

Are Blood Lead Levels in the United States Still Declining? The US National Health Nutrition and Examination Survey (NHANES) 1999-2016

Kelvin KW Lui

University of Hong Kong

Man-Fung Tsoi

University of Hong Kong <https://orcid.org/0000-0002-9892-0125>

Tommy Tsang Cheung

University of Hong Kong

Ching-Lung Cheung

University of Hong Kong

Bernard MY Cheung (✉ mycheung@hku.hk)

Department of Medicine, The University of Hong Kong, Hong Kong, China <https://orcid.org/0000-0001-9106-7363>

Research

Keywords: blood lead level, United States, NHANES

Posted Date: March 17th, 2020

DOI: <https://doi.org/10.21203/rs.3.rs-17481/v1>

License: © ⓘ This work is licensed under a Creative Commons Attribution 4.0 International License. [Read Full License](#)

Abstract

Background: Lead is toxic without a safe limit. The current upper reference blood lead level (BLL), 5 µg/dL, came from the 97.5th percentile in children aged 1-5 years in NHANES 2007-2010.

Objectives: We studied the latest trend in BLL in US NHANES and estimated the proportion of children with BLL ≥5 µg/dL, which would inform the setting of an upper reference level.

Methods: We analyzed 68877 participants (aged 1 to 85 years) with BLL measurements in NHANES 1999-2016 using SPSS complex sample module v25.0.

Results: In NHANES 2011-2012, 2013-2014, and 2015-2016, the mean and 95% confidence intervals (CIs) of BLLs (µg/dL) were 0.97 (0.96, 0.99), 0.86 (0.85, 0.87), and 0.82 (0.81, 0.83), respectively ($P < 0.0001$). The estimated proportion (95% CI) of children aged 1-5 years with elevated BLL (EBLL) in 2011-2012, 2013-2014, and 2015-2016 were 2.0% (1.3, 3.0), 0.5% (0.4, 0.7), and 1.3% (0.8, 2.3), respectively ($P = 0.267$). In 2015-2016, the proportion of children with EBLL was similar in high- and low-income groups ($P = 0.9979$). The estimated 97.5th percentile of BLL in children was 3.71 µg/dL in NHANES 2015-2016.

Conclusions: BLL continued to decline in the overall US population. The disparity in BLL in children from higher and lower income families has decreased. Our findings support a reduction in the reference BLL, continual monitoring of population BLL and continual efforts to reduce environmental exposure to lead.

Introduction

Lead is a well-documented environmental toxin. Environmental lead exposure is mainly from water pipe, soil, paint, and gasoline. Several mechanisms of lead poisoning have been proposed, including enzyme inhibition, oxidative stress, inflammation and epigenetic modifications [1, 2]. In adults, lead can affect multiple organ systems. A recent systemic review and meta-analysis has shown that lead exposure is associated with increased risk of cardiovascular disease, coronary heart disease, and stroke [3]. Even at low blood lead levels (BLLs), blood pressure and other cardiovascular outcomes are linked to lead exposure [4, 5]. Lead in blood is also related to renal dysfunction [6–8] and has an additive effect with hypertension [9]. Lead toxicity in children is particularly worrying as they suffer from more serious consequences. In children, high BLLs is a risk factor for cognitive deficits [10–13] and attention-deficit hyperactivity disorder [14, 15]; and chelation therapy seems to be unable to correct the damage [16]. For this reason, efforts have been focusing on reducing environmental exposure to lead through monitoring and regulation.

In 2012, the US Centers for Disease Control and Prevention (CDC) set, as the upper reference BLL, the 97.5th percentile of BLLs in children aged 1 to 5 years in the National Health Nutrition and Examination Survey (NHANES) 2007-2010 [17]. This reference limit of 5 µg/dL has been adopted by the US and many other places to identify children with elevated BLLs. However, a recent US population-based cohort study suggests that even among individuals with BLLs lower than 5 µg/dL, an increase of BLL from 1.0 µg/dL to 5.0 µg/dL was associated with 38% increase in all-cause mortality, almost two-fold increase in cardiovascular disease mortality, and more than 150% increase in mortality related to ischemic heart disease [18]. This finding has provided another important piece of evidence to support the notion that there is no safe limit of BLL and there is no room for complacency [19].

However, 132 (79%) schools and 95 (13%) childcare providers in Vermont had elevated tap water lead level²⁰. The testing of this water lead level is conducted in accordance to Act 66 which requires all Vermont schools, supervisory unions, independent schools and childcare providers to measure water lead level²¹. The government provided the resources to replace the affected water pipes and taps. All affected water taps are not allowed to use²⁰. In the current study, we aim to reveal the latest trend in BLLs in the United States by analyzing data from NHANES 1999-2016 on BLL in the US population and estimate the proportion of children with elevated BLL, defined as ≥5 µg/dL, which would inform the setting of a new upper reference level.

Methods

NHANES is conducted by the National Center for Health Statistics (NCHS) of the CDC. The survey uses a complex multistage probability design. The main feature of this survey is that it involves both household questionnaires and medical examination for a representative sample of the US civilian non-institutionalized population. Approximately 5,000 participants were examined annually and each represents about 50,000 Americans. Oversampling of Mexican Americans, Hispanics and non-Hispanic blacks, Asians, elderly and low-income white was done in order to increase the reliability of subgroup estimates [20].

Out of 77225 participants in NHANES 1999-2016, 8348 (10.8%) participants with missing BLL were excluded. The final dataset included 68877 participants.

Collection of venous whole blood samples and measurements of BLLs were carried out under standard procedures. Specimens were stored at -30°C before being shipped to the National Center for Environmental Health of CDC, where they were analyzed using Inductively Coupled Plasma Mass Spectrometer (ICP-MS) with Dynamic Reaction Cell Technology (ELAN® DRC II) (PerkinElmer Norwalk, CT) [21]. The limits of detection (LOD) of blood lead were 0.6 µg/dL, 0.25 µg/dL and 0.07 µg/dL in 1999-2002, 2003-2012, and 2013-2016 respectively. For BLLs below the LOD, an imputed value of LOD/√2 was assigned prior to analysis.

Statistical Analysis

The model we used accounted for the stratification and clustering of the complex sampling design of NHANES. Sample weights were used to adjust for biases arising from oversampling, differential probability of selection, non-response, and post-stratification. BLLs were natural logarithm-transformed to correct for the skewness in the data. Estimates were adjusted for age, gender, and ethnicity.

The estimated proportions of pregnant women and children 1-5 years old with elevated BLL (EBLL) (defined as ≥ 5 µg/dL), and other categorical variables were presented as percentages with 95% confidence intervals (CIs). BLLs were expressed as geometric means and 95% CIs. Results were analyzed by socio-demographic subgroups, e.g. age, gender, ethnicity, poverty income ratio (PIR). The trend of mean BLLs and proportions of EBLLs in different subgroups across the survey years were assessed by multiple regression. Comparisons of mean BLLs between subgroups in the same survey year were conducted with analysis of variance (ANOVA) or 2-tailed Student's *t*-test. A 2-sided *P*-value of <0.05 was considered as statistically significant. The estimated 97.5th percentile of BLL in children 1-5 years old in NHANES 2015-2016 was also computed. All statistical analyses were conducted using SPSS 25.0 complex sample module (IBM Corp., Armonk, NY).

Results

Socio-demographic characteristics of participants in NHANES 1999-2016 included in the current study are summarized in Table 1. Overall, there was a continual decline in BLLs across the years, from mean (95% CI) 1.66 (1.60, 1.72) µg/dL in 1999-2000 to 0.82 (0.78, 0.87) µg/dL in 2015-2016 ($P < 0.0001$). The decline was substantial and statistically significant in all age groups ($P < 0.0001$) (Figure 1) and in both males and females of all age groups ($P < 0.001$) (Online Supplementary Table 1). This was also observed in all ethnic/racial groups reported ($P < 0.0001$) (Online Supplementary Table 2) apart from the 'Other Non-Hispanic' ethnic groups (Non-Hispanic Asian and Other race – including multi-racial, $P = 0.0550$ and $P = 0.1573$, respectively), which were subdivided from 'Other Race' since NHANES 2011-2012. Likewise, there was a gradual decrease in the proportion of the US population with EBLL regardless of age, gender, ethnicity, and poverty income ratio (Online Supplementary Table 3 and 4). There was a significant increase in the proportion of females and Mexican Americans with EBLLs between 2013-2014 and 2015-2016 (0.3% vs 0.6% ($P < 0.01$) and 0.9% vs 1.7% ($P < 0.001$), respectively).

Figure 2 shows the trend in the proportion of children 1-5 years old with EBLL in NHANES 1999-2016. The proportion of children 1-5 years old with EBLLs decreased steadily across the years, from 9.7% (95%CI: 7.2, 12.8) in 1999-2000 to 1.3% (95% CI: 0.8, 2.3) in 2015-2016 ($P < 0.0001$). There was no significant increase in the percentage of children with EBLLs in 2015-2016 compared with 2013-2014 (1.3% vs 0.5%, $P = 0.267$). This was also observed in other age groups (Online Supplementary Table 3). The proportion of children aged 1-5 with EBLL declined significantly across the years in both lower-income (PIR <1.3) and higher-income (PIR ≥ 1.3) groups ($P < 0.0001$). A significantly higher proportion of children in the lower-income group had EBLLs than the higher income group each year ($P < 0.001$), but the difference was no longer significant in 2015-2016 ($P = 0.9979$). The estimated 97.5th percentile of BLL in children 1-5 years old, was 3.71 µg/dL in NHANES 2015-2016.

Discussion

Our results show a persistent decline in BLLs in the US from 1999-2000 to 2015-2016 irrespective of age, gender, ethnicity, and poverty-income ratio. The decline is consistent with a number of previous population-based studies [22–28], including our previous study [29]. This has once again demonstrated the effectiveness of public health efforts to abate environmental exposure to lead in the US. Nevertheless, the rate of decline has been diminishing in recent years, which may indicate that there are other environmental lead sources yet to be identified and controlled.

Despite the substantial decline in BLLs, disparities between different income and ethnic groups remain. From our analysis, we found in NHANES 2015-2016, for the first time, that there was no longer a disparity between high- and low-income families in the proportion of children aged 1 to 5 years with EBLLs. This is a very encouraging finding. Lead affects neurocognitive development in children and BLL may partly lead to a poverty trap in which children from poor families will remain poor when they grow up. The elimination of income disparity in EBLLs is likely to be a result of targeted screening in high-risk children. Until 2014, black children (below age of 18) were twice as likely to live in poverty as white children [30]. Black children in poverty are also more likely to live in substandard housing and hence, at a higher risk of exposure to lead paint at home and lead pollutants from nearby factories [31, 32]. The racial and income disparity has been historically important in blood lead monitoring. In 1982, Mahaffey and colleagues first reported higher BLLs in young children who were black and from low-income families in a national estimate (NHANES II, 1976-1980) [33]. Since then, the observation was confirmed in numerous studies and the disparities persisted over the past four

decades [22–4, 27, 33–36]. In 2013-2014, the mean BLLs in black children was still 36% higher than in white children [29]. Children at the age of 1-5 years that were non-Hispanic black, from a low-income family and living in housing built before 1950 were in the highest risk groups for lead poisoning [24, 34, 35]. The black-white disparity in higher BLLs has been found to be independent of income level, housing quality and environmental conditions [38]. Targeted prevention strategies have been implemented such as increased screening on these high-risk groups and identification of high-risk environment [17]. the percentage of non-Hispanic black children with EBLL is still higher compared to other ethnicities, and so more efforts to reduce the environmental exposure to lead, particularly by removing lead paint and lead plumbing in houses, are necessary.

The proportion of EBLL among children aged 1-5 years in NHANES 2015-2016 increased numerically, albeit insignificantly. This should be interpreted with caution. 30.9% of these participants had missing blood lead measurements. The sample size was 790 and only 12 children had EBLL. Caldwell et al. [39] reported an estimated 35% of lot screening failures due to lead contamination, which could falsely elevate BLL in the NHANES 2015-2016 cycle. However, a study based on a large national clinical laboratory database [23] reported a slight increase in the rate of children <6 years old with EBLLs in 2014-2015 after consecutive years of decline. Our analysis of NHANES 2015-2016 data does not dispel this disturbing finding. These may just be random fluctuations along an overall decreasing trend, so the next two-year NHANES cycle would clarify the underlying trend better. Continual monitoring of BLL is thus as critical as ever. However, as BLL in the general population and in healthy people becomes lower and lower, advances in analytical methods are needed. This is a reason why the CDC is cautious about lowering the reference level of BLL further to a level where measurements become inaccurate.

Since the well-publicized crisis of lead in drinking water in Flint, Michigan, there have been increased awareness and testing of lead level in drinking water. Recently in Vermont, elevated tap water lead level was found. This is potentially harmful to children. Children absorb 40-50% of water-soluble lead⁴⁰. Every increase in 1 ppb in water lead level increases 35% in BLL⁴¹. The effect of water lead level on BLL is well demonstrated in Flint, Michigan. The switching of water source caused a significant increase in EBLL in children^{42,43}. The switching back for the source of tap water reduced the BLL^{44,45}.

Although we analyzed the same dataset from which US reference levels of BLL are derived, there are limitations to our analysis. There were high rates of missing blood lead measurements particularly among children aged 1-5 years in NHANES 1999-2016 (ranging from 24-38%), which may affect the representativeness of the survey to estimate the BLL of children in the population. While the sampling in US NHANES was random, not all Americans could be included in the sampling frame. Institutionalized, people without fixed addresses and people who refused were not included. As with any analysis of subgroups, power is reduced and confidence intervals are wider, and multiple comparisons are possible. Our new findings in NHANES 2015-2016 generate a new hypothesis that requires confirmation in future cycles of NHANES and other national estimates of BLL.

In conclusion, our latest analysis of BLL in US NHANES showed that BLL continued to decline overall in the US population. The disparity in BLL in children in high and low- income households has diminished. Black children still have higher BLLs than white children. In young children aged 1 to 5 years in the 2015-2016 survey, BLL did not decline and appeared to increase. Our data suggest that monitoring the trend in BLL in the population is as necessary as ever and that efforts to reduce environmental exposure to lead must not be relaxed.

Declarations

Ethical Approval and Consent to participate

All participants gave informed consent before participation and ethics approval of the study was granted by the Research Ethics Review Board at the National Center for Health Statistics of the Centers for Disease Control and Prevention, USA.

Consent for publication

Not applicable

Availability of supporting data

Data used in this study can be downloaded from: <https://wwwn.cdc.gov/nchs/nhanes/Default.aspx>

Competing interests

BMYC reports personal fees from Amgen, Pfizer and Roche, outside the submitted work.

The remaining authors declare no potential conflicts of interest.

Funding

No specific funding was received for this work.

Authors' contributions

BMJ Cheung, KKWL and MFT designed the study. KKWL and MFT performed the data analysis. KKWL wrote the first draft of the manuscript. All authors contributed to the interpretation of results. The corresponding author had full access to the data and had final responsibility for the decision to submit for publication. All authors read and approved the final manuscript.

Acknowledgements

KWK Lui was awarded a Summer Research Internship Scheme by the Li Ka Shing Faculty of Medicine, the University of Hong Kong.

References

1. Mitra P, Sharma S, Purohit P, Sharma P. Clinical and molecular aspects of lead toxicity: an update. *Crit Rev Clin Lab Sci*. 2017;54(7-8):506-28.
2. Wani AL, Ara A, Usmani JA. Lead toxicity: a review. *Interdiscip Toxicol*. 2015;8(2):55-64.
3. Chowdhury R, Ramond A, O'Keeffe L, Shahzad S, Kunutsor SK, Muka T, et al. Environmental toxic metal contaminants and risk of cardiovascular disease: systematic review and meta-analysis. *BMJ*. 2018;362:p. k3310.
4. Lee KR, Ko KD, Hwang IC, Suh HS, Kim KK. Association between blood lead levels and blood pressures in a non-smoking healthy Korean population. *Postgrad Med J*. 2017;93(1103):513-8.
5. Scinicariello F, Abadin HG, Murray HE. Association of low-level blood lead and blood pressure in NHANES 1999-2006. *Environ Res*. 2011;111(8):1249-57.
6. Harari F, Sallsten G, Christensson A, Petkovic M, Hedblad B, Forsgard N, et al. Blood lead levels and decreased kidney function in a population-based cohort. *Am J Kidney Dis*. 2018;72(3):381-9.
7. Fadrowski JJ, Navas-Acien A, Tellez-Plaza M, Guallar E, Weaver VM, Furth SL. Blood lead level and kidney function in US adolescents: the third National Health and Nutrition Examination Survey. *Arch Intern Med*. 2010;170(1):75-82.
8. Muntner P, He J, Vupputuri S, Coresh J, Batuman V. Blood lead and chronic kidney disease in the general United States population: results from NHANES III. *Kidney Int*. 2003;63(3):1044-50.
9. Wang X, Liang H, Wang Y, Cai C, Li J, Li X, et al. Risk factors of renal dysfunction and their interaction in level-low lead exposure paint workers. *BMC Public Health*. 2018;18(1):526.
10. Sobolewski M, Varma G, Adams B, Anderson DW, Schneider JS, Cory-Slechta DA. Developmental lead exposure and prenatal stress result in sex-specific reprogramming of adult stress physiology and epigenetic profiles in brain. *Toxicol Sci*. 2018;163(2):478-89.
11. Reuben A, Caspi A, Belsky DW, Broadbent J, Harrington H, Sugden K, et al. Association of childhood blood lead levels with cognitive function and socioeconomic status at age 38 years and with IQ change and socioeconomic mobility between childhood and adulthood. *JAMA*. 2017;317(12):1244-51.
12. Geier DA, Kern JK, Geier MR. Blood lead levels and learning disabilities: a cross-sectional study of the 2003-2004 National Health and Nutrition Examination Survey (NHANES). *Int J Environ Res Public Health*. 2017;14(10):1202.
13. Canfield RL, Henderson CR, Cory-Slechta DA, Cox C, Jusko TA, Lanphear BP. Intellectual impairment in children with blood lead concentrations below 10 µg per deciliter. *N Engl J Med*. 2003;348(16):1517-26.
14. He J, Ning H, Huang R. Low blood lead levels and attention-deficit hyperactivity disorder in children: a systematic review and meta-analysis. *Environ Sci Pollut Res Int*. 2017; <http://doi.org/10.1007/s11356-017-9799-2>.
15. Nigg JT, Nikolas M, Mark K, Knottnerus G, Cavanagh K, Friderici K. Confirmation and extension of association of blood lead with attention-deficit/hyperactivity disorder (ADHD) and ADHD symptom domains at population-typical exposure levels. *J Child Psychol Psychiatry*. 2010;51(1):58-65.
16. Rogan WJ, Dietrich KN, Ware JH, Dockery D, Salganik M, Radcliffe J et al. The effect of chelation therapy with succimer on neuropsychological development in children exposed to lead. *N Engl J Med*. 2001;344(19):1421-6.
17. Centers for Disease Control and Prevention. CDC response to Advisory Committee on Childhood Lead Poisoning Prevention recommendations in "Low level lead exposure harms children: a renewed call of primary prevention". 2012. https://www.cdc.gov/nceh/lead/acclpp/cdc_response_lead_exposure_recs.pdf. Accessed Aug 5 2018.

18. Lanphear BP, Rauch S, Auinger P, Allen RW, Hornung RW. Low-level lead exposure and mortality in US adults: a population-based cohort study. *The Lancet Public Health*. 2018;3(4):e177-e184.
19. Cheung BMY, Cheung TT. No lead is better than a little lead. *Postgrad Med J*. 2017;93(1103):512.
20. State of Vermont. Lead in School and Child Care Drinking Water Results <https://leadresults.vermont.gov/summary>. Accessed Feb 2020.
21. Act 66, Vermont government. <https://legislature.vermont.gov/Documents/2020/Docs/ACTS/ACT066/ACT066%20As%20Enacted.pdf> Accessed Feb 2020.
22. Johnson CL, Dohrmann SM, Burt VL, Mohadjer LK. National Health and Nutrition Examination Survey: sample design, 2011-2014. *Vital Health Stat*. 2014; 2(162):1-33.
23. Centers for Disease Control and Prevention. NHANES 2015-2016 laboratory procedure manual: blood metals panel in whole blood. 2018. https://wwwn.cdc.gov/nchs/data/nhanes/2015-2016/labmethods/PBCD_I_met.pdf. Accessed July 27 2018.
24. Jain RB. Trends and variability in blood lead concentrations among US adults aged 20-64 years and senior citizens aged ≥ 65 years. *Environ Sci Pollut Res Int*. 2016;23(14):14056-67.
25. McClure LF, Niles JK, Kaufman HW. Blood lead levels in young children: US, 2009-2015. *J Pediatr*. 2016;175:173-81.
26. Jones RL, Homa DM, Meyer PA, et al. Trends in blood lead levels and blood lead testing among US children aged 1 to 5 years, 1988-2004. *Pediatrics*. 2009;123(3):e376-85.
27. Wheeler W, Brown MJ. Blood lead levels in children aged 1-5 years - United States, 1999-2010. *MMWR Morb Mortal Wkly Rep*. 2013;62(13):245-8.
28. Raymond J, Brown MJ. Childhood blood lead levels in children aged <5 years - United States, 2009-2014. *MMWR Surveill Summ*. 2017;66(3):1-10.
29. Watson CV, Lewin M, Ragin-Wilson A, Jones R, Jarrett JM, Wallon K, Ward C, Hiliard N, Irvin-Barnwell E. Characterization of trace elements exposure in pregnant women in the United States, NHANES 1999–2016. *Environ Res*. 2020;183:109208.
30. Ettinger AS, Egan KB, Homa DM, Brown MJ. Blood Lead Levels in U.S. Women of Childbearing Age, 1976-2016. *Environ Health Perspect*. 2020;128(1):17012.
31. Tsoi MF, Cheung CL, Cheung TT, Cheung BMY. Continual decrease in blood lead level in Americans: United States National Health Nutrition and Examination Survey 1999-2014. *Am J Med*. 2016;129(11):1213-8.
32. Health, United States, 2015: with special feature on racial and ethnic health disparities. Hyattsville (MD): National Center for Health Statistics (US). 2016. <https://www.cdc.gov/nchs/data/hs/hs15.pdf>. Accessed 5 August 2018.
33. Wengrovitz AM, Brown MJ. Recommendations for blood lead screening of Medicaid-eligible children aged 1-5 years: an updated approach to targeting a group at high risk. *MMWR Recomm Rep*. 2009;58(Rr-9):1-11.
34. Raymond J, Wheeler W, Brown MJ. Inadequate and unhealthy housing, 2007 and 2009. *MMWR Suppl*. 2011;60(1):21-7.
35. Mahaffey KR, Annett JL, Roberts J, Murphy RS. National estimates of blood lead levels: United States, 1976-1980: association with selected demographic and socioeconomic factors. *N Engl J Med*. 1982;307(10):573-9.
36. Pirkle JL, Brody DJ, Gunter EW, Kramer RA, Paschai DC, Flegal KM, et al. The decline in blood lead levels in the United States: the National Health and Nutrition Examination Surveys (NHANES). *JAMA*. 1994;272(4):284-91.
37. Bernard SM, McGeehin MA. Prevalence of blood lead levels ≥ 5 $\mu\text{g}/\text{dL}$ among US children 1 to 5 years of age and socioeconomic and demographic factors associated with blood of lead levels 5 to 10 $\mu\text{g}/\text{dL}$, third National Health and Nutrition Examination Survey, 1988–1994. *Pediatrics*. 2003;112(6):1308-13.
38. Meyer PA, Pivetz T, Dignam TA, Homa DM, Schoonover J, Brody D. Surveillance for elevated blood lead levels among children—United States, 1997-2001. *MMWR Surveill Summ*. 2003;52(10):1-21.
39. Richter PA, Bishop EE, Wang J, Kaufmann R. Trends in tobacco smoke exposure and blood lead levels among youths and adults in the United States: the National Health and Nutrition Examination Survey, 1999-2008. *Prev Chronic Dis*. 2013;10:E213.
40. Sampson RJ, Winter AS. The racial ecology of lead poisoning. *Du Bois Review: Social Science Research on Race* 2016; 13(02): 261-83.
41. Caldwell KL, Cheng P-Y, Jarrett JM, Makhmudov A, Vance K, Ward CD, et al. Measurement challenges at low blood lead levels. *Pediatrics* 2017;140(2):e20170272.
42. Toxicological profile for lead. US Department of Health and Human Services, Public Health Service, Agency for Toxic Substances and Diseases Registry. 2007. Available at: <http://www.atsdr.cdc.gov/toxprofiles/tp13.pdf>. Accessed 26 Feb 2020.
43. Ngueta G, Abdous B, Tardif R, St-Laurent J, Levallois P. Use of a Cumulative Exposure Index to Estimate the Impact of Tap Water Lead Concentration on Blood Lead Levels in 1- to 5-Year-Old Children (Montréal, Canada). *Environ Health Perspect*. 2016;124(3): 388–395.

44. Hanna-Attisha M, LaChance J, Sadler RC, Schnepf AC. Elevated Blood Lead Levels in Children Associated With the Flint Drinking Water Crisis: A Spatial Analysis of Risk and Public Health Response. *Am J Public Health*. 2016;106(2): 283–290.
45. Kennedy C, Yard E, Dignam T, et al. Blood Lead Levels Among Children Aged <6 Years — Flint, Michigan, 2013–2016. *MMWR Morb Mortal Wkly Rep* 2016;65(25):650-4.
46. Zahran S, McElmurry SP, Sadler RC. Four Phases of the Flint Water Crisis: Evidence from Blood Lead Levels in Children. *Environ Res*. 2017;157:160-172
47. Gómez HF, Borgialli DA, Sharman M, Shah KK, Scolpino AJ, Oleske JM, Bogden JD. Blood Lead Levels of Children in Flint, Michigan: 2006–2016. *J Pediatr*. 2018;197:158-164.

Tables

Table 1 Characteristics of Participants Included in Analysis

Year	1999-2000	2001-2002	2003-2004	2005-2006	2007-2008	2009-2010	2011-2012	2013-2014	2015-2016	<i>P</i>
N	7970	8945	8373	8407	8266	8793	7920	5215	4988	
Age, yr	36.2 ± 0.39	36.7 ± 0.63	37.5 ± 0.39	37.9 ± 0.77	38.5 ± 0.44	38.7 ± 0.48	39.1 ± 0.78	39.1 ± 0.49	39.8 ± 0.73	0.7463
Female (%)	4057 (50.9%)	4606 (51.5%)	4241 (50.7%)	4315 (51.3%)	4119 (49.8%)	4427 (50.3%)	3952 (49.9%)	2628 (50.4%)	2500 (50.1%)	0.9972
No. of children aged <20 yr	3763 (47.2%)	4173 (46.7%)	3848 (46.0%)	3898 (46.4%)	2902 (35.1%)	3028 (34.4%)	2890 (36.5%)	2520 (48.3%)	2378 (47.7%)	0.0561
Ethnicity										
Mexican Americans (%)	2742 (34.4%)	2268 (25.4%)	2085 (24.9%)	2236 (26.6%)	1712 (20.7%)	1966 (22.4%)	1077 (13.6%)	969 (18.6%)	994 (19.9%)	
Other Hispanics (%)	471 (5.9%)	403 (4.5%)	274 (3.3%)	277 (3.3%)	980 (11.9%)	949 (10.8%)	854 (10.8%)	512 (9.8%)	670 (13.4%)	
Non-Hispanic White (%)	2670 (33.5%)	3768 (42.1%)	3436 (41.0%)	3310 (39.4%)	3461 (41.9%)	3760 (42.8%)	2493 (31.5%)	1848 (35.4%)	1511 (30.3%)	
Non-Hispanic Black (%)	1807 (22.7%)	2174 (24.3%)	2225 (26.6%)	2193 (26.1%)	1746 (21.1%)	1593 (18.1%)	2195 (27.7%)	1119 (21.5%)	1070 (21.5%)	0.9999
Others (%)	280 (3.5%)	332 (3.7%)	353 (4.2%)	391 (4.7%)	367 (4.4%)	525 (6.0%)	1301 (16.4%)	767 (14.7%)	743 (14.9%)	
Non-Hispanic Asian (%)							1005 (12.7%)	510 (9.8%)	479 (9.6%)	
Other race, including multiracial (%)							296 (3.7%)	257 (4.9%)	264 (5.3%)	
Pregnancy (%)	267 (3.4%)	322 (3.6%)	256 (3.1%)	353 (4.2%)	50 (0.6%)	65 (0.7%)	51 (0.6%)	28 (0.5%)	24 (0.5%)	<0.0001
Mean blood lead level (µg/dL)	1.66 (1.60, 1.72)	1.46 (1.41, 1.51)	1.43 (1.37, 1.50)	1.29 (1.23, 1.36)	1.27 (1.22, 1.33)	1.12 (1.08, 1.16)	0.97 (0.92, 1.03)	0.86 (0.82, 0.90)	0.82 (0.78, 0.87)	<0.0001

Table 2 Estimated Proportion of Pregnant Women and Children Aged 1 to 5 Years of Different Ethnicities and Poverty Income Ratios with Elevated Blood Lead Level in 1999-2016

Year	1999-2000	2001-2002	2003-2004	2005-2006	2007-2008	2009-2010	2011-2012	2013-2014	2015-2016	P
Pregnant women	0.5% (0.5, 0.6)	0.2% (0.0, 1.2)	0.9% (0.1, 6.0)	0.0%	0.0%	0.0%	0.0%	0.0%	13.1% (2.0, 52.1)	0.0002
Children aged 1-5 yr										
Stratified by genders										
Male	8.8% (5.2, 14.5)	9.2% (6.8, 12.2)	4.4% (3.1, 6.2)	3.5% (2.3, 5.1)	3.2% (2.4, 4.3)	1.8% (1.2, 2.9)	2.8% (1.7, 4.6)	0.7% (0.5, 1.0)	1.3% (0.5, 3.1)	<0.0001
Female	10.7% (7.7, 14.6)	5.7% (4.0, 8.0)	6.4% (4.5, 8.9)	2.3% (1.5, 3.5)	3.1% (2.3, 4.1)	2.4% (1.6, 3.6)	1.1% (0.4, 2.7)	0.4% (0.3, 0.4)	1.4% (0.7, 3.0)***	<0.0001
Stratified by Ethnicities										
Mexican Americans	10.2% (7.2, 14.3)	4.3% (2.3, 7.9)	3.7% (2.0, 6.8)	1.4% (0.5, 3.8)	1.2% (0.5, 3.2)	2.6% (1.2, 5.6)	0.0%	0.5% (0.1, 3.1)	0.5% (0.1, 3.1)	<0.0001
Other Hispanics	3.2% (1.1, 9.1)	6.6% (2.7, 15.5)	3.8% (0.5, 22.4)	6.9% (2.8, 15.8)	1.0% (0.2, 6.2)	1.7% (0.4, 6.7)	1.1% (0.2, 7.0)	0.7% (0.1, 4.8)	0.0%	0.0686
Non-Hispanic White	9.3% (5.3, 15.7)	5.6% (3.4, 8.9)	2.9% (1.5, 5.4)	1.6% (0.8, 3.3)	3.2% (1.5, 6.3)	1.6% (0.8, 3.0)	2.8% (1.1, 6.8)	0.0%	1.8% (0.7, 4.3)	<0.0001
Non-Hispanic Black	18.6% (13.7, 24.8)	17.9% (13.7, 22.9)	16.2% (11.7, 22.1)	7.8% (5.1, 11.9)	7.1% (4.4, 11.3)	3.8% (2.0, 7.3)	3.2% (1.6, 6.4)	1.6% (0.6, 4.4)	2.2% (0.8, 6.0)	<0.0001
Others	1.5% (0.3, 6.5)	8.8% (2.7, 24.7)	5.5% (1.9, 15.0)	4.0% (1.3, 11.8)	1.2% (0.2, 8.2)	1.7% (0.4, 6.7)	0.0%	1.4% (0.3, 5.8)	0.9% (0.1, 5.9)	0.0373
Non-Hispanic Asian							0.0%	1.5% (0.2, 9.7)	2.7% (0.4, 17.2)	0.5828
Other race, including multiracial							0.0%	1.4% (0.2, 9.2)	0.0%	0.5111
Stratified by poverty income ratio										
< 1.3	11.9% (9.1, 15.3)	13.2% (10.8, 16.2)	8.9% (7.3, 10.9)	7.2% (6.1, 8.6)	4.8% (3.6, 6.2)	4.1% (3.1, 5.5)	3.2% (1.9, 5.4)	0.9% (0.8, 0.9)	1.2% (0.8, 1.7)	<0.0001
≥ 1.3	6.2% (3.9, 9.6)	2.7% (1.5, 4.8)	2.5% (1.5, 4.1)	0.9% (0.4, 1.8)	1.9% (1.7, 2.2)	0.6% (0.3, 1.1)	0.8% (0.8, 0.9)	0.0%	1.2% (0.5, 2.6)	<0.0001
Stratified by age										
1-2 yr	11.8% (8.1, 16.8)	12.1% (8.9, 16.3)	7.7% (5.4, 10.8)	3.6% (2.4, 5.4)	3.4% (2.1, 5.4)	2.9% (1.8, 4.6)	3.3% (1.1, 9.7)	0.9% (0.3, 2.2)	2.6% (1.0, 6.5)	<0.0001
3-5 yr	8.4% (4.7, 14.6)	4.5% (2.5, 7.7)	3.6% (2.0, 6.4)	2.5% (1.6, 3.7)	3.0% (1.3, 6.6)	1.6% (1.0, 2.6)	1.3% (0.4, 4.4)	0.3% (0.1, 1.0)	0.6% (0.2, 1.6)	<0.0001

*** : significant difference (P<0.0001) from 2013-2014

Figures

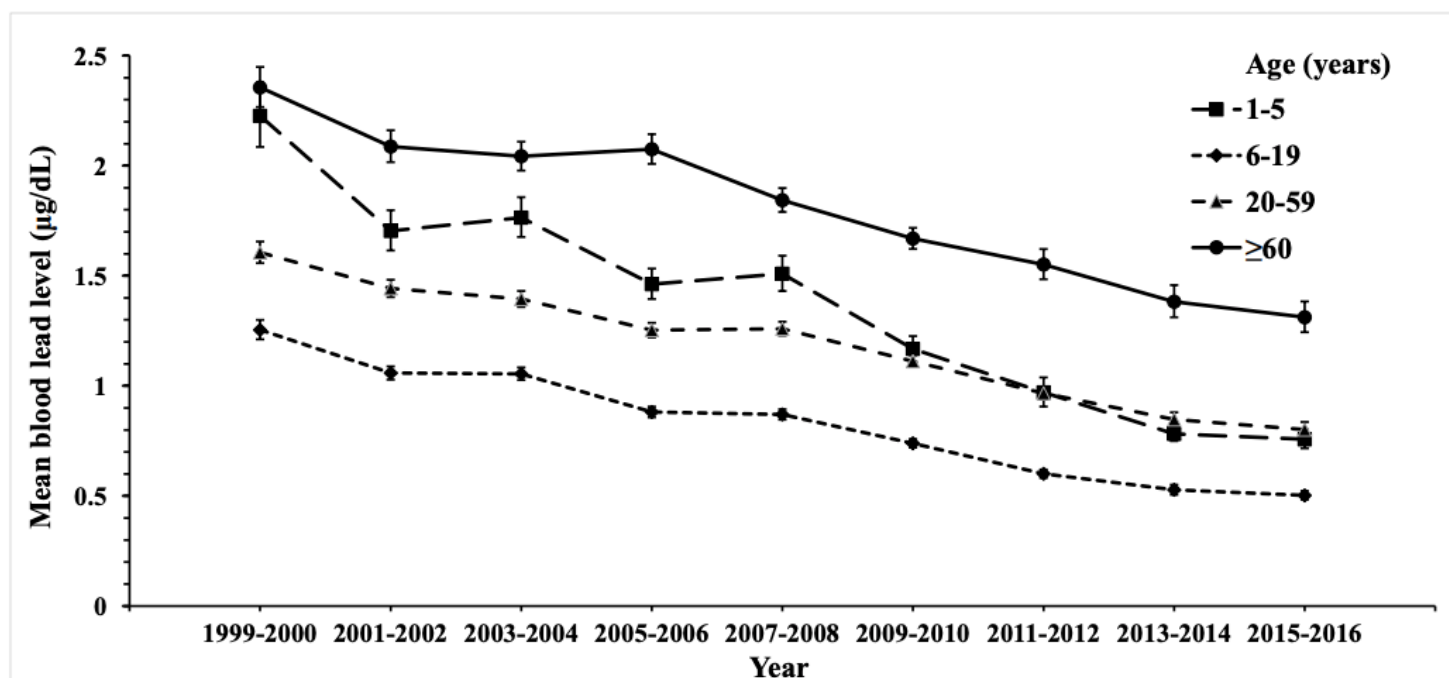


Figure 1

Estimated mean blood lead level in participants of different age groups in 1999-2016

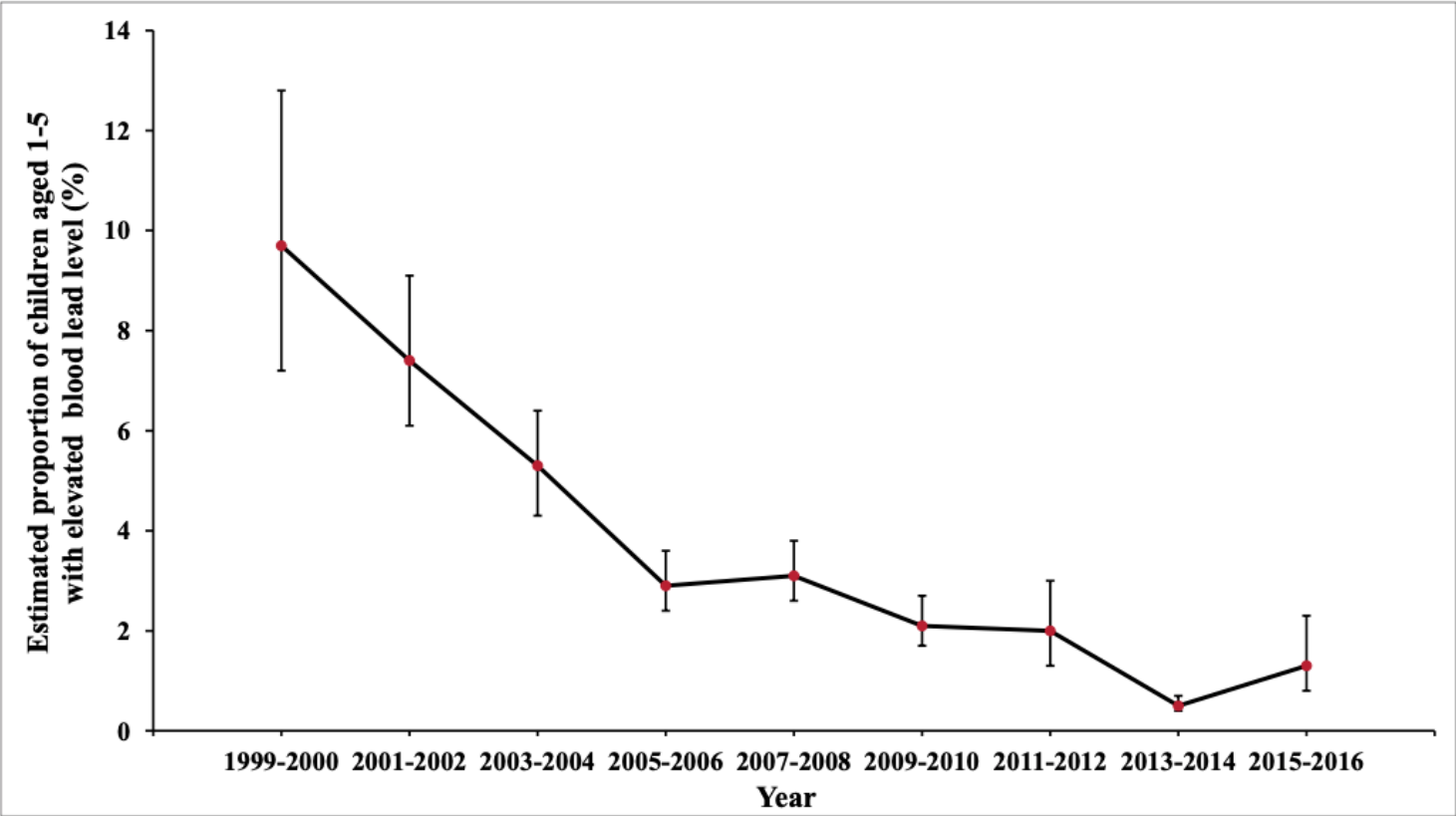


Figure 3

Estimated Proportion of children 1-5 years old with Elevated Blood Lead Level in 1999-2016