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

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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 µg/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 µg/dL have been associated with increased risk for hypertension and increased all-cause mortality (ATSDR, 1999; Menke et al., 2006). BLLs > 75 µg/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 µg/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 µg/dL among children 1–5 years of age in the United States has decreased over time. Since 1988 percent of BLLs ≥ 10 µg/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 µg/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

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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 μg/dL and the prevalence of BLLs ≥ 10 μg/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 ≥ 5 μg/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 ≥ 10 μg/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 (lead 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 quantifiable BLL reported was 2 μg/dL. For BLLs < 2 μg/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 μg/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, 1996).

One test per child was included 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 sampling 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 ≥ 5 μg/dL as the highest.

CDC review of CLPPP records identified that the number of tests reported in 2003 was approximately 30% 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 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 wereitusen. Of these 63,854 children, 10,850 were identified as having been tested at least once during the study period. The remaining children 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 specified above, these houses were coded as 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, 2000; 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 multinominal 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 μg/dL) to determine the presence of a dose–response relationship. In the second we calculated the odds of a BLL ≥ 10 μg/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 μg/dL and BLL ≥ 10 μg/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 μg/dL, 10,197 children (16%) had BLLs 5–9 μg/dL, and 2065 children (3%) had BLLs ≥ 10 μg/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 ratios (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 μg/dL [OR = 1.9 (95% CI, 1.5–2.3)] and BLL ≥ 10 μg/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 μg/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 μg/dL, n = 769, mean = 323 days; BLL 5–9 μg/dL, n = 120, mean = 344 days; BLL ≥ 10 μg/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 μg/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 was also attenuated in analyses that controlled for age of housing. The risk for BLL ≥ 10 μg/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 μg/dL) was more than doubled in the chloramine alone period compared to either of the other disinfection periods.

When BLLs ≥ 10 μg/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).
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 2

<table>
<thead>
<tr>
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<tbody>
<tr>
<td></td>
<td>Model 1 OR (95% CI) for LSL (n=17,509)</td>
<td>Model 2 OR (95% CI) for LSL, controlling for age of housing (n=9,860)</td>
<td>Model 1 OR (95% CI) for LSL (n=23,837)</td>
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<td>BLDa</td>
<td>1.0</td>
<td>1.0</td>
<td>1.0</td>
</tr>
<tr>
<td>2–&lt; 3</td>
<td>0.9 (0.7, 1.2)</td>
<td>1.0 (0.7, 1.3)</td>
<td>1.1 (1.0, 1.3)</td>
</tr>
<tr>
<td>3–&lt; 5</td>
<td>1.0 (0.8, 1.2)</td>
<td>1.1 (0.8, 1.4)</td>
<td>1.6 (1.4, 1.8)</td>
</tr>
<tr>
<td>≥ 5</td>
<td>1.6 (1.3, 2.0)</td>
<td>1.4 (1.1, 1.9)</td>
<td>3.0 (2.7, 3.4)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Blood lead level (µg/dL)c</th>
<th>Address validated (n = 63,854)</th>
<th>Address not validated (n = 3977)</th>
</tr>
</thead>
<tbody>
<tr>
<td>BLDa</td>
<td>1.0</td>
<td>1.0</td>
</tr>
<tr>
<td>2–&lt; 3</td>
<td>0.9 (0.7, 1.2)</td>
<td>1.0 (0.7, 1.3)</td>
</tr>
<tr>
<td>3–&lt; 5</td>
<td>1.0 (0.8, 1.2)</td>
<td>1.1 (0.8, 1.4)</td>
</tr>
<tr>
<td>≥ 5</td>
<td>1.6 (1.3, 2.0)</td>
<td>1.4 (1.1, 1.9)</td>
</tr>
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</table>

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


Table 3
Odds of having a blood lead level (BLL) 5–9 μg/dL or ≥ 10 μg/dL, comparing partial replacement* 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.

<table>
<thead>
<tr>
<th>BLL</th>
<th>Partial replacement vs. no LSL</th>
<th>Partial replacement vs. LSL not replaced</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Partial replacement</td>
<td>No LSL</td>
</tr>
<tr>
<td>&lt; 5 μg/dL</td>
<td>598</td>
<td>17,029</td>
</tr>
<tr>
<td>5–9 μg/dL</td>
<td>105</td>
<td>1502</td>
</tr>
<tr>
<td>≥ 10 μg/dL</td>
<td>27</td>
<td>236</td>
</tr>
<tr>
<td>Total</td>
<td>730</td>
<td>18,857</td>
</tr>
</tbody>
</table>

* 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.

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<tbody>
<tr>
<td></td>
<td>Model 1 (OR (95% CI) for LSL (n=3711))</td>
<td>Model 2 (OR (95% CI) for LSL, controlling for age of housing (n=2180))</td>
<td>Model 1 (OR (95% CI) for LSL (n=7232))</td>
</tr>
<tr>
<td>BLL*</td>
<td></td>
<td></td>
<td>Model 1 (OR (95% CI) for LSL (n=3781))</td>
</tr>
<tr>
<td>2 &lt; 3</td>
<td>1.0</td>
<td>1.0</td>
<td>1.0</td>
</tr>
<tr>
<td>3 &lt; 5</td>
<td>0.9 (0.6, 1.4)</td>
<td>1.0 (0.6, 1.5)</td>
<td>1.4 (1.2, 1.7)</td>
</tr>
<tr>
<td>≥ 5</td>
<td>1.0</td>
<td>1.1 (1.1, 2.4)</td>
<td>2.1 (1.7, 2.5)</td>
</tr>
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</table>

**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), by 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.

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<tbody>
<tr>
<td></td>
<td>Model 1 (OR (95% CI) for LSL (n=3711))</td>
<td>Model 2 (OR (95% CI) for LSL, controlling for age of housing (n=2180))</td>
<td>Model 1 (OR (95% CI) for LSL (n=3781))</td>
</tr>
<tr>
<td>Children &gt; 16 months to &lt; 6 years of age</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt; 10</td>
<td>1.0</td>
<td>1.0</td>
<td>1.0</td>
</tr>
<tr>
<td>≥ 10</td>
<td>2.3 (2.0, 2.7)</td>
<td>1.5 (1.3, 1.8)</td>
<td>3.0 (2.6, 3.6)</td>
</tr>
<tr>
<td>Children ≤ 16 months of age</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt; 10</td>
<td>N=3711*</td>
<td>N=2180*</td>
<td>N=3781*</td>
</tr>
<tr>
<td>≥ 10</td>
<td>3.4 (2.3, 5.0)</td>
<td>3.7 (2.2, 6.2)</td>
<td>3.1 (2.3, 4.3)</td>
</tr>
</tbody>
</table>

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

Includes children living in homes where the LSL was fully replaced prior to their BLL test.

Includes only children for whom there was data on LSL.

Includes only children for whom there were data on both LSL and age of housing.

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 trend or inter-laboratory or intra-laboratory 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, substandard 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 µg/dl, although not for BLLs ≥ 10 µg/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 µg/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 time as likely to have had BLLs ≥ 10 µg/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 µg/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 moulting 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 are exposed. The consequences of changes in water disinfection practices on a range of health issues including exposure to lead are.


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


References


