

Continued Decline in Blood Lead Levels Among Adults in the United States

The National Health and Nutrition Examination Surveys

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Background: Declines in blood lead levels between 1976 and 1991 among US adults have been previously reported. More recent trends in blood lead levels and the association of lower blood lead levels with chronic disease have not been reported.

Methods: Data from 2 nationally representative cross-sectional surveys, the Third National Health and Nutrition Examination Survey conducted in 1988-1994 (n=16 609) and the National Health and Nutrition Examination Survey conducted in 1999-2002 (n=9961) were analyzed.

Results: The geometric mean blood lead level declined 41% from 2.76 $\mu\text{g/dL}$ (0.13 $\mu\text{mol/L}$) in 1988-1994 to 1.64 $\mu\text{g/dL}$ (0.08 $\mu\text{mol/L}$) in 1999-2002. The percentage of adults with blood lead levels of 10 $\mu\text{g/dL}$ (0.48 $\mu\text{mol/L}$) or higher declined from 3.3% in 1988-1994 to 0.7% in 1999-2002 ($P<.001$). In 1999-2002, the multivariable-adjusted odds ratio of having a blood lead level of 10 $\mu\text{g/dL}$ (0.48 $\mu\text{mol/L}$) or higher was 2.91 (95% confidence interval [CI], 1.74-4.84) and 3.26 (1.83-

5.81) for non-Hispanic blacks and Mexican Americans, respectively, compared with non-Hispanic whites. After multivariable adjustment, persons in the highest quartile (≥ 2.47 $\mu\text{g/dL}$ [≥ 0.12 $\mu\text{mol/L}$]) compared with those in the lowest quartile (< 1.06 $\mu\text{g/dL}$ [< 0.05 $\mu\text{mol/L}$]) of blood lead levels were 2.72 (95% CI, 1.47-5.04) and 1.92 (95% CI, 1.02-3.61) times more likely to have chronic kidney disease and peripheral arterial disease, respectively. In addition, higher blood lead levels were associated with a higher multivariable-adjusted odds ratio of hypertension among non-Hispanic blacks and Mexican Americans.

Conclusions: Blood lead levels continue to decline among US adults, but racial and ethnic disparities persist. Higher blood lead levels remain associated with a higher burden of chronic kidney and peripheral arterial diseases among the overall population and with hypertension among non-Hispanic blacks and Mexican Americans.

Arch Intern Med. 2005;165:2155-2161

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THE PRESENCE OF LEAD IN the environment has decreased since the 1970s when the US government enacted regulations banning the use of lead in gasoline, paint, and the soldering of cans.¹⁻³ Nonetheless, lead remains present because of its wide dispersion over many decades and continuing industrial uses.^{4,5} Studies have demonstrated associations between environmental lead exposure and an increased risk of high blood pressure, chronic kidney disease, hyperuricemia, gout, and peripheral arterial disease.⁶⁻⁹

In 1994, Pirkle et al³ published data from the National Health and Nutrition Examination Survey (NHANES) II and the first phase of NHANES III showing a substantial decline in blood lead levels between 1976-1980 and 1988-1991. Among indi-

viduals aged 20 to 74 years, the geometric mean blood lead level decreased 78%, from 13.1 to 3.0 $\mu\text{g/dL}$ (from 0.63 to 0.14 $\mu\text{mol/L}$). Despite such a marked decline, 6.6% of those studied still had blood lead levels of 10 $\mu\text{g/dL}$ (0.48 $\mu\text{mol/L}$) or higher in 1988-1991. In addition, using data from NHANES III, we observed associations between these lower levels of lead exposure and morbidity, including hypertension and chronic kidney disease.^{10,11}

Recent studies¹² suggest that because there is no threshold below which the adverse effects of lead are not experienced, reducing environmental sources of lead exposure remains a public health priority. The goal of the present study is to characterize trends in blood lead levels for the adult population of the United States between 1988-1994 and 1999-2002 using data from NHANES III and NHANES

1999-2002, respectively. In addition, we determined the association between demographic factors, socioeconomic characteristics, and elevated blood lead levels in 1999-2002. Finally, we also determined the association of the blood lead levels observed in NHANES 1999-2002 with hypertension, chronic kidney disease, and peripheral arterial disease.

METHODS

NHANES SURVEYS

NHANES III and NHANES 1999-2002 were nationally representative cross-sectional surveys of the civilian noninstitutionalized population of the United States.^{13,14} The procedures involved in these studies have been published in detail and are available online.^{13,14} In brief, each of these studies included a stratified multistage probability sample based on selection of counties, blocks, households, and persons within households. To provide stable subgroup estimates, Mexican Americans, non-Hispanic blacks, and adults 60 years or older were oversampled in NHANES III and NHANES 1999-2002.

Each participant's NHANES information was obtained from an in-home interview followed by a medical evaluation and blood sample collection at a mobile examination center. Variables collected during the in-home interview that are of relevance to the present analysis are age, race/ethnicity, sex, cigarette smoking, alcohol consumption, a history of diabetes mellitus, pharmacologic treatment for hypertension, health insurance status, household income, having a high school education, and the year each participant's current residence was constructed. Participants who reported having smoked at least 100 cigarettes during their lifetime were classified as current or former smokers depending on whether they answered the question "Do you smoke cigarettes now?" affirmatively or negatively, respectively. Alcohol consumption was defined as having an average of 1 or more drinks per week during the previous year. Diabetes mellitus status was based on self-report.

The NHANES 1999-2002 examination procedures included measurements of height, weight, blood pressure, and ankle-brachial index (ABI). Height was measured using a fixed stadiometer and weight by using a digital scale (Toledo Scale Corp, Toledo, Ohio) with participants wearing underwear, a disposable gown, and foam slippers. Body mass index was calculated as weight in kilograms divided by height in meters squared.

During a single examination visit, a physician took up to 3 blood pressure measurements using the standard protocol of the American Heart Association.¹⁵ Blood pressure was measured with the participant in a seated position following 5 minutes of quiet rest. Based on the average of all available blood pressure measurements, hypertension was defined as systolic or diastolic blood pressure of at least 140 mm Hg or 90 mm Hg, respectively, and/or self-reported current use of blood pressure-lowering medication.

For individuals with at least 1 arm and weighing 180 kg (400 lb) or less, systolic blood pressure for the ABI was measured via blood pressure cuffs on the right brachial artery and both posterior tibial arteries. For individuals aged 40 to 59 years, 2 measurements were taken and averaged at each site, whereas for individuals aged 60 years or older, 1 measurement was taken at each site. For individuals with conditions that precluded measurement of the right arm, the left brachial artery systolic blood pressure was taken. For each ankle, the ABI was calculated as the ratio of the average ankle systolic blood pressure to arm systolic blood pressure. The smaller of the 2 measurements was

considered the ABI for this study. Peripheral arterial disease was defined as an ABI of less than 0.9.

Serum creatinine levels were measured by the Jaffe modified kinetic method using a Hitachi 917 analyzer (Boehringer Mannheim Corp, Indianapolis, Ind). We added 0.13 mg/dL (11 μ mol/L) to each NHANES 1999-2000 participant's measured serum creatinine concentration and 0.02 mg/dL (2 μ mol/L) to each NHANES 2001-2002 participant's concentration to align the concentrations with the assays employed in the development of the Modification of Diet in Renal Disease study equation.¹⁶ Kidney function was assessed by estimating the glomerular filtration rate with the simplified prediction equation from the Modification of Diet in Renal Disease study.¹⁷ Specifically, estimated glomerular filtration rate was calculated as follows:

$$186.3 \times (\text{Serum Creatinine in Milligrams per Deciliter})^{-1.154} \times \text{Age}^{-0.203} \times (0.742 \text{ if Female}) \times (1.21 \text{ if African American}).$$

Individuals with an estimated glomerular filtration rate lower than 60 mL/min per 1.73 m² were considered to have chronic kidney disease.

MEASUREMENT OF BLOOD LEAD LEVELS

All blood samples were venous specimens. They were shipped on dry ice to the NHANES laboratory at the National Centers for Environmental Health at the Centers for Disease Control and Prevention in Atlanta, Ga. Identical methods for measuring blood lead levels were used for NHANES III and NHANES 1999-2002 and are described in detail online.¹⁸ Specifically, blood lead levels were measured using whole blood and graphite furnace atomic absorption spectrophotometry as described by Sassa et al.¹⁹ An elevated blood lead level was defined a priori as 10 μ g/dL (0.48 μ mol/L) or higher. Because there was a low number of NHANES 1999-2002 participants with blood lead levels of at least 10 μ g/dL (0.48 μ mol/L) ($n=113$), persons with blood lead levels of at least 5 μ g/dL (0.24 μ mol/L) ($n=655$) were categorized as having elevated blood lead levels in secondary analyses.

The protocols for NHANES III and NHANES 1999-2002 were approved by the National Center for Health Statistics of the Centers for Disease Control and Prevention institutional review board.

STATISTICAL ANALYSIS

Because of the skewed distribution, blood lead levels were log-transformed, and the age-standardized geometric mean and 95% confidence interval were calculated by age grouping (18-39, 40-59, 60-74, and ≥ 75 years), sex, and race/ethnicity (non-Hispanic white, non-Hispanic black, or Mexican American) for NHANES III and NHANES 1999-2002. Age standardization was made to the year 2000 US population. Next, we calculated the age-standardized prevalence of elevated blood lead levels for 1988-1994 and for 1999-2002 (≥ 5 μ g/dL [≥ 0.24 μ mol/L] and ≥ 10 μ g/dL [≥ 0.48 μ mol/L] respectively), overall and by age grouping, sex, and race/ethnicity. We compared differences in the prevalence estimates across the 2 surveys using the Wald χ^2 test, taking into account the complex survey design used in NHANES III and NHANES 1999-2002. We calculated these test statistics as the difference in prevalence estimates divided by the standard error of the difference, calculated as the square root of the sum of each estimate's variance.

Then, we determined the association of age grouping, race/ethnicity, and sex with elevated blood lead levels (≥ 5 μ g/dL [≥ 0.24 μ mol/L] and ≥ 10 μ g/dL [≥ 0.48 μ mol/L]) using multivariable logistic regression models and data from NHANES 1999-2002. The initial multivariable models included age group, race/ethnicity, and sex, and a subsequent model provided additional adjustment for current and former cigarette smoking,

alcohol consumption, having a high school education, and having health insurance. Next, the age-standardized geometric mean blood lead level and the prevalence of a blood lead level of 5 µg/dL (0.24 µmol/L) or higher was calculated by household income (>\$20 000 vs <\$20 000 per year), health insurance status, living in housing built before or after 1978 (the year the US government banned lead-based paint from housing), having a high school education, and smoking status (current, former, or never). The association between each of these variables and a blood lead level of 5 µg/dL (0.24 µmol/L) or higher was also calculated using logistic regression after adjusting for age, race/ethnicity, and sex.

Next, the NHANES 1999-2002 population was divided into quartiles by blood lead level. The prevalences of hypertension, chronic kidney disease, and peripheral arterial disease were separately calculated by blood lead quartile with trends across quartiles assessed using the χ^2 test for trend. Because the effect of environmental lead exposure on blood pressure has been noted to differ by race/ethnicity, the prevalence and adjusted odds ratios of hypertension were calculated for non-Hispanic whites, non-Hispanic blacks, and Mexican Americans, separately.¹¹ The race/ethnicity-stratified odds ratios of hypertension were determined after adjustment for age, sex, body mass index, diabetes mellitus, current and former cigarette smoking, health insurance status, and having a high school education. The adjusted odds ratios of chronic kidney disease and peripheral arterial disease were determined after adjustment for these potential confounders and for race/ethnicity.

We applied sample weights that account for the unequal probabilities of selection, oversampling, and nonresponse for all analyses using SUDAAN statistical software (version 8.0; Research Triangle Institute, Research Triangle Park, NC). We estimated standard errors using the Taylor series linearization method.

RESULTS

Between 1988-1994 and 1999-2002, the age-standardized geometric mean blood lead level declined by 41% from 2.76 µg/dL (1.33 µmol/L) to 1.64 µg/dL (0.08 µmol/L) (**Table 1**). Substantial declines in blood lead levels occurred in each age group, among men and women, and among non-Hispanic whites, non-Hispanic blacks, and Mexican Americans. The age-standardized prevalence of blood lead levels of 5 µg/dL (0.24 µmol/L) or higher declined from 20.5% to 5.0% between 1988-1994 and 1999-2002 (**Figure, A**; $P<.001$). Large declines in the prevalence of blood lead levels of 5 µg/dL (0.24 µmol/L) or higher were also present within each age group, among men and women, and among non-Hispanic whites, non-Hispanic blacks, and Mexican Americans ($P<.001$ for all). In addition, the age-standardized prevalence of blood lead levels of 10 µg/dL (0.48 µmol/L) or higher decreased from 3.3% in 1988-1994 to 0.7% in 1999-2002 (**Figure, B**; $P<.001$). The age-standardized prevalence of blood lead levels of 10 µg/dL (0.48 µmol/L) or higher decreased to less than 1.0% of the population for each age group, women, and non-Hispanic whites in 1999-2002 ($P<.001$ for all). In addition, the age-standardized prevalence of blood lead levels of 10 µg/dL (0.48 µmol/L) or higher declined from 5.7% to 1.2% among men, from 7.3% to 1.8% among non-Hispanic blacks, and from 4.5% to 1.7% among Mexican Americans between 1988-1994 and 1999-2002 ($P<.001$ for all).

Table 1. Age-Adjusted Geometric Mean Blood Lead Levels Among NHANES III (1988-1994) and NHANES 1999-2002 Participants

Variable	Blood Lead Level, µg/dL, Mean (95% CI)	
	NHANES III (1988-1994)	NHANES 1999-2002
Overall	2.76 (2.61-2.92)	1.64 (1.59-1.68)
Age group, y		
18-39	2.21 (2.07-2.36)	1.28 (1.23-1.33)
40-59	2.97 (2.79-3.15)	1.81 (1.75-1.87)
60-74	3.61 (3.42-3.80)	2.17 (2.09-2.25)
≥75	3.75 (3.57-3.94)	2.32 (2.20-2.44)
Sex		
Men	3.61 (3.40-3.83)	2.08 (2.02-2.14)
Women	2.13 (2.02-2.25)	1.31 (1.27-1.35)
Race		
Non-Hispanic white	2.65 (2.49-2.82)	1.58 (1.54-1.62)
Non-Hispanic black	3.29 (3.12-3.46)	1.85 (1.76-1.94)
Mexican American	2.96 (2.78-3.14)	1.86 (1.74-1.98)

Abbreviations: CI, confidence interval, NHANES, National Health and Nutrition Examination Survey.

SI conversion factor: To convert lead to micromoles per liter, multiply by 0.0483.

In a multivariable model including age, race/ethnicity, and sex, persons in the older age groups, men, and non-Hispanic blacks and Mexican Americans were more likely to have blood lead levels of 5 µg/dL (0.24 µmol/L) or higher (**Table 2**; $P<.001$ for all). Also, men, non-Hispanic blacks, and Mexican Americans were more likely than women and non-Hispanic whites, respectively, to have blood lead levels of 10 µg/dL (0.48 µmol/L) or higher ($P<.001$ for all). Each of these associations remained significant after further adjustment for current and former cigarette smoking, alcohol consumption, having a high school education, and having health insurance. Also, after multivariable adjustment, the odds ratios of having blood lead levels of 10 µg/dL (0.48 µmol/L) or greater were higher for persons in the older age groups, but this trend was not significant ($P=.16$).

Age-standardized geometric mean blood lead levels and the prevalence of blood lead levels of at least 5 µg/dL (0.24 µmol/L) were higher among persons with an income of less than \$20 000 per year, no health insurance, living in a house built prior to 1978, and not having a high school education, and among former and current smokers (**Table 3**; $P<.001$ for all).

The prevalence of chronic kidney disease and peripheral arterial disease were each progressively higher at the higher quartile of blood lead level (**Table 4**; $P<.001$). After adjustment for age, race/ethnicity, sex, diabetes mellitus, body mass index, current and former cigarette smoking, alcohol consumption, having a high school education, and having health insurance, the multivariable-adjusted odds ratios of chronic kidney disease and peripheral arterial disease were progressively higher at higher lead quartiles (each P value for trend, $<.001$). In these multivariable-adjusted models, persons in the highest quartile of blood lead level (≥ 2.47 µg/dL [≥ 0.12 µmol/L]) were 2.72 (1.47-5.04) times more likely to have

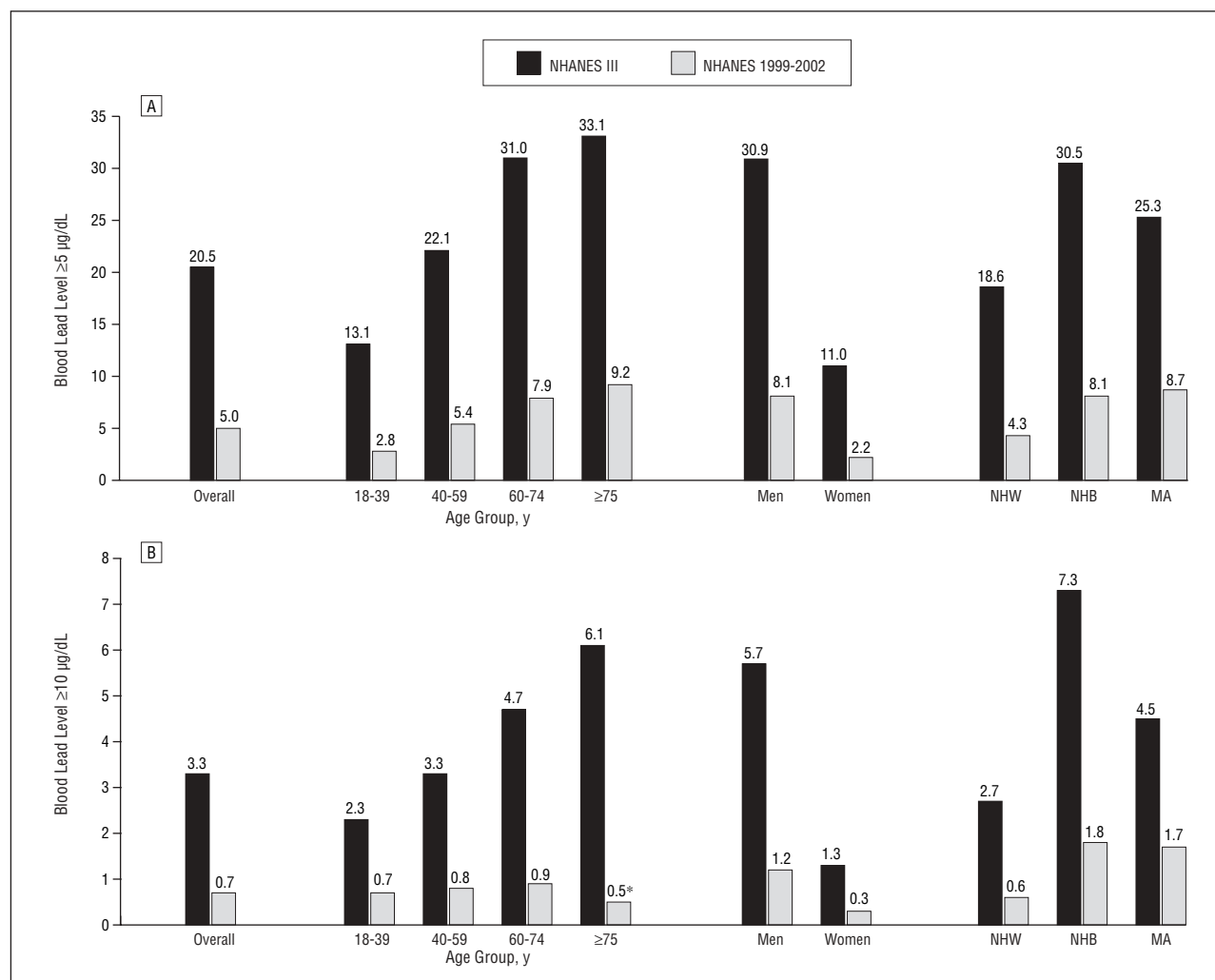


Figure. Age-specific and age-standardized prevalence of blood lead levels 5 µg/dL or higher (A) and 10 µg/dL or higher (B) among US adults during 1988-1994 (National Health and Nutrition Examination Survey [NHANES] III) and 1999-2002 (NHANES 1999-2002). MA indicates Mexican Americans; NHW, non-Hispanic whites; NHB, non-Hispanic blacks. The asterisk denotes an estimate that is unstable due to a small number of participants with blood lead level ≥ 10 µg/dL. To convert lead to micromoles per liter, multiply by 0.0483.

chronic kidney disease, and 1.92 (1.02-3.61) times more likely to have peripheral arterial disease compared with their counterparts in the lowest quartile of blood lead level (<1.06 µg/dL [<0.05 µmol/L]).

Hypertension was also progressively more common at higher quartiles of blood lead level among each race/ethnicity subgroup (**Table 5**; each $P < .001$). After multivariable adjustment, no association was present between higher lead levels and hypertension among non-Hispanic whites (P value for trend, .61). In contrast, the multivariable-adjusted odds ratios of hypertension were higher with each increasing quartile of blood lead for non-Hispanic blacks (P value for trend, .06) and Mexican Americans (P value for trend, .04).

COMMENT

The data presented in this study, derived from 2 national surveys, document the continued decline in environmental lead exposure in the US population. Substantial declines in blood lead levels occurred between

1988-1994 and 1999-2002 in the overall US adult population in every age group; for non-Hispanic whites, non-Hispanic blacks, and Mexican Americans; and among men and women. The geometric mean blood lead level and prevalence of blood lead levels of 10 µg/dL (0.48 µmol/L) or higher declined by 41% and 79%, respectively, between 1988-1994 and 1999-2002. In 1999-2002, the geometric mean blood lead level of adults in the United States was 1.64 µg/dL (0.08 µmol/L), and 0.7% of adults had a blood lead level of 10 µg/dL (0.48 µmol/L) or higher. However, non-Hispanic blacks and Mexican Americans remain disproportionately affected by exposure to environmental lead and are 3 times more likely to have blood lead levels of 10 µg/dL (0.48 µmol/L) or higher compared with non-Hispanic whites.

The continued decline in blood lead levels among all demographic groups investigated provides evidence of the benefits from public health initiatives aimed at removing lead from population-wide sources. However, lead exposure remains an environmental health problem among low socioeconomic populations in the United

Table 2. Adjusted Odds Ratios (ORs) of Blood Lead Levels ≥ 5 $\mu\text{g}/\text{dL}$ and ≥ 10 $\mu\text{g}/\text{dL}$ Associated With Age Grouping, Sex, and Race/Ethnicity Among Participants of NHANES 1999-2002

Variable	Adjusted OR (95% CI)			
	Blood Lead ≥ 5 $\mu\text{g}/\text{dL}$		Blood Lead ≥ 10 $\mu\text{g}/\text{dL}$	
	Demographic*	Multivariable Adjusted†	Demographic*	Multivariable Adjusted†
Age group, y				
18-39	1.00 (Reference)	1.00 (Reference)	1.00 (Reference)	1.00 (Reference)
40-59	2.33 (1.71-3.17)	2.61 (1.90-3.59)	1.29 (0.64-2.58)	1.33 (0.66-2.67)
60-74	3.45 (2.43-4.89)	4.67 (3.18-6.85)	1.19 (0.53-2.70)	1.53 (0.65-3.61)
≥ 75	5.11 (3.30-7.91)‡	8.54 (5.24-13.92)‡	1.19 (0.45-3.10)	1.93 (0.70-5.38)
Sex				
Women	1.00 (Reference)	1.00 (Reference)	1.00 (Reference)	1.00 (Reference)
Men	4.02 (2.96-5.46)‡	3.28 (2.30-4.67)‡	4.38 (2.13-8.97)‡	3.43 (1.59-7.40)‡
Race/ethnicity				
Non-Hispanic white	1.00 (Reference)	1.00 (Reference)	1.00 (Reference)	1.00 (Reference)
Non-Hispanic black	2.07 (1.60-2.69)‡	1.96 (1.51-2.54)‡	3.12 (1.78-5.48)‡	2.91 (1.74-4.84)‡
Mexican American	2.55 (1.93-3.36)‡	2.24 (1.67-2.98)‡	3.54 (2.09-6.01)‡	3.26 (1.83-5.81)‡

Abbreviations: CI, confidence interval; NHANES, National Health and Nutrition Examination Survey.

SI conversion factor: To convert lead to micromoles per liter, multiply by 0.0483.

*Adjusted for age, race/ethnicity, and sex.

†Adjusted for age, race/ethnicity, sex, current and former cigarette smoking, alcohol consumption, having a high school education, and having health insurance.

‡ $P < .001$ (P value for trend, $< .001$ across age groups).

Table 3. Blood Level Values Among Participants of NHANES 1999-2002

Variable	Age-Adjusted Geometric Mean (95% CI)	Blood Lead Level ≥ 5 $\mu\text{g}/\text{dL}$, % (SE)	Adjusted OR (95% CI)*
Household income, \$			
$\geq 20\,000$	1.59 (1.54-1.64)	4.3 (0.4)	1.00 (Reference)
$< 20\,000$	1.76 (1.68-1.85)	7.2 (0.8)†	1.74 (1.23-2.46)†
Health Insurance			
Yes	1.55 (1.51-1.59)	4.1 (0.3)	1.00 (Reference)
No	2.01 (1.88-2.15)	9.4 (1.0)†	2.62 (1.95-3.52)†
Year current residence was built			
In 1978 or after	1.48 (1.43-1.53)	2.7 (0.3)	1.00 (Reference)
Prior to 1978	1.69 (1.62-1.75)	5.7 (0.5)†	2.17 (1.61-2.93)†
High school education			
Yes	1.56 (1.51-1.60)	3.6 (0.3)	1.00 (Reference)
No	1.98 (1.92-2.04)	9.6 (0.9)†	2.67 (1.83-3.90)†
Smoker			
Never	1.41 (1.37-1.45)	2.7 (0.3)	1.00 (Reference)
Former	1.68 (1.60-1.77)	5.0 (0.5)†	1.63 (1.21-2.20)†
Current	2.21 (2.11-2.30)	10.2 (1.0)†	3.78 (2.63-5.42)†

Abbreviations: CI, confidence interval; NHANES, National Health and Nutrition Examination Survey.

SI conversion factor: To convert lead to micromoles per liter, multiply by 0.0483.

*Adjusted for age, race/ethnicity, and sex.

† $P < .001$.

States.^{20,21} A continued diligent effort to eliminate lead pollution in the United States, especially targeting vulnerable communities, is crucial. Given the association between low-level lead exposure and chronic disease, further efforts to reduce environmental lead exposure are warranted. Available strategies for the reduction of lead exposure include use of safe removal practices for lead-based paint and clean-up of lead-contaminated dust and soil.

Despite being one of the most pervasive and persistent heavy metals in the environment, lead has no known necessary function in humans. Instead, high levels of lead exposure have been repeatedly documented to cause sig-

nificant neurological defects and several chronic diseases. Many people believe that lead-abatement interventions have all but eliminated the major health consequences of lead poisoning. However, several studies^{6,8,22} have demonstrated that even low-level lead exposure results in increased risk for hypertension, chronic kidney disease, and peripheral arterial disease. For example, in the population-based Cadmibel study, Staessen et al⁶ showed that each 10-fold increase in blood lead level was associated with a 13-mL/min (0.22-mL/s) and 30-mL/min (0.50-mL/s) lower estimated creatinine clearance rate among men and women, respectively. In the present study, higher blood lead levels conferred an in-

Table 4. Prevalence and Adjusted* Odds Ratios (ORs) of Hypertension, Chronic Kidney Disease, and Peripheral Arterial Disease by Quartile of Blood Lead Level Among Adult Participants of NHANES 1999-2002

Variable	Quartile of Blood Lead Level (Range, µg/dL)				P Value for Trend
	Quartile 1 (<1.06)	Quartile 2 (1.06-1.63)	Quartile 3 (1.63-2.47)	Quartile 4 (≥2.47)	
Chronic kidney disease†					
Prevalence, % (SE)	1.8 (0.4)	3.4 (0.5)	5.6 (0.6)	8.1 (0.7)	<.001
Multivariable-adjusted OR (95% CI)*	1.00 (Reference)	1.49 (0.75-2.98)	1.89 (1.09-3.30)	2.72 (1.47-5.04)	<.001
Peripheral arterial disease‡					
Prevalence, % (SE)	2.6 (0.6)	3.0 (0.5)	4.8 (0.6)	7.7 (0.6)	<.001
Multivariable-adjusted OR (95% CI)*	1.00 (Reference)	1.00 (0.45-2.22)	1.21 (0.66-2.23)	1.92 (1.02-3.61)	<.001

Abbreviations: CI, confidence interval; NHANES, National Health and Nutrition Examination Survey.

SI conversion factor: To convert lead to micromoles per liter, multiply by 0.0483.

*Adjusted for age; race/ethnicity; sex; diabetes mellitus; body mass index (calculated as weight in kilograms divided by the square of height in meters); current cigarette smoking; alcohol consumption; having a high school education; and having health insurance.

†Chronic kidney disease was defined as an estimated glomerular filtration rate lower than 60 mL/min.

‡Peripheral arterial disease was defined as an ankle-brachial index lower than 0.9.

Table 5. Race/Ethnicity-Specific Prevalence and Multivariable-Adjusted Odds Ratios (ORs) of Hypertension* by Quartile of Blood Lead Level Among Adult Participants of NHANES 1999-2002

Race/Ethnicity	Quartile of Blood Lead Level (Range, µg/dL)				P Value for Trend
	Quartile 1 (<1.06)	Quartile 2 (1.06-1.63)	Quartile 3 (1.63-2.47)	Quartile 4 (≥2.47)	
Non-Hispanic white					
Prevalence, % (SE)	18.7 (1.6)	26.4 (1.6)	32.0 (1.7)	38.9 (1.9)	<.001
Multivariable-adjusted OR†	1.00 (Reference)	1.12 (0.83-1.50)	1.03 (0.78-1.37)	1.10 (0.87-1.41)	.61
Non-Hispanic black					
Prevalence, % (SE)	20.9 (3.0)	29.1 (2.4)	37.5 (3.5)	47.2 (2.0)	<.001
Multivariable-adjusted OR (95% CI)†	1.00 (Reference)	1.03 (0.63-1.67)	1.12 (0.77-1.64)	1.44 (0.89-2.32)	.06
Mexican American					
Prevalence, % (SE)	10.3 (1.8)	16.8 (1.7)	17.8 (2.1)	18.2 (1.7)	<.001
Multivariable-adjusted OR (95% CI)†	1.00 (Reference)	1.42 (0.75-2.71)	1.48 (0.89-2.48)	1.54 (0.99-2.39)	.04

Abbreviations: CI, confidence interval; NHANES, National Health and Nutrition Examination Survey.

SI conversion factor: To convert lead to micromoles per liter, multiply by 0.0483.

*Hypertension was defined as systolic/diastolic blood pressure of 140/90 mm Hg or higher and/or current antihypertensive medication usage.

†Adjusted for age; sex; diabetes mellitus; current cigarette smoking; body mass index (calculated as weight in kilograms divided by the square of height in meters); alcohol consumption; having a high school education; and having health insurance.

creased risk of chronic kidney disease and peripheral arterial disease.

In this study, we found that higher blood lead levels were associated with higher rates of hypertension among non-Hispanic blacks and Mexican Americans. However, the association between low-level lead exposure and hypertension has become increasingly controversial.^{23,24} Most previous studies^{11,22,25-28} reported lead exposure to be significantly and positively associated with an elevated blood pressure and an increased risk of hypertension. However, other studies^{24,29,30} have disputed the presence of a relation between low-level lead exposure and hypertension. The adverse effect of lead on blood pressure has been reported to be stronger among non-Hispanic blacks than among non-Hispanic whites.^{11,31} We decided a priori to analyze the association between blood lead levels and hypertension stratified by race/ethnicity. The association between blood lead levels and hypertension was stronger for non-Hispanic blacks and Mexican Americans than for non-Hispanic whites, which is consistent with previous findings. Documentation that en-

vironmental lead exposure may be a contributing factor in the occurrence of hypertension among minority populations is important because it may provide further understanding into the disparities in hypertension in the United States. Hypertension is more common, more severe, and usually appears earlier in life in non-Hispanic blacks.^{32,33} Understanding the role of low-level environmental lead exposure in the pathogenesis of hypertension should remain an important public health priority.

A limitation of our study is that blood lead concentration is not an optimal biomarker. Data indicate that bone lead is the most valuable measurement 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 over a long period of time.^{34,35} However, bone lead measurements are impractical for large-scale population studies. Given that mean blood lead levels reported in the present study fell dramatically between 1988-1994 and 1999-2002, it is possible that the association between higher blood lead levels and chronic kidney disease and

peripheral arterial disease may be the result of much higher blood lead levels at some previous time. Unfortunately, data were not available in the present study to determine previous sources or levels of lead exposure. Finally, it has been proposed³⁶ that kidney dysfunction may result in the decreased excretion of lead, and this may explain the higher lead levels among patients with chronic kidney disease. Given the cross-sectional design of the present study, data were not available to examine this hypothesis. However, results from most previous studies³⁷⁻⁴⁰ suggest that higher blood lead levels are the cause, rather than the result, of lower renal function.

In conclusion, the virtual disappearance of overt lead poisoning may have caused complacency toward the hazards of low-level chronic lead exposure. Despite major overall reductions in the United States, blood lead levels remain associated with hypertension among non-Hispanic blacks and Mexican Americans, who continue to have higher blood lead concentrations compared with non-Hispanic whites. In addition, blood lead levels are significantly associated with chronic kidney disease and peripheral arterial disease in the overall population. Given the strong associations between higher blood lead levels and several chronic diseases observed in the present study, continued efforts to reduce environmental lead exposure are warranted.

Accepted for Publication: May 31, 2005.

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Financial Disclosure: None.

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