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Elevated Blood Lead in Young Children Due to Lead-Contaminated Drinking Water: Washington, DC, 2001−2004

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Incidence of EBL (blood lead ≥10 μg/dL) for children aged ≤1.3 years in Washington, DC increased more than 4 times comparing 2001−2003 when lead in water was high versus 2000 when lead in water was low. The incidence of EBL was highly correlated (R^2 = 0.81) to 90th percentile lead in water lead levels (WLLs) from 2000 to 2007 for children aged ≤1.3 years. The risk of exposure to high water lead levels varied markedly in different neighborhoods of the city. For children aged ≤30 months there were not strong correlations between WLLs and EBL, when analyzed for the city as a whole. However, the incidence of EBL increased 2.4 times in high-risk neighborhoods, increased 1.12 times in moderate-risk neighborhoods, and decreased in low-risk neighborhoods comparing 2003 to 2000. The incidence of EBL for children aged ≤30 months also deviated from national trends in a manner that was highly correlated with 90th percentile lead in water levels from 2000 to 2007 (R^2 = 0.83) in the high-risk neighborhoods. These effects are consistent with predictions based on biokinetic models and prior research.

Introduction

The Washington, DC "lead in drinking water crisis" was triggered by a change in disinfectant from free chlorine to chloramine in November 2000 (*1*). The switch in disinfectant reduced the concentration of potential carcinogens (a byproduct of chlorine disinfection) to levels below those specified by the U.S. Environmental Protection Agency (EPA). However, the chloramine also altered the water chemistry and unexpectedly caused lead to leach from lead service line pipes (*1, 2*) and other plumbing materials such as leaded brass and solder (*1*). The resulting contamination affected water lead levels (WLLs) in homes throughout the city.

Two previous studies of blood lead levels (BLLs) relative to the high WLLs in Washington, DC have been published (*3, 4*). While the high WLLs appeared to have some impact on the incidence of BLLs ≥ 5 μg/dL (*3*), no evidence was found of increased incidence over the 10 μg/dL level of concern set by the Centers for Disease Control and Prevention (CDC) for children aged <6 years. Blood lead levels exceeding the CDC level of concern are termed "elevated blood lead" (EBL) in this work.

A close examination of the two prior studies reveals noteworthy limitations. Neither study focused on infants, who are most vulnerable to harm from lead in water (*5*−*7*) due to their small body weight and heavy reliance on water as a major component of their diet in the case of infants using reconstituted formula. Moreover, both studies lumped all the blood lead data for Washington, DC together, an approach that can "mask disparities among communities and camouflage pockets of high risk" relative to smaller area analysis at the neighborhood or zip code level (*8*). This research addressed these limitations.

Methodology and Data

Environmental Data. *Water Lead Data.* Measurements of "total lead (*9*)" in potable water were collected by the local water utility using EPA-approved methodology. A "first draw" sample refers to a 1 L sample collected from a tap after greater than 6 h holding time in the household plumbing. After first draw samples are collected, water is flushed for a short time period (typically 30 s to 5 min) and a 1 L "second draw" sample is collected.

Two data sets of potable water lead concentrations were used throughout this research. Data on WLLs in homes with lead pipe during 2003 were collected by the local water utility from over 6000 Washington, DC homes with lead service line pipe. The WLL EPA Monitoring Data (2000−2007) were collected by the water utility specifically for compliance with EPA regulations. Compliance is determined by using the "90th percentile lead," which is the 90th percentile of the cumulative distribution of first draw lead samples collected within a given time period. The monitoring data were reorganized into calendar year time periods for which corresponding blood lead measurements were compiled. For example, the official 2002 EPA monitoring round at the utility included water samples collected between July 1, 2001 and June 30, 2002. The samples collected between July 1, 2001 and December 31, 2001 from that round were used in calculations of the 90th percentile WLLs for the second half of 2001. The remaining water samples from that round were included in calculations of 90th percentile WLLs for calendar year 2002. Several audits have been conducted on the utility's EPA monitoring data (*10*), and trends in 90th percentile lead used in this study are not strongly impacted by remaining unresolved errors in the data.

Lead Pipes by Zip Code and other Demographic Data. The number of lead pipes in each zip code was determined using a database provided by the CDC (*3*). Demographic data within each zip code were obtained from the U.S. Census.

Identification of Sensitive Population. *Predicted Impact of WLLs on BLLs.* In April 2004 the US EPA National Center for Environmental Assessment (NCEA) modeled the impact of high WLLs on the BLLs of children in the city (See Supporting Information Reports 1−3). The NCEA results and additional assumptions were used to make predictions of EBL incidence for children who had consumed formula reconstituted with tap water during their first year of life, and children aged 1−6 years who did not consume formula but drank tap water (see Supporting Information 1). A one-year-old infant living in a Washington, DC home with lead service line pipe and consuming formula made from tap water was predicted to have a 21% likelihood of EBL in 2003. The overall prediction was that there would be 600−700 cases of EBL for children under 6 years of age in 2003 due to the high WLLs. This estimate of 600−700 cases represents only

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0.1% of the total city population and only 1.5% of the population under age 6 years.

Any attempt to correlate WLLs with incidence of EBL is also confounded by the fact that incidence of EBL in the US for children aged <6 years declined from 3.96% in 2000 to 2.0% in 2003 (*11*). This 1.96% decline is of the same order, or even higher, than the predicted 1.5% increase in Washington, DC due to the high WLLs. If the impacts of the high WLLs are to be quantified, methods that can account for the reduction in the national incidence of EBL must be considered.

For this work, additional modeling was conducted using the International Commission for Radiation Protection (ICRP) biokinetic model, to more precisely identify the population(s) most sensitive to lead in water. The ICRP biokinetic model has been successfully used to predict seasonal or weekly trends in BLLs (*12, 13*). We confirmed that the population most sensitive to EBL from high WLL is children aged ≤1 year consuming reconstituted infant formula. Moreover, the modeling indicates that some evidence of EBL due to consumption of formula in the first year of life should persist until age ≤30 months (Supporting Information 1). This result is consistent with expectations based on other research (*5*−*7*). Thus, children aged ≤1 year and children aged ≤30 months were selected as target populations for this research.

Blood Lead Data. *CDC Database.* A blood lead database from the 2004 CDC study (*3*) was obtained through the Freedom of Information Act.

Children's National Medical Center (CNMC) Blood Lead Database. A study of blood lead was reviewed and approved by the Institutional Review Board at Children's National Medical Center. The CNMC data containing >28,000 records from 1999−2007 were sorted and data for children aged ≤30 months were extracted. If there were multiple measurements of BLL for the same individual, a convention was followed in which the highest recorded blood lead for each child was retained and all other measurements were deleted (*14, 15*). This approach ensures that calculations of EBL incidence in the population are not skewed by multiple measurements from the same individual.

The 1999 CNMC data are treated differently in this work because no 1998 data are available. The convention of removing multiple blood lead measurements per child makes the 1998 data influential on the 1999 data set (children often have blood lead measurements at 1 and 2 years). Thus, with one exception, only CNMC data from 2000−2007 are used in this work.

Results

After discussing temporal trends in WLLs throughout the city from 2000 to 2007, the effects of WLLs on EBL for children aged ≤1.3 years are examined. Thereafter, a neighborhood analysis is presented for children aged ≤30 months.

Temporal Trends in WLLs in Washington, DC. The 90th percentile WLLs (Figure 1) increased after the switch to chloramine disinfectant in November 2000 (*1*). The exact point at which the WLLs began to rise after the switch in disinfectant cannot be precisely determined. Therefore, 2001 is considered a transition year and data are divided into halves (data from January to June 2001 are termed 2001a and data from July to December are termed 2001b). Other support for dividing 2001 in half is presented in Supporting Information 2. The 90th percentile WLLs remained higher than the EPA regulatory "action level" of 15 ppb from 2001 to 2004 (Figure 1) before dropping back below the action level in 2005. The drop in WLLs in 2005 is temporally linked to dosing of an orthophosphate corrosion inhibitor (from August 2004 onward) to mitigate high WLLs.

Following a January 31, 2004 front page *Washington Post* article that revealed the widespread problem with elevated

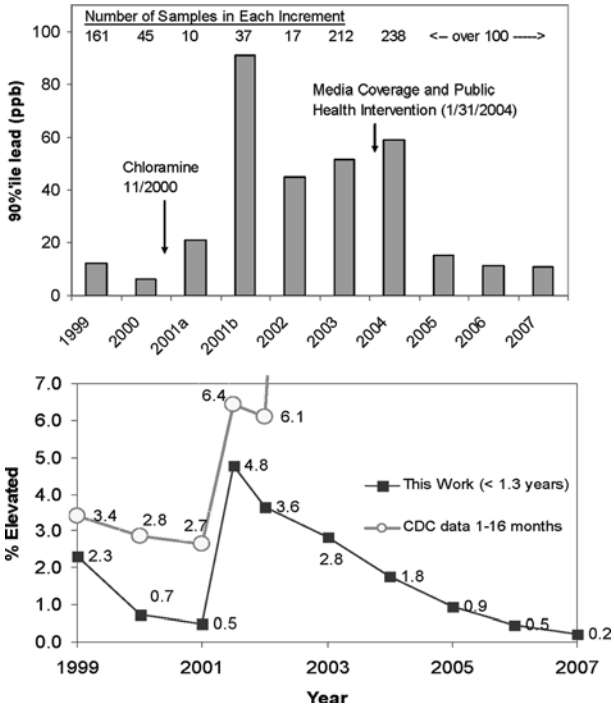

FIGURE 1. Temporal variation of lead in water (90th percentile water lead) and key events related to lead exposure in Washington, DC (top). Trends in EBL incidence for children aged ≤1.3 years (bottom).

WLLs, the public was eventually instructed to flush their water lines >10 min before collecting water for cooking and drinking. More than 20,000 lead filters were also mailed to homes with high risk of elevated WLLs in early 2004. Assuming these strategies were effective in largely abating human exposure to elevated WLLs, mid-2001 to early 2004 is the time period of greatest unprotected exposure to high WLLs.

Correlation Between EBL and WLLs for Children ≤1.3 Years of Age. Although the most highly impacted population is children aged ≤1 year there are insufficient data for this population group to support a statistically valid analysis. Only 0.62% of the overall CNMC data are for children aged ≤9 months and only 6.6% of the data are for children ≤1 year of age. The age group closest to the target population with adequate data (27% of the overall data) is children aged ≤1.3 years.

The incidence of EBL for children aged ≤1.3 years continued its decades long decline from 1999 through the first half of 2001 (Figure 1). But in the second half of 2001 the incidence of EBL abruptly increased by 9.6 times versus the first half of 2001. This 4.3% increase (from 0.5% to 4.8%) is not inconsistent with expectations presented in Supporting Information 1, especially considering that 90th percentile WLLs were higher in late 2001 than in 2003 (rough predictions in Table S1 are based on 2003 data). In 2002 and 2003, the incidence of EBL was ≥4 times higher than in 2000. In fact, EBL incidence did not return to levels observed in 2000 until about 2005, when lead in water once again met EPA standards. A proportions test in *R* (*16*) determined that the EBL incidence in the years 2001, 2002 and 2003 is greater than in 2000 with >95% confidence. A linear correlation between the incidence of EBL and the 90th percentile lead from 2000 to 2007 (see Supporting Information 5, Figure S7) is very strong (R^2 = 0.81).

The CDC database (*3*) was analyzed for the same trends. The incidence of EBL for children aged 1−16 months showed trends similar to the CNMC data (Figure 1, bottom). The absolute values of the CNMC data and the CDC data

TABLE 1. Summary Data for Neighborhoods of High, Moderate, and Low Relative Risk of Exposure to High Elevated WLLs

relative exposure risk	est. lead pipes	% of total pop. in city	pop. (1000)	% pop. with lead pipe	% 1st draw over 100 ppb	% pop. above indicated WLL (ppb)		
						1st draw >100	2nd draw >100	1st draw >400
high	10086	22	126.3	17.6	15.0	2.63	3.43	0.13
moderate	14743	55	314.3	10.3	9.4	0.97	1.59	0.02
low	1318	23	131.4	2.2	12.8	0.28	0.37	0.00

are not expected to be in agreement, because the CDC included multiple measurements of blood lead for children which tends to skew the EBL incidence higher. CDC data for 2003 are not plotted on the graph, because only 90 children were identified as age 1−16 months for that year, of which 31 had elevated blood lead (34% EBL incidence).

Data from the CDC study (*3*) were then compared to the blood lead data (>28,000 records) from CNMC. In theory, the CNMC data are a subset of the more expansive data compiled and maintained by the DC Department of Health and which were used in the CDC study. However, a comparison of records between the two databases for the year 2003 revealed an error rate of more than 50%. That is, there was less than a 50% chance that a given record in the CNMC database matched a record in the CDC data in 5 domains: sample collection date, subject age, sample recording date, zip code, and BLL. Because repeated attempts to resolve this and other discrepancies in the CDC data were not successful, only the CNMC data were used for analyses and conclusions in this work.

Correlation Between EBL and WLLs for Children Aged ≤30 Months. No strong temporal trends or correlations between EBL incidence and the varying WLLs were observed for children aged ≤30 months if the data were analyzed across the entire city (data not shown). A neighborhood analysis of the data was then conducted.

High-, Moderate-, and Low-Exposure-Risk Neighborhoods. During 2003, the local utility conducted intensive sampling in Washington, DC homes with lead service pipe. Contrary to the popular perception that lead leaching to water is a fairly reproducible phenomenon from home to home, WLLs present in the first and second draw (flushed) samples from home to home vary dramatically (*9, 17*). For instance, in homes known to have lead service line pipe the second draw samples collected from 33% of homes had WLLs below the 15 ppb EPA action level. But 17% were above 100 ppb, 1% were above 1,000 ppb, and one sample contained 48,000 ppb.

A Freedom of Information Act request of the water utility revealed that a "geographic phenomena" was identified that played a key role in the observed variability of water lead in homes throughout the city (Supporting Information 3). Specifically, certain neighborhoods were "hot spots" for high water lead. While the utility would not provide documentation of the neighborhood analysis, their 2003 lead in water data were scrutinized for geographic trends based on zip code.

The analysis demonstrated that relative risk of exposure to high lead in water was a strong function of zip code (see Supporting Information 4). To capture the risk of exposure to high WLLs for the different neighborhoods, while also pooling data to maintain sufficient statistical power, the city was demarcated into neighborhoods that had relatively high risk (22% of the population), moderate risk (55% of the population), and low risk (23% of the population). In the high-risk part of the city, 2.63% of the population had first draw WLLs above 100 ppb (Table 1). This is 9.4 times higher than the 0.28% of the population having first draw WLLs above 100 ppb in the low-risk part of the city, and 2.7 times higher than in the moderate-risk part of the city (Table 1). The population living in the high-risk neighborhoods also had much greater likelihood of exposure to second draw lead over 100 ppb or to first draw lead over 400 ppb when compared to the moderate- and low-risk neighborhoods (Table 1).

Temporal Trends in EBL. The incidence of EBL for children aged ≤30 months had strong temporal trends that differed based on neighborhood risk level (Figure 2). In the high-risk neighborhoods EBL incidence increased from 2.5% in 2000 when WLLs were low, to 6% in 2003 after WLLs had been high for a few years. Thus, the incidence of EBL cases increased 2.4 times in 2003 versus 2000 in the high risk neighborhoods. The incidence of EBL dropped rapidly in the high-risk neighborhoods beginning in 2004. In the moderate-risk part of the city the EBL incidence was higher in each of the years 2001−2003 when water lead levels were high, relative to 2000 when water lead levels were low. But in neighborhoods of the city with the lowest risk of exposure to high WLLs, the percentage of children aged ≤30 months with EBL dropped steadily from 2000 to 2007.

Comparing the high-risk part of the city to the low-risk part of the city using a proportions test in R shows no significant difference in EBL incidence for the year 2000 (before WLLs were high) or for 2001 ($p = 0.544$ and 0.330, respectively). But utilizing the same test in 2002, 2003, and 2004 shows a statistically higher incidence of EBL in high-risk neighborhoods relative to low-risk neighborhoods ($p =$ 0.024 for 2002, 0.037 for 2003, and 0.006 for 2004). This analysis shows that the high WLLs had a very significant impact on EBL incidence for children aged ≤30 months in the neighborhoods with high WLLs.

Comparison of EBL in Washington, DC to the U.S. Trend in BLLs, 2000−2007. National trends in EBL incidence from 2000 to 2006 (*11*) are reasonably fit by an exponential decay model with an annual rate constant of −0.1867/year ($R^2 =$ 0.99). Extrapolation of this trendline using the year 2000 as time = 0 provides a basis for relating the Washington, DC blood lead data to the national trend. For example, the calculated "Δ high risk 2003" (Figure 2), is the difference between the U.S. trendline and the DC data. This represents the increased incidence of EBL in the high-risk DC neighborhoods in 2003, compared to what would have occurred if the national trend had been followed.

Correlation between WLL and Deviations from National BLL Trends. The correlation between the increased incidence of EBL in DC children aged ≤30 months versus national trends, and the reported 90th percentile WLL concentrations for the city, was dependent on the neighborhood risk level (Figure 2). In neighborhoods with the highest WLLs a strong positive linear correlation was established between the increased incidence of EBL and the 90th percentile WLL concentration ($R^2 = 0.82$). In the moderate-risk section of the city the slope and correlation were slightly lower ($R^2 =$ 0.71). The weak correlation ($R^2 = 0.50$) in the low-risk section of the city is to be expected, because the population in these neighborhoods had relatively low likelihood of exposure to high WLLs (Table 1). The slope of the trend-line in the highest risk part of the city is approximately double that observed

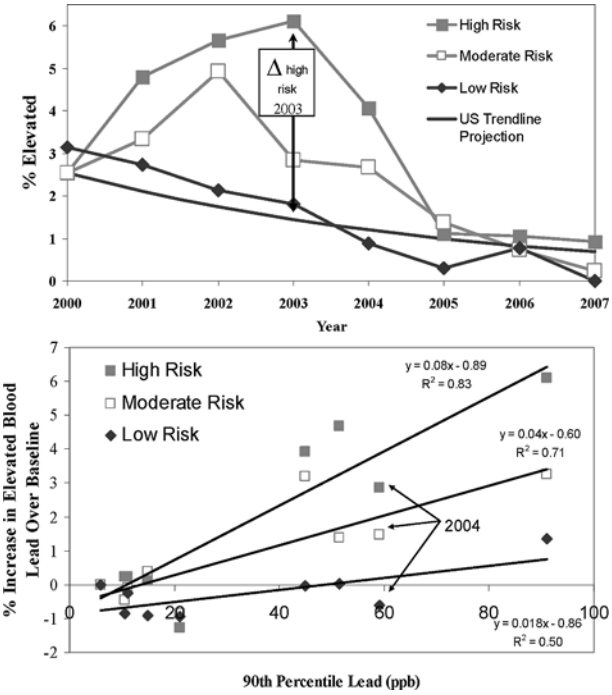

FIGURE 2. Temporal trends in incidence of EBL for children age ≤30 months. The deviation from the U.S. trendline is determined by the difference between the actual data and the projected U.S. trendline (top). Correlation between increased incidence of elevated blood lead in Washington, DC children aged ≤30 months and 90th percentile lead (bottom).

in the moderate-risk part of the city, and 4.4 times higher than in the low-risk part of the city.

If the 2001 data are not split into a first and second half, R^2 in the high-risk part of the city drops from 0.83 to 0.65, R^2 in the moderate-risk part of the city drops from 0.71 to 0.45, and R^2 in the low-risk part of the city drops from 0.50 to 0.18. (Supporting Information 5). The 2004 data also deviate significantly from the trendline (Figure 2 bottom), in that the high WLLs did not increase the percentage of children aged ≤30 months with EBL to the same extent as they did in 2001−2003. This is to be expected, since public health interventions were implemented in early 2004. If 2004 were treated as a transitional year and excluded from the analysis, R^2 would increase for the correlations (Supporting Information 5).

Discussion

EBL Cases Attributed to High WLLS Versus Predictions of Bio-Kinetic Model. The estimated number of children with EBL in Washington, DC due to the lead-contaminated water can be roughly estimated using the results of Figure 2 and the population of children aged ≤30 months in each part of the city (low-, moderate-, and high-risk neighborhoods). It is estimated from the CNMC analysis that 342 children in DC aged ≤30 months had EBL in 2003 due to high WLLs, and that 517 additional children aged ≤30 months had EBL from high WLLs in 2002. The corresponding exposure model predictions including all 1 year old, all 2 year old, and 50% of the 3 year old category (to approximate children aged ≤30 months) is for 170 cases in 2003 (Supporting Information 1). The discrepancy (342 estimated cases of EBL using the CNMC analysis vs 170 predicted for children aged ≤30 months) is not large given the model assumptions. It is not even unexpected, since the exposure model predictions did not include cases for which BLLs would be raised above 10 μg/dL from a combination of sources that include water. The most significant impacts of the high WLLs on EBL incidence probably occurred in the second half of 2001 (Figure 1, Figure 2, Supporting Information Figures S13 and S14), but calculating an increased number of EBL cases in that time period is beyond the scope of this work.

Lack of Monitoring Data for the Population Most Vulnerable to High WLLs. CDC recommends that BLL blood lead of children be screened at 1 and 2 years of age, "based on the fact that children's blood lead levels increase most rapidly at 6−12 months age and peak at 18−24 months (*18*)." These guidelines were developed from studies conducted in Cincinnati and elsewhere, where lead dust and lead paint were the predominant sources of exposure and water lead levels were low (*19*). In contrast, previous research has demonstrated that BLLs begin to rise rapidly when infant formula contains elevated lead ((*7*), see Supporting Information 2 Figure S6). Thus, when lead contaminated water is the sole or main source of lead exposure for infants, it is logical to expect that blood lead levels would tend to peak at ages much younger than 18−24 months (Supporting Information 1).

In earlier research on effects of high WLL on EBL for Washington, DC residents, it was stated that the blood lead monitoring was "focused on identifying children at highest risk for lead exposure (*3*)." This statement is correct from the perspective of lead paint and lead dust, but it is not necessarily accurate from the perspective of exposure to lead from water. Indeed, because so little blood lead data had been collected in Washington, DC for the population most vulnerable to high WLLs, no statistically valid conclusions are possible relative to incidence of EBL for children aged ≤1 year. The data presented herein for children aged ≤1.3 years (which are actually mostly data for children aged 1−1.3 years) supports the prediction that the impacts would be highly significant.

Other Considerations and Biases. A significant number of children aged ≥30 months was likely to have EBL during 2000−2003 because of exposure to high WLLs (Supporting Information 1). Moreover, even in the low-risk neighborhoods many children probably had EBL due to exposure to high WLLs. But the increased incidence of these cases cannot be readily detected in the BLL monitoring data in this work for reasons discussed previously. It is also inevitable that some misclassification of children's addresses will occur in a study of this nature, in that some children in high-risk neighborhoods would be misclassified as living in low-risk neighborhoods and vice versa. To the extent that such random bias occurred, it would tend to make the reported correlations between EBL and WLLs less significant than they actually were.

The Literature Revisited. Differences in conclusions between this work and the earlier CDC study (*3*) are mostly attributed to the type of analysis and interpretation, as opposed to discrepancies between the two databases discussed previously. In a recent discussion of the original CDC results, Levin et al. (2008) noted that the percentage of BLL measurements ≥5 μg/dL declined by 70% from 2000−2003 across the U.S., but did not decline at all in Washington, DC during the period of high WLLs (*20*). The obvious implication is that the high WLLs in Washington, DC countered the expected decline in BLLs that would have otherwise occurred, even for the general population that was analyzed in the CDC report.

Applying the Levin et al. (2008) logic to a closer examination of the CDC (2004) data suggests that the rate of decline in BLL measurements >10 μg/dL across the city was also reduced during the time period that WLLs were high. For example, the CDC study reported that from 2000 to 2003, the incidence of BLL measurements >10 μg/dL in homes with lead pipe declined by 28%, whereas

incidence of EBL declined 50% nationally in the same time period (*11*). Indeed, the original CDC study did find a slight (but insignificant) increase in incidence of EBL in 2001 versus 2000 for residents living in homes with lead pipe (*3*). When the CDC 2001 data are broken into halves according to the approach of this work, the second half of 2001 has an anomalous increase in EBL incidence relative to what occurred in 1999 or 2000 for all ages tested (Figure 1; Supporting Information 6). The results for second half of 2001 are deserving of increased scrutiny in light of the very high WLLs throughout the city in July and August 2001 (Supporting Information Figures S4 and S14).

There are two other studies that examined the impact of WLLs on BLLs of DC residents. Guidotti et al. (2007) report a low incidence of EBL in a population tested well after high WLLs were front page news (*4*). Another portion of the CDC (2004) study reported no cases of EBL in 2004 for residents living in homes where second draw WLLs were over 300 ppb (*3*). In both of these studies there was a delay of months to a year between the time that consumers were first informed of hazardous WLLs and the actual measurement of their BLL (*21*). Since the half-life of lead in blood is 28−36 days, these results cannot be construed to indicate lack of harm from exposure to the lead contaminated water (*22*).

The Guidotti et al. (2007) study also erroneously identified critical dates and facts regarding the lead in water contamination event that skewed interpretations (*4*). For example the authors state that:

(1) chloramine was first added to the water supply in November 2002 [the actual date for addition of chloramines was November 2000 [see Supporting Information 7]
(2) WLLs showed an "abrupt rise" in 2003 [the WLLs had risen by the second half of 2001 as per Figure 1]
(3) the Washington, DC population had been protected by "massive public health interventions" starting in 2003 [the significant public health intervention did not begin until after the story was front page news in early 2004, see Supporting Information 7].

This may explain why the conclusions of Guidotti et al. (2007) differ from those of Miranda et al. (2006), who found a significant correlation between children's BLLs and a switch to chloramine disinfection in North Carolina (*14*).

Overall, this research demonstrates that the experience in Washington, DC is consistent with decades of research linking elevated WLLs to higher BLL and EBL (*23, 24*). Studies in France (*25*), Scotland (*26*) and Germany (*27*) correlated WLLs to adult BLLs, even for adults drinking water after corrosion control markedly reduced water lead levels. Lanphear has also noted a correlation between BLLs and higher WLLs in a U.S. city in which no system-wide problem with WLLs was occurring (*28*). Lead in potable water is therefore a viable explanation for some of the 30% of elevated BLL cases that occur nationally for which no paint source can be found (*20*), and may even be a significant contributor to EBL in cases where lead paint is identified as a hazard in the home. Assumptions by the CDC that high WLLs are rarely the cause of EBL in children should be re-evaluated.

Acknowledgments

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Supporting Information Available

Seven supporting analyses and three reports (EPA, 2004). This material is available free of charge via the Internet at http://pubs.acs.org.

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