Effects of Particulate Matter and Antioxidant Dietary Intake on Blood Pressure

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Regulatory actions reducing fine particulate matter less than 2.5 micrometers in diameter ($PM_{2.5}$) are associated with improvements in life expectancy in the United States.[1,2] However, levels of $PM_{2.5}$ remain high and continue to be positively associated with risk of high blood pressure (BP), a precursor for many adverse cardiovascular outcomes, including coronary heart disease, myocardial infarction, and heart failure.[3–6] In the United States overall, medical expenses associated with the nearly 1 in 3 adults with hypertension[7] are estimated at approximately \$131 billion annually.[8] Cardiovascular disease is the leading cause of death in the United States and accounts for one third of the excess risk of death experienced by non-Hispanic Black in comparison with non-Hispanic White Americans.[9,10] Non-Hispanic Blacks, Hispanics, and individuals of low income in the United States are disproportionately likely to reside in communities with excess exposure to environmental hazards, including $PM_{2.5}$.[11–13] Continued investigation of strategies to reduce exposure to $PM_{2.5}$, and its adverse effects on BP, are essential to efforts to reduce racial and ethnic disparities in cardiovascular risk.

Oxidative stress may be one molecular pathway linking $PM_{2.5}$ to BP.[14-17] $PM_{2.5}$ compounds, whose composition largely depends on their source (e.g., industry, transportation), typically contain organic chemicals, metals, soot, soil, dust, allergens, and acids on their surface. When inhaled, these particles, alone or through chemical reactions, may initiate the creation of reactive oxygen species (ROS), commonly referred to as free radicals, resulting in various physiological responses in lung, heart, and vascular tissue.[18] Specifically, ROS can contribute to vasoconstriction, endothelial dysfunction, and hypertrophy, among other mechanisms that can ultimately contribute to hypertension.[19]

Oxidative stress may be mitigated when antioxidants absorb ROS in the airways and

Objectives. We assessed 2 pathways through which dietary antioxidants may counter adverse effects of exposure to particulate matter less than 2.5 micrometers in diameter ($PM_{2.5}$) on blood pressure (BP): main (compensatory) and modifying (protective) models.

Methods. We used 2002 to 2003 data from the Detroit Healthy Environments Partnership community survey conducted with a multiethnic sample of adults (n = 347) in low- to moderate-income, predominantly Hispanic and non-Hispanic Black neighborhoods in Detroit, Michigan. We used generalized estimating equations to test the effects of ambient exposure to $PM_{2.5}$ and dietary antioxidant intake on BP, with adjustment for multiple confounders.

Results. Dietary antioxidant intake was inversely associated with systolic BP (b = –0.5; *P* < .05) and pulse pressure (b = –0.6; *P* < .05) in neighborhoods closest to major sources of air pollutants. Adverse effects of $PM_{2.5}$ remained significant after accounting for antioxidant intakes. Exploratory analyses suggested potential modifying effects of antioxidant intake on associations between ambient $PM_{2.5}$ exposure and BP.

Conclusions. Interventions to improve access to antioxidant-rich foods in polluted urban areas may be protective of cardiovascular health. However, efforts to reduce $PM_{2.5}$ exposure remain critical for cardiovascular health promotion. (*Am J Public Health*. 2015;105:1254–1261. doi:10.2105/AJPH.2014. 302176)

inhibit oxidation.[20] Antioxidants are available through dietary intake of foods or supplements (e.g., vitamins A, C, and E and selenium) and may protect against adverse effects of oxidative stress. The majority of studies addressing the effects of antioxidants on cardiovascular health have examined the modifying (protective) or main (compensatory) role of antioxidant intake from supplements, rather than from whole foods captured through dietary intakes. These effects remains unsettled, however, with several meta-analyses reporting minimal or no main effects of supplements on the incidence of major cardiovascular effects across study designs.[21,22] Romieu et al. conducted a substantial review of air pollution, oxidative stress, and various health outcomes and concluded that antioxidant supplements may modify air pollution's adverse effects on cardiovascular health.[23]

A few clinical studies have noted deleterious effects of antioxidant supplement use.[24,25] Many factors compromise or complicate comparison

of these studies' outcomes. For example, study design varies by antioxidant type, dose, duration, and the health status of study participants.[26–28] Reflecting these inconclusive findings, the American Heart Association's scientific position recommends against antioxidant supplement use.[29]

By contrast, on the basis of modest evidence of reductions in aging-related illnesses,[30] the Institute of Medicine provides recommended dietary allowances for many well-known antioxidants, including selenium (400 mg) and vitamins A (900 μg), C (90 mg), and E (15 mg). Despite the uncertainties in the evidence base, several scholars recommend direct dietary intake of antioxidants through healthy food (i.e., fruit, vegetables, whole grains) or beverage sources to mitigate the adverse effects of ROS on cardiovascular health.[30–34]

Antioxidant intakes are not consistent across diverse populations. Chun et al.[31] used food consumption and supplement use data from National Health and Nutritional Examination

Survey (1999–2002)[35] to estimate overall antioxidant intake in the United States, deriving antioxidant values from the US Department of Agriculture Database for the Flavonoid Content of Selected Food.[36] They concluded that overall intake appeared to be higher among women, older adults, non-Hispanic Whites, and higher-income and physically active individuals. For some antioxidants, including vitamin C and carotenes, intake appeared to be higher among nonsmokers and those who did not consume alcohol.[31] Researchers have used various clinical indicators to detect antioxidant deficiency among those with chronic illnesses, including asthma, chronic obstructive lung diseases, diabetes, and cardiovascular disease,[13,23,37,38] which have well-established disparities by race, ethnicity, and income.[39,40]

The unequal distribution of exposure to $PM_{2.5}$ and unequal access to antioxidant-rich foods[41] raise questions about their contributions to racial, ethnic, and socioeconomic health inequities. Residents of urban communities of color and low-income communities are more likely to experience excess exposure to $PM_{2.5}$.[11,42] Emerging research also suggests racial differences in oxidative stress, with persons of color experiencing higher levels.[43–45] Access to stores that sell fresh produce, an important source of dietary antioxidants, is low in some urban communities, particularly lower-income communities composed predominantly of people of color.[46–50] Together, excess exposure to air pollutants and psychosocial stress may increase levels of oxidative stress in low-income, urban communities of color, at the same time that these communities experience reduced access to foods rich in protective antioxidants. Few studies have examined the question of whether dietary antioxidant intake (DAI) may counter the adverse effects of exposure to $PM_{2.5}$ on blood pressure in a community sample.

We previously reported adverse effects of $PM_{2.5}$ on blood pressure[4,51] and associations between neighborhood availability of fruits and vegetables and dietary intakes of those foods.[41,50] We built on those findings to specifically examine, in data from Detroit, Michigan, the extent to which DAI is inversely associated with BP and whether it may partially compensate for or counter adverse effects of $PM_{2.5}$ on BP. If higher levels of DAI inhibit oxidation

through absorption of ROS, thus reducing levels of oxidative stress, adverse effects of $PM_{2.5}$ on BP may be contingent on DAI levels. Thus, we also examined protective models, exploring the extent to which DAI modifies adverse effects of exposure to $PM_{2.5}$ on BP. We considered the implications of our findings for understanding and intervening to reduce excess risk of cardiovascular disease among residents of predominantly non-Hispanic Black and Hispanic low- to moderate-income urban communities. Our research questions were (1) Is DAI associated with reduced BP? (2) Does DAI reduce adverse effects of $PM_{2.5}$ on BP? and (3) Does DAI modify the association between $PM_{2.5}$ and BP?

METHODS

The Healthy Environments Partnership (HEP) is a community-based participatory research collaboration established in 2000 to investigate and address social and environmental factors that contribute to disparities in cardiovascular disease.[52] HEP examines racial and socioeconomic inequalities in cardiovascular risk and the role of social and physical environmental exposures in this process, as well as disseminating and translating findings to inform new and established intervention and policy efforts. HEP engages academic researchers and representatives from health service organizations, community-based organizations, and the community at large in a collaborative effort to address these questions. Representatives of these partner organizations compose the HEP Steering Committee, which meets monthly to oversee all aspects of the research process.

Data

Our data came from 3 sources: (1) the HEP 2002 to 2003 community survey[53]; (2) a modified Block Food Frequency Questionnaire (Berkeley Nutrition Services, Berkeley, CA), implemented as part of the community survey; and (3) community-level ambient exposure measures collected in 2002 to 2003.

The HEP community survey had a stratified 2-stage probability sample of occupied housing units, designed for 1000 completed interviews with adults aged 25 years or older in 3 parts of Detroit, allowing for comparisons across

geographic areas of the city.[52] The survey collected self-reported demographic and health data, including age, gender, race, ethnicity, household income, education, smoking behavior, hypertension medication use, and dietary intake. The survey also collected anthropometric clinical measures (height, weight, BP) during the interviews. For a subset of 347 participants, the survey measured BP a second time, along with additional clinical measures (e.g., triglycerides, fasting blood glucose). All survey participants completed the Block Food Frequency Questionnaire.

Measures

Dependent variables were systolic blood pressure (SBP), diastolic blood pressure (DBP), and pulse pressure (PP). Certified phlebotomists measured BP by the method used by the National Health and Nutritional Examination Survey,[35] with a portable cuff device (Omron model HEM 711AC, Omron Healthcare Inc, Lake Forest, IL) that passed Association for the Advancement of Medical Instrumentation standards.[54] Phlebotomists used a large cuff for participants whose arm circumference was greater than 15 inches. They took 3 consecutive measures of SBP and DBP, separated by about 1 minute, at each of the 2 time points, with the mean of the second and third measures used for all data analysis. PP, an indicator of arterial stiffness, was calculated as the difference between SBP and DBP.[55]

Independent variables were DAI and $PM_{2.5}$. We created DAI from self-reported dietary intakes in the Block Food Frequency Questionnaire. We assigned antioxidant levels according to estimates for specific foods and quantities established by Halvorsen et al.[56] Between 2002 and 2003, we assessed daily community-level $PM_{2.5}$ in study communities with tapered element oscillating microbalances (TEOM model 1400, Rupprecht and Patashnick Inc, East Greenbush, NY).[57] We used a monitoring site established by the Michigan Department of Environmental Quality and 2 additional sites to capture $PM_{2.5}$ levels in each of the 3 study communities. All participants in the 2002 HEP survey resided within 5 kilometers of 1 of 3 monitors.[4] We also collected the following meteorological data: daily temperature, atmospheric pressure, relative humidity, wind speed, and wind direction, at each site.

Covariates were age, gender, race/ethnicity, household income, education, body mass index (defined as weight in kilograms divided by the square of height in meters), smoking behavior, doctor-diagnosed diabetes, total cholesterol, and medication use for hypertension. We also estimated models that controlled for meteorological variables (temperature, atmospheric pressure, relative humidity).

Analyses

Our study built on previously reported findings demonstrating associations between $PM_{2.5}$ and BP in a multiethnic urban community.[4] We used the same statistical modeling technique, the PROC SURVEYREG procedure of SAS for Windows version 9.13 (SAS Institute, Cary, NC), to test for associations between DAI and BP and for the joint effect of ambient exposure $PM_{2.5}$ and DAI on BP. These procedures are specifically designed for analysis of complex sample survey data and incorporate the complex sample weights (final weights, strata, and primary sampling unit) for standard error estimates.

To temporally align $PM_{2.5}$ measures with BP measures, we examined lagged exposure with individual 24-hour daily spans from 1 day before (lag 1) through 4 days before (lag 4) and larger spans of 48 (2 days average), 72 (3 days average), up to 120 (5 days average) hours average prior. After removing outliers, the final sample for these analyses ranged from 270 to 300, depending on lag of exposure considered.

To test for mediation effects, we used the method described by Judd and Kenny, which involves computing the difference between 2 parameter estimates (with and without the mediator) and then testing for the significance of the difference.[58] To assess whether the slope of the association between DAI and BP varied by area, we ran models that incorporated an interaction between area and DAI. Similarly, in models assessing the joint effects of DAI and $PM_{2.5}$, we included interaction terms for DAI and area and for $PM_{2.5}$ and area. Results reported are from models with these interaction terms. All models adjusted for covariates.

RESULTS

Table 1 summarizes baseline demographic and health data for study participants (n = 347).

The mean SBP was 129.7 millimeters of mercury (SE = 1.3 mm/Hg), mean DBP was 78.9 millimeters of mercury (SE = 0.07 mm/Hg), and mean PP was 50.9 millimeters of mercury (SE = 1.1 mm/Hg). A majority (22%) of participants had been prescribed medication to treat hypertension. The mean level of $PM_{2.5}$ was 15.7 micrograms per cubic meter (SE = 0.7 $\mu g/m^3$), at the US Environmental Protection Agency's former standard (15 $\mu g/m^3$) and above

the new annual National Ambient Air Quality Standards attainment level (12 $\mu g/m^3$). Mean DAI was 7.11 millimoles per day (SE = 0.3 mmol/day), with average intake of 6.1 millimoles per day (SE = 4.1 mmol/day), in eastside, 6.9 millimoles per day (SE = 4.2 mmol/day) in northwest, and 7.9 millimoles per day (SE = 5.8 mmol/day) in southwest Detroit.

The results for associations between DAI and BP indicated an inverse association of DAI

TABLE 1—Baseline Demographic and Health Characteristics of Study Participants: Detroit Healthy Environments Partnership; Detroit, MI; 2002–2003

Characteristic	% or Mean ±SE (95% CI)
Age, y	21.3 ±1.1 (19.1, 23.5)
Female	55.6
Race/ethnicity	
Hispanic	18.0
White	20.1
Black	58.5
Annual household income, $	
< 10 000	35.0
10 000–19 999	27.9
20 000–34 999	22.3
≥ 35 000	14.8
Education	
< high school diploma	27.3
High school diploma	22.3
Some college	29.5
≥ college diploma	20.9
BMI	30.9 ±0.5 (30, 31.9)
Hypertension medication	22.2
Smoking status	
Never	34.0
Current	43.3
Former	22.7
Antioxidant dietary intake, mmol/d	7.11 ±0.29 (6.5, 7.7)
Baseline blood pressure measures	
Systolic	128.8 ±1.3 (126.2, 131.5)
Diastolic	80.1 ±0.7 (78.6, 81.5)
Pulse	48.8 ±0.9 (46.9, 50.6)
Blood pressure measures at time 2	
Systolic	129.7 ±1.3 (127.0, 132.4)
Diastolic	78.9 ±0.7 (77.4, 80.4)
Pulse	50.9 ±1.1 (48.6, 53.2)
Ambient exposure	
$PM_{2.5}$ (lag 1)	309 15.7 ±0.7 (14.4, 17.1)
$PM_{2.5}$ (at time 2, lag 1)	291 14 ±0.4 (13.1, 14.9)

Note. BMI = body mass index; CI = confidence interval; $PM_{2.5}$ = particulate matter < 2.5 micrometers in diameter. The sample size was n = 347.

TABLE 2—Joint Effects of Particulate Air Pollution and Dietary Antioxidant Intake on Blood Pressure Outcomes: Detroit Healthy Environments Partnership; Detroit, MI; 2002–2003

b	Lag 1, b (95% CI)	Lag 2, b (95% CI)	Lag 3, b (95% CI)	Lag 4, b (95% CI)
Systolic BP				
$PM_{2.5}$	-2.6 (-3.0, -2.2)	4.2 (3.9, 4.5)	3.0 (2.7, 3.3)	7.3 (6.7, 7.9)
DAI	-0.5 (-1.0, 0.0)	-0.5* (-1, -0.1)	-0.6* (-1.1, -0.1)	-0.5* (-0.9, -0.1)
Diastolic BP				
$PM_{2.5}$	-2.1 (-2.3, -1.8)	-1.1 (-1.4, -0.8)	0.6 (0.3, 0.9)	2.7 (2.1, 3.3)
DAI	0.1 (-0.7, 0.8)	0.0 (-0.7, 0.8)	0.1 (-0.6, 0.8)	0.0 (-0.7, 0.8)
Pulse pressure				
$PM_{2.5}$	-0.4 (-0.8, 0.0)	5.4 (5, 5.8)	2.5 (2.2, 2.7)	4.8 (4.5, 5.2)
DAI	-0.6* (-1.0, -0.1)	-0.6* (-1.1, -0.1)	-0.7** (-1.2, -0.2)	-0.6* (-1.1, 0)

Note. BP = blood pressure; CI = confidence interval; DAI = dietary antioxidant intake; $PM_{2.5}$ = particulate matter < 2.5 micrometers in diameter.
*P < .05; **P < .01; ***P < .001.

with SBP (b = −0.42; 95% confidence interval [CI] = −0.83, −0.01; P = .049) and PP (b = −0.55; 95% CI = −0.88, −0.22; P = .003), but not DBP (b = 0.12; 95% CI = −0.27, 0.51; P = .548).

Results from models testing the joint effects of $PM_{2.5}$ and DAI on BP are shown in Table 2. Results are presented for each of four 24-hour lags of $PM_{2.5}$. Because differences in associations between $PM_{2.5}$ and BP by area of the city were reported previously,[4,52] we also tested for differences across areas of the city in the joint effects of $PM_{2.5}$ and DAI. These models showed that associations between $PM_{2.5}$ and BP remained significant after accounting for DAI in southwest Detroit, the area of the city with the greatest proximity to multiple stationary and mobile sources of $PM_{2.5}$. Associations were not significant for eastside and northwest Detroit. For residents of southwest Detroit, DAI was significantly and inversely associated with SBP at lags 2 (b = −0.52; 95% CI = −1.0, −0.1; P = .03), 3 (b = −0.59; 95% CI = −1.1, −0.1; P = .02), and 4 (b = −0.49; 95% CI = −0.9, −0.1; P = .03) and with PP at lags 1 (b = −0.57; 95% CI = −1.0, −0.1; P = .01), 2 (b = −0.59; 95% CI = −1.1, −0.1; P = .02), 3 (b = −0.74; 95% CI = −1.2, −0.1; P = .01), and 4 (b = −0.56; 95% CI = −1.1, −0.1; P = .05), after accounting for the effect of ambient exposure of $PM_{2.5}$ (results not shown). We also observed antioxidant effects combined with effects of multiday averaged exposure to $PM_{2.5}$ on BP outcomes

in the models. Results were similar, with significant antioxidant effects on SBP (2-, 3-, 4-, and 5-day averages) and PP (2-, 3-, 4-, and 5-day averages; results not shown).

Parameter estimates for $PM_{2.5}$ in Table 2 were somewhat reduced from those previously reported in models that did not account for DAI.[4] Figure 1 shows these differences for each measure of BP, with model 1 showing previously reported levels not accounting for DAI[4] and model 2 showing estimates for associations between $PM_{2.5}$ and SBP and PP after adjustment for DAI.

To assess whether the reductions in associations between $PM_{2.5}$ and BP, with adjustment for DAI in model 2, were statistically significant, we ran formal tests of mediation, with methods proposed by Friedman and McAdam[59] (see also Zhang et al.[60]). Results from these analyses suggested that DAI exerted a small but statistically significant effect, reducing adverse effects of $PM_{2.5}$ on SBP and PP. The test statistics for this comparison were notable for lags 2 to 4 for SBP (P < .001) and for PP (P = .001). These findings were consistent with a hypothesized reduction in ROS through absorption by antioxidants.

Finally, we ran exploratory models assessing whether associations between $PM_{2.5}$ exposure and blood pressure differed among participants with high and low DAI. Although not statistically significant, our results suggested a potential modifying effect of DAI on associations between $PM_{2.5}$ and BP. Specifically, we found

some suggestion that, at higher levels of DAI, the adverse effects of $PM_{2.5}$ on SBP were dampened somewhat. Because of our relatively small sample size and the multiple interaction terms in these final models, our confidence in reporting these results is relatively low. Further study is needed on this effect.

DISCUSSION

Two key findings emerged from our examination of whether antioxidant dietary intakes counter adverse effects of exposure to $PM_{2.5}$ on BP in a multiethnic community sample. First, our findings were generally consistent with the hypothesis that DAI offers some protection against adverse effects of $PM_{2.5}$ on BP. Our finding of an inverse association between DAI and SBP and PP was consistent and extended results reported elsewhere, in studies that used dietary supplements rather than our DAI measures.[26,61] This effect was significant in the study community that hosts the greatest number of point and mobile sources of $PM_{2.5}$. The inclusion of antioxidants in the model only slightly attenuated the main effect of $PM_{2.5}$ on SBP and PP in southwest Detroit.

Our second finding, on whether DAI modifies associations between $PM_{2.5}$ and BP, although exploratory, was suggestive that adverse effects of $PM_{2.5}$ on BP may be weakened for those with higher DAI. However, these analyses were underpowered, and further analyses with larger data sets are warranted.

Effects of Dietary Antioxidant Intake

Our results supported the hypothesis that DAI is inversely associated with indicators of SBP and PP. Associations remained statistically significant in models that included $PM_{2.5}$, suggesting that these effects occurred above and beyond effects of $PM_{2.5}$ and may serve to partially compensate for adverse effects of $PM_{2.5}$ on SBP. An individual with average DAI in our sample (7.4 mmol/d) would realize a 3.5–millimeters of mercury decrease in SBP. We detected no significant associations with DBP, but dietary antioxidants similarly reduced adverse effects of $PM_{2.5}$ on PP. In other words, residents who reported higher dietary intakes of antioxidant-rich foods slightly reduced adverse effects of $PM_{2.5}$ on SBP and on PP. Our results were consistent with the idea that $PM_{2.5}$
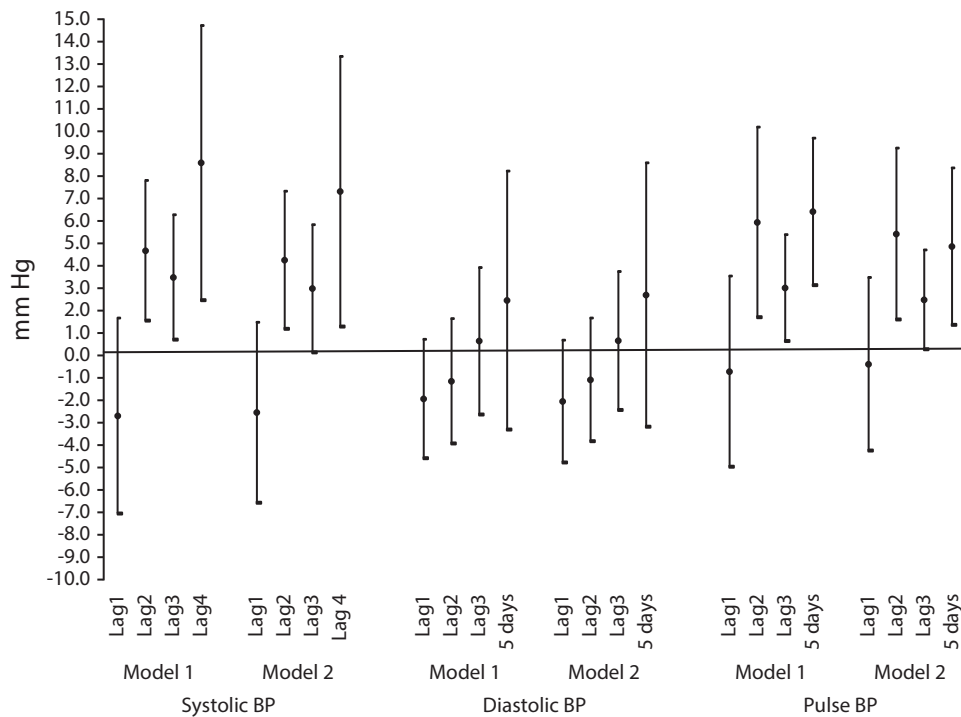
FIGURE 1—Associations between particulate matter < 2.5 micrometers in diameter and blood pressure without (model 1) and with (model 2) dietary antioxidant intake: Detroit Healthy Environments Partnership, 2002–2003.

Note. BP = blood pressure.

influences BP through the production of ROS and that DAI may reduce these adverse effects through absorption of free radicals. $PM_{2.5}$ retained a significant adverse association with SBP and PP, even after accounting for DAI. Thus, our findings suggest that DAI may reduce but, at the level of DAI we found, not eradicate adverse effects of $PM_{2.5}$ on BP.

Our tests of whether DAI modifies associations between $PM_{2.5}$ and BP must be considered exploratory, because of the limited sample size and number of covariates in our models. Our findings are suggestive of reductions in associations between $PM_{2.5}$ and BP for individuals reporting higher levels of DAI, but require further study.

Our tests of both main and modifying effects suggested that DAI is likely insufficient to protect against adverse effects of $PM_{2.5}$ on BP. Our findings support the importance of continued efforts to strengthen the existing monitoring network to include near-roadway monitoring of $PM_{2.5}$ as well as reductions in the National Ambient Air Quality Standards for fine particles from 15 to 12 micrograms per cubic meter to promote health. Such efforts may be particularly important to protect the health of residents in neighborhoods near point and mobile sources of pollution, who are disproportionately likely to be members of racial and ethnic groups that experience excess vulnerability caused by cumulative exposures to adverse social and economic conditions.[11,12,42]

Limitations and Strengths

We relied on self-reported indicators of dietary intake, to which we assigned estimated antioxidant values. Although it is unlikely these biases were systematically patterned so as to skew results, these measures had a degree of imprecision. Our data set did not allow assessment of biological indicators of oxidative stress, individual sensitivity to oxidative stress, or gene–environment interactions that may moderate antioxidant levels present in blood and tissues.[23,62,63] Our study focused on low- to moderate-income communities of color, which may experience higher baseline levels of oxidative stress[43–45] as well as higher exposures to $PM_{2.5}$. Such communities have been underrepresented in previous studies of antioxidant intake.

Levels of DAI in our sample were low relative to estimates from other investigations. For example, the Health Professionals Follow-Up Study reported daily DAI of approximately 10.76 millimoles[64]; our sample averaged 7.4 millimoles. Chun et al. estimated DAI as well as supplemental antioxidant intake from National Health and Nutritional Examination Survey data.[31] Their reported consumption of vitamins A, C, and E and selenium translates to about 32 millimoles per day, substantially higher than our estimates. Thus, the DAI derived from our data may have underestimated the compensatory or protective effects that may operate in populations with higher antioxidant intake from diet or supplements.

Despite these limitations, our study had several unique strengths and contributions. It was among only a handful of studies to examine the joint effects of $PM_{2.5}$ and DAI in

a community-dwelling population, rather than in a controlled, clinical setting. Our data provided measured (rather than self-reported) BP and ambient measures of air quality recorded over a 3-year period. We used measures of daily intake of antioxidants derived from whole foods, rather than supplements. Our study also highlighted the potential of a long-term community–academic partnership to advance new research questions that address cumulative impacts of community environmental conditions on health.

Conclusions

Our findings are consistent with, and build upon, previously reported results suggesting that residents of some Detroit neighborhoods experience excess cardiovascular risk in part through exposure to poor air quality.[4,51,65–67] Our finding that DAI was associated with reduced blood pressure and may partially mitigate adverse effects of $PM_{2.5}$ on hemodynamic indicators is particularly relevant in light of previous research reporting limited access to healthy food in some Detroit neighborhoods[48,50,68] and linking food access to dietary intakes.[41,53] Aligning with extensive, ongoing work to improve equity of food environments and nutrition throughout the United States,[69–72] our findings emphasize the need to ensure availability of foods rich in antioxidants in food stores, with particular attention to such availability in areas in which residents are exposed to air pollution.

Although our findings suggested beneficial effects of DAI, large and adverse effects of $PM_{2.5}$ on SBP and PP remained. Our findings suggest that these potential protective effects, although helpful, are unlikely to eliminate adverse effects of $PM_{2.5}$ exposure on cardiovascular health or the disproportionate risk of such exposures on the health of low- to moderate-income urban communities. Attention to land-use decisions that shape the exposures of residents of low- to moderate-income communities and communities of color to particulate pollutants is critical to efforts to reduce health inequities.[73,74] Such efforts should consider these cumulative effects and devise strategies to address underlying social, political, and economic dynamics that may place marginalized communities at disproportionate risk. In recognition of the disproportionate effects of

such cumulative exposures for residents of low- to moderate-income urban communities, continued investment should be made to improve mechanisms to better quantify the cumulative effects of social, economic, and chemical exposures and to incorporate these assessment tools into regulatory decision-making processes.[75–77] ∎

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Contributors

A. J. Schulz conceptualized the study and oversaw the data analysis. G. B. Mentz helped analyze data, oversaw the compilation of data for the antioxidant scales, and created the antioxidant scales. N. R. Sampson helped analyze data and conducted relevant literature reviews. J. T. Dvonch oversaw the portions of the analysis related to air quality amd helped review and interpret findings. A. G. Reyes helped conceptualize the study. B. Izumi helped construct the antioxidant scale, review the literature, and interpret findings. All authors contributed to and reviewed the article.

Human Participant Protection

The University of Michigan institutional review board approved the HEP study. The survey was conducted in accordance with ethical standards for treatment of human participants and the Helsinki Declaration of 1975, as revised in 2000. All survey participants gave written informed consent prior to inclusion in the study.

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