

Trends and variability in blood lead concentrations among US children and adolescents

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Abstract Using data from the National Health and Nutrition Examination Survey for the period 2003–2012, the objective of this study was to evaluate trends in blood lead levels (BLL) among children aged 1–5 and 6–11 years and smoker and nonsmoker adolescents aged 12–19 years. Regression models with log₁₀ transformed values of BLLs as dependent variable were fitted to evaluate how gender, race/ethnicity, smoking, and exposure to secondhand smoke at home affect BLLs. Irrespective of age, gender, and race/ethnicity, BLLs declined over the study period ($p \leq 0.01$). Overall, adjusted BLLs declined by 0.00114 $\mu\text{g}/\text{dL}$ for every 2 years. Children aged 1–5 years had about 50 % higher BLLs than smoker adolescents, about 75 % higher BLLs than nonsmoker adolescents, and about 45 % higher BLLs than children aged 6–11 years. While overall, children aged 1–5 years with $\text{BLL} \geq 5 \mu\text{g}/\text{dL}$ made up 3.24 %, 7.8 % non-Hispanic Black children aged 1–5 years had $\text{BLL} \geq 5 \mu\text{g}/\text{dL}$. Males were found to have higher adjusted BLLs than females, and non-Hispanic Blacks were found to have higher adjusted BLLs than non-Hispanic Whites. Higher poverty income ratio was associated with lower adjusted BLLs ($\beta = -0.02916$, $p < 0.01$). Children living in owner-occupied homes had lower adjusted BLLs than children living in renter-occupied homes. BLLs increased with increase in number of smokers smoking inside the home

($\beta = 0.02496$, $p = 0.02$). In conclusion, while BLLs have declined for all age groups, genders, and races/ethnicities, certain races/ethnicities like non-Hispanic Blacks continue to have substantially higher BLLs than non-Hispanic Whites.

Keywords NHANES · Blood lead · Children · Smoking · Adolescents · Trends

Introduction

Lead is a heavy, low-melting, bluish-gray metal that occurs naturally in the earth's crust at about 15–20 mg/kg (<http://www.atsdr.cdc.gov/toxprofiles/tp13.pdf>, p. 1, 19). Lead and its alloys are commonly found in pipes, storage batteries, weights, shots and ammunitions, cable covers, and sheets used to shield from radiation, and lead compounds are used as a pigment in paints, dyes, and ceramic glazes and caulk (<http://www.atsdr.cdc.gov/toxprofiles/tp13.pdf>, p. 1–2). Lead is released into the air during burning of coal, oil, or waste, and before its use was banned in gasoline, most of the lead released into the US environment came from vehicle exhaust. Rain removes lead from the air and its particles fall to land or into surface water (<http://www.atsdr.cdc.gov/toxprofiles/tp13.pdf>, p. 2–3). Some lead compounds are changed into other forms of lead by sunlight, air, and water.

Exposure to lead from house dust (Bi et al., 2015, Swaddiwudhipong et al., 2014), street dust (Han et al., 2015), and roof dust (Yu et al., 2015) has also been reported. Babies and children can swallow and breathe lead in dirt, dust, or sand while they lie on the floor or ground, and they can swallow nonfood items such as paint chips which may contain a large amount of lead (<http://www.atsdr.cdc.gov/toxprofiles/tp13.pdf>, p. 9–10). Children can swallow lead-contaminated

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dirt from their hands and toys in normal hand-to-mouth activities (<http://www.atsdr.cdc.gov/toxprofiles/tp13.pdf>, p. 363).

People who live near hazardous waste sites and those who work in lead smelting and refining industries; brass/bronze foundries; rubber products and plastics industries; soldering, steel welding, and cutting operations; battery manufacturing plants; and lead compound manufacturing industries may be exposed to lead and chemicals that contain lead by breathing air, drinking water, eating foods, or swallowing dust or dirt that contain lead (<http://www.atsdr.cdc.gov/toxprofiles/tp13.pdf>, p. 4). Other routes of exposure to lead include drinking water in houses that contain lead pipes and eating foods contained in improperly glazed pottery or ceramic dishes and from leaded-crystal glassware (<http://www.atsdr.cdc.gov/toxprofiles/tp13.pdf>, p. 4). Breathing in and swallowing airborne dust and dirt are associated with exposure to lead. At home, children may be exposed to lead if they use certain types of home remedy medications that contain lead. Health care costs attributable to lead poisoning have been estimated to be \$43.4 billion (Landrigan et al., 2002). Based on the analysis of National Health and Nutrition Examination (NHANES) III data among those aged ≥ 40 years, there was a suggestion that higher blood lead levels (BLLs) may be associated with increased cancer mortality (van Bommel et al., 2011). Cheung (2013) used data from NHANES III and reported a positive association between BLLs and all cause, all cancer, and lung cancer mortality among adults. According to Bellinger (2008), no level of lead appears to be safe and even the currently defined low levels of exposure among children are associated with neurodevelopmental deficits.

Having reviewed data from 19 surveys in general population and 12 studies in occupationally exposed groups, Nawrot et al. (2002) concluded that a twofold increase in BLLs was associated with a 1.0-mmHg rise in systolic blood pressure and a 0.6-mmHg rise in diastolic blood pressure. Navas-Acien et al. (2007) found sufficient evidence to conclude a causal relationship between BLLs and hypertension.

Among adults aged ≥ 18 years, increasing blood lead levels were found to be associated ($p = 0.014$) with increased prevalence of elevated serum alanine aminotransferase used as a biomarker for nonalcoholic fatty liver disease (Cave et al., 2010). Trzeciakowski et al. (2014) used a somewhat complicated statistical methodology to show a negative relationship between serum lead and renal function. Luo and Hendryx (2014), based on data from NHANES 2007–2010, reported BLLs and cadmium levels to be a disrupter of thyroid function. Kresovich et al. (2015) used data from NHANES for the period 1999–2004 for adult males and found blood lead levels to be positively associated with the levels of testosterone and sex-hormone-binding globulin.

Hu et al. (2014) used NHANES data for the period 2005–2010 for participants aged 20–59 years and reported a dose-

response-like relationship between BLLs and substance use, risky sexual behaviors, self-reported sexually transmitted disease, and biologically confirmed herpes simplex virus type 2. Min et al. (2012) suggested an association between BLLs and balance and vestibular dysfunction in adults aged ≥ 40 years. Blood lead concentration was associated with decreased walking speed in females aged ≥ 50 years (Ji et al., 2013). In a pilot study of 47 healthy subjects aged 55–67 years, an analysis adjusted for age, education, and smoking status found higher bone lead values with increased memory impairment (van Wijngaarden et al., 2009). Past occupational exposure to lead was found to be associated with a progressive decline in cognitive function (Schwartz et al., 2000). Behavior and mental development of children aged 2–4 years were found to be adversely affected by lead poisoning (Hou et al., 2013). As compared to healthy children, children with lead poisoning had higher levels of abnormal behaviors like social withdrawal, depression, atypical body movements, aggressions, and destruction (Hou et al., 2013).

An inverse association between BLLs and/or urinary lead levels and bone mineral density (BMD) has been investigated by several authors. An inverse association between BLLs and total hip BMD among non-Hispanic White males aged ≥ 50 years was reported by Campbell and Auinger (2007). Postmenopausal females have been found to be more susceptible to high BLLs and low BMD as compared with premenopausal females (Lee and Kim, 2012). In a Chinese study of males and females aged ≥ 27 years (Chen et al., 2014), low BMD was found to be associated with blood lead levels ≥ 10 $\mu\text{g}/\text{dL}$ both in males and females.

Lead and mercury can easily cross the placenta and accumulate in fetal tissues (Iyengar and Rapp, 2001) and as such have the potential to affect birth outcomes. In a study of newborns conducted in Nepal (Parajuli et al. 2014), cord BLLs were found to be inversely associated with the neurodevelopment of newborns, suggesting prenatal exposure to lead may induce retardation. Wells et al. (2014) used data from NHANES 2005–2006 for children aged 2–12 years and found an 11.1 % increase in immunoglobulin E and a 4.9 % increase in eosinophils for a 1- $\mu\text{g}/\text{dL}$ increase in blood levels, and associations were stronger among children who lived with smokers. In a study among low-income predominantly African-American children in Saginaw, MI (Smith and Nriagu, 2011) under the age of 12 years, elevated blood lead levels (≥ 10 $\mu\text{g}/\text{dL}$) were over five times more likely to be found among asthmatic than nonasthmatic children. The Centers for Disease Control and Prevention (CDC) (2013), based on NHANES data for 1999–2010, reported 2.6 % children aged 1–5 years have blood lead levels ≥ 5 $\mu\text{g}/\text{dL}$.

Prenatal exposure to even very low BLLs was found to enhance sensitization to common aeroallergens at the age of 5 years (Jedrychowski et al., 2011). Prenatal exposure to very low BLLs (< 5 $\mu\text{g}/\text{dL}$) as measured in cord blood was found to

be negatively associated with the cognitive function (Jedrychowski et al., 2009a) at the age of 3 years in boys ($p < 0.01$) but not in girls ($p = 0.43$), suggesting that there may be no threshold for lead toxicity among children. In addition, statistically significant negative associations between cord BLLs and scores on the Bayley Mental Development Index were also found at the ages of 12, 24, and 36 months (Jedrychowski et al., 2009b) and average deficit in cognitive development over 3 years was also statistically significant. In a retrospective study of 262 mother-infant pairs, Jelliffe-Pawlowski et al. (2006) reported females with a maximum BLL of ≥ 10 $\mu\text{g}/\text{dL}$ have a threefold risk of having a preterm birth and a more than fourfold risk of giving birth to an infant small for gestational age when compared with females with a maximum pregnancy BLL of < 10 $\mu\text{g}/\text{dL}$. They (Jelliffe-Pawlowski et al., 2006) also found a negative association between second trimester maximum BLLs ≥ 10 $\mu\text{g}/\text{dL}$ and duration of gestation. In a retrospective cohort study of 43,288 mother-infant pairs in upstate New York, Zhu et al. (2010) reported a negative association between birth weight and maternal BLLs but BLLs were not found to be related to preterm or small for gestational age delivery.

There have been a few studies that have reported data on BLLs using NHANES. The results from some of these studies are described below. Dixon et al. (2009) analyzed NHANES data for the years 1999–2004 for children aged 1–5 years and concluded (i) BLLs were affected by the levels of lead in the floor and the surface type and condition of the floor, (ii) non-Hispanic Black (NHB) children had higher BLLs than non-Hispanic White children (NHW), (iii) children born in Mexico had higher BLLs than children born in the USA, (iii) houses built before 1940 were associated with higher BLLs, (iv) children living in houses where someone smoked had higher BLLs than when no one smoked inside the house, and (v) the odds of NHB children having BLLs > 5 $\mu\text{g}/\text{dL}$ and > 10 $\mu\text{g}/\text{dL}$ were more than twice that of NHW children. Richter et al. (2013) used data from NHANES for the years 1999–2008 for those aged ≥ 3 years and concluded (i) youths aged 3–18 years with exposure to secondhand smoke (SHS) had higher BLLs than youths without exposure to SHS, (ii) BLLs among adults aged ≥ 19 years were in the order smokers $>$ nonsmokers with exposure to SHS $>$ nonsmokers without exposure to SHS, (iii) people living below the poverty level and in houses built in 1949 or earlier and born outside of the USA had relatively higher BLLs, and (iv) there was a decreasing trend in BLLs over the period covered by the study. Richter et al. (2013) presented some of their results for those aged 3–18 years labeled as “youth” and for those aged ≥ 19 years labeled as “adults.” In the opinion of this author, grouping all those under the age of 19 years is not appropriate because of the lifestyle changes associated with various ages in this group that can affect exposure to lead and as such BLLs. Children aged under 5 years remain at home

under the protection of their parents, and being very low to the ground, they are likely to have much higher levels of exposure to lead-contaminated dust and floors than other children. Children at the age of 6 years start going to school “full time” on a regular basis and as such are likely to be exposed to relatively lower levels of lead at home but may also be exposed to lead in the outdoor environment, for example, school buildings and the playground. Once these children turn 12 years of age, they spend more and more time out of the home in middle and high schools and engage in other personal and recreational activities like driving, going to movies, and sports. Consequently, the level of exposure to lead for adolescents aged 12–19 years is likely to be at a level substantially different than that of children aged < 6 and 6–11 years. Richter et al. (2013) arbitrarily defined no exposure to SHS as being equivalent to serum cotinine levels < 0.035 ng/mL , low exposure to SHS as having serum cotinine levels between 0.035 and 0.256 ng/mL , and high exposure to SHS as having serum cotinine levels between 0.256 and 10 ng/mL . In addition, since data on serum cotinine were not available for children under the age of 3 years, these children were excluded from the study. While decreasing trends over time in BLLs have been previously reported among children (Jones et al., 2009), it is essential that any adverse deviation from these decreasing trends be monitored not only among children but also among adolescents and adults since elevated BLLs affect all ages. In brief, the objective of this study is to study trends in BLLs of children aged 1–11 years and adolescents aged 12–19 years over the period 2003–2012. In addition, the impact of race/ethnicity, gender, age, exposure to secondhand smoke (SHS), and other relevant factors that may affect BLLs will be studied. The data from NHANES for the years 2003–2012 were selected for this purpose. It should be noted that this study will be limited to children and adolescents. Resources permitting, results of a separate study will be reported for adults.

Materials and methods

Data for the years 2003–2010 from NHANES on those aged ≤ 19 years from files on demographics, body measure, blood lead, serum cotinine, family smoking questionnaire, and housing characteristics were downloaded and match merged. NHANES uses a complex, stratified, multistage, probability sampling designed as representative of the civilian, noninstitutionalized US population based on age, gender, and race/ethnicity (<http://www.nchs/nchs/nhanes.htm>). Sampling weights are created in NHANES to account for the complex survey design, including oversampling, survey nonresponse, and post-stratification. All those aged < 12 years were considered nonsmokers. Adolescents were defined as nonsmokers if their serum cotinine levels were below 10 $\text{ng}/$

mL and smokers if their serum cotinine levels were ≥ 10 ng/mL. This classification scheme to define smokers and non-smokers has previously been used by Jain (2013a), among others. Same laboratory methods were used to measure blood lead throughout the study period of 2003–2012 and are provided elsewhere (http://wwwn.cdc.gov/Nchs/Nhanes/2011-2012/PbCd_G.htm#Description_of_Laboratory_Methodology). For blood lead (Pb), concentrations were found to be at or above the limit of detection (LOD) for at least 97.5 % of samples irrespective of age, gender, race/ethnicity, and smoking status (Supplementary Table S1). Traditionally, at least for the data reported from NHANES, when concentrations are below the LOD, they are imputed as $\text{LOD} \div \sqrt{2}$ before proceeding to analyze data. This approach has been used by Jain (2013a) among other authors, and therefore, in this study, I adopted this approach.

All analyses were done using SAS version 9.3 (<http://www.sas.com>, SAS, Cary, NC, USA). All analyses used appropriate sampling weights and information on sampling design such as stratification and clustering as provided in NHANES data files. The dependent variable in the regression model was the log₁₀ transformed values of Pb measured in nanograms per deciliter. While NHANES provides data for BLLs in micrograms per deciliter, to avoid working with small values which may result in unstable and unreliable estimates of statistical parameters like slopes, BLLs were converted into nanograms per deciliter. For the purpose of this study, four age-smoking categories were created, namely children aged 1–5 years (Y15), children aged 6–11 years (Y611), nonsmoker adolescents aged 12–19 years (Y1219NSM), and smoker adolescents aged 12–19 years (Y1219SM). In the regression model, the categorical independent variables used were age-smoking categories (Y15, Y611, Y1219NSM, and Y1219SM), gender (male, female), race/ethnicity (non-Hispanic White (NHW), non-Hispanic Black (NHB), Mexican American (MA), and other unclassified races/ethnicities (OTH)), exposure to secondhand smoke (yes, no), housing living arrangements (owned (OWN), renting (RENT)), and other arrangements like living with someone else (OA)), and continuous independent variables used were number of smokers smoking inside the home, number of cigarettes smoked inside the home every day, number of rooms in the house, poverty to income ratio (PIR), and NHANES survey year in two-year groups coded as 3 (for 2003–2004) through 7 (for 2011–2012) to adjust for changes in BLLs over time. It should be noted that the NHANES survey year was used as a continuous ordinal variable, not as a categorical variable. First-order interaction terms between gender and race/ethnicity, race/ethnicity and housing arrangements, and race/ethnicity and exposure to SHS were considered but were retained in the final models only if they were statistically significant at $\alpha = 0.05$.

A total of 16,373 participants were available for analysis. Detailed sample sizes by age-smoking categories, gender, and race/ethnicity are given in Table 1. However, actual sample sizes used for regression models were smaller because of missing values for one or more independent variables like PIR, etc. Sample sizes used in regression model are presented as Supplementary Table S2. Briefly, total sample size used in regression models for those aged 1–5 years was 4182, and for those aged 6–11 years, it was 4775. The sample size for non-smoker adolescents was 6256, and for smoker adolescents, it was 870.

Results

Time trends

Irrespective of age-smoking category, gender, and race/ethnicity, BLLs decreased over the study period of 2003–2012 ($p \leq 0.02$) and for each survey period from 2003–2004 to 2011–2012; unadjusted geometric means (UGM) for BLLs for every gender and race/ethnicity category were in the order $Y15 > Y611 > Y1219SM > Y1219NSM$ (Table 2). For example, for 2003–2004, the UGMs for BLLs for Y15 were almost double of what they were for Y1219NSM (1.76 vs. 0.92 $\mu\text{g}/\text{dL}$, Table 2).

Overall variability in unadjusted BLLs by age smoking, gender, and race/ethnicity

For the study period of 2003–2012, for both males and females and every racial/ethnic group, Y15 had statistically significantly higher UGMs for BLL (Table 3) than Y611 ($p < 0.01$), Y1219NSM ($p < 0.01$), and Y1219SM ($p < 0.01$). For NHB, UGMs for BLL for Y15 were about 80 % higher than those for Y1219NSM (1.89 vs. 1.05 $\mu\text{g}/\text{dL}$). Also, irrespective of gender and race/ethnicity, Y611 had statistically significantly higher UGMs for BLL ($p < 0.01$, Table 3) than Y1219NSM. In addition, Y611 also had statistically significantly higher UGMs for BLL than Y1219SM for females and NHB ($p < 0.01$, Table 3). Finally, Y1219SM had statistically significantly higher UGMs for BLL than Y1219NSM ($p < 0.01$, Table 3).

Categorical distribution of BLLs

As compared with other age-smoking categories, Y15 had the highest BLLs between 5 and 9.9 $\mu\text{g}/\text{dL}$ and above ≥ 10 $\mu\text{g}/\text{dL}$ irrespective of gender and race/ethnicities (Table 4). For NHB, the differences were particularly noteworthy. For NHB, percent BLLs ≥ 5 $\mu\text{g}/\text{dL}$ for Y15 was 7.8 % as compared to 2.69, 0.75, and 0.40 % for Y611, Y1219NSM, and Y1219SM, respectively.

Table 1 Unweighted sample sizes by age, gender, race/ethnicity, and smoking status. Data from National Health and Nutrition Examination Survey 2003–2012

	Age group with smoking status							
	1–5 years		6–11 years		12–19 years, nonsmokers		12–19 years, smokers	
	<i>N</i>	%	<i>N</i>	%	<i>N</i>	%	<i>N</i>	%
Total	4244	100.0	4858	100.0	6376	100.0	895	100.0
Males	2190	51.6	2437	50.2	3214	50.4	595	66.5
Females	2054	48.4	2421	49.8	3162	49.6	300	33.5
Non-Hispanic Whites	1190	28.0	1298	26.7	1625	25.5	357	39.9
Non-Hispanic Blacks	1105	26.0	1341	27.6	1956	30.7	277	30.9
Mexican Americans	1258	29.6	1417	29.2	1883	29.5	153	17.1
Other races/ethnicities	691	16.3	802	16.5	912	14.3	108	12.1

Multivariate analysis—adjusted blood lead levels

The only interaction term that was found to be statistically significant at $\alpha=0.05$ was between race/ethnicity and

residential living arrangement. R^2 for the regression model was 32.5 %.

Males had statistically significantly higher adjusted geometric mean (AGM) BLLs than females ($p<0.01$, Table 5).

Table 2 Unadjusted geometric means (UGM) with 95 % confidence intervals in micrograms per deciliter for blood lead by age, gender, race/ethnicity, smoking status, and survey year with regression slope (β) for time trends. Data from National Health and Nutrition Examination Survey 2003–2012

	Age group	UGM with 95 % confidence intervals in $\mu\text{g}/\text{dL}$					β	P_{trends}
		2003–2004	2005–2006	2007–2008	2009–2010	2011–2012		
Total	1–5 years	1.76 (1.61–1.93)	1.46 (1.37–1.56)	1.51 (1.38–1.65)	1.17 (1.09–1.25)	0.97 (0.88–1.07)	–0.061	<0.01
	6–11 years	1.25 (1.13–1.38)	1.02 (0.95–1.1)	0.99 (0.92–1.06)	0.84 (0.79–0.88)	0.68 (0.63–0.74)	–0.061	<0.01
	12–19 years, nonsmokers	0.92 (0.85–1)	0.77 (0.73–0.82)	0.78 (0.72–0.84)	0.66 (0.62–0.7)	0.54 (0.5–0.58)	–0.055	<0.01
	12–19 years, smokers	1.07 (0.97–1.18)	0.96 (0.87–1.06)	0.95 (0.86–1.05)	0.88 (0.79–0.98)	0.72 (0.62–0.84)	–0.037	<0.01
Males	1–5 years	1.75 (1.6–1.93)	1.47 (1.35–1.6)	1.54 (1.4–1.68)	1.17 (1.06–1.29)	1.03 (0.9–1.17)	–0.056	<0.01
	6–11 years	1.29 (1.15–1.45)	1.03 (0.95–1.12)	0.99 (0.92–1.08)	0.88 (0.82–0.95)	0.72 (0.66–0.79)	–0.057	<0.01
	12–19 years, nonsmokers	1.07 (0.98–1.16)	0.91 (0.84–0.99)	0.91 (0.83–1)	0.78 (0.73–0.83)	0.62 (0.57–0.67)	–0.055	<0.01
	12–19 years, smokers	1.32 (1.17–1.48)	1.1 (0.98–1.22)	1.05 (0.96–1.15)	0.94 (0.83–1.06)	0.8 (0.63–1.01)	–0.049	<0.01
Females	1–5 years	1.77 (1.57–2.01)	1.46 (1.36–1.56)	1.48 (1.33–1.66)	1.17 (1.1–1.25)	0.91 (0.84–0.99)	–0.067	<0.01
	6–11 years	1.2 (1.09–1.33)	1.01 (0.94–1.1)	0.98 (0.91–1.06)	0.79 (0.74–0.85)	0.64 (0.58–0.71)	–0.065	<0.01
	12–19 years, nonsmokers	0.8 (0.73–0.87)	0.65 (0.61–0.7)	0.67 (0.61–0.72)	0.56 (0.53–0.6)	0.47 (0.42–0.51)	–0.053	<0.01
	12–19 years, smokers	0.83 (0.7–0.97)	0.8 (0.71–0.9)	0.73 (0.62–0.87)	0.76 (0.65–0.88)	0.6 (0.52–0.71)	–0.029	0.01
Non-Hispanic Whites	1–5 years	1.56 (1.43–1.7)	1.34 (1.24–1.44)	1.42 (1.23–1.64)	1.1 (0.98–1.23)	0.93 (0.78–1.12)	–0.052	<0.01
	6–11 years	1.08 (1–1.16)	0.94 (0.85–1.04)	0.91 (0.83–1)	0.77 (0.7–0.85)	0.62 (0.55–0.71)	–0.056	<0.01
	12–19 years, nonsmokers	0.83 (0.77–0.9)	0.72 (0.66–0.78)	0.73 (0.66–0.81)	0.6 (0.55–0.67)	0.52 (0.47–0.58)	–0.048	<0.01
	12–19 years, smokers	0.97 (0.85–1.1)	0.9 (0.79–1.04)	0.91 (0.82–1.02)	0.87 (0.74–1.01)	0.68 (0.57–0.83)	–0.03	0.01
Non-Hispanic Blacks	1–5 years	2.73 (2.24–3.34)	2.12 (1.87–2.4)	1.94 (1.71–2.2)	1.59 (1.42–1.77)	1.29 (1.11–1.51)	–0.078	<0.01
	6–11 years	1.89 (1.6–2.24)	1.35 (1.2–1.53)	1.31 (1.17–1.47)	1.15 (1–1.34)	0.9 (0.78–1.03)	–0.073	<0.01
	12–19 years, nonsmokers	1.17 (1.03–1.32)	0.88 (0.77–1)	0.89 (0.8–0.99)	0.76 (0.69–0.83)	0.57 (0.55–0.6)	–0.069	<0.01
	12–19 years, smokers	1.37 (1.22–1.53)	1.11 (0.95–1.3)	1.13 (0.94–1.35)	1.01 (0.81–1.27)	0.77 (0.73–0.82)	–0.054	<0.01
Mexican Americans	1–5 years	1.77 (1.6–1.96)	1.39 (1.25–1.53)	1.46 (1.28–1.67)	1.13 (1.03–1.23)	0.91 (0.78–1.05)	–0.067	<0.01
	6–11 years	1.45 (1.33–1.57)	1.05 (0.92–1.19)	1.01 (0.87–1.16)	0.86 (0.76–0.97)	0.65 (0.58–0.72)	–0.079	<0.01
	12–19 years, nonsmokers	1.07 (1–1.16)	0.87 (0.75–1.01)	0.78 (0.65–0.93)	0.69 (0.61–0.77)	0.5 (0.46–0.55)	–0.077	<0.01
	12–19 years, smokers	1.53 (1.25–1.88)	1.04 (0.92–1.18)	0.91 (0.54–1.52)	0.78 (0.6–1.01)	0.92 (0.61–1.37)	–0.057	0.02
Other races/ethnicities	1–5 years	1.78 (1.49–2.14)	1.6 (1.39–1.83)	1.49 (1.34–1.65)	1.11 (0.97–1.27)	0.88 (0.78–0.99)	–0.078	<0.01
	6–11 years	1.16 (0.96–1.4)	1.08 (0.92–1.26)	1.04 (0.94–1.16)	0.82 (0.75–0.89)	0.73 (0.65–0.82)	–0.053	<0.01
	12–19 years, nonsmokers	1.04 (0.91–1.19)	0.88 (0.75–1.02)	0.89 (0.78–1.01)	0.77 (0.68–0.87)	0.58 (0.51–0.66)	–0.059	<0.01
	12–19 years, smokers	1.45 (1.14–1.85)	1.12 (0.82–1.53)	1.11 (0.95–1.29)	0.86 (0.7–1.05)	0.75 (0.65–0.86)	–0.067	<0.01

Table 3 Unadjusted geometric means in micrograms per deciliter with 95 % confidence intervals for blood lead by age, gender, race/ethnicity, and smoking status for years 2003–2012. Data from National Health and Nutrition Examination Survey

	Age group with smoking status				Statistically significant differences
	1–5 years (Y15)	6–11 years (Y611)	12–19 years, nonsmokers (Y1219NSM)	12–19 years, smokers (Y1219SM)	
Total	1.36 (1.31–1.42)	0.94 (0.9–0.97)	0.72 (0.69–0.74)	0.92 (0.88–0.97)	Y15 > Y611 ($p < 0.01$), Y15 > Y1219NSM ($p < 0.01$), Y15 > Y1219SM ($p < 0.01$), Y611 > Y1219NSM ($p < 0.01$), Y1219SM > Y1219NSM ($p < 0.01$)
Males	1.38 (1.32–1.44)	0.97 (0.93–1.01)	0.84 (0.81–0.87)	1.04 (0.98–1.1)	Y15 > Y611 ($p < 0.01$), Y15 > Y1219NSM ($p < 0.01$), Y15 > Y1219SM ($p < 0.01$), Y611 > Y1219NSM ($p < 0.01$), Y1219SM > Y1219NSM ($p < 0.01$)
Females	1.34 (1.28–1.41)	0.9 (0.87–0.94)	0.61 (0.59–0.64)	0.75 (0.7–0.81)	Y15 > Y611 > Y1219SM > Y1219NSM ($p < 0.01$)
Non-Hispanic Whites	1.28 (1.21–1.35)	0.86 (0.81–0.9)	0.67 (0.64–0.7)	0.88 (0.82–0.94)	Y15 > Y611 ($p < 0.01$), Y15 > Y1219NSM ($p < 0.01$), Y15 > Y1219SM ($p < 0.01$), Y611 > Y1219NSM ($p < 0.01$), Y1219SM > Y1219NSM ($p < 0.01$)
Non-Hispanic Blacks	1.89 (1.75–2.04)	1.29 (1.19–1.4)	0.83 (0.78–0.88)	1.05 (0.97–1.15)	Y15 > Y611 > Y1219SM > Y1219NSM ($p < 0.01$)
Mexican Americans	1.3 (1.22–1.38)	0.95 (0.89–1.02)	0.74 (0.69–0.79)	1.02 (0.87–1.2)	Y15 > Y611 ($p < 0.01$), Y15 > Y1219NSM ($p < 0.01$), Y15 > Y1219SM ($p < 0.01$), Y611 > Y1219NSM ($p < 0.01$), Y1219SM > Y1219NSM ($p < 0.01$)
Other races/ethnicities	1.3 (1.22–1.38)	0.93 (0.88–0.99)	0.78 (0.74–0.84)	1.03 (0.91–1.16)	Y15 > Y611 ($p < 0.01$), Y15 > Y1219NSM ($p < 0.01$), Y15 > Y1219SM ($p < 0.01$), Y611 > Y1219NSM ($p < 0.01$), Y1219SM > Y1219NSM ($p < 0.01$)

NHW had statistically significantly lower AGMs for BLL than NHB ($p=0.03$, Table 5) by about 15 %. Y15 had statistically significantly higher AGMs for BLL than Y611, Y1219NSM as well as Y1219SM ($p \leq 0.02$). Y15 had more than 50 % higher AGMs than Y1219SM (1.46 vs. 0.94 $\mu\text{g/dL}$, Table 5). Lower BLLs were observed when the home was owned as compared to rented ($p=0.02$, Table 5). However, when interaction between race/ethnicity and residential living arrangement was taken into account, only for NHB, MA, and OTH, living in an owned home was associated with lower BLLs than renting a home ($p \leq 0.01$, Fig. 1) or having other living arrangements ($p \leq 0.02$, Fig. 1), and renting a home was associated with higher BLLs than having other living arrangements ($p \leq 0.02$, Fig. 1).

Multivariate analysis—association of blood lead levels with continuous independent variables

Number of rooms in the house did not affect BLLs ($p=0.08$, Table 6). The same was true for the number of cigarettes smoked inside the home every day ($p=0.28$, Table 6). Increase in number of smokers smoking inside the home was positively associated with increased BLLs ($p=0.02$,

Table 6). Poverty income ratio was negatively associated with BLLs ($p < 0.01$, Table 6). Adjusted BLLs decreased over time ($p=0.02$).

Discussion

Based on the review of the existing literature, this is the first time that BLLs for younger children aged 1–5 years are being compared with those of older children aged 6–11 years and both nonsmoker and smoker adolescents in a nationally representative sample. There was strong evidence to conclude that children aged 1–5 years had higher BLLs than not only children aged 6–11 ($p < 0.01$) but also both nonsmoker ($p=0.01$) and smoker ($p=0.01$, Table 5) adolescents. Children aged 1–5 years had about 50 % higher BLLs than smoker adolescents, about 75 % higher BLLs than nonsmoker adolescents, and about 45 % higher BLLs than older children aged 6–11 years (Table 5). It may be that younger children aged 1–5 years had a higher level of exposure to Pb than both older children and adolescents or the metabolism of Pb may accelerate with age. Children aged 1–5 years do spend relatively more time at home than children aged 6–11 years since

Table 4 Percent distribution of blood lead levels by age, race/ethnicity, and smoking status. Data from National Health and Nutrition Examination Survey 2003–2012

	Blood lead level	Age and smoking status			
		1–5 years	6–11 years	12–19 years, nonsmokers	12–19 years, smokers
Total	<5 µg/dL	96.86 (96.04 to 97.68)	99.22 (98.85 to 99.59)	99.42 (99.2 to 99.64)	99.65 (99.34 to 99.96)
	5–9.9 µg/dL	2.35 (1.77 to 2.92)	0.7 (0.37 to 1.04)	0.5 (0.29 to 0.7)	0.21 (–0.01 to 0.44)
	≥10 µg/dL	0.79 (0.38 to 1.2)	0.07 (–0.02 to 0.17)	0.08 (–0.02 to 0.19)	0.14 (–0.14 to 0.42)
Males	<5 µg/dL	96.83 (95.83 to 97.83)	99.14 (98.67 to 99.61)	99.15 (98.76 to 99.54)	99.45 (98.95 to 99.94)
	5–9.9 µg/dL	2.56 (1.8 to 3.32)	0.81 (0.35 to 1.27)	0.8 (0.42 to 1.18)	0.33 (–0.02 to 0.69)
	≥10 µg/dL	0.61 (0.2 to 1.02)	0.05 (–0.05 to 0.15)	0.05 (0 to 0.1)	0.22 (–0.22 to 0.66)
Females	<5 µg/dL	96.89 (96.04 to 97.74)	99.31 (98.96 to 99.65)	99.69 (99.51 to 99.88)	100 (100 to 100)
	5–9.9 µg/dL	2.12 (1.53 to 2.71)	0.59 (0.28 to 0.9)	0.2 (0.06 to 0.33)	0 (0 to 0)
	≥10 µg/dL	0.99 (0.45 to 1.54)	0.1 (–0.07 to 0.27)	0.11 (–0.09 to 0.31)	0 (0 to 0)
Non-Hispanic Whites	<5 µg/dL	97.59 (96.89 to 98.29)	99.49 (99.15 to 99.83)	99.73 (99.52 to 99.95)	99.65 (99.35 to 99.95)
	5–9.9 µg/dL	1.58 (1.04 to 2.12)	0.39 (0.14 to 0.65)	0.27 (0.05 to 0.48)	0.15 (–0.15 to 0.45)
	≥10 µg/dL	0.83 (0.48 to 1.18)	0.12 (–0.05 to 0.28)	0 (0 to 0)	0.2 (0.17 to 0.23)
Non-Hispanic Blacks	<5 µg/dL	92.2 (89.33 to 95.07)	97.31 (95.51 to 99.11)	99.25 (98.82 to 99.68)	99.6 (99 to 100.19)
	5–9.9 µg/dL	5.88 (3.8 to 7.96)	2.69 (0.89 to 4.49)	0.66 (0.24 to 1.08)	0.4 (–0.19 to 1)
	≥10 µg/dL	1.92 (0.75 to 3.09)	0 (0 to 0)	0.09 (0.01 to 0.17)	0 (0 to 0)
Mexican Americans	<5 µg/dL	98.18 (97.26 to 99.09)	99.53 (99.19 to 99.87)	98.95 (98.41 to 99.49)	99.15 (99.07 to 99.22)
	5–9.9 µg/dL	1.76 (0.85 to 2.66)	0.41 (0.1 to 0.73)	0.92 (0.44 to 1.4)	0.85 (0.78 to 0.93)
	≥10 µg/dL	0.07 (–0.07 to 0.21)	0.06 (–0.06 to 0.17)	0.13 (–0.02 to 0.28)	0 (0 to 0)
Other races/ethnicities	<5 µg/dL	97.43 (95.98 to 98.87)	99.83 (99.58 to 100.07)	98.71 (97.58 to 99.85)	100 (100 to 100)
	5–9.9 µg/dL	2.19 (0.82 to 3.55)	0.17 (–0.07 to 0.42)	0.89 (0.05 to 1.74)	0 (0 to 0)
	≥10 µg/dL	0.39 (0 to 0.77)	0 (0 to 0)	0.39 (–0.38 to 1.16)	0 (0 to 0)

Table 5 Adjusted geometric means with 95 % confidence intervals for blood lead in micrograms per deciliter by age, race/ethnicity, exposure to secondhand smoke (SHS), smoking status, and residential status. Data from National Health and Nutrition Examination Survey 2003–2012

	AGM (95 % CI)	Statistically significant differences
Gender		
Male (M)	1.14 (0.91–1.43)	M > F ($p < 0.01$)
Female (F)	0.95 (0.79–1.13)	
Race/ethnicity		
Non-Hispanic White (NHW)	0.96 (0.75–1.21)	NHW < NHB ($p = 0.03$)
Non-Hispanic Black (NHB)	1.15 (1.03–1.28)	
Mexican American (MA)	1 (0.68–1.46)	NHB > OTH ($p < 0.01$)
Other races/ethnicities (OTH)	1.06 (0.98–1.15)	
Age and smoking status		
1–5 years (Y15)	1.46 (1.2–1.79)	Y15 > Y611 > Y1219NSM > Y1219SM ($p \leq 0.02$)
6–11 years (Y611)	1.04 (0.84–1.28)	
12–19 years, nonsmokers (Y1219NSM)	0.82 (0.65–1.04)	
12–19 years, smokers (Y1219SM)	0.94 (0.8–1.1)	
Exposure to SHS		
Not exposed to SHS (NoSHS)	1 (0.91–1.1)	OWN < RENT ($p = 0.02$)
Exposed to SHS (SHS)	1.08 (0.8–1.47)	
Residential status		
Owned (OWN)	1.02 (0.87–1.2)	OWN < RENT ($p = 0.02$)
Renting (RENT)	1.07 (0.89–1.28)	
Other arrangements (OA)	1.03 (0.79–1.33)	

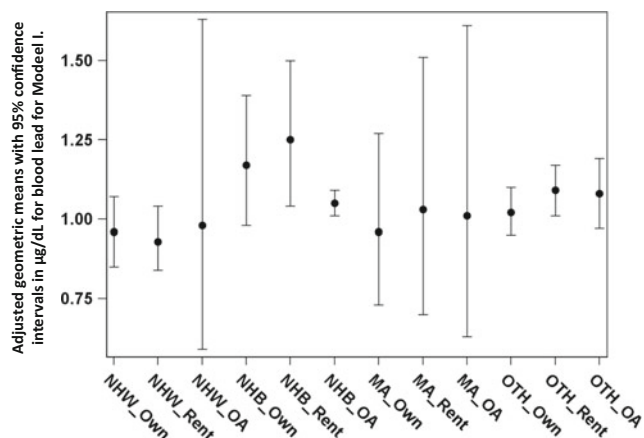


Fig. 1 Adjusted geometric means with 95 % confidence intervals for blood lead in micrograms per deciliter by race/ethnicity (NHW=non-Hispanic White, NHB=non-Hispanic Black, MA=Mexican American, OTH= other races/ethnicities) and residential living housing status (Own = owned, Rent=renting, OA= other living arrangements like living with someone else)

the latter start going to primary school. But then, children aged 6–11 years may be exposed to Pb outside the home, for example, at school, in the playground, and even in the absence of lead in gasoline, while traveling in the school bus or private transportation, possibly because of exposure to tobacco smoke. Consequently, it is hard to make a definitive conclusion that younger children are exposed to higher levels of Pb than older children. As such, the possibility that younger children excrete Pb more slowly than older children do cannot be ruled out but more research will be needed in this area.

Adolescents who enter middle school and high school do not spend much time at home, but a whole lot more time is spent in the outside environment. This outside environment provides them substantial opportunities to be exposed to Pb, for example, when driving, traveling for pleasure, participating in entertainment activities like shopping, and joining clubs and sororities where there are ample opportunities to engage in alcohol use and abuse and even recreational drug use. But, in the absence of any exposure data, it is not possible to make a statement that the sum total of exposure to Pb for adolescents is any less or more than it is

for children. Consequently, the possibility that adolescents may be excreting Pb faster than children cannot be ruled out. Higher levels of BLLs among adolescent smokers when compared with nonsmokers are in concordance with the results for adults provided by Jain (2013b) and Hu et al. (2014), among others.

A higher number of smokers inside the home was associated with higher adjusted BLLs ($\beta=0.025, p=0.02$, Table 6). When the number of smokers inside the home was controlled for, neither the number of cigarettes smoked in the home ($p=0.28$, Table 6) nor the report of secondhand smoke was associated with BLLs ($p>0.05$). These findings suggest that the number of smokers inside the home is a more reliable measure than either the number of cigarettes or the report of exposure to SHS.

While overall, 3.14 % children aged 1–5 years had BLLs $\geq 5 \mu\text{g/dL}$, 7.8 % NHB children aged 1–5 years had had BLLs $\geq 5 \mu\text{g/dL}$. Overall, NHW had statistically significantly lower BLLs than NHB which is in conformity with what has been reported by other authors (Jain, 2013b, Hu et al., 2014, Richter et al., 2013).

Living in an owned home vs. a rented home was found to be associated with lower BLLs because a rented home is more likely to be older with leaded paint still in use, with lead pipes still carrying water into the house, and with greater likelihood of coming in contact with lead-infected dirt and dust. NHANES used to provide data on the age of the homes until the 2005–2006 cycle but these data are no longer available, and as such, the age of the residence could not be used as a variable in regression models. As has also been reported by Hu et al. (2014), the poverty income ratio used as a surrogate measure of socioeconomic status was found to be inversely associated with BLLs. This may be because low SES (and as such, low poverty income ratio) is associated with living in a rented house, older house, apartment building, and residential area with a greater likelihood of providing exposure to Pb.

It should be noted that there may be variability in BLLs by individual ages within each age group, for example, 1–5 years. It has been shown that 1–2-year-olds have higher BLLs than 3–5-year-olds, using NHANES data (Jones et al., 2009,

Table 6 Regression slopes with associated standard errors and p values for log10 transformed values of blood lead measured in nanograms per deciliter for various continuous variables used as independent variables. Data from National Health and Nutrition Examination Survey 2003–2012

Independent variable	Regression slopes with p values			Change in blood levels in ng/dL per unit change
	β	SE	p	
No. of rooms in the house	0.00301	0.000907007	0.08	1.01
No. of cigarettes smoked inside home every day	0.00043	0.000294002	0.28	1.00
No. of cigarette smokers smoking inside home	0.02496	0.00379466	0.02	1.06
Poverty income ratio	-0.02916	0.000427111	<0.01	-1.07
NHANES year	-0.05632	0.007235729	0.02	-1.14
R^2	32.50 %			

Wheeler, 2013). For NHANES 2007–2010, the unadjusted geometric mean for 1–2-year-olds was reported to be 1.5 and 1.2 $\mu\text{g}/\text{dL}$ for 3–5-year-olds. In a sub-analysis of the data used for this study, the unadjusted geometric mean for 1–2-year-olds was found to be 1.55 (1.49–1.62) $\mu\text{g}/\text{dL}$ and 1.26 (1.20–1.32) $\mu\text{g}/\text{dL}$ for 3–5-year-olds and the adjusted geometric means for those aged 1, 2, 3, 4, and 5 years were found to be 1.70 (1.63–1.78), 1.59 (1.51–1.67), 1.43 (1.32–1.54), 1.39 (1.31–1.46), and 1.25 (1.18–1.32) $\mu\text{g}/\text{dL}$, respectively. Thus, there may be a downward trend in BLLs for the age group 1–5 years. Whether this age-based downward trend is due to a downward trend in exposure to Pb or due to age-based metabolic differences should be a subject for future study. Similar trends within the age groups 6–11 and 12–19 may be possible but, to the best of my knowledge, have not been studied.

In summary, (i) children aged 1–5 years had higher BLLs than children aged 6–11 years and both smoker and nonsmoker adolescents aged 12–19 years, (ii) the percent non-Hispanic Black children aged 1–5 years with $\text{BLL} > 5 \mu\text{g}/\text{dL}$ was 7.8 %, (iii) adjusted levels of Pb declined over 2003–2012, (iv) BLLs were negatively associated with poverty income ratio, and (v) males had statistically significantly higher BLLs than females.

The limitations of this study should be carefully considered. This study was based on cross-sectional data. Neither the magnitude nor the source of exposure to Pb was available. The timing of the exposure was also not available. A relatively long-term follow-up study (or maybe a short-term well-designed study) that can keep track of the ongoing Pb exposure and observed BLLs may better be able to evaluate the association between Pb exposure and other relevant factors.

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Compliance with ethical standards

Conflict of interest The author declares that he has no competing interests.

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