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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 in 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).
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 2.94 mmHg (95% CI: −0.85, 6.72) increase in SBP for each 10 $\mu g/m^3$ increase in 2-day prior $PM_{2.5}$ exposure. Those who reported high stress showed 9.05 mmHg (95% CI: 3.29, 14.81) increase in SBP for each 10 $\mu g/m^3$ 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.
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1. 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).

Abbreviations: BMI, body mass index; DBP, diastolic blood pressure; HEP, healthy environments; PartnershipNHANES, National Health and Nutrition Examination Survey; PIR, poverty income ratio$PM_{2.5}$, particulate matter air pollution of 2.5 μm in diameter; SBP, systolic blood pressure; SES, socioeconomic status

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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; Dvonch et al., 2009). For example, previous results from the Healthy Environments Partnership (HEP) Detroit-based study reported that 10 μg/m^3 increase in $PM_{2.5}$ was associated with 3.25 mmHg increase in systolic blood pressure (SBP) ($p<0.05$) two days later (Dvonch et al., 2009). This association was particularly pronounced in the Southwest Detroit neighborhood, which experienced 4.66 mmHg increase in systolic blood pressure ($p=0.01$) two days later – and up to 8.6 mmHg increase in systolic blood pressure ($p=0.01$) four days later (Dvonch 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 the best of 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 (Dvonch 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.

2. Methods

2.1. Dataset

We used data from 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.

2.2. 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-h averaged particulate matter ambient air pollution $\geq$2.5 μm in aerodynamic diameter ($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 (Dvonch 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 μg/m^3(SD=8.2 μg/m^3) (Dvonch et al., 2009). Furthermore, the mean $PM_{2.5}$ level for the Eastside and Northwest neighborhoods were nearly identical at approximately 15 μg/m^3, while the mean $PM_{2.5}$ level for the Southwest neighborhood was approximately 20% higher (Dvonch et al., 2009). Following from previous work (Dvonch et al., 2009), we examined $PM_{2.5}$ 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 $PM_{2.5}$ exposure and blood pressure indicates 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 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 included age, gender, race/ethnicity (non-Hispanic White, non-Hispanic Black, Hispanic, other), education ($<$high school (HS), HS, $>$HS), and poverty income ratio (PIR).

2.3. 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 (Dvonch 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 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 (Dvonch 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).

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