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Fine particulate matter air pollution and blood pressure: The modifying role of psychosocial stress

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Abstract

Background—Consensus is growing on the need to investigate the joint effects of psychosocial stress and environmental hazards on health. Some evidence suggests that psychosocial stress may be an important modifier of the association between air pollution respiratory outcomes, but few have examined cardiovascular outcomes.

Objectives—We examined the modifying effect of psychosocial stress on the association between fine particulate matter air pollution (PM_{2.5}) and blood pressure (BP).

Methods—Our data came from the Detroit Healthy Environments Partnership (HEP) 2002–2003 survey. Of 919 participants, BP was collected at two time points for a subset of 347. Building on previous work reporting associations between PM_{2.5} and BP in this sample, we regressed systolic (SBP) and diastolic (DBP) BP and pulse pressure (PP), in separate linear models, on the interaction among psychosocial stress, PM_{2.5}, and HEP neighborhood (Southwest, Eastside, Northwest).

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Results—The association between PM_{2.5} and SBP was stronger for those who reported high levels of stress, but this interaction was significant only in the Southwest Detroit neighborhood. Southwest Detroit residents who reported low stress showed a 2.94 mmHg (95%CI: -0.85, 6.72) increase in SBP for each 10µg/m³ increase in 2-day prior PM_{2.5} exposure. Those who reported high stress showed a 9.05 mmHg (95%CI: 3.29, 14.81) increase in SBP for each 10µg/m³ increase in PM_{2.5} exposure.

Conclusions—These results suggest that psychosocial stress may increase vulnerability to the hypertensive effects of PM_{2.5}. This work contributes to an understanding of the ways in which the social and physical environments may jointly contribute to poor health and to health disparities.

Keywords

disease susceptibility; air pollution; stress; psychological; blood pressure

Introduction

A growing literature has documented the health impacts of psychosocial stress, and environmental hazards – although generally in separate disciplinary literatures. However, consensus is growing on the need to investigate their joint effects as they are often spatially correlated, may operate through common biological mechanisms, and may act synergistically to affect health (Clougherty and Kubzansky 2009; Evans and Pilyoung 2010; Gee and Payne-Sturges 2004; Morello-Frosch and Lopez 2006; Pope and Dockery 2006; Schulz et al. 2005). Indeed, the US Clean Air Act requires that the National Ambient Air Quality Standards (NAAQS) protect populations that may be particularly vulnerable to the health effects of air pollution ([Anonymous] 1970). As such, it is a public health imperative to understand the factors that increase vulnerability to the health effects of air pollution.

Specifically, there is some evidence suggesting that psychosocial stress may be an important modifier of associations between air pollution and health (Clougherty et al. 2007; Clougherty and Kubzansky 2009). For example, some report that the association between air pollution and asthma is stronger in children who either have high exposure to violence or whose parents report high levels of stress (Clougherty et al. 2007; Shankardass et al. 2009). Others report that the association between air pollution and clinical asthma symptoms is stronger among asthmatic children who also report high levels of chronic family stress (Chen et al. 2008). Moreover, animal models support the notion that stress increases susceptibility to the respiratory effects of air pollution (Clougherty et al. 2010).

To date, most of the research on the modifying effects of psychosocial stress on the association between air pollution and health has focused on respiratory health outcomes. Yet, a growing body of work has documented positive associations between fine particulate matter air pollution (PM_{2.5}) and cardiovascular disease (CVD) (Brook et al. 2004; Brook 2008; Pope and Dockery 2006; Sun et al. 2010), and blood pressure in particular (Auchincloss et al. 2008; Brook and Rajagopalan 2009; Dvorchak et al. 2009). For example, previous results from the Healthy Environments Partnership (HEP) Detroit-based study reported that a 10µg/m³ increase in PM_{2.5} was associated with a 3.25 mmHg increase in systolic blood pressure (SBP) ($p<0.05$) two days later (Dvorchak et al. 2009). This association

was particularly pronounced in the Southwest Detroit neighborhood, which experienced a 4.66 mmHg increase in systolic blood pressure ($p=0.01$) two days later – and up to an 8.6 mmHg increase in systolic blood pressure ($p=0.01$) four days later (Dvorchak et al. 2009).

As with respiratory outcomes, it may be that the impact of air pollution on cardiovascular outcomes is modified by social stressors or psychosocial stress. One study showed that proximity to high-traffic roads is associated with coronary artery calcification, but only in those who live in neighborhoods with high unemployment (Dragano et al. 2009), suggesting that social factors and air pollution act synergistically to affect cardiovascular health. However, to our knowledge, there are no studies that have examined modifying effects of psychosocial stress on the association between $PM_{2.5}$ and cardiovascular outcomes, including blood pressure.

We used data from the HEP Community Survey to investigate the extent to which short-term exposures to $PM_{2.5}$ and psychosocial exposures may act together to affect blood pressure in an urban sample. This work builds on findings of an association between $PM_{2.5}$ and blood pressure in HEP, that was particularly strong in the Southwest neighborhood of Detroit (Dvorchak et al. 2009) by exploring effect modification by psychosocial stress. Specifically we examined whether psychosocial stress modified the association between $PM_{2.5}$ and blood pressure within each of the three Detroit neighborhoods.

Methods

Dataset

We used data from the 2002–2003 Community Survey of the Detroit HEP (Schulz et al. 2005). The details of this survey are found elsewhere (Schulz et al. 2005). Briefly, a stratified probability sample of 919 residents, ages 25 and older, of the three HEP neighborhoods participated in the survey with blood pressure measurement (community survey = time 1 (t_1)). Of that 919 at t_1 , 347 completed a follow-up survey (t_2) with an additional blood pressure measurement. The mean time between the first and second blood pressure measurements was four weeks. We excluded those with missing information on any of the key variables, which reduced our sample size to 313.

Variables

Our outcome variables, systolic (SBP) and diastolic blood pressure (DBP) were measured from the right arm of seated participants using a portable cuff device (Omron model HEM 711AC). Three measurements were collected with approximately one minute between measures. Blood pressure variables were calculated as the average of the second and third measurements. Blood pressure measurements were taken at both t_1 and t_2 . Pulse pressure (PP) was calculated as $SBP-DBP$.

Our air pollution exposure variables, 24-hour averaged particulate matter ambient air pollution 2.5 microns in aerodynamic diameter ($PM_{2.5}$), were collected in the three HEP communities, with each monitor located within a five-kilometer radius of all study participants in each of the three communities (Dvorchak et al. 2009). Published results on these data showed that mean $PM_{2.5}$ levels across all three neighborhoods for the study

period (2000–2003) was $15.0\mu\text{g}/\text{m}^3$ ($\text{SD}=8.2\mu\text{g}/\text{m}^3$) (Dvornch et al. 2009). Furthermore, the mean $\text{PM}_{2.5}$ level for the Eastside and Northwest neighborhoods were nearly identical at approximately $15\mu\text{g}/\text{m}^3$, while the mean $\text{PM}_{2.5}$ level for the Southwest neighborhood was approximately 20% higher (Dvornch et al. 2009). Following from previous work (Dvornch et al. 2009), we examined $\text{PM}_{2.5}$ exposures at 2, 3, and 4 days prior to blood pressure measurement as a proxy for acute exposure to ambient air pollution. While the literature outlining the biological mechanisms linking $\text{PM}_{2.5}$ exposure and blood pressure indicate that blood pressure responses be seen on the same day as exposure, the epidemiological literature using population-level samples suggests that the error in ambient air pollution measurement provides for effects seen with longer exposure times (Auchincloss et al. 2008; Hicken et al. 2013).

The psychosocial stress variable was created as an index of six psychosocial stress variables created from information collected at t_1 , as has been done previously in the literature (Evans and Pilyoung 2010; Lee and Hicken 2013; Sternthal et al. 2011). Scores were created for each so that higher scores represented higher stress. The neighborhood environment stress measure was composed of 13 questions pertaining to perceptions of the physical (e.g., litter, noise) and social (e.g., gang activity) aspects of one's neighborhood. Regarding perceptions of the physical characteristics, participants were asked to respond on a Likert scale the extent to which they agreed (1-strongly agree to 5-strongly disagree) with a series of statements such as "The houses in my neighborhood are generally well maintained." Regarding perceptions of the social characteristics, participants were asked to respond on a Likert scale about the frequency (1-never to 5-always) of such activities as "Gang activity in your neighborhood." A score was developed as the mean of all responses ($\alpha=0.75$). We used an adapted version of the Duke acute life events scale (Hughes et al. 1988) that incorporated additional items based on focus group results in Detroit (Israel et al. 2002; Schulz et al. 2001). Participants were asked if, in the last 12 months, they had experienced any of nine events, such as death of a loved one, a relative or close friend going to jail. A score was developed as the sum of affirmative responses. The family caregiving stress measure included responses, on a Likert scale, to three questions about the frequency (1- never to 5-always) over the past 12 months of caregiving to adult family members. Participants were asked how often they were responsible for care, were burdened by caregiving problems, and were worried about caregiving problems. A score was developed as the mean of all responses ($\alpha=0.70$). The financial vulnerability measure was created as the mean of two questions on financial strain (Kessler et al. 1987; Schulz et al. 2008; Vinokur and Caplan 1987). Participants were asked how long they could live at their current address and standard of living if they lost all sources of income (1-less than one month to 5-more than one year). Participants were also asked how difficult it is to provide for basics such as food, clothing, medical care, and housing (1-very difficult to 4-not at all difficult). The responses to the latter question were reverse-coded and the responses to both questions were standardized before combining. For major unfair treatment, participants were asked if they had experienced any of seven situations in which they had received unfair treatment, such as in school, by police, or at work (Krieger 1990). A score was developed as the sum of affirmative responses. For everyday unfair treatment, participants were asked to respond on a Likert scale regarding the frequency (1-never to 5- always) of five situations including:

less courteous treatment by others, poorer service as restaurants and stores, people acting as if participant is not smart, people acting as if afraid of participant, and threats or harassment from others (Williams et al. 1997). A score was created as the mean of the responses ($\alpha=0.77$). To create the overall index, each scale was transformed into a z-score and the dichotomized into the high quartile and lower three quartiles. The index is the sum of the number of high scores of each of the six transformed scales.

Information on sociodemographics and other potential confounders was collected at t_1 and include: age, gender, race/ethnicity (non-Hispanic White, non-Hispanic Black, Hispanic, other), education (<high school (HS), HS, >HS), and poverty income ratio (PIR).

Analytic approach

We examined sample descriptive statistics by estimating the means and standard deviations for continuous variable and percentages within categories for categorical variables. We examined these descriptive statistics in the total sample and by HEP neighborhood.

We built on published results using these HEP data, where blood pressure at t_2 was regressed on the interaction between neighborhood and $PM_{2.5}$ at t_2 (Dvorchak et al. 2009). First, we regressed BP at t_2 on the interaction between neighborhood and $PM_{2.5}$ at t_2 , adjusting for psychosocial stress. Second, we regressed BP at t_2 on the three-way interaction between psychosocial stress, neighborhood and $PM_{2.5}$ at t_2 . All models were adjusted for age, gender, race/ethnicity, education, PIR, BP at t_1 and $PM_{2.5}$ at t_1 .

Each blood pressure outcome (SBP, DBP, PP) was analyzed in separate models. Each of the $PM_{2.5}$ exposure measures (2-, 3-, and 4-days prior to blood pressure measurement) was analyzed in separate models. $PM_{2.5}$ was mean-centered before all interactions. Temperature and season were not included due to multicollinearity resulting in nonconvergence of those models (Dvorchak et al. 2009). Because the sociodemographic, stress, and air pollution measures may be highly correlated particularly by neighborhood, we computed and examined the variance inflation factors (VIF) for each of these measures and the models overall. High multicollinearity was defined as an average VIF greater than or equal to five (Belsley et al. 2005).

We performed several sensitivity analyses. First, we examined different average $PM_{2.5}$ exposure estimates, including: the maximum one-hour average for the 2-, 3-, and 4-day lag periods, the maximum 8-hour average for the 2-, 3-, and 4-day lag periods, and 48-, 72-, and 96-hour periods. We also examined models that further adjusted for anti-hypertensive medication use, body mass index (BMI), current smoking status, sodium intake, and diabetes diagnosis. While these are likely mediators in the linkages among neighborhood, stress, and blood pressure, they may also account for some confounding between neighborhood and blood pressure. Because there are racial/ethnic or sex composition differences among the neighborhoods, we examined the possibility that race/ethnicity or sex either modified the association between $PM_{2.5}$ and blood pressure independently of neighborhood or that race/ethnicity mediated the modifying effects of neighborhood using approaches outlined in the literature (Muller et al. 2005).

All analyses were conducted in STATA MP 11.0 (StataCorp) and include survey weights developed to account for the complex survey design of the HEP Community Survey (Dvorchak et al. 2009).

Results

While there were neighborhood differences in $PM_{2.5}$ (discussed in the Methods section), there were no neighborhood differences in blood pressure, as shown in Table 1. While there were no neighborhood differences in the mean stress index, there were differences in the percent in each neighborhood that were in the high stress quartile. Forty-two percent of those in the Eastside neighborhood were in the high stress quartile of the overall stress index distribution. In general, each neighborhood differed from the others in sociodemographic characteristics.

In Tables 2 through 5, we provide the parameter estimates for the components of the interactions and post-estimation calculations for the association between $PM_{2.5}$ and blood pressure in each neighborhood at low and high stress for SBP, DBP, and PP, respectively. In Table 2, Model 1 under each of the $PM_{2.5}$ exposure periods is the association between $PM_{2.5}$ and SBP, adjusting for covariates, location, and stress. For example, for each ten $\mu g/m^3$ increase in $PM_{2.5}$ three days prior, there was a 1.18 mmHg (95% CI: -1.21, 3.57) increase in SBP for the entire sample.

Model 2 under each of the $PM_{2.5}$ exposure periods is the association between the interaction among neighborhood, $PM_{2.5}$, and stress with SBP. Furthermore in the lower panel, for Model 2, we include the post-estimation calculations for the association between $PM_{2.5}$ and SBP for both the low and high stress groups in each of the neighborhoods. For example, for those in the low stress group in the Southwest neighborhood, there was a 1.69 mmHg (95% CI: -1.31, 4.70) increase in SBP for each ten $\mu g/m^3$ increase in $PM_{2.5}$ three days prior. However, for those in the high stress group in the Southwest neighborhood, there was an *additional* 7.16 mmHg (95% CI: 0.21, 14.11) increase in SBP for each ten $\mu g/m^3$ in $PM_{2.5}$ three days prior. In the lower panel, we show that this means that for the high stress group in the Southwest neighborhood, there is a total 8.86 mmHg increase in SBP for each ten $\mu g/m^3$ in $PM_{2.5}$ three days prior. This difference in $PM_{2.5}$ -SBP association between the low and high stress groups is statistically significant as shown by the interaction term. This pattern of results was observed at each of the $PM_{2.5}$ exposure periods, but the interaction was statistically significant only with a $PM_{2.5}$ exposure 3- and 4-days prior to the exam.

All information in Tables 2 through 5 is presented in the same manner. A similar pattern of results was observed for DBP, although the interaction was statistically significant only with a $PM_{2.5}$ exposure 4 days prior to the exam. Finally, a similar pattern of results was observed for PP, with significant interaction terms with a $PM_{2.5}$ exposure 3 days prior to the exam.

We did not find evidence of problematic multicollinearity in our models. Average VIFs ranged from 2.46 for 2-day lagged exposure with DBP to 3.42 for the 4-day lagged exposure with SBP. Our model results were similar when we used alternate exposure averaging periods. When we adjusted for anti-hypertensive medication use, BMI, current smoking

status, sodium intake, and diabetes diagnosis, the pattern of results did not change, with several of the interaction terms increasing, suggesting a stronger modifying effect of stress after adjustment for these additional hypertension risk factors. We did not find evidence that race/ethnicity modified the association between PM_{2.5} and blood pressure or that race/ethnicity mediated the modifying effects of neighborhood on the association between PM_{2.5} and blood pressure.

We did find, however, that men but not women exhibited an association between PM_{2.5} and blood pressure. For example, for each 10 µg/m³ increase in PM_{2.5} 4 days prior, men showed a 11.07 mmHg increase in SBP (95%CI: 5.25,16.89) while women showed a 0.87 decrease in SBP (95%CI: -4.04,2.30; results not shown in table form). However, we did not find evidence that the differences in the sex composition mediated the modifying effect of neighborhood.

Discussion

We set out to examine the potentially modifying role of psychosocial stress on the association between PM_{2.5} and blood pressure. We found evidence to suggest that there is, in fact, a modifying effect of stress on the association between PM_{2.5} and SBP, but this modifying effect varied by location. While the pattern of results was similar across the time-lagged exposure measures in the Southwest area, the most pronounced associations were for exposure four days prior to blood pressure measurement. The literature suggests that PM_{2.5} would increase blood pressure within the first day (Brook et al. 2002; Brook et al. 2009) rather than four days later. It is likely that this apparent delayed reaction is really due to measurement issues. Others who have examined ambient PM_{2.5} in relation to blood pressure have reported similar associations with longer-term acute measures of PM_{2.5} (Auchincloss et al. 2008; Hicken et al. 2013).

Researchers have theorized that psychosocial stress increases vulnerability to the health effects of environmental hazards. However, there is still a dearth of empirical evidence. In most of the empirical work to date, researchers have examined the modifying role of stress on the association between air pollution and respiratory outcomes. For example, some report that the association between air pollution and asthma is stronger in children who either have high exposure to violence or whose parents report high levels of stress (Clougherty et al. 2007; Shankardass et al. 2009). Others report that the association between air pollution and clinical asthma symptoms is stronger among asthmatic children who also report high levels of chronic family stress (Chen et al. 2008). Although this study is one of the first examination of a modifying effect of stress on the association between PM_{2.5} and blood pressure, one study showed that the association between proximity to high-traffic roads and coronary artery calcification was stronger for those in neighborhoods with high unemployment, which can be considered a social source of stress (Dragano et al. 2009). (Babisch et al. 2001; Babisch 2002, 2003; Combined Environmental Exposures: Noise Air Pollutants and Chemicals participants 2007; Huang et al. 2013) However, another recent study examined the modifying effect of social and psychosocial adversity on the association between PM_{2.5} and BP and found no evidence of effect modification (Hicken et al. 2013). However, in that study, the authors did not use an index of multiple sources and types of

stress. Alternatively, noise has been framed as a source of stress (Babisch 2002) and has been shown to activate the stress response systems (Babisch et al. 2001; Babisch 2003). Furthermore, noise has been shown to enhance the association between air pollution and heart rate variability (Huang et al. 2013).

Some have found evidence of a modifying effect of psychosocial stress on the association between other environmental hazards and hypertension. For example, researchers reported that men who report high levels of perceived stress showed a stronger association between bone lead and blood pressure compared to those who reported low levels of perceived stress (Peters et al. 2007). Similarly, researchers found the association between blood lead and hypertension is stronger in adults with high, compared to low, allostatic load scores, a measure of the wear and tear on the physiological systems due to psychosocial stress (McEwen 1998; Zota et al. 2010). Our work extends this literature by examining the modifying effect of psychosocial stress on the association between PM_{2.5} and blood pressure.

That the evidence of a modifying effect was isolated to the Southwest neighborhood is consistent with research suggesting that location is important for the health effects of air pollution (Brook et al. 2009). It may be that the chemical composition of PM_{2.5} is important to this effect of stress. While in our particular study the Southwest neighborhood did not experience greater PM_{2.5} levels due to seasonal confounding with regard to data collection in each neighborhood, others have shown that the southwest area of Detroit has consistently elevated levels of PM_{2.5} compared to other areas of Detroit (Keeler et al. 2002). It has been observed that the PM_{2.5} in the Southwest neighborhood is derived from numerous nearby emission sources, including traffic-related (particularly diesel) and industrial point sources (Hammond et al. 2008; Morishita et al. 2006; Pancras et al. 2013) and that the chemical composition is different between neighborhoods (Hammond et al. 2008). It may be that the chemical composition of PM_{2.5} in Southwest Detroit is particularly harmful to cardiovascular health. The Southwest area of Detroit is at the nexus of numerous high-traffic roadways and at the point of the largest commercial border crossing between the U.S. and Canada. This suggests that motor vehicle, including diesel exhaust pollution, may be higher in this area of Detroit compared to the other two neighborhoods (Morishita et al. 2006). Authors recently reported that, at the time of the HEP Community Survey, 35% of the PM_{2.5} mass in the area was due to motor vehicle exhaust – with 12% alone due to diesel exhaust (Hammond et al. 2008). Research has shown that diesel exhaust pollution results in increased blood pressure (Cosselman et al. 2012) inflammation and oxidative stress in both animal and human studies (Lodovici and Bigagli 2011). Furthermore, using animal models, researchers have shown elevated levels of an endogenous NO synthase inhibitor (Dvornchik et al. 2004) and inflammatory responses (Morishita et al. 2004) after exposure to the particulate matter (PM) air pollution in Southwest Detroit. They further showed that the inflammatory responses were not due simply to the level but the chemical composition of the PM in Southwest Detroit (Morishita et al. 2004). These pathways through oxidative stress and inflammatory processes are thought to be an important mechanism linking PM_{2.5} and cardiovascular outcomes (Brook and Rajagopalan 2009). Future research should focus, not only on PM_{2.5} level, but also on the chemical composition in relation to health.

Similarly, it may be that the proximity of the Southwest neighborhood to unique forms of traffic-related noise (i.e., the movement and idling of thousands of commercial trucks per day) is responsible for the modifying effect of PM_{2.5}. As we mentioned earlier, research suggests that noise may modifying the cardiovascular health effects of air pollution (Huang et al. 2013).

Finally, a growing literature documents the link between overactivation of the biological stress process and oxidative stress and inflammatory processes (Dimsdale 2008; McEwen 1998). While there have been no animal studies specifically on the causal mechanistic linkages among PM_{2.5}, stress, and blood pressure, there are animal studies to support these linkages among PM_{2.5}, stress, and respiratory outcomes (Clougherty et al. 2010). Our understanding of the pathways through which PM_{2.5} and stress synergistically affect blood pressure would benefit from future research in animal models.

While we had hypothesized that stress would modify the association between PM_{2.5} and blood pressure, our results in the Southwest neighborhood may be due to differences in neighborhood composition. Some studies suggest that Black and White adults exhibit different mechanisms in the development of hypertension (Morris et al. 1999). In general, however, there are not racial/ethnic differences in the association between PM_{2.5} and cardiovascular outcomes, including blood pressure (Adar et al. 2010; Adar et al. 2013; Hicken et al. 2013), although one study showed a stronger association between air pollution and self-reported cardiovascular outcomes for White compared to Black or Hispanic adults (Johnson and Parker 2009). It may also be that the differences in the sex composition of the neighborhoods are driving the differences in the modifying effects of stress, as there are fewer women in the Southwest neighborhood than in the other two. Estrogen has been shown to be cardioprotective and may provide a resiliency to the joint cardiovascular effects of PM_{2.5} and stress. While we found that sex (but not race/ethnicity) modified the association between PM_{2.5} and blood pressure, we did not find that either sex or race/ethnicity mediated the modifying effect of neighborhood.

While our research is one of the first contributions to the literature on the modifying effects of psychosocial stress on the association between PM_{2.5} and blood pressure, it is not without limitations. The HEP sample is of relatively low socioeconomic status (SES) and psychosocial stress, PM_{2.5} exposure, and hypertension have been shown to be related to SES (Adler et al. 1994; Kaplan and Nunes 2003; O'Neill et al. 2003). As such, our results may provide a conservative estimate of the associations among these factors. Future research should examine these associations in samples with broader ranges of socioeconomic characteristics.

Conclusion

We show evidence to suggest that psychosocial stress increases vulnerability to the hypertensive effects of PM_{2.5}. Our work expands the empirical literature on the synergistic effects of psychosocial stress and environmental hazards on health. More importantly, our work contributes to an understanding of the ways in which the social and physical environments may jointly contribute to poor health and, potentially, health disparities.

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Abbreviations

BMI	body mass index
DBP	diastolic blood pressure
HEP	Healthy Environments Partnership
NHANES	National Health and Nutrition Examination Survey
PIR	poverty income ratio
PM_{2.5}	particulate matter air pollution of 2.5 microns in diameter
SBP	systolic blood pressure
SES	socioeconomic status

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Highlights

- Work suggests that psychosocial stress increases vulnerability to the effects of air pollution in certain contexts.
- We examined the modifying role of psychosocial stress on the hypertensive effects of PM_{2.5}.
- In Southwest Detroit, high stress was associated with stronger PM_{2.5}-BP associations.

Table 1

Characteristics of the Healthy Environments Partnerships survey sample, by neighborhood

	Total sample (n=313)			Southwest (n=127)			Eastside (n=106)			Northwest (n=80)			Neighborhood difference ^a			
	mean	SD	Mean	SD	Mean	SD	mean	SD	mean	SD	S-E	S-N	E-N			
SBP, mmHg	128.7	20.8	128.5	20.5	129.7	21.3	127.7	20.9								
DBP, mmHg	78.7	11.9	77.9	11.9	79.3	12.0	79.3	11.7								
PP, mmHg	50.0	15.8	50.6	17.4	50.5	17.4	48.1	13.3								
Stress	1.9	1.5	1.7	1.4	2.1	1.7	2.0	1.5								
High stress, %	32		25		42		30				*					
Age, years	46.1	13.6	45.2	13.1	46.4	13.4	47.0	14.6								
Female, %	79		59		79		80				***	**				
Race																
White, %	20		33		1		24				***		***			
Black, %	62		26		97		73				***	***	***			
Hispanic, %	17		39		1		1				***	***	***			
Other, %	2		2		1		3									
Education																
<HS, %	34		45		27		25				**	**				
=HS, %	39		28		33		26									
>HS, %	37		28		40		49				*	**				
Poverty income ratio	1.7	2.0	1.5	1.5	1.8	2.7	1.8	1.5								

Results reported are means and standard deviations, SD, unless otherwise noted. Category percentages of a single variable may not add to 100 due to rounding. Blood pressure values are for the second time point. Abbreviations: SD, standard deviation; HS, high school; SBP, systolic blood pressure; DBP, diastolic blood pressure; PP, pulse pressure; PM_{2.5}, 2.5micron diameter particulate matter air pollution; S, southwest; E, east; N, northwest.

^aTests for differences among neighborhoods:

* p<0.05;

** p<0.01;

*** p<0.001

Table 2

Linear regression estimates for systolic blood pressure regressed on the interaction among neighborhood, stress, and 24-hour averaged PM_{2.5} exposure

24-hour average PM _{2.5} exposure, number of days prior to exam						
	2 days prior		3 days prior		4 days prior	
	--1--	--2--	--1--	--2--	--1--	--2--
Neighborhood						
Southwest	ref	ref				
East	-3.90 (-9.05,1.26)	-4.71 (-9.87,0.45)	-4.14 (-9.76,1.49)	-3.58 (-8.96,1.80)	-4.17 (-10.13,1.79)	-4.96 (-11.29,1.37)
Northwest	-4.51* (-8.76,-0.25)	-1.57 (-5.52,2.39)	-4.20 (-9.18,0.78)	-1.58 (-6.10,2.94)	-5.54* (-11.00,-0.08)	-2.36 (-7.88,3.17)
PM _{2.5}	3.06* (0.00,6.12)	2.94 (-0.85,6.72)	1.18 (-1.21,3.57)	1.69 (-1.31,4.70)	3.72 (-0.29,7.73)	5.84* (0.45,11.24)
Stress level						
Low stress	ref	ref	ref	ref	ref	ref
High stress	0.37 (-3.28,4.01)	3.76 (-2.90,10.42)	0.94 (-2.65,4.53)	6.67* (1.23,12.10)	0.43 (-3.26,4.12)	9.79*** (4.68,14.89)
NeighborhoodXstress						
SouthwestXstress		ref		ref		ref
EastXstress		-0.78 (-9.62,8.06)		-4.53 (-11.75,2.68)		-7.21 (-15.03,0.61)
NorthwestXstress		-8.76* (-16.16,-1.36)		-11.64*** (-18.18,-5.11)		-16.41** (-25.95,-6.86)
NeighborhoodXPM _{2.5}						
SouthwestXPM _{2.5}		ref		ref		ref
EastXPM _{2.5}		4.33 (-4.89,1.355)		0.11 (-4.86,5.08)		-4.39 (-13.45,4.67)
NorthwestXPM _{2.5}		-4.23 (-14.68,6.22)		-6.08 (-16.24,4.09)		-8.01 (-19.29,3.27)
StressXPM _{2.5}						
Low stressXPM _{2.5}		ref		ref		ref
High stressXPM _{2.5}		6.11 (-2.41,14.63)		7.16* (0.21,14.11)		14.85*** (0.74,2.23)
NeighborhoodXstressXPM _{2.5}						
SouthwestXstressXPM _{2.5}						
EastXstressXPM _{2.5}		-12.22 (-26.45,2.01)		-7.83 (-18.02,2.36)		-6.94 (-18.48,4.60)
NorthwestXstressXPM _{2.5}		-11.20 (-29.03,6.63)		-8.33 (-22.49,5.83)		-11.57 (-34.94,11.81)
Southwest neighborhood						

	24-hour average PM _{2.5} exposure, number of days prior to exam					
	2 days prior		3 days prior		4 days prior	
	--1--	--2--	--1--	--2--	--1--	--2--
Low stress		2.94 (-0.85,6.72)		1.69 (-1.31,4.70)		5.84* (0.45,11.24)
High stress		9.05** (3.29,14.81)		8.86** (3.20,14.51)		20.69*** (14.47,26.92)
East neighborhood						
Low stress		7.26 (-1.01,15.53)		1.80 (-2.03,5.63)		1.45 (-5.20,8.10)
High stress		1.16 (-7.91,10.22)		1.14 (-5.12,7.39)		9.36 (-1.97,20.68)
Northwest neighborhood						
Low stress		-1.29 (-11.27,8.68)		-4.39 (-14.10,5.33)		-2.17 (-12.27,7.94)
High stress		-6.38 (-19.26,6.50)		-5.55 (-15.49,4.39)		1.12 (-13.00,15.23)

Results reported in the upper panel are regression coefficients (95% confidence intervals) for PM_{2.5}, stress, and PM_{2.5}-by-stress interaction terms. Results reported in the lower panel are post-estimation coefficients (95% confidence intervals) for the association between PM_{2.5} and systolic blood pressure at low (lower three quartiles of the stress distribution) and high (high quartile of the stress distribution) variable. PM_{2.5} coefficients are interpreted as change in systolic blood pressure per 10 µg/m³ increase in PM_{2.5}. All estimates are adjusted for: age, sex, race, education, and poverty income ratio. PM_{2.5} was mean-centered before interaction.

* p<0.05;

** p<0.01;

*** p<0.001 for difference from zero.

Table 3

Linear regression estimates for diastolic blood pressure regressed on the interaction among neighborhood, stress, and 24-hour averaged PM_{2.5} exposure

	24-hour average PM _{2.5} exposure, number of days prior to exam					
	2 days prior		3 days prior		4 days prior	
	--1--	--2--	--1--	--2--	--1--	--2--
Neighborhood						
Southwest	ref	ref				
East	-2.22 (-7.04,2.60)	-3.65 (-8.10,0.80)	-2.08 (-7.37,3.21)	-2.84 (-7.93,2.25)	-2.48 (-7.96,3.00)	-3.86 (-9.87,2.15)
Northwest	-0.73 (-4.49,3.02)	0.45 (-3.31,4.20)	-0.68 (-5.07,3.71)	0.99 (-3.56,5.53)	-1.48 (-6.29,3.32)	0.41 (-4.76,5.58)
PM _{2.5}	-1.51 (-3.79,0.77)	-2.32 (-5.24,0.60)	-0.68 (-2.81,1.45)	0.21 (-2.83,3.25)	1.23 (-1.93,4.38)	1.49 (-5.08,8.06)
Stress level						
Low stress		ref		ref		ref
High stress		2.19 (-2.60,6.99)		4.80 (-0.64,10.24)		8.08** (3.19,12.96)
NeighborhoodXstress						
SouthwestXstress		ref		ref		ref
EastXstress		1.77 (-4.91,8.45)		-0.75 (-7.33,5.83)		-3.07 (-9.86,3.73)
NorthwestXstress		-4.82 (-11.65,2.01)		-7.37 (-14.96,0.22)		-12.28*** (-17.95,-6.62)
NeighborhoodXPM _{2.5}						
SouthwestXPM _{2.5}		ref		ref		ref
EastXPM _{2.5}		2.03 (-2.04,6.09)		-2.04 (-5.89,1.82)		-3.20 (-10.67,4.28)
NorthwestXPM _{2.5}		1.15 (-4.90,7.19)		-1.71 (-7.59,4.17)		-2.20 (-9.63,5.22)
StressXPM _{2.5}						
Low stressXPM _{2.5}		ref		ref		ref
High stressXPM _{2.5}		3.16 (-3.49,9.81)		1.87 (-5.40,9.14)		10.31* (0.14,20.49)
NeighborhoodXstressXPM _{2.5}						
SouthwestXstressXPM _{2.5}						
EastXstressXPM _{2.5}		-4.52 (-13.62,4.58)		-0.23 (-9.09,8.62)		-3.16 (-17.21,10.89)
NorthwestXstressXPM _{2.5}		-4.89 (-15.75,5.97)		-3.18 (-12.40,6.04)		-7.04 (-20.96,6.89)
Southwest neighborhood						

	24-hour average PM _{2.5} exposure, number of days prior to exam					
	2 days prior		3 days prior		4 days prior	
	--1--	--2--	--1--	--2--	--1--	--2--
Low stress	-2.32 (-5.24,0.60)			0.21 (-2.83,3.25)		1.49 (-5.08,8.06)
High stress	0.84 (-5.05,6.73)			2.08 (-5.53,9.69)		11.80** (3.80,19.80)
East neighborhood						
Low stress	-0.30 (-3.27,2.68)			-1.83 (-4.27,0.62)		-1.71 (-4.68,1.26)
High stress	-1.66 (-8.45,5.13)			-0.19 (-5.17,4.79)		5.45 (-1.99,12.88)
Northwest neighborhood						
Low stress	-1.18 (-6.61,4.26)			-1.50 (-6.44,3.44)		-0.71 (-4.23,2.80)
High stress	-2.90 (-10.43,4.62)			-2.81 (-7.74,2.12)		2.56 (-5.42,10.55)

Results reported in the upper panel are regression coefficients (95% confidence intervals) for PM_{2.5}, stress, and PM_{2.5}-by-stress interaction terms. Results reported in the lower panel are post-estimation coefficients (95% confidence intervals) for the association between PM_{2.5} and diastolic blood pressure at low (lower three quartiles of the stress distribution) and high (high quartile of the stress distribution) variable. PM_{2.5} coefficients are interpreted as change in diastolic blood pressure per 10 µg/m³ increase in PM_{2.5}. All estimates are adjusted for: age, sex, race, education, and poverty income ratio. PM_{2.5} was mean-centered before interaction.

* p<0.05;
 ** p<0.01;
 *** p<0.001 for difference from zero.

Table 4

Linear regression estimates for pulse pressure regressed on the interaction among neighborhood, stress, and 24-hour averaged PM_{2.5} exposure

24-hour average PM _{2.5} exposure, number of days prior to exam							
	2 days prior		3 days prior		4 days prior		
	--1--	--2--	--1--	--2--	--1--	--2--	
Neighborhood							
Southwest	ref	ref	ref	ref	ref	ref	ref
East	-1.39 (-5.28,2.51)	-0.89 (-5.20,3.41)	-1.69 (-6.38,3.01)	-0.58 (-5.86,4.69)	-1.44 (-6.16,3.28)	-0.99 (-6.40,4.43)	
Northwest	-3.58 (-7.63,0.47)	-1.83 (-6.08,2.42)	-3.34 (-8.02,1.35)	-2.37 (-7.46,2.72)	-3.94 (-9.08,1.20)	-2.65 (-8.63,3.34)	
PM _{2.5}	4.47** (1.82,7.12)	5.30* (1.22,9.37)	1.84* (0.34,3.34)	1.56 (-0.60,3.73)	2.62 (-0.18,5.43)	4.49 (-0.18,9.15)	
Stress level							
Low stress		ref		ref			ref
High stress		2.04 (-1.49,5.57)		2.39 (-2.03,6.81)		2.07 (-3.38,7.51)	
NeighborhoodXstress							
SouthwestXstress		ref		ref			ref
EastXstress		-2.71 (-6.92,1.50)		-3.96 (-9.05,1.13)		-4.216 (-10.19,1.76)	
NorthwestXstress		-4.14 (-11.95,3.67)		-4.56 (-12.16,3.04)		-4.22 (-15.81,7.38)	
NeighborhoodXPM _{2.5}							
SouthwestXPM _{2.5}		ref		ref			ref
EastXPM _{2.5}		1.85 (-5.95,9.66)		2.05 (-1.22,5.31)		-1.23 (-8.84,6.38)	
NorthwestXPM _{2.5}		-5.50 (-13.94,2.94)		-4.57 (-10.66,1.53)		-5.76 (-14.48,2.96)	
StressXPM _{2.5}							
Low stressXPM _{2.5}		ref		ref			ref
High stressXPM _{2.5}		3.41 (-1.16,7.98)		5.47* (0.83,10.10)		4.42 (-4.24,13.08)	
NeighborhoodXstressXPM _{2.5}							
SouthwestXstressXPM _{2.5}							
EastXstressXPM _{2.5}		-7.88 (-16.72,0.96)		-7.80* (-13.89,-1.71)		-3.95 (-13.53,5.63)	
NorthwestXstressXPM _{2.5}		-7.04 (-21.47,7.38)		-5.42 (-17.65,6.81)		-4.68 (-22.08,12.73)	
Southwest neighborhood							

		24-hour average PM _{2.5} exposure, number of days prior to exam			
		2 days prior		3 days prior	
		--1--	--2--	--1--	--2--
Low stress		5.30 [*] (1.22,9.37)		1.56 (-0.60,3.73)	
					4.49 (-0.18,9.15)
High stress		8.71 ^{***} (4.74,12.67)		7.03 ^{**} (2.37,11.69)	
					8.91 [*] (2.02,15.80)
East neighborhood					
Low stress		7.15 [*] (0.36,13.94)		3.61 ^{**} (1.17,6.05)	
					3.26 (-2.50,9.01)
High stress		2.68 (-0.83,6.20)		1.28 (-0.87,3.43)	
					3.73 (-2.15,9.61)
Northwest neighborhood					
Low stress		-0.20 (-7.66,7.26)		-3.00 (-8.71,2.70)	
					-1.28 (-9.10,6.55)
High stress		-3.83 (-15.59,7.93)		-2.96 (-12.13,6.21)	
					-1.53 (-10.57,7.52)

Results reported in the upper panel are regression coefficients (95% confidence intervals) for PM_{2.5}, stress, and PM_{2.5}-by-stress interaction terms. Results reported in the lower panel are post-estimation coefficients (95% confidence intervals) for the association between PM_{2.5} and pulse pressure at low (lower three quartiles of the stress distribution) and high (high quartile of the stress distribution) variable. PM_{2.5} coefficients are interpreted as change in pulse pressure per 10 µg/m³ increase in PM_{2.5}. All estimates are adjusted for: age, sex, race, education, and poverty income ratio. PM_{2.5} was mean-centered before interaction.

* p<0.05;
 ** p<0.01;
 *** p<0.001 for difference from zero.