a significant predictor of increased BP effects, partially explained by an apparent mitigating effect of taking BP medication, with older participants more likely to be using medication. Our findings corroborate and extend previous much smaller studies and demonstrate that $PM_{2.5}$ within individual communities of an urban area may have varying effects on BP. There is substantial evidence that low-income communities of color are more likely to be exposed to sources of air pollutants. Given that the differentials in exposure to and BP impact of $PM_{2.5}$ are associated with variations in the racial-ethnic and socioeconomic composition of community populations, future research should further explore not only the pollution emission sources contributing to and mechanisms producing these effects, but also their implications for understanding and potentially alleviating racial-ethnic and socioeconomic disparities in health.

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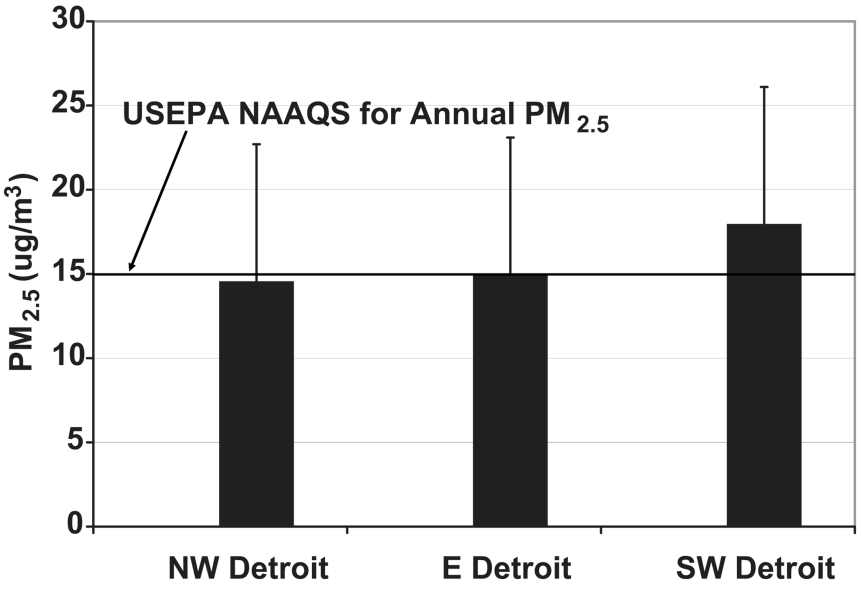

Figure 1.
Mean $PM_{2.5}$ measured in each HEP study community, 2000-2003 (error bars represent standard deviation).

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Table 1

Baseline Demographics of HEP Biomarker Study Participants.

		Full Biomarker Sample				Eastside Detroit				Northwest Detroit				Southwest Detroit			
Variable		**N**	**%**	**Mean**	**SD**	**N**	**%**	**Mean**	**SD**	**N**	**%**	**Mean**	**SD**	**N**	**%**	**Mean**	**SD**
Systolic BP		347		129.2	20.8	116		130.1	20.5	96		128.9	21.8	135		128.7	20.5
Diastolic BP		347		79.4	12.7	116		80.2	11.9	96		80.7	13.8	135		77.9	12.4
Pulse BP		347		49.8	14.7	116		49.9	15.1	96		48.2	12.9	135		50.8	15.6
BMI		347		30.9	7.9	116		31.0	7.2	96		31.1	8.3	135		30.8	8.3
Age		347		46.2	13.7	116		47.7	14.1	96		45.4	13.8	135		45.7	13.3
	< 55 years	236	75.4			86	73.4			69	72.1			107	79.3		
	55 years or more	85	24.6			31	26.6			27	27.9			28	20.7		
Sex	Males	99	44.5			21	40			21	33.6			53	55.8		
Location	East	116	31.8														
	South	135	40.0														
	North	96	28.2														
Race-Ethnicity	Hispanic	55	18.0			1				1				53	43.3		
	White	70	20.1			1				25	22.2			44	33.6		
	Black	212	58.4			112	94.3			67	72.1			33	20.1		
Household Income	<$10,000	124	35.0			43	34.9			34	37.1			47	33.5		
	$10,000-19,999	93	28.0			31	31.8			21	21.8			41	29.4		
	$20,000-34,999	81	22.2			30	21.6			22	20.0			29	24.2		
	$35,000+	49	14.8			12	11.7			19	21.1			18	12.9		
Education	Less than 8th grade	37	11.3			9	7.8			3	2.4			25	20.4		
	Some High School	78	22.4			24	19.7			19	20.4			35	25.8		
	High School Graduate	97	29.6			36	33.7			23	27.0			38	28.1		
	Some College	79	20.8			32	25.8			27	26.2			20	13.0		
	College or More	50	13.9			13	11.0			21	19.3			16	12.3		
Hypertension Medication Use		259	77.9			74	66.3			71	78.2			114	86.9		

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Table 2

(a and b) Individual day lag effects of $PM_{2.5}$ on BP outcomes (per 10 μg/m^3 increase in $_{2.5}$) assessing community location interaction.

a.) Total Sample - Lags

Blood Pressure	Exposure	Lag 1		Lag 2		Lag 3		Lag 4	
		Δ mm Hg	p-value	Δ mm Hg	p-value	Δ mm Hg	p-value	Δ mm Hg	p-value
Systolic	$PM_{2.5}$	-0.33	0.83	**3.24**	**0.05**	1.37	0.32	3.75	0.11
Diastolic	$PM_{2.5}$	-1.42	0.14	-0.92	0.41	-0.13	0.91	1.54	0.34
Pulse	$PM_{2.5}$	1.10	0.29	**4.16**	**0.01**	1.53	0.10	2.36	0.11

b.) Location Interaction (Southwest Detroit) - Lags

Blood Pressure	Exposure	Lag 1		Lag 2		Lag 3		Lag 4	
		Δ mm Hg	p-value	Δ mm Hg	p-value	Δ mm Hg	p-value	Δ mm Hg	p-value
Systolic	$PM_{2.5}$	-2.71	0.23	**4.66**	**0.01**	**3.47**	**0.02**	**8.58**	**0.01**
Diastolic	$PM_{2.5}$	-1.95	0.16	-1.16	0.42	0.63	0.71	2.44	0.41
Pulse	$PM_{2.5}$	-0.73	0.74	**5.93**	**0.01**	**3.00**	**0.02**	**6.40**	**0.01**

Note: The following variables are controlled/included in all equations: age, sex, race-ethnicity, household income, education, body mass index, doctor diagnosed diabetes, smoking behavior, total cholesterol, and medication use for hypertension.

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Table 3

(a and b) Combined day lag effects of $PM_{2.5}$ on BP outcomes (per 10 $\mu g/m^3$ increase in $PM_{2.5}$) assessing community location interaction.

a.) Total Sample - Averages

Blood Pressure	Exposure	2 Days		3 Days		4 Days		5 Days	
		Δ mm Hg	p-value	Δ mm Hg	p-value	Δ mm Hg	p-value	Δ mm Hg	p-value
Systolic	$PM_{2.5}$	1.19	0.56	2.17	0.26	3.87	0.08	4.73	0.05
Diastolic	$PM_{2.5}$	-1.89	0.15	-1.27	0.38	-0.59	0.75	0.89	0.64
Pulse	$PM_{2.5}$	**3.15**	**0.04**	**3.56**	**0.01**	**4.62**	**0.01**	**4.04**	**0.02**

b.) Location Interaction (Southwest Detroit) - Averages

Blood Pressure	Exposure	2 Days		3 Days		4 Days		5 Days	
		Δ mm Hg	p-value	Δ mm Hg	p-value	Δ mm Hg	p-value	Δ mm Hg	p-value
Systolic	$PM_{2.5}$	0.07	0.98	3.27	0.08	**5.65**	**0.01**	**5.93**	**0.01**
Diastolic	$PM_{2.5}$	-2.09	0.16	0.09	0.96	1.57	0.51	2.02	0.36
Pulse	$PM_{2.5}$	2.49	0.39	**3.55**	**0.04**	**4.50**	**0.02**	**4.24**	**0.02**

Note: The following variables are controlled/included in all equations: age, sex, race-ethnicity, household income, education, body mass index, doctor diagnosed diabetes, smoking behavior, total cholesterol, and medication use for hypertension.

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Table 4

Individual day lag effects of $PM_{2.5}$ on BP outcomes (per 10 μg/m^3 increase in $PM_{2.5}$) assessing effect modification by age.

Total Sample - Lags

Blood Pressure	Exposure	Effect Modification	Lag 1		Lag 2		Lag 3		Lag 4	
			Δ mm Hg	p-value	Δ mm Hg	p-value	Δ mm Hg	p-value	Δ mm Hg	p-value
Systolic	$PM_{2.5}$	55+	3.33	0.30	1.23	0.78	0.55	0.87	-1.25	0.73
		25-54 years	-1.23	0.44	**4.24**	**0.02**	1.50	0.26	**6.28**	**0.02**
Diastolic	$PM_{2.5}$	55+	-1.70	0.20	-3.76	0.06	-1.67	0.40	-0.93	0.64
		25-54 years	-1.36	0.20	0.25	0.84	0.44	0.71	2.74	0.17
Pulse	$PM_{2.5}$	55+	5.07	0.08	4.78	0.17	2.20	0.38	-0.29	0.92
		25-54 years	0.08	0.94	**4.02**	**0.02**	1.11	0.17	**3.61**	**0.01**

Note: The following variables are controlled/included in all equations: sex, race-ethnicity, household income, education, body mass index, doctor diagnosed diabetes, smoking behavior, total cholesterol, and medication use for hypertension.

Table 5

(a and b) Individual day lag effects of $PM_{2.5}$ on BP outcomes (per 10 μg/m^3 increase in $PM_{2.5}$) assessing effect modification by prevalence of BP medication use and including community location interaction.

a.) Total Sample - Lags

Blood Pressure	Exposure	Effect Modification	Lag 1		Lag 2		Lag 3		Lag 4	
			Δ mm Hg	p-value	Δ mm Hg	p-value	Δ mm Hg	p-value	Δ mm Hg	p-value
Systolic	$PM_{2.5}$	Taking BP Med.	3.75	0.41	5.76	0.32	1.45	0.68	0.67	0.89
		Not Taking BP Med.	-1.30	0.32	2.93	0.07	1.88	0.17	**6.01**	**0.01**
Diastolic	$PM_{2.5}$	Taking BP Med.	1.42	0.58	-0.70	0.84	0.04	0.99	-1.58	0.59
		Not Taking BP Med.	**-2.27**	**0.01**	-0.93	0.39	0.06	0.96	3.42	0.06
Pulse	$PM_{2.5}$	Taking BP Med.	2.35	0.40	5.85	0.18	1.31	0.53	2.39	0.42
		Not Taking BP Med.	0.92	0.41	**3.94**	**0.01**	1.87	0.08	**2.72**	**0.04**

b.) Location Interaction (Southwest Detroit) - Lags

Blood Pressure	Exposure		Lag 1		Lag 2		Lag 3		Lag 4	
		Effect Modification	Δ mm Hg	p-value	Δ mm Hg	p-value	Δ mm Hg	p-value	Δ mm Hg	p-value
Systolic	$PM_{2.5}$	Taking BP Med.	2.11	0.67	7.64	0.20	4.02	0.29	4.55	0.31
		Not Taking BP Med.	-2.84	0.23	**4.71**	**0.01**	**3.18**	**0.04**	**10.25**	**0.01**
Diastolic	$PM_{2.5}$	Taking BP Med.	1.22	0.67	-1.36	0.70	1.31	0.63	-0.71	0.84
		Not Taking BP Med.	-2.30	0.07	-1.09	0.45	0.55	0.74	4.00	0.16
Pulse	$PM_{2.5}$	Taking BP Med.	0.96	0.78	8.65	0.07	2.94	0.30	**5.55**	**0.02**
		Not Taking BP Med.	-0.54	0.81	**5.98**	**0.01**	**2.85**	**0.04**	**6.54**	**0.01**

Note: The following variables are controlled/included in all equations: age, sex, race-ethnicity, household income, education, body mass index, doctor diagnosed diabetes, smoking behavior, and total cholesterol.