Blood Lead Level Is Associated With Elevated Blood Pressure in Blacks

Suma Vupputuri, Jiang He, Paul Muntner, Lydia A. Bazzano, Paul K. Whelton, Vecihi Batuman

Abstract—Chronic lead exposure has been associated with elevated blood pressure in epidemiological studies. It is not known whether the previously observed relation between blood lead and hypertension persists after significant reductions have been made in environmental lead contamination. We examined the relation between blood lead levels and blood pressure in a representative sample of 14,952 whites and blacks aged 18 years or older who participated in the Third National Health and Nutrition Examination Survey. Blood lead was measured by atomic absorption spectrophotometry and blood pressure by standard sphygmomanometry. Mean blood lead levels were significantly higher for black men and women (5.4 and 3.4 μg/dL, respectively) compared with white men and women (4.4 and 3.0 μg/dL, respectively). After multivariate adjustment for important covariables, each standard deviation higher blood lead (3.3 μg/dL) was associated with a 0.82 (95% confidence interval [CI], 0.19 to 1.44) mm Hg and a 1.55 (95% CI, 0.47 to 2.64) mm Hg higher systolic blood pressure among black men and women, respectively. In contrast, blood lead level was not associated with blood pressure among white men or women. The multivariate-adjusted odds ratio (95% CI) of hypertension associated with a 1-SD higher level of blood lead was 1.08 (95% CI, 0.99 to 1.19) for black men and 1.39 (95% CI, 1.21 to 1.61) for black women. These findings suggest that increased levels of blood lead remain an important environmental risk factor for elevated blood pressure in blacks. (Hypertension. 2003;41:1114–1120.)

Key Words: blood pressure ■ ethnic groups ■ blood lead ■ blacks ■ epidemiology

Most,1–10 but not all,11–16 studies have indicated that environmental exposure to lead is associated with an increased risk of hypertension and renal disease. A strong association between blood lead level and prevalence of hypertension was noted in the Second National Health and Nutrition Examination Survey (NHANES II).4,17,18 The NHANES II was conducted during 1976 and 1980, when many of the current environmental measures, such as reduction in use of leaded gasoline, had not been adopted or had not been in place long enough to have a measurable effect on lead pollution.17–19 In recent years, a strong secular trend toward decreasing blood lead concentrations has been noted in the general population. For example, among the US population aged 1 to 74 years, mean blood lead concentrations dropped by 78%, from 12.8 μg/dL to 2.8 μg/dL between 1976 to 1980 (NHANES II) and 1988 to 1991 (the first phase of the Third National Health and Nutrition Examination Survey [NHANES III]).8,19–21 Whether the relation between blood lead level and blood pressure (BP) is still present after this marked decline in environmental lead exposure is unknown. The present study examined the relation between blood lead level and BP in a large representative sample of blacks and whites who participated in NHANES III.

Study Participants
The NHANES III was conducted by the National Center for Health Statistics between 1988 and 1994. Details of the NHANES III study participants and methods have been published elsewhere.22 In brief, a stratified multistage probability design was used to obtain a representative sample of the civilian noninstitutionalized US general population. To improve the precision of estimates in certain subgroups, the design included oversampling of the very young, the elderly, blacks, and Mexican-Americans. A total of 19,618 participants 18 years of age and older were included in NHANES III. Participants were excluded from the current analysis when their self-reported race was not white or black (n=635) or when data on blood lead (n=3936) or BP (n=95) were missing. A total of 10,548 whites and 4,404 blacks were included in the main analysis where hypertension was the outcome of interest. Participants who were taking antihypertensive medications (n=2,496) were excluded from the linear regression analysis examining BP as a continuous variable.

Measurements
The NHANES III data collection included a standardized home interview followed by a detailed physical examination in a mobile examination center or the participant’s home. Information on a wide variety of sociodemographic, medical history, nutritional history, and family history questions, such as self-reported age, race/ethnicity, gender, years of education completed, usual level of physical activity, smoking status, and personal and family history of hypertension was recorded. Blood pressure was measured on the upper arm using a conventional mercury sphygmomanometer, after 5 minutes of lying supine. Total body weight was measured while participants were wearing light clothing and no shoes. Height was measured with a mechanical stadiometer and the body mass index (BMI) was calculated. The blood lead level was determined by atomic absorption spectrophotometry of an aliquot of blood on a spectrophotometer using an atomic absorbance method. The association between blood pressure and blood lead was assessed after adjusting for potential confounders such as age, gender, race, education level, smoking status, and BMI.

Received August 15, 2002; first decision September 6, 2002; revision accepted December 18, 2002.
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Hypertension is available at http://www.hypertensionaha.org

DOI: 10.1161/01.HYP.0000055015.39788.29
activity, history of smoking, hypertension, diabetes, alcohol intake, and 24-hour dietary recall, were obtained at the home interview.\textsuperscript{22}

BP was measured 3 times during the home interview and another 3 times at the mobile examination center. BP for individual participants was calculated as the average of all available systolic and diastolic readings. Hypertension was defined as the presence of a mean systolic BP $\geq$140 mm Hg and/or diastolic BP $\geq$90 mm Hg and/or taking antihypertensive medication. Body weight and height were measured according to a standard protocol, and body mass index was calculated as an index for obesity.

A blood sample was collected by venipuncture during the physical examination. Blood lead concentration was measured at the National Center for Environmental Health of the Centers for Disease Control and Prevention in Atlanta, Ga, by graphite furnace atomic absorption spectrophotometry.\textsuperscript{23,24}

Statistical Analyses

Baseline characteristics were calculated as means and SEs for continuous variables and percentages for categorical variables. \textsuperscript{*}Percentage of participants who drank alcohol at least 12 times during the past year. \textsuperscript{†}Percentage of participants who reported no regular physical activity including walking, jogging/running, bicycling, swimming, dancing, calisthenics, gardening and weight-lifting. \textsuperscript{‡}Includes persons on antihypertensive medication.


<table>
<thead>
<tr>
<th>Variable</th>
<th>White (n = 5,360)</th>
<th>Black (n = 2,104)</th>
<th>P</th>
<th>White (n = 5,188)</th>
<th>Black (n = 2,300)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, y</td>
<td>43.9 ± 0.5</td>
<td>39.9 ± 0.5</td>
<td>&lt;0.001</td>
<td>47.4 ± 0.6</td>
<td>42.0 ± 0.4</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>High school education, %</td>
<td>74.6</td>
<td>64.1</td>
<td>&lt;0.001</td>
<td>74.7</td>
<td>67.0</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Body mass index, kg/m$^2$</td>
<td>26.6 ± 0.1</td>
<td>26.3 ± 0.1</td>
<td>0.08</td>
<td>26.3 ± 0.2</td>
<td>28.4 ± 0.2</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Alcohol consumption, %</td>
<td>65.9</td>
<td>58.7</td>
<td>0.002</td>
<td>44.5</td>
<td>30.6</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>No leisure-time physical activity, %†</td>
<td>14.7</td>
<td>21.8</td>
<td>&lt;0.001</td>
<td>25.6</td>
<td>41.2</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Dietary sodium, mg/d</td>
<td>4291 ± 55</td>
<td>4201 ± 84</td>
<td>0.25</td>
<td>2838 ± 35</td>
<td>2941 ± 44</td>
<td>0.05</td>
</tr>
<tr>
<td>Dietary potassium, mg/d</td>
<td>3451 ± 34</td>
<td>2832 ± 45</td>
<td>&lt;0.001</td>
<td>2505 ± 26</td>
<td>2080 ± 26</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Total energy, kcal/d</td>
<td>2684 ± 28</td>
<td>2602 ± 38</td>
<td>0.03</td>
<td>1760 ± 19</td>
<td>1782 ± 20</td>
<td>0.37</td>
</tr>
<tr>
<td>Systolic BP, mm Hg‡</td>
<td>124.6 ± 0.5</td>
<td>126.1 ± 0.5</td>
<td>0.02</td>
<td>121.1 ± 0.6</td>
<td>122.4 ± 0.6</td>
<td>0.11</td>
</tr>
<tr>
<td>Diastolic BP, mm Hg‡</td>
<td>76.2 ± 0.2</td>
<td>77.5 ± 0.4</td>
<td>0.01</td>
<td>71.8 ± 0.2</td>
<td>73.5 ± 0.4</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Blood lead, $\mu$g/d</td>
<td>4.4 ± 0.1</td>
<td>5.4 ± 0.2</td>
<td>&lt;0.001</td>
<td>3.0 ± 0.1</td>
<td>3.4 ± 0.1</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

Data are mean ± SE for continuous variables and percentage for categorical variables.

Results

Characteristics of the study participants by race and gender are presented in Table 1. Black participants were younger than their white counterparts and were less likely to have a high school education. Mean body mass index was higher among black women compared with black men, white men, or white women. Whites and black men were more likely to have reported consumption of alcohol at least 12 times during the preceding year than black women. Dietary sodium, potassium, and total energy intake were higher among men compared with women, and dietary potassium intake was higher among whites than among blacks. Systolic and diastolic BP levels were higher in blacks compared with whites and higher in men compared with women. Blood lead levels were higher for blacks compared with whites and for men compared with women.

Tests for interaction terms (lead-race and lead-gender) in multivariate logistic and linear regression models revealed that the relation between blood lead level and BP differed significantly by race as well as gender (each $P<0.001$).

In age-adjusted linear regression analyses (Table 2), blood lead level was not associated with systolic BP among white men and women but was significantly and positively associated among black men and women. After multivariate adjustment for important covariates, each SD higher blood lead (3.3 $\mu$g/dL) was associated with a 0.82 (95% CI, 0.70 to 0.95) mm Hg and a 1.55 (95% CI, 0.47 to 2.64) mm Hg higher systolic BP among black men and women, respectively. Furthermore, in age-adjusted models, blood lead level was significantly associated with diastolic BP only among black women. After further controlling for education, body mass index, alcohol consumption, leisure-time physical activity, dietary intake of sodium, potassium, and total energy, higher blood lead was statistically significantly associated with higher diastolic BP for black men and women but not whites.

The age-adjusted prevalence of hypertension was higher among blacks compared with whites. In age-adjusted logistic regression analysis, blood lead levels were positively related with increased odds of hypertension for black women (Table...
Multivariate-adjusted differences in systolic and diastolic blood pressure for persons with a blood lead concentration ≥5 μg/dL compared with those with a blood lead concentration <5 μg/dL by race and gender. Data were adjusted for age, education, body mass index, use of alcohol, physical activity, sodium, potassium, and total calories. *P<0.05; **P<0.01.

for white men, 1.48 (1.13 to 1.93) for white women, 1.22 (0.93 to 1.60) for black men, and 1.73 (1.24 to 2.43) for black women.

Discussion

Our study shows that, in addition to having higher mean blood lead levels, blacks demonstrated consistent, significant associations between blood lead level and systolic and diastolic BPs. In contrast, blood lead level was not significantly associated with BP among whites. Furthermore, blacks with blood lead levels ≥5 μg/dL had a significant increase in mean BP (systolic BP of 1.66 mm Hg and 2.48 mm Hg in men and women, respectively). The association of blood lead with hypertension in this study was significant in black women but only of borderline significance in black men.

It is important to distinguish between the implications of individual and population changes in BP. For an individual, the increases in BP that we observed were slight (systolic BP of 0.82 mm Hg and 1.55 mm Hg in black men and women, respectively) and have limited importance. However, at the population level, even a small downward shift in BP would be expected to result in a substantial reduction in cardiovascular disease. It is estimated that a population-wide reduction in systolic BP of as little as 2 mm Hg would result in a 17% reduction in the prevalence of hypertension as well as a 15% reduction in the risk of stroke and transient ischemic attacks and a 6% reduction in the risk of coronary heart disease.

### Table 2. Differences and 95% Confidence Interval of Blood Pressures Associated With a 1-SD Higher Level of Blood Lead Concentration† Among NHANES III Participants by Race and Gender

<table>
<thead>
<tr>
<th>Race/Gender Group</th>
<th>Age-Adjusted</th>
<th>Multivariate-Adjusted‡</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Systolic BP, mm Hg</td>
<td></td>
</tr>
<tr>
<td>White Men</td>
<td>0.37 (−0.18, 0.93)</td>
<td>0.29 (−0.24, 0.83)</td>
</tr>
<tr>
<td>Women</td>
<td>0.27 (−0.59, 1.13)</td>
<td>0.34 (−0.49, 1.17)</td>
</tr>
<tr>
<td>Black Men</td>
<td>0.83 (0.19, 1.47)*</td>
<td>0.82 (0.19, 1.44)*</td>
</tr>
<tr>
<td>Women</td>
<td>1.15 (0.08, 2.21)*</td>
<td>1.55 (0.47, 2.64)**</td>
</tr>
<tr>
<td></td>
<td>Diastolic BP, mm Hg</td>
<td></td>
</tr>
<tr>
<td>White Men</td>
<td>−0.20 (−0.54, 0.14)</td>
<td>0.01 (−0.38, 0.40)</td>
</tr>
<tr>
<td>Women</td>
<td>−0.27 (−0.76, 0.23)</td>
<td>−0.04 (−0.56, 0.47)</td>
</tr>
<tr>
<td>Black Men</td>
<td>0.48 (−0.08, 1.04)</td>
<td>0.64 (0.08, 1.20)*</td>
</tr>
<tr>
<td>Women</td>
<td>0.93 (0.20, 1.66)*</td>
<td>1.07 (0.37, 1.77)**</td>
</tr>
</tbody>
</table>

†1-SD Blood lead=3.3 μg/dL; ‡adjusted for age, education, body mass index, alcohol consumption, no leisure time physical activity, sodium, potassium, and total calories. *P<0.05; **P<0.01, ***P<0.001.

3). After multivariate adjustment for important covariables, blood lead level was significantly associated with the odds of hypertension among white women (odds ratio [OR]=1.32; 95%CI, 1.14 to 1.52) and black women (OR=1.39; 95%CI, 1.21 to 1.61), marginally significantly associated with hypertension among black men (OR=1.08; 95%CI, 0.99 to 1.19; P=0.08), and not associated with hypertension among white men (OR=1.04; 95% CI, 0.93 to 1.16).

In multivariate-adjustment models, a blood lead concentration ≥5 μg/dL was associated with higher systolic and diastolic BP among blacks but not whites (Figure). Compared with their counterparts with a blood lead <5 μg/dL, systolic and diastolic BP was 1.67 and 1.68 mm Hg higher, respectively, among black men and 2.48 and 2.22 mm Hg higher, respectively, among black women with a blood lead concentration ≥5 μg/dL (each P<0.05). Also, the multivariate-adjusted OR (95% CI) of hypertension associated with a blood lead concentration ≥5 μg/dL was 1.06 (0.81 to 1.38)

### Table 3. Odds Ratios and 95% Confidence Intervals of Hypertension Associated With a 1-SD Higher Level of Blood Lead Concentration Among NHANES III Participants by Race and Gender

<table>
<thead>
<tr>
<th>Race/Gender Group</th>
<th>Prevalence</th>
<th>OR (95% CI)</th>
<th>P</th>
<th>OR (95% CI)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Age-Adjusted</td>
<td>Multivariate-Adjusted‡</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>White Men</td>
<td>18.2</td>
<td>0.99 (0.89, 1.11)</td>
<td>0.91</td>
<td>1.04 (0.93, 1.16)</td>
<td>0.47</td>
</tr>
<tr>
<td>Women</td>
<td>16.0</td>
<td>1.16 (0.99, 1.36)</td>
<td>0.07</td>
<td>1.32 (1.14, 1.52)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Black Men</td>
<td>31.8</td>
<td>1.06 (0.99, 1.14)</td>
<td>0.10</td>
<td>1.08 (0.99, 1.19)</td>
<td>0.08</td>
</tr>
<tr>
<td>Women</td>
<td>28.5</td>
<td>1.23 (1.08, 1.39)</td>
<td>0.002</td>
<td>1.39 (1.21, 1.61)</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

‡Adjusted for age, education, body mass index, alcohol consumption, physical activity, sodium, potassium, and total calories.
Our findings are consistent with the experience in a majority of other studies suggesting that exposure to lead (including low-level exposure) is significantly and positively associated with an elevated BP level and risk of hypertension. Furthermore, our findings are similar to those of others who identified higher blood lead levels in blacks and a stronger association between blood lead and hypertension in blacks, compared with whites. The biological mechanisms for the observed racial differences in the association between blood lead level and elevated BP are not entirely clear. Our results suggest that blacks have a higher mean blood lead level compared with whites. In addition, different patterns of exposure in different environments, ie, inner city tenements, where blacks are disproportionately represented, may have divergent consequences. Epidemiological studies also indicate that blacks are more likely to be exposed to lead in an occupational setting. Furthermore, racial differences in bone metabolism and, specifically, in bone handling of lead may contribute to this apparent discrepancy in the association between blood lead level and elevated BP. Blacks might also be more sensitive to the effects of lead exposure on BP owing to environmental and genetic factors. Residual confounding due to factors related to social-economic status might also contribute to a stronger association among blacks.

Although we observed no significant association between blood lead level and systolic or diastolic BP in white women, we did find a significant association between blood lead and hypertension among both white and black women. Several other studies have suggested that blood lead is more strongly related to elevated BP and hypertension in women compared with men. Both environmental and occupational exposure to lead has long been associated with hypertension and renal disease. Animal studies suggest that even low levels of exposure to lead can result in hypertension. Blood lead levels have dramatically declined in the US general population since efforts to reduce environmental lead exposure were emphasized in the 1970s. The virtual disappearance of overt lead poisoning may have caused complacency towards the hazards of low-level asymptomatic lead poisoning. Many believe that the lead-abatement interventions have all but eliminated the major health consequences of lead poisoning, and the association between low-level lead poisoning and hypertension has become increasingly controversial. Several studies have disputed the presence of a relation between low-level lead exposure and hypertension. Staessen and colleagues identified a significant association between blood lead levels and impairment of renal function in the general population in the Cadmibel study but failed to recognize a significant relation between blood lead and hypertension. Although it is possible that the latter does not exist, the findings in the Cadmibel study are consistent with the hypothesis that renal microvascular disease caused by lead precedes the occurrence of hypertension.

Since NHANES II, there has been a significant reduction in the mean blood lead concentration in the general population. With more stringent exposure guidelines in the workplace and environmental measures, such as reducing lead content in gasoline and house paint, the average blood lead concentration has declined dramatically in the general population, and symptomatic overt lead poisoning has become a rare event. Although the overall lowering of blood lead levels in the United States may represent a public health success in primary prevention efforts, disturbing disparities in exposure to environmental lead continue to be noted. Higher blood lead levels are still being seen in older versus younger adults, males versus females, inner-city residents versus non–inner-city residents, and blacks versus whites. In addition, higher-than-average blood lead levels are being noted in those with a low income, low educational attainment, and residence in the northeastern United States.

The possibility that environmental lead exposure may be a contributing factor in the occurrence of hypertension among blacks is very important. This may explain, in part, the unusually high burden of illness due to hypertension and renal disease among blacks. Hypertension is more common, more severe, and usually appears earlier in life in blacks. The higher blood lead levels and associated higher BP levels in blacks raise the possibility that chronic exposure to environmental lead is partially responsible for the strikingly higher incidence of both hypertension and renal disease in this population.

A continued diligent effort to eliminate lead pollution in the United States, especially targeting high-risk black communities, is crucial. Unfortunately, the cost of lead abatement is high, and the number of properly trained lead-abatement professionals is low. Many interventions that have been implemented to reduce environmental lead exposure have focused on secondary prevention, targeting homes with children who already have elevated blood lead levels. However, the number of these children with elevated blood lead levels has decreased rapidly, and the focus can, appropriately, be placed on primary prevention strategies with continuing efforts to adequately remove lead pollutants (such as lead paint) in high-risk communities.

Our study has a number of important strengths. Because NHANES III surveyed a large probability sample of the general population, our findings can be generalized to US adults. This study also provides the opportunity to reevaluate the association of blood lead level and BP after lead-abatement procedures were put in place since NHANES II. Additionally, the large sample size of NHANES III provided ample power to detect a small but important association between blood lead level and elevated BP. A limitation of the present study is the use of a cross-sectional study design. Although this restricts the inferences that can be made based on our findings, there are numerous prospective studies that support the causal role of lead exposure in raising BP.

Another limitation of our study is that blood lead concentration is not an optimal biomarker and may underestimate the internal dose of lead. Current evidence points to bone lead as the most valuable measure of internal dose because it represents a cumulative exposure, and thus it can accurately assess persons who are exposed to chronic low-level environmental lead pollutants. However, currently, the most effective way to measure bone lead involves in vivo K x-ray fluorescence, which is expensive, time-intensive, and thus
impractical for large-scale population studies. Furthermore, blood lead is a good measure of recent exposure to environmental lead and has been shown to be strongly associated with bone lead.46,61 Therefore, although bone lead is a more sensitive marker for environmental lead exposure, the majority of epidemiological studies to date have assessed lead exposure by means of blood lead concentration measurements.

Perspectives
Despite major overall reductions in the United States, blood lead levels remain significantly and positively related to elevated BP and hypertension among blacks, who continue to have higher blood lead concentrations compared with whites. This observation suggests that continuous efforts to reduce environmental lead exposure in the US general population, especially among blacks, may be an important approach to prevent hypertension in the community.

Acknowledgment
This study was supported in part by grant R01HL60300 from the National Heart, Lung, and Blood Institute of the National Institutes of Health, Bethesda, Md.

References


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_Hypertension_, published online February 3, 2003;
_Hypertension_ is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0194-911X. Online ISSN: 1524-4563

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