



ELSEVIER

Contents lists available at ScienceDirect

Environmental Research

journal homepage: www.elsevier.com/locate/envres

Association between children's blood lead levels, lead service lines, and water disinfection, Washington, DC, 1998–2006

Mary Jean Brown^{a,*}, Jaime Raymond^a, David Homa^a, Chinaro Kennedy^a, Thomas Sinks^b

^a Lead Poisoning Prevention Branch, National Center for Environmental Health, Centers for Disease Control and Prevention, 4770 Buford Highway NE, Atlanta, GA 30348, USA

^b National Center for Environmental Health/Agency for Toxic Substances and Disease Registry, Centers for Disease Control and Prevention, Atlanta, GA, USA

ARTICLE INFO

Article history:

Received 15 June 2009

Received in revised form

1 October 2010

Accepted 7 October 2010

Keywords:

Lead

Lead service line

Childhood lead poisoning

Drinking water

Blood lead level

Elevated blood lead levels

ABSTRACT

Objective: Evaluate the effect of changes in the water disinfection process, and presence of lead service lines (LSLs), on children's blood lead levels (BLLs) in Washington, DC.

Methods: Three cross-sectional analyses examined the relationship of LSL and changes in water disinfectant with BLLs in children < 6 years of age. The study population was derived from the DC Childhood Lead Poisoning Prevention Program blood lead surveillance system of children who were tested and whose blood lead test results were reported to the DC Health Department. The Washington, DC Water and Sewer Authority (WASA) provided information on LSLs. The final study population consisted of 63,854 children with validated addresses.

Results: Controlling for age of housing, LSL was an independent risk factor for BLLs ≥ 10 $\mu\text{g}/\text{dL}$, and ≥ 5 $\mu\text{g}/\text{dL}$ even during time periods when water levels met the US Environmental Protection Agency (EPA) action level of 15 parts per billion (ppb). When chloramine alone was used to disinfect water, the risk for BLL in the highest quartile among children in homes with LSL was greater than when either chlorine or chloramine with orthophosphate was used. For children tested after LSLs in their houses were replaced, those with partially replaced LSL were > 3 times as likely to have BLLs ≥ 10 $\mu\text{g}/\text{dL}$ versus children who never had LSLs.

Conclusions: LSLs were a risk factor for elevated BLLs even when WASA met the EPA water action level. Changes in water disinfection can enhance the effect of LSLs and increase lead exposure. Partially replacing LSLs may not decrease the risk of elevated BLLs associated with LSL exposure.

© 2010 Published by Elsevier Inc.

1. Introduction

The adverse health effects of lead exposure are well known. For children, these include developmental delay, behavior disorders at low lead levels, seizures, and, in rare cases, death at very high levels. The public health impacts of lead exposure are substantial. For example, it is estimated that as BLLs increase from 0 to 10 $\mu\text{g}/\text{dL}$ the fraction of individuals with an IQ > 120 decreases from 9% to 3% (US EPA, 2006a). No blood lead level (BLL) threshold for adverse health effects in children has been identified (Canfield et al., 2003; Lanphear et al., 2000; Bellinger and Needleman, 2003). For adults, BLLs > 1.9 but < 10 $\mu\text{g}/\text{dL}$ have been associated with increased risk for hypertension and increased all-cause mortality (ATSDR, 1999; Menke et al., 2006). BLLs > 75 $\mu\text{g}/\text{dL}$ in adults can cause poor

pregnancy outcomes, intellectual impairment, and death (ATSDR, 1999). In addition, an estimate of hypertension-related risk for serious cardiovascular events indicates that a decrease in BLLs from 10 to 5 $\mu\text{g}/\text{dL}$ could result in an annual decrease of 27 events per 100,000 women and 39 events per 100,000 men (US EPA, 2006a).

Although lead remains a pervasive environmental toxicant, a significant and sustained decrease in BLLs in the United States has been shown, particularly among African American and low-income children. The prevalence of BLLs of ≥ 10 $\mu\text{g}/\text{dL}$ among children 1–5 years of age in the United States has decreased over time. Since 1988 percent of BLLs ≥ 10 $\mu\text{g}/\text{dL}$ has decreased from 8.6% to 1.4% during 1999–2004, an 84% decline (Jones et al., 2009). In the United States, most children with BLLs ≥ 10 $\mu\text{g}/\text{dL}$ have been exposed to residential lead paint hazards in older homes or lead-contaminated house dust and soil (CDC, 2002). However, lead in drinking water is known to contribute to children's BLLs and cases of childhood lead poisoning have been associated with drinking water in the United States (Shannon and Graef, 1989; Cosgrove et al., 1989; CDC, 1994). Before enactment of the Lead and Copper Rule, the US Environmental Protection Agency (US EPA, 1986) estimated that 10–20% of total exposure to lead among the general population might have come from drinking water. The contribution of water

Abbreviations: LSL, Lead service line; WASA, District of Columbia Water and Sewer Authority; BLD, Below the Limit of Detection; BLLs, Blood lead levels; EPA, Environmental Protection Agency; CLPPP, District of Columbia Childhood Lead Poisoning Prevention Program; CDC, Centers for Disease Control and Prevention; DC, District of Columbia; DOH, Department of Health

* Corresponding author. Fax: +1 770 488 3635.

E-mail address: mjb5@cdc.gov (M.J. Brown).

lead to total lead exposure would be far greater for an infant whose dietary intake is primarily infant formula mixed with lead contaminated household tap water (Shannon and Graef, 1989; CDC, 1994). In one study, an increase in drinking water lead levels from 0.5 part per billion (ppb) to 15 ppb – EPA's action level for lead in water – was estimated to increase children's BLLs an average of 1.9 $\mu\text{g}/\text{dL}$ and the prevalence of BLLs $\geq 10 \mu\text{g}/\text{dL}$ by 14% (Lanphear et al., 1998).

Lead is rarely found in water at the distribution point or well head. It most commonly enters finished water through corrosion of plumbing materials containing lead (Chin and Karalekas, 1985). Three factors that influence the level of lead in drinking water are the presence of lead in plumbing materials, the pH of finished water, and the presence or absence of mineral scale in plumbing. Leaded service lines (LSL) connect homes to a central water main or run from the water meter to the home and are known to contribute to lead found in household tap water (AWWA, 1990; Schock et al., 1996). Homes built before the 1980s may have LSLs or copper pipes with lead-soldered joints. Homes built after the 1986 Safe Drinking Water Amendments have "lead-free" plumbing that may contain up to 8% lead (SDWA Amendments, 1986). Lead dissolves more readily in soft water than hard water resulting in lead leaching from lead-soldered copper water pipes or LSLs. Changes to the water supply that increase the water's pH decrease water lead levels (Goldberg et al., 1981). Mineral scale on the inner surface of older plumbing prevents lead from leaching into drinking water. However, when mineral scale is removed or has yet to develop, lead may be leached into drinking water even from "lead-free" pipes and fixtures.

In 1994, EPA proposed enhanced surface water treatment rules designed not only to protect the public from dangerous microbes in drinking water but also to limit the levels of disinfectants and disinfection byproducts that are classified as possible carcinogens (Tibbets, 1995; US EPA, 2006b). Chloramine, a chlorine–ammonia combination, produces fewer disinfection by-products than chlorine alone, thus a number of water suppliers adopted chloramine water treatment (Tibbets, 1995). In the absence of specific anti-corrosive treatments such as orthophosphate, chloramine degrades accumulated mineral scale resulting in lead leaching into drinking water (Switzer et al., 2006). Increases in the average BLL of children after water disinfectant was changed from chlorine to chloramine have been reported (Miranda et al., 2007).

In Washington, DC, from November 2000 to June 2004, chloramine without orthophosphate was used as a water disinfectant. During this period, one study found that the percent of BLL test results $\geq 5 \mu\text{g}/\text{dL}$ increased above expected levels among persons living in homes with an LSL (CDC, 2004). A second study estimated that 859 D.C. children had BLLs $\geq 10 \mu\text{g}/\text{dL}$ in 2002 and 2003 because of exposures to high water-lead levels (Edwards et al., 2009).

In July 2004, WASA began adding orthophosphate in conjunction with chloramine to prevent corrosion of pipes and also began replacing LSLs. WASA was responsible for the costs of replacing lines from the street to the water meter of residences, while property owners were responsible for the costs of replacing the LSL between the water meter and the interior plumbing. In many cases, property owners declined to pay these costs, thus only the length of the line from the water main to the meter was replaced (partial replacement) rather than the entire length of line between the water meter and the residence (full replacement).

In this study, we examined BLL results among children tested for lead in Washington, DC, between 1998 and 2006. We assessed how the BLLs of tested children were affected by water disinfectant type and LSL while adjusting for the effect of housing age. We further examined the effect of both partial and no replacement of LSLs on the BLLs of children tested between 2004 and 2006.

2. Methods and materials

Three cross-sectional analyses were conducted. In this study the type of water disinfectant, the extent of LSL replacement (partial or none), and the type of service line (leaded or nonleaded) at the residence were the primary exposure variables of interest. BLL was the outcome of interest. Model 1 included LSL as the dependent variable. Model 2 included both LSL and age of housing as dependent variables. Model 2 is used to control for the potential confounding effect of age of housing, a proxy for household lead sources such as paint, house dust, and soil.

2.1. Blood lead levels

The study population was derived from the Washington, DC, Childhood Lead Poisoning Prevention Program (CLPPP) blood lead surveillance system that collected laboratory-based reports of the BLL results of individuals who were tested and whose results were reported to the CLPPP between January 1, 1998, and December 31, 2006. Blood lead tests were analyzed at various laboratories across the United States and were reported as whole numbers to the CLPPP. DC legally required that all BLLs be reported to the CLPPP beginning in 2002 (*Title XX of the Fiscal Year 2003 Budget Support Action of 2002*). The minimum quantitative BLL reported was 2 $\mu\text{g}/\text{dL}$. For BLLs $< 2 \mu\text{g}/\text{dL}$, we adopted the National Health and Nutritional Examination Survey (NHANES) strategy for coding laboratory results below the limit of detection. The results of tests below the limits of detection (BLD) are replaced with a value equal to the detection limit divided by the square root of 2, in this case the value 1.4 $\mu\text{g}/\text{dL}$ (CDC, 2006). This method has been demonstrated to provide an accurate estimation of geometric mean and standard deviation when data are not highly skewed (Hornung and Reed, 1990).

Only one test per child was used in the analyses because repeated blood lead measures for an individual are not independent. For children with multiple tests, an algorithm consistent with CDC recommendations for follow up was used to select the most accurate result for analyses (CDC, 1997). According to this algorithm, if a child's blood lead test consisted solely of capillary samples, the lowest result was used to reduce the potential for positive bias caused by lead from skin contaminating blood when capillary tests were conducted. When the blood lead tests included venous samples, the highest venous BLL was used in analyses. Venous samples are unlikely to be contaminated. The highest test is used because subsequent tests could be influenced by efforts to reduce lead exposures. If sample type was not reported, it was presumed to be capillary. Data-cleaning included checking for duplicate observations and examining inclusion criteria, ranges of variables, and consistency of coding. A total of 67,831 unique children were identified as having been tested at least once during the study period. A categorical BLL variable based on the quartiles of the BLL distribution in the sample population was constructed with BLD as the lowest quartile and $\geq 5 \mu\text{g}/\text{dL}$ as the highest.

CDC review of CLPPP records identified that the number of tests reported in 2003 was approximately 50% lower than the number of tests reported in either 2002 or 2004. The number of tests reported in the surveillance system was otherwise consistent from 1 year to the next. These missing tests were not entered into the DC DOH lead surveillance system because laboratories did not report all BLLs during that year or tests received by DC DOH were not entered into the surveillance system.

In 2009, CDC acquired approximately 12,000 missing 2003 test results that had been unavailable in 2004 (Brown et al., 2010). We analyzed two datasets; one with and one without the missing 2003 blood lead level test data. The previously missing 2003 test results were collected using methods inconsistent with all other years. Therefore, we planned to provide results for both datasets only if the results differed. Otherwise, we provide here the results from the dataset without the missing 2003 tests.

2.2. Lead service lines

WASA provided the CLPPP and the Centers for Disease Control and Prevention (CDC) with a list of 26,155 homes presumed by WASA to have an LSL using criteria established by the Lead and Copper Rule (40 CFR Part 141). Street addresses from blood lead tests reported to CLPPP and the WASA address data were standardized using Centrus Desktop™ software version 4.02 (Sagent Technology, Mountain View, CA) and matched to the complete street address. Of the 67,831 unique children with at least one BLL reported, complete street address could be found for 63,854 children who comprise our final study population. The houses of 10,859 of these 63,854 children were identified by WASA as having LSLs. The remaining houses did not appear on the WASA LSL list and were initially coded as not having an LSL.

WASA provided CDC with a list of 14,121 residential houses in which the water service line had been partially or fully replaced between 2004 and 2006. The BLL tests of 738 children were conducted after LSL was partially removed. Except as described below, these houses were coded as not having an LSL. Houses remained coded as having an LSL if the LSL replacement occurred after the blood lead test was conducted. WASA reported replacing a water service line in some homes that were

not included in the original WASA data file of houses with an LSL. We recoded these houses as having had an LSL if the BLL occurred before the line was replaced.

2.3. Water disinfectant type

We designed a categorical variable, water disinfectant type coded as (1) chlorine if the BLL test was conducted between January 1, 1998, and October 31, 2000; (2) chloramine if the BLL test was conducted between November 1, 2000, and June 30, 2004; or (3) chloramine with orthophosphate if the BLL test was conducted between July 1, 2004, and December 31, 2006. These periods correspond to the dates when these types of water disinfection were used by WASA.

2.4. Study population

The final sample consisted of 63,854 uniquely identified children < 6 years of age who had a BLL reported to DC DOH during the 9-year study period and a validated address.

Additional variables used in the analyses included age of housing and the child's age at the time of the BLL test. Age of housing was coded as pre-1950, 1950–1978, and post-1978. These periods coincide with changes in lead concentration in residential paint. The greatest amount of leaded paint was used pre-1950, and moderate use of leaded paint occurred from 1950 through 1978. Leaded residential paint was banned after 1978. To determine the age of houses, blood lead data were linked with tax assessor data. Data for age of houses were obtained for 37,322 (58.5%) children with validated addresses. We also categorized the child's age into ≤ 16 months of age and > 16 months – 6 years of age to allow comparison to other published studies.

2.5. Data analysis

We examined the difference in distribution of children with or without validated addresses on the following variables: age, sex, blood sample type (venous, capillary, or not reported and assumed capillary), categorical BLL, and water disinfectant in use at time of the BLL test. We tested the relationship between BLL and LSL using two approaches. We used polychotomous logistic models that assumed a multinomial dependent variable. We categorized children into blood lead levels quartiles. The models estimated the risk of falling into the second, third, or fourth quartile of blood lead level compared to the lowest quartile of blood lead level given the presence or absence of a lead service line stratified by water disinfection type.

In the first approach we computed odds of BLL quartiles (BLD, 2- < 3, 3- < 5, and ≥ 5 $\mu\text{g}/\text{dL}$) to determine the presence of a dose-response relationship. In the second we calculated the odds of a BLL ≥ 10 $\mu\text{g}/\text{dL}$. We tested the association between BLLs and LSL for each water disinfectant type, controlling for age of housing. We further stratified these analyses by focusing on children ≤ 16 months of age. Finally, we computed odds ratios of BLL ≥ 5 $\mu\text{g}/\text{dL}$ and BLL ≥ 10 $\mu\text{g}/\text{dL}$ by intact or partially replaced LSL.

Logistic regression was used to model the relationship between BLL quartile, LSL, and age of housing. Standard statistical methods were used to compute odds ratios (OR) and 95% confidence intervals (CI) for all effects studied (Rothman and Greenland, 1998). The Statistical Analysis System (SAS) (SAS Institute, Cary, NC) was used for generating descriptive statistics, and regression models.

3. Results

3.1. Demographics

Among the 63,854 children in the study population 22,719 (36%) children were tested before their second birthday; 17,509 (27%) children were tested when chlorine was used to disinfect water, 23,837 (37%) when chloramine alone was used, and 22,508 (35%) when chloramine and orthophosphate were used. A total of 51,592 children (81%) had BLLs < 5 $\mu\text{g}/\text{dL}$, 10,197 children (16%) had BLLs 5–9 $\mu\text{g}/\text{dL}$, and 2065 children (3%) had BLLs ≥ 10 $\mu\text{g}/\text{dL}$. As Table 1 indicates, children with a valid address were more likely than other children to have been tested by venous samples and had a slightly higher BLL distribution than children without a valid address. Of the 37,322 children in the study for whom age of housing was available, 28,238 (44%) lived in housing built before 1950. Of the children whose data were used in the analysis, 9938 (16%) lived in housing where an LSL was present and had been not replaced before the BLL test was conducted.

3.2. Stratified analyses of BLL quartile and LSLs

A relationship was observed between BLL quartile status and probability of living in a house with an LSL for every year between 1998 and 2006 including those years when WASA was in compliance with the EPA action level of 15 ppb. However, this relationship was attenuated when age of housing was entered into the models. For the period when chloramine alone was used as a water disinfectant, the adjusted odds ratio (OR) of a BLL in the highest versus the lowest quartile for children living in homes with an LSL was 2.5 (95% CI, 2.2–2.9), controlling for age of housing. The risk was greatest in 2003 when the adjusted OR of a BLL in the highest versus the lowest quartile for children living in homes with a LSL was 3.2 (95% CI 2.4, 4.4; data not shown). In models that included the nearly 6000 children whose 2003 BLLs were received in 2009, the adjusted OR of a BLL in the highest versus the lowest quartile for children living in homes with a LSL was 3.0 (95% CI 2.3, 3.8; data not shown). When chloramine with orthophosphate was used as the disinfectant (2004–2006), the odds of a BLL in the highest quartile relative to the lowest remained elevated, but these odds were somewhat lower than when chloramine was used alone (Table 2).

3.3. LSL replacement

Chloramine with orthophosphate was the water disinfectant used during the period of time when the WASA LSL replacement program was conducted. Compared to households with no LSL, partial LSL replacement was associated with elevated OR for a child's BLL 5–9 $\mu\text{g}/\text{dL}$ [OR=1.9 (95% CI, 1.5–2.3)] and BLL ≥ 10 $\mu\text{g}/\text{dL}$ [OR=3.3 (95% CI, 2.2–4.9)] (Table 3). Conversely no significant difference in risk was found between children in households with partially replaced LSL compared to intact LSL for either BLL ≥ 5 or 10 $\mu\text{g}/\text{dL}$ (Table 3). The number of days between lead service line replacement and BLL for 921 children where LSL were replaced was unrelated to BLL (BLL < 5 $\mu\text{g}/\text{dL}$, $n=769$, mean=323 days; BLL 5–9 $\mu\text{g}/\text{dL}$, $n=120$, mean=344 days; BLL ≥ 10 $\mu\text{g}/\text{dL}$, $n=32$, mean=307 days; $p=0.6$). When models where children with partially replaced LSL were removed from the analyses during the time period when chloramine and orthophosphate were used for water disinfection LSL remain strongly associated with BLL in the highest quartile [OR 1.6 (95% CI, 1.4–1.9)] for children < 6 years of age and [OR 1.5 (95% CI 1.2, 2.0)] for children ≤ 16 months of age. These values are not significantly different from the OR estimates of models that include these children.

3.4. Age

In a subsample of 17,181 children ≤ 16 months of age, the odds of a BLL ≥ 5 $\mu\text{g}/\text{dL}$ was 1.7 (95% CI, 1.1–2.6) in the period of chlorine disinfection; 3.6 (95% CI, 2.8–4.6) for the chloramine disinfection period and 1.6 (95% CI, 1.3–2.0) for the chloramine with orthophosphate period in models that controlled for age of housing (Table 4). A dose response relationship between living in a house with an LSL and BLL quartile was identified for all three disinfectant periods. Consistent with previous analyses, this relationship also was attenuated in analyses that controlled for age of housing. The risk for BLL ≥ 10 $\mu\text{g}/\text{dL}$ was remarkably similar for younger and older children during the chloramine alone and chloramine with orthophosphate disinfection periods. However, for younger children the risk for BLL in the highest BLL quartile (≥ 5 $\mu\text{g}/\text{dL}$) was more than doubled in the chloramine alone period compared to either of the other disinfection periods.

When BLLs ≥ 10 $\mu\text{g}/\text{dL}$ are considered, in models that control for age of housing, living in a house with an LSL was an independent risk factor for the entire study period (Table 5).

Table 1

Comparison of children < 6 years of age with validated^a and non-validated address on blood lead variables and selected demographic characteristics, Washington, DC, 1998–2006.

Characteristic	Address validated (n=63,854)		Address not validated (n=3977)		p-value
	n	% ^b	n	% ^b	
Gender					
Male	31,534	49.4	1945	48.9	< 0.0001
Female	30,529	47.8	1820	45.8	
Not reported	1791	2.8	212	5.3	
Age					
0–11 months	8414	13.2	556	14.0	< 0.0001
12–23 months	14,305	22.4	1034	26.0	
24–35 months	13,645	21.4	747	18.8	
36–47 months	10,830	17.0	547	13.8	
48–72 months	16,660	26.1	1093	27.5	
Sample type					
Not reported, assumed capillary	12,166	19.1	2770	69.7	< 0.0001
Capillary	4183	6.6	343	8.6	
Venous	47,505	74.4	864	21.7	
Blood lead level (µg/dL) ^c					
< 5	51,592	80.8	3321	83.5	< 0.0001
5– < 10	10,197	16.0	576	14.5	
≥ 10	2065	3.2	80	2.0	
Age of housing					
Pre-1950	28,238	44.2			NA
1950–1978	7651	12.0			
Post 1978	1433	2.2			
Data not available	26,532	41.6	3977	100.0	
Lead service line (LSL) ^d					NA
Partial replacement	738	1.2			
Full replacement	183	0.3			
LSL, not replaced	9938	15.6			
No LSL	52,995	83.0			
Data not available			3977	100.0	
Water disinfectant at time of blood test					
Chlorine (1/1/1998 to 10/31/2000)	17,509	27.4	1137	28.6	< 0.0001
Chloramine only (11/1/2000–6/30/2004)	23,837	37.3	2706	68.0	
Chloramine with orthophosphate (7/1/2004–12/31/2006)	22,508	35.3	134	3.4	

^a Address considered validated if data available for complete street address (including house number, street name, and street suffix (e.g., St., Dr., Pl)).

^b Percents may not add to 100.0% due to rounding.

^c Service line defined as replaced only if date of replacement was before date of blood lead test.

^d Statistics regarding age of housing and LSLs cannot be computed for non-validated addresses.

Table 2

Odds ratios (with 95% CIs) for having a LSL for BLL quartiles, relative to lowest quartile, by time periods corresponding to water disinfection type, children < 6 years of age, in Washington, DC, 1998–2006.

Blood Lead Quartile Cutpoints (µg/dL)	Chlorine (1/1/1998–10/31/2000)		Chloramine (11/1/2000–6/30/2004)		Chloramine with Orthophosphate (7/1/2004–12/31/2006)	
	Model 1 OR (95% CI) for LSL (n=17,509)	Model 2 OR (95% CI) for LSL, controlling for age of housing (n=9860)	Model 1 OR (95% CI) for LSL (n=23,837)	Model 2 OR (95% CI) for LSL, controlling for age of housing (n=13,898)	Model 1 OR (95% CI) for LSL (n=22,508)	Model 2 OR (95% CI) for LSL, controlling for age of housing (n=13,564)
BLD ^a	1.0	1.0	1.0	1.0	1.0	1.0
2– < 3	0.9 (0.7, 1.2)	1.0 (0.7, 1.3)	1.1 (1.0, 1.3)	1.2 (1.0, 1.4)	1.0 (0.9, 1.2)	1.1 (0.9, 1.2)
3– < 5	1.0 (0.8, 1.2)	1.1 (0.8, 1.4)	1.6 (1.4, 1.8)	1.6 (1.4, 1.8)	1.4 (1.3, 1.5)	1.3 (1.1, 1.4)
≥ 5	1.6 (1.3, 2.0)	1.4 (1.1, 1.9)	3.0 (2.7, 3.4)	2.5 (2.2, 2.9)	2.1 (1.9, 2.3)	1.7 (1.5, 1.9)

^a BLD—Below the limit of detection (1.4 µg/dL).

4. Discussion

In this study of children's BLLs in Washington, DC, LSLs were associated with increased odds of having elevated BLLs even during

time periods when WASA was in compliance with the EPA action level of 15 ppb. The association was stronger when chloramine alone rather than chlorine was used as a disinfectant, particularly among younger children. Adding orthophosphate to chloramine

Table 3
Odds of having a blood lead level (BLL) 5–9 µg/dL or ≥ 10 µg/dL, comparing partial replacement^a of lead service line (LSL) to no LSL and partial replacement to LSL not replaced when water disinfectant was chloramine with orthophosphate (7/1/2004–12/31/2006), Children < 6 years of age, Washington, DC, 2004–2006.

BLL	Partial replacement vs. no LSL			Partial replacement vs. LSL not replaced		
	Partial replacement ^a	No LSL	Odds ratio (95% CI)	Partial replacement ^a	LSL Not replaced	Odds ratio (95% CI)
< 5 µg/dL	598	17,029	1.0	598	2434	1.0
5–9 µg/dL	105	1592	1.9 (1.5, 2.3)	105	406	1.1 (0.8, 1.3)
≥ 10 µg/dL	27	236	3.3 (2.2, 4.9)	27	81	1.4 (0.9, 2.1)
Total	730	18,857		730	2921	

^a Service line defined as replaced only if date of replacement was before date of blood lead test.

Table 4
Odds ratios (with 95% CIs) for having a LSL for BLL quartiles, relative to lowest quartile, by time periods corresponding to water disinfection type, children ≤ 16 months of age, in Washington, DC, 1998–2006.

Blood lead quartile cutpoints (µg/dL)	Chlorine (1/1/1998–10/31/2000)		Chloramine (11/1/2000–6/30/2004)		Chloramine with orthophosphate (7/1/2004–12/31/2006)	
	Model 1 OR (95% CI) for LSL (n=3711)	Model 2 OR (95% CI) for LSL, controlling for age of housing (n=2180)	Model 1 OR (95% CI) for LSL (n=6238)	Model 2 OR (95% CI) for LSL, controlling for age of housing (n=3781)	Model 1 OR (95% CI) for LSL (n=7232)	Model 2 OR (95% CI) for LSL, controlling for age of housing (n=4536)
BLD ^a	1.0	1.0	1.0	1.0	1.0	1.0
2– < 3	0.9 (0.6, 1.4)	1.0 (0.6, 1.5)	1.4 (1.2, 1.7)	1.6 (1.2, 2.0)	1.0 (0.8, 1.2)	1.0 (0.8, 1.2)
3– < 5	1.0 (0.7, 1.5)	1.1 (0.7, 1.7)	2.1 (1.7, 2.5)	2.2 (1.7, 2.7)	1.3 (1.1, 1.5)	1.2 (1.0, 1.4)
≥ 5	1.6 (1.1, 2.4)	1.7 (1.1, 2.6)	4.1 (3.4, 5.0)	3.6 (2.8, 4.6)	2.0 (1.6, 2.4)	1.6 (1.3, 2.0)

^a BLD—below the limit of detection (1.4 µg/dL).

Table 5
Odds of having a blood lead level (BLL) ≥ 10 µg/dL, relative to a BLL of < 10 µg/dL, in presence of lead service line (LSL)^{a,b} at time of blood lead test and controlling for age of housing, by water disinfectant type, children ages > 16 months to < 6 years of age and children ≤ 16 months of age, Washington, DC, 1998–2006.

Blood lead level (BLL) (µg/dL)	Chlorine (1/1/1998–10/31/2000)		Chloramine only (11/1/2000–6/1/2004)		Chloramine with orthophosphate (7/1/2004–12/31/2006)	
	Model 1 OR (95% CI) for LSL	Model 2 OR (95% CI) for LSL, controlling for age of housing	Model 1 OR (95% CI) for LSL	Model 2 OR (95% CI) for LSL, controlling for age of housing	Model 1 OR (95% CI) for LSL	Model 2 OR (95% CI) for LSL, controlling for age of housing
Children > 16 months to < 6 years of age	N=13,798 ^c	N=7680 ^d	N=17,599	N=10,117 ^d	N=15,276 ^c	N=9028 ^d
< 10	1.0	1.0	1.0	1.0	1.0	1.0
≥ 10	2.3(2.0, 2.7)	1.5 (1.3, 1.8)	3.0 (2.6, 3.6)	2.2 (1.8, 2.7)	2.4 (1.8, 3.1)	1.7 (1.2, 2.3)
Children ≤ 16 months of age	N=3711 ^c	N=2180 ^d	N=6238 ^c	N=3781 ^d	N=7232 ^c	N=4536 ^d
< 10	1.0	1.0	1.0	1.0	1.0	1.0
≥ 10	3.4 (2.3, 5.0)	3.7 (2.2, 6.2)	3.1 (2.3, 4.3)	2.3 (1.6, 3.4)	2.7 (1.7, 4.2)	1.9 (1.1, 3.1)

^a Includes children living in homes where the LSL was not replaced or where the LSL was only partially replaced prior to their BLL test.

^b No LSL includes children living in homes where the LSL was fully replaced prior to their BLL test.

^c N includes children for whom there was data on LSL.

^d N includes only children for whom there were data on both LSL and age of housing.

attenuated, but did not eliminate the association. These data suggest that changes in water disinfectants that increase the leaching of lead into water can increase BLLs of children.

We also found that children living in housing where an LSL was partially replaced after 2003, were more likely to have BLLs 5–9 or ≥ 10 µg/dL than children living in housing without an LSL. The risk for BLL elevation for children living in homes with partial LSL replacement was consistent with the risk for BLL elevation among children living in homes with an intact LSL. Virtually all of the homes having lead service lines replaced were built before 1950.

Continued exposure to lead in water, lead from other sources, or a combination of factors may explain these risks. However, partial LSL replacement was not effective in decreasing risk for BLL 5–9 µg/dL or ≥ 10 µg/dL.

4.1. Limitations

This study is subject to a number of limitations. First, we could not control for water consumption patterns at the individual level.

We have no reason to suspect that water consumption patterns at the population level changed in concert with the use of chlorine or chloramine alone until the public became aware of the high levels of lead in D.C. drinking water in January 2004. However, public health interventions such as distribution of water filters and widespread information including instructions not to drink unfiltered tap water that occurred after January 2004 may have changed drinking water habits. This public information may have partially attenuated the association we reported in the chloramine alone and with orthophosphate water disinfection time periods, although the association remained strong. We also could not evaluate the extent to which children living in houses with or without an LSL may have obtained their drinking water in other places (e.g., other houses, schools, etc.) or from bottled drinking water. Random misclassification introduced as a result of this limitation would have attenuated the estimates of an association between BLLs and LSLs.

Because BLLs were analyzed at several laboratories and reported to DOH, inter-laboratory variability may have resulted in misclassification. Measurement error in BLLs would tend to increase the standard error and result in an attenuation of the association between exposure and outcome. However, a 1998 review of commercial laboratories certified to analyze BLLs found strong reproducibility within and among laboratories, without any overall time trend or interlaboratory or intralaboratory variance (Jobanputra et al., 1998). A subsequent study of commercial laboratories found that differences in laboratory performance on blinded BLL samples were clinically insignificant (Parsons et al. 2001).

BLL surveillance data were subject to errors as well as failure to report or enter results throughout the study period. Random error would have attenuated the estimates of association reported here. Non-random error or differential misclassification can affect the odds ratio in various ways depending on the direction of the misclassification (Armstrong et al., 1992).

We assessed the impact of the 2003 missing data on the results presented here. No substantive differences were noted. For example the risk of BLL in the highest quartile compared to the lowest quartile given a LSL for the chloramine period alone without the missing data was OR=3.0 (95% CI, 2.7–3.4) versus with the missing data OR=2.9 (95% CI, 2.6–3.2).

Addresses with an LSL were derived from a list of addresses provided by WASA as highly suspected to have LSLs. Some misclassification of houses with and without LSLs is likely, given that direct inspection, which requires digging down to the LSL, is necessary to determine whether the service line is indeed made of lead. We identified that 1338 (1.5%) of the children's houses originally coded by WASA as not having LSLs were later reported by WASA as having a service line replacement and these homes were reclassified as having a LSL. Additional random misclassification of LSLs would attenuate the estimates of an association between BLLs and LSLs. Coding of the 738 children whose blood lead levels were drawn after partial replacement of LSL as having no LSL also may have introduced misclassification and attenuated the strength of the association. However, models in which these children were removed were not statistically different from those that included these children.

Control for the influence of lead exposures other than LSLs was limited to using a categorical variable for age of housing. This variable is a well established predictor of lead paint hazards in housing (CDC, 1997). We did not control for exposure to other important sources in the environment. Also, since LSLs were not perfectly assessed by WASA, age of housing may serve not only as a proxy for the presence of lead paint hazards but also for unidentified LSLs. Models controlling for age of housing may underestimate the association between LSL and BLL.

It is unlikely that lead exposure sources are distributed randomly in the population, given that children living in old, sub-standard housing face a variety of other lead exposure sources. Future studies that collect environmental samples, such as house dust, soil, and water would address this weakness.

4.2. Strengths

This study has several strengths that enhance the interpretation of these findings. First, the study uses laboratory-based reports of 63,854 children's blood lead level tests. The large number of tests facilitated our analysis of the association between BLL, LSL, and water disinfectant, enabling us to control for the effect of the temporal decline in BLL that has occurred across the United States during the study period. Secondly, the lab-based reports of BLLs included the listing of the child's address at the time of the test. These addresses could be matched to the data provided by WASA that identified housing considered most likely to be connected to drinking water mains by an LSL. Additionally, we could adjust for the potential confounding caused by leaded paint using the surrogate variable, age of housing. The correlation between elevated BLL in children and housing age is well established (CDC, 1997). Third, only 1338 homes originally considered as not having an LSL, were reclassified once information became available that an LSL had been partially or fully replaced. Finally, the analysis of children ≤ 16 months of age provided us the opportunity to evaluate how the association between BLL, LSL, and disinfectant affected very young children, thought to be the most vulnerable segment of the population.

4.3. Comparison to previous work

In 2004, CDC conducted a rapid assessment of reported BLL testing data in response to concerns about high concentrations of lead in water and concluded that the expected trajectory of BLLs decreasing over time was reversed for children living in houses with LSLs for BLLs ≥ 5 $\mu\text{g}/\text{dL}$, although not for BLLs ≥ 10 $\mu\text{g}/\text{dL}$, during the period when chloramine alone was used as a water disinfectant (CDC, 2004). The 2004 assessment was subject to a number of limitations including the use of multiple blood lead tests per person, the inclusion of individuals > 6 years of age and not specifically investigating the effects on the youngest children. This study addressed all those limitations. We found that LSLs increased the odds of elevated BLLs during both time periods when chloramine was used as a water disinfectant, and that this finding is independent of age of housing, which is a proxy for the presence of lead paint.

Guidotti et al. (2007) found that 5.3% of children living in homes with LSL had BLL ≥ 10 $\mu\text{g}/\text{dL}$ based in part, on 2342 children tested for lead between February and July 2004. In a correction to the study the authors concluded that public health measures instituted in 2004 may have prevented more frequent blood lead elevations. The authors attributed the blood lead elevations to other environmental sources in the community (Guidotti et al., 2007).

In contrast, we found that LSL was a risk factor for r increased BLL independent of age of housing throughout the study period. The association was strongest during the period when chloramine alone was used as a water disinfectant.

Edwards et al. (2009) reported that D.C. children ≤ 16 months of age were more than four times as likely to have had BLLs ≥ 10 $\mu\text{g}/\text{dL}$ during the period 2001–2003 compared to 2000. Both Edwards et al. (2009) and this study examined the association between water disinfection methods and BLLs ≥ 10 $\mu\text{g}/\text{dL}$. We found that the strength of the association (size of the odds ratio) was greater for younger children than for older children during all three time

periods (Table 5). This difference was greatest during the earliest time period when chlorine was used to disinfect drinking water. The stronger associations reported for young children may reflect their intake of formula mixed with tap water and mouthing behaviors.

5. Conclusions and recommendations

In Washington, DC, between November 2000 and December 2006, children living in homes with an LSL were at increased risk of having higher BLLs than children living in homes without an LSL. This association was strongest during 2003 when chloramine alone was used for water disinfection. The association persisted after controlling for the age of housing. Finally, partial replacement of LSLs did not result in a decrease in the association between LSL and elevated BLL.

For the majority of children in the United States with elevated BLLs, lead paint and lead-contaminated house dust and soil are the primary routes of lead exposure (Levin et al., 2008). But children are exposed to multiple lead exposure sources – including water – and evidence suggests that, particularly for children with BLLs 5–10 µg/dL, no exposure source may dominate (Bernard and McGeehin, 2003). Thus the contribution of lead in drinking water to children's BLLs, particularly at BLLs < 10 µg/dL may be underestimated.

The most effective strategy to reduce BLLs remains controlling or eliminating sources of lead in children's environments before they are exposed. The consequences of changes in water disinfection practices on a range of health issues including exposure to lead should be carefully considered by water utilities before they are adopted. A summary of internal corrosion control of water distribution systems concluded that appropriate corrosion control is essential in water distribution systems where lead is present (AWWA, 1996). Had appropriate corrosion control been in place in DC in 2001–2004, it would have prevented the increase in water lead concentrations seen (US EPA, 2007).

Residents of properties where plumbing work has been done, including partial replacement of LSL, should take precautions such as using bottled or filtered water until they are sure that the water lead levels are below the EPA action level of 15 ppb. Finally, given that no safe blood lead level threshold has been identified for children, and that lead in water contributes to BLLs, prompt and effective action by utilities to rapidly comply with existing drinking water standards is warranted.

Acknowledgments

We would like to thank Mr. Barry Brooks for his tireless effort in seeing us through this process. We also thank the Washington DC Childhood Lead Poisoning Prevention Program and the DC Water and Sewer Authority for providing data.

Appendix A. Supporting information

Supplementary data associated with this article can be found in the online version at doi:10.1016/j.envres.2010.10.003.

References

Agency for Toxic Substances and Disease Registry, 1999. Toxicological profile for lead. Department of Health and Human Services, Agency for Toxic Substances and Disease Registry, Atlanta, Georgia.

Armstrong, B.K., White, E., Saracci, R., 1992. Exposure measurement error and its effects. Principles of Exposure Measurement in Epidemiology, Oxford University, NY.

AWWA (American Water Works Association) Research Foundation, 1996. Internal Corrosion of Water Distribution Systems—Cooperative Research Report. AWWA Research Foundation and American Water Works Association, Denver, CO.

AWWA (American Water Works Association) Research Foundation, 1990. Lead Control Strategies. AWWA Research Foundation and American Water Works Association, Denver, CO.

Bellinger, DC, Needleman, HL, 2003. Intellectual impairment and blood lead levels. *N. Engl. J. Med.* 349, 500–502.

Bernard, SM, McGeehin, MA, 2003. Prevalence of blood lead levels ≥ 5 µg/dL among US children 1 to 5 years of age and socioeconomic and demographic factors associated with blood lead level 5 to 10 µg/dL. Third national health and nutrition examination survey, 1988–1994. *Pediatrics* 112 (6), 1308–1313.

Brown M.J., Blanton C., Sinks T., 2010. Examining the Effect of Previously Missing Blood Lead Level (BPb) Surveillance Data on Results Reported in the MMWR (April 2, 2004/53(12):268–270). Available at <www.cdc.gov/nceh/lead>.

Canfield, RL, Henderson Jr., CR, Cory-Slechta, DA, Cox, C, Jusko, TA, Lamphear, BP, 2003. Intellectual impairment in children with blood lead concentrations below 10 µg per deciliter. *N. Engl. J. Med.* 348, 1517–1526.

CDC (Centers for Disease Control and Prevention), 2004. Blood lead levels in residents of homes with elevated lead in tap water—District of Columbia. *Morb. Mortal. Wkly. Rev.* 53, 268–270.

CDC (Centers for Disease Control and Prevention), 2002. Managing Elevated Blood Lead Levels Among Young children: Recommendations from the Advisory Committee on Childhood Lead Poisoning Prevention. CDC, Atlanta.

CDC (Centers for Disease Control and Prevention), 1997. Screening Young Children for Lead Poisoning: Guidance for State and Local public Health officials. CDC, Atlanta.

CDC (Centers for Disease Control and Prevention), 1994. Lead-contaminated drinking water in bulk-water storage tanks—Arizona and California, 1993. *Morb. Mortal. Wkly. Rev.* 43 (751), 757–758.

CDC (Centers for Disease Control and Prevention), 2006. General Information about the NHANES 2003–2004., Laboratory Methodology and Public Data Files, January 2006. US Department of Health and Human Services, Atlanta, Georgia.

Chin, D., Karalekas, P.C.J., 1985. Lead product use survey of public water supply distribution systems throughout the United States. In: Plumbing Materials and Drinking Water Quality: Proceedings of a Seminar Held at Cincinnati, Ohio, May 16–17, 1984. EPA 600/9-85-007, US Environmental Protection Agency, Washington, DC, pp. 110–123.

Cosgrove, E, Brown, MJ, Madigan, P, McNulty, P, Okonski, L, Schmidt, J, 1989. Childhood lead poisoning: case study traces source to drinking water. *J. Environ. Health* 52 (1), 346–349.

Edwards, M, Triantafyllidou, S, Best, D, 2009. Elevated blood lead in young children due to lead-contaminated drinking water: Washington, DC, 2001–2004. *Environ. Sci. Technol.* 43, 1618–1623.

Goldberg, MM, Fyfe, WM, Richards, WN, 1981. Maternal lead levels after alterations to the water supply. *Lancet* 283, 203–204.

Guidotti, T, Calhoun, T, Davies-Cole, J, Knuckles, M, Stokes, L, Glymph, C, Lum, G, Moses, M, Goldsmith, D, Ragain, L, 2007. Elevated lead in drinking water in Washington, DC, 2003–2004: the public health response. *Environ. Health Perspect.* 115, 695–701.

Hornung, RW, Reed, LD, 1990. Estimation of average concentration in the presence of nondetectable values. *Appl. Occup. Environ. Hyg.* 5, 46–51.

Jobanputra, NK, Jones, R, Bukler, G, et al., 1998. Accuracy and reproducibility of blood lead testing in commercial laboratories. *Arch. Pediatr. Adolesc. Med.* 152, 548–553.

Jones, R, Homa, D, Meyer, P, Brody, D, Caldwell, K, Pirkle, J, et al., 2009. Trends in blood lead levels and blood lead testing among US children aged 1 to 5 years: 1998–2004. *Pediatrics* 123, e376–e385.

Lanphear, BP, Dietrich, KN, Auinger, P, Cox, C, 2000. Cognitive deficits associated with blood lead concentrations < 10 µg/dL in US children and adolescents. *Public Health Rep.* 115, 521–529.

Lanphear, BP, Burgoon, DA, Rust, SW, Eberly, S, Galke, W, 1998. Environmental exposures in lead and urban children's blood lead levels. *Environ. Res.* 76, 120–130.

Levin, R, Brown, MJ, Kashtock, ME, Jacobs, DE, Whelan, EA, Rodman, J, Schock, MR, Padilla, A, Sinks, T, 2008. US children's lead exposures, 2008: implications for prevention. *Environ. Health Perspect.* 116, 1285–1293.

Menke, A, Muntner, P, Batuman, Silbergeld, EK, Guallar, E, 2006. Blood lead below 0.48 µmol/L (10 µg/dL) and mortality among US adults. *Circulation* 114, 1388–1394.

Miranda, ML, Kim, D, Jull, AP, Paul, CJ, Overstreet Galeano, MA, 2007. Changes in blood lead levels associated with use of chloramines in water treatment systems. *Environ. Health Perspect.* 115, 221–225.

Parsons, PJ, Reilly, AA, Esernio-Jensen, D, et al., 2001. Evaluation of blood lead proficiency testing: comparison of open and blind paradigms. *Clin. Chem.* 47, 322–330.

Rothman, KJ, Greenland, S, 1998. Modern Epidemiology second ed. Lippincott-Raven, Philadelphia, PA.

Safe Drinking Water Act Amendments (SDWA), 1986. PL 99–339.

Schock M.R., Wagner, I., Oliphant, R., 1996. The corrosion and solubility of lead in drinking water. In: Internal Corrosion of Water Distribution Systems, second ed., AWWA Research Foundation/TZW, Denver, CO, p. 131–230.

- Shannon, M, Graef, JW, 1989. Lead intoxication from lead-contaminated water used to reconstitute infant formula. *Clin. Pediatr.* 28 (8), 380–382.
- Switzer, JA, Rajasekharan, VV, Boonsales, S, Kulp, EA, Bohannon, EW, 2006. Evidence that monochloramine disinfectant could lead to elevated Pb levels in drinking water. *Environ. Sci. Technol.* 40 (10), 3384–3387.
- Tibbets, J, 1995. What's in the water? *Environ. Health Perspect.* 103 30–34.
- US EPA, 2007. Elevated lead in drinking water—a study of potential causative events. Final Summary Report, US Environmental Protection Agency, EPA 815-R-07-021, Washington, DC.
- US EPA, 2006a. Air quality criteria for lead. Final Report, US Environmental Protection Agency, Washington, DC, EPA/600/R-05/144aF-bF <http://oaspub.epa.gov/eims/eimscomm.getfile?p_download_id=459555>.
- US EPA (US Environmental Protection Agency), 2006b. National primary drinking water regulations: stage 2 disinfectants and disinfectant by product rule (40 CFR Parts 9, 141 and 142). *Fed. Regis.* 71 (2), 387–484.
- US EPA (US Environmental Protection Agency). 1986. Environmental concentrations and potential pathways to human exposure. *Air Quality Criteria for Lead*, vol. 2, US Environmental Protection, Washington, DC.