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

J. Timothy Dvonch, Srimathi Kannan, Amy J. Schulz, Gerald J. Keeler, Graciela Mentz, James House, Alison Benjamin, Paul Max, Robert L. Bard, Robert D. Brook

Abstract—Recent studies have suggested a link between exposure to ambient particulate matter <2.5 μm 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 2 time points for 347 adults residing in 3 distinct communities of Detroit, Michigan. 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 3 of the communities, PM$_{2.5}$ was significantly associated with systolic blood pressure; a 10-μg/m$^3$ increase in daily PM$_{2.5}$ was associated with a 3.2-mm Hg increase in systolic blood pressure ($P=0.05$). However, in models that added a location interaction, larger effects were observed for systolic blood pressure within the community with highest PM$_{2.5}$ levels; a 10-μg/m$^3$ increase in daily PM$_{2.5}$ was associated with a 8.6-mm Hg increase in systolic blood pressure ($P=0.01$). 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, because those participants <55 years of age 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. (Hypertension. 2009; 53:853-859.)

Key Words: air pollution ■ particulate matter ■ blood pressure ■ urban ■ cardiovascular outcomes

Several observational studies have demonstrated that short-term exposure to fine particulate matter (PM) <2.5 μm in diameter (PM$_{2.5}$) can acutely raise blood pressure (BP). However, not all studies have been positive. 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. 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 to identify potentially sensitive subpopulations and/or particularly toxic local PM environments. This is important because the prohypertensive 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 a 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 3 distinct Detroit communities with differing levels of exposure to ambient PM$_{2.5}$. Because 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 were collected as part of the Detroit Healthy Environments Partnership (HEP), an affiliated project of the Detroit Community-Academic Urban Research Center. 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 east-side, northwest, and southwest Detroit. These 3 communities differ in racial, ethnic, and socioeconomic compositions. As a community-based participatory research effort, 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 compose 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 Institutional Review Board for Protection of Human Subjects.

BP Measures and Covariates

A stratified probability sample of 919 residents of the 3 Detroit study communities (northwest, southwest, and east side) 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. All of the 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 4 weeks between each measurement time point). The measures included systolic and diastolic BPs collected using a portable cuff device (Omron model HEM 711AC) that passed Association for the Advancement of Medical Instrumentation standards. Self-reports included age, sex, race-ethnicity, household income, education, body mass index, smoking behavior, doctor-diagnosed diabetes mellitus, 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 nonparticipants. A slightly higher percentage of biomarker participants had an annual household income of less than $10 000 (32% versus 26%; P<0.01), and fewer biomarker participants were characterized as having “never smoked” (34% versus 45%; P=0.02).

BP was measured following the methodology used by the National Health and Nutrition Examination Survey, in a seated position using the right arm, with a large cuff used in instances where arm circumference was >15 in. Three consecutive measures of systolic and diastolic pressures, separated by 1 minute, were taken at each of the 2 time points, with the mean of the second and third measures used for all of the data analyses. Pulse pressure was calculated as systolic minus diastolic BP.

Community-Level Characterization of PM$_{2.5}$

Levels of ambient PM$_{2.5}$ were characterized in the 3 Detroit communities during the years 2000–2003 using tapered element oscillating microbalances (TEOM Model 1400a, Rupprecht and Patashnick, Inc). Two of the 3 monitoring sites were established for the sole purpose of conducting this study, and the northwest site was established previously by the state of Michigan. Each monitoring site was located within a 5-km radius of all of the 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 years.
studies. For days in which PM$_{2.5}$ was not available from the northwest site, data were interpolated using regression with data from the east-side 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 2 sites to be nearly identical (Figure), allowing the study days. Concentrations observed at the southwest Detroit site were significantly elevated (by $\approx$20%) over those measured at the northwest and east-side monitoring locations. These levels are above the US Environmental Protection Agency National Ambient Air Quality Standard 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 to 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 modifications of these associations were observed for community location, age, and medication use (data presented below), whereas no significant effects were found for baseline BP (data not presented).

Effects of Community Location

Table 2 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 lag 2 ($P=0.05$), because 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 2 communities. For example, as is seen in Table 2, a significant increase in systolic pressure (as well as pulse pressure) was observed for lags 2, 3, and 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 (eg, 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

<table>
<thead>
<tr>
<th>Blood Pressure</th>
<th>Exposure</th>
<th>Lag 1</th>
<th></th>
<th>Lag 2</th>
<th></th>
<th>Lag 3</th>
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<tr>
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<td>$\Delta$, mm Hg</td>
<td>$P$</td>
<td>$\Delta$, mm Hg</td>
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<td>0.83</td>
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<tr>
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<td>PM$_{2.5}$</td>
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<td>Pulse</td>
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</table>

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

Results

The mean (SD) level of PM$_{2.5}$ measured across all 3 of the community-level monitoring sites for the period 2000–2003 was 15.0 $\mu$g/m$^3$ (8.2 $\mu$g/m$^3$; mean levels at each individual site are shown in the Figure). Concentrations observed at the southwest Detroit site were significantly elevated (by $\approx$20%) over those measured at the northwest and east-side monitoring locations. These levels are above the US Environmental Protection Agency National Ambient Air Quality Standard of 15 $\mu$g/m$^3$ for annual PM$_{2.5}$.
Diabetes mellitus, smoking behavior, total cholesterol, and medication use for hypertension.

Table 4. Individual Day Lag Effects of PM$_{2.5}$ on BP Outcomes (per 10-µg/m$^3$ Increase in PM$_{2.5}$) Assessing Community Location Interaction

<table>
<thead>
<tr>
<th>Blood Pressure</th>
<th>Exposure</th>
<th>2 Days</th>
<th>3 Days</th>
<th>4 Days</th>
<th>5 Days</th>
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<tr>
<td></td>
<td></td>
<td>$\Delta$, mm Hg</td>
<td>$P$</td>
<td>$\Delta$, mm Hg</td>
<td>$P$</td>
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<td>Total sample, averages</td>
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<td></td>
<td></td>
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</tr>
<tr>
<td>Systolic</td>
<td>PM$_{2.5}$</td>
<td>1.19</td>
<td>0.56</td>
<td>2.17</td>
<td>0.26</td>
</tr>
<tr>
<td>Diastolic</td>
<td>PM$_{2.5}$</td>
<td>$-1.89$</td>
<td>0.15</td>
<td>$-1.27$</td>
<td>0.38</td>
</tr>
<tr>
<td>Pulse</td>
<td>PM$_{2.5}$</td>
<td>3.15</td>
<td>0.04</td>
<td>3.56</td>
<td>0.01</td>
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<td></td>
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<td></td>
<td></td>
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<tr>
<td>Systolic</td>
<td>PM$_{2.5}$</td>
<td>0.07</td>
<td>0.98</td>
<td>3.27</td>
<td>0.08</td>
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<tr>
<td>Diastolic</td>
<td>PM$_{2.5}$</td>
<td>$-2.09$</td>
<td>0.16</td>
<td>0.09</td>
<td>0.96</td>
</tr>
<tr>
<td>Pulse</td>
<td>PM$_{2.5}$</td>
<td>2.49</td>
<td>0.39</td>
<td>3.55</td>
<td>0.04</td>
</tr>
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</table>

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

Effects of Age and Medication Use

Table 4 presents analysis results for the 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 pressures for lag 2 and lag 4). Because our data showed increased medication use among older study participants, we then analyzed for effect modification by prevalence of BP medication use. These results confirm and extend previous epidemiological studies to a broad population of adults by demonstrating these effects in a multiethnic 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, on the likely cardiovascular risk as well.

Even relatively small increases in systolic and/or pulse pressures of similar magnitudes found in this study are well-established to substantially increase the long-term risk for both coronary and cerebrovascular events. However, was associated with a 10.3-mm Hg increase in systolic pressure for lag 4 ($P=0.01$). Among those taking BP medications, the PM$_{2.5}$ effect on BP appeared to be mitigated, partially explaining the age effect, because those participants <55 years of age were less likely to use BP medications.

Discussion

In this study of 347 adults in 3 Detroit communities, short-term increases in exposure to PM$_{2.5}$ levels less than the current daily US Environmental Protection Agency National Ambient Air Quality Standard (65 µg/m$^3$) were significantly associated with an increase in systolic and pulse pressures. These results confirm and extend previous epidemiological studies to a broad population of adults by demonstrating these effects in a multiethnic 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, on the likely cardiovascular risk as well.

Even relatively small increases in systolic and/or pulse pressures of similar magnitudes found in this study are well-established to substantially increase the long-term risk for both coronary and cerebrovascular events. However,
Total sample, lags

<table>
<thead>
<tr>
<th>Blood Pressure</th>
<th>Exposure</th>
<th>Effect Modification</th>
<th>Δ, mm Hg</th>
<th>P</th>
<th>Δ, mm Hg</th>
<th>P</th>
<th>Δ, mm Hg</th>
<th>P</th>
<th>Δ, mm Hg</th>
<th>P</th>
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<tr>
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<td>5.76</td>
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<td>0.68</td>
<td>0.67</td>
<td>0.89</td>
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<td>PM2.5</td>
<td>Taking BP medication</td>
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<td>0.58</td>
<td>-0.70</td>
<td>0.84</td>
<td>0.04</td>
<td>0.99</td>
<td>-1.58</td>
<td>0.59</td>
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<td></td>
<td>Not taking BP medication</td>
<td>-1.30</td>
<td>0.32</td>
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<td>1.88</td>
<td>0.17</td>
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<td>-0.93</td>
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<td>0.06</td>
<td>0.96</td>
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<td>0.06</td>
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<td>Taking BP medication</td>
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<td>0.01</td>
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<td>0.08</td>
<td>2.72</td>
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Location interaction (southwest Detroit), lags

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<th>Exposure</th>
<th>Effect Modification</th>
<th>Δ, mm Hg</th>
<th>P</th>
<th>Δ, mm Hg</th>
<th>P</th>
<th>Δ, mm Hg</th>
<th>P</th>
<th>Δ, mm Hg</th>
<th>P</th>
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<tbody>
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<td>Taking BP medication</td>
<td>2.11</td>
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<td>7.64</td>
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<td>4.02</td>
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<td>0.31</td>
</tr>
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<td>Diastolic</td>
<td>PM2.5</td>
<td>Taking BP medication</td>
<td>1.22</td>
<td>0.67</td>
<td>-1.36</td>
<td>0.70</td>
<td>1.31</td>
<td>0.63</td>
<td>-0.71</td>
<td>0.84</td>
</tr>
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<td>Not taking BP medication</td>
<td>-2.84</td>
<td>0.23</td>
<td>4.71</td>
<td>0.01</td>
<td>3.18</td>
<td>0.04</td>
<td>10.25</td>
<td>0.01</td>
</tr>
<tr>
<td></td>
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<td>Not taking BP medication</td>
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<td>0.07</td>
<td>-1.09</td>
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<td>0.55</td>
<td>0.74</td>
<td>4.00</td>
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<td>0.96</td>
<td>0.78</td>
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<td>0.07</td>
<td>2.94</td>
<td>0.30</td>
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<td>0.01</td>
<td>2.85</td>
<td>0.04</td>
<td>6.54</td>
<td>0.01</td>
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The following variables are controlled/included in all of the equations: age, sex, race-ethnicity, household income, education, body mass index, doctor-diagnosed diabetes mellitus, smoking behavior, and total cholesterol.

Table 5. Individual Day Lag Effects of PM2.5 on BP Outcomes (per 10-μg/m3 Increase in PM2.5) Assessing Effect Modification by Prevalence of BP Medication Use and Including Community Location Interaction

these associations are presumably related to sustained BP elevations. It is not clear whether the differences in BP because of 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 prohypertensive change has been consistently implicated as one of the major triggers of cardiovascular events in vulnerable individuals. It is conceivable that, in susceptible people, a rapid prohypertensive response (or the underlying mediating hemodynamics responsible, e.g, 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 because of increases in cardiac afterload and oxygen demand. Moreover, the relation between BP increase and PM2.5 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 fifth and 95th percentile PM2.5 pollution days for the southwest Detroit community for our study period were 4.9 and 35.1 μg/m3, respectively. Based on results in Table 5, an individual residing in southwest 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-μg/m3 increase in daily PM2.5, lag 4) from PM2.5 exposure on a fifth-percentile pollution day to a 95th-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 PM2.5 have been reported for southwest Detroit and attributed to the density of traffic and industrial facilities present in this community relative to other areas of the city. Results of the community location analysis in this study suggest that increased levels of PM2.5 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 been conducted previously in southwest Detroit and have observed impacts of nearby emission sources. One study assessed levels of plasma asymmetrical dimethyl arginine, an endogenous inhibitor of NO synthase, in rats after 3 days of exposure to concentrated ambient PM2.5 and found a significant increase of asymmetrical dimethyl arginine in rats exposed to PM compared with a control group exposed to filtered air. The measured meteorologic conditions and the elemental tracers observed in the PM2.5 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 PM2.5 in this study. Another animal-based study conducted in southwest Detroit found that the chemical composition of PM, rather than the PM2.5 mass concentration, was most indicative of adverse effects. These analyses determined that increased pulmonary retention of specific chemical components of PM2.5 were associated with the enhancement of airway inflammation, specifically in rodents with increased eosinophilic infiltrates in lungs of allergic rats. In addition, 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 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 2 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 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}$. Because there was higher medication use among older study participants, we then analyzed for effect modification by prevalence of medication use for hypertension. These results clearly showed that not taking medication was a strong predictor of increased BP effects (both systolic and pulse pressures). Among those taking BP medications, the PM$_{2.5}$ effect on BP appeared to be mitigated, partially explaining the age effect, because participants $<$55 years were less likely to take BP medications.

BP medications appeared to be protective in our study against the effects of PM exposure. Although we were not able to assess whether 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. β-Blockers may be most protective by blocking sympathetic nervous system responses, or perhaps angiotensin-converting enzyme inhibitors and angiotensin receptor blockers may be most protective because of their antioxidant and anti-inflammatory responses. Controlled studies with hypertensive versus normotensive participants not on BP medications (looking at β-blockers versus angiotensin-converting enzyme inhibitors or angiotensin receptor blockers versus calcium blockers versus diuretics in responses, each separately) could assess whether there are differences in responses after PM exposure.

**Potential Mechanisms**

Several biological mechanisms could be responsible for affecting cardiovascular hemodynamics in response to PM$_{2.5}$. Although 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 proinflammatory/oxidative mediators from pulmonary cells and/or translocated PM constituents affecting the function of the system arterial circulation.$^{25}$ A third hypothesis is that PM within the lung may promote arterial vasoconstriction via altering cardiovascular 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 the cardiovascular sympathetic/parasympathetic balance.$^{30}$ Several studies have shown that PM rapidly affects cardiovascular 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 biological 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 US Environmental Protection Agency National Ambient Air Quality Standard was associated with potentially clinically meaningful increases in systolic and pulse pressures. 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 compositions 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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Disclosures

None.

References

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J. Timothy Dvonch, Srimathi Kannan, Amy J. Schulz, Gerald J. Keeler, Graciela Mentz, James House, Alison Benjamin, Paul Max, Robert L. Bard and Robert D. Brook

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