

Predicting Children's Blood Lead Levels From Exposure to School Drinking Water in Seattle, Washington, USA

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Objective.—Lead exposure through drinking water is of increasing interest with little known about its potential childhood health impact. In 2004, school testing in Seattle, Washington, found lead concentrations in drinking water that exceeded national guidelines (>20 ppb). On the basis of these data, we estimated potential blood lead levels (BLLs) in elementary school children to better understand the potential health risks posed by these exposures.

Methods.—We used the US Environmental Protection Agency Integrated Uptake Biokinetic Model for Lead in Children to predict geometric mean BLLs. We modeled typical-case and worst-case scenarios for children in 71 elementary schools on the basis of drinking water lead concentrations results from 2004.

Results.—The estimated geometric mean BLLs under a typical scenario for each school ranged from 1.6 to 2.5 $\mu\text{g}/\text{dL}$. The worst-case scenario predicted geometric mean BLLs ranging

from 1.7 to 5.0 $\mu\text{g}/\text{dL}$. All modeling yielded predicted BLLs well below the Centers for Disease Control and Prevention's public health goal of <10 $\mu\text{g}/\text{dL}$.

Conclusions.—Our modeling suggests drinking water exposures up to 10–15 times the Environmental Protection Agency guideline are unlikely to result in BLLs exceeding the current guidelines of the Centers for Disease Control and Prevention in the absence of other significant exposure sources. In Seattle, elevated school drinking water lead concentrations are not a significant source of lead exposure in school-age children. Further characterization of drinking water impacts are merited only if younger-age children are consuming water or if water lead concentrations are higher than those in this study.

KEY WORDS: children; lead exposure; lead poisoning; school drinking water

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Low-dose lead exposure in early childhood affects early neurodevelopment and produces measurable cognitive deficits later in life.^{1–3} The Centers for Disease Control and Prevention (CDC) recommend screening efforts to identify children with blood lead levels (BLLs) exceeding 10 $\mu\text{g}/\text{dL}$. However, recognizing that studies suggest no threshold of effect exists, the CDC continues to highlight the importance of primary prevention of childhood lead exposures.^{4–6}

Lead exposure through drinking water has not been identified as a major source of childhood lead exposure and has not been the focus of primary prevention efforts that largely target lead contaminated dust and soil.⁷ Recent water sampling efforts in communities and schools demonstrate a wide range of drinking water lead concentrations, including very high concentrations in some settings.^{8–10} This has fueled public and scientific interest in potential adverse health effects,⁸ but reports in the lay

press and published literature rarely translate lead contaminated drinking water exposures into public health risks through modeling efforts.

The US Environmental Protection Agency (EPA) regulates public water supplies under the Safe Drinking Water Act, which sets a lead action level of 15 ppb. However, their jurisdiction does not extend to drinking water in public school buildings.¹¹ The EPA does provide a non-enforceable guideline for schools that recommends drinking water lead not exceed 20 ppb.¹² The difference in the Safe Drinking Water Act action level of 15 ppb and the EPA guideline of 20 ppb reflects different sample collection methods for residential and school sites. Lead content in most water supplies reflects corrosion of lead-based plumbing materials rather than primary contamination of source water.¹³ In the Seattle, Washington, school system, lead-based plumbing and drinking fountain components are the primary contributors to lead-contaminated drinking water.¹⁴

In Seattle, elevated water lead concentrations were brought to public attention by parental concerns regarding discolored drinking water from school fountains. These concerns were followed by comprehensive testing by the Seattle Public Schools that found elevated lead concentrations in drinking water (>20 ppb) in many schools.¹⁵ In response, the school district provided bottled water to affected schools and initiated extensive remediation efforts.

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We sought to provide a health risk perspective for the available lead exposure information in the Seattle Public Schools' drinking water. Currently, the best clinical tool for diagnosing overexposure to lead is the BLL. However, this testing was not part of the response in Seattle. Historically, elevated BLLs are uncommon in the Seattle pediatric population, and screening is not routinely included in preventive care. Therefore, we modeled predicted BLLs in children exposed to lead contaminated school drinking water.

METHODS

Population

Census data from the 2004–2005 school year estimated elementary school enrollment at 21 874 students, and the school district conducted sampling in 71 elementary schools.¹⁶ We predicted BLLs for 5–6-year-old children because this is the youngest school-age group in the Seattle Public Schools. In addition, these children are vulnerable, given their young age among the childhood school population. Most childhood lead exposure studies have focused on preschool-age children because the studies use a paradigm that lead neurotoxicity is greatest during early life events (fetal to age 5) of neurological development. However, recent data suggest that neurotoxicity also occurs in children in older age groups.^{17,18} We therefore focused on the youngest school-age population that is likely to be in attendance for a full school day.

Seattle School Water Sampling Data (2004)

From April to June 2004, the Seattle Public Schools collected drinking fountain and faucet water samples from 71 elementary schools to determine lead concentrations. The Economic and Engineer Services Corporation collected first-draw and running water samples for each fountain or faucet. First-draw samples were collected after nonuse of the faucet or drinking fountain for 8–18 hours before sample collection, and running samples were collected after the faucet or drinking fountain water ran for 30 seconds. Collection volume was 250 mL for both standing and running samples. The Economic and Engineer Services Corporation used standardized sampling techniques as outlined in EPA guidelines for water testing.¹¹ The Seattle Public Schools published results of this testing on their Web site for public review.¹⁵ We used these publicly available data to estimate BLLs for 5–6-year-old children in elementary school as described below.

BLL Predictive Modeling

We used the EPA Integrated Exposure Uptake Biokinetic Model for Lead in Children (IEUBK) to predict geometric mean BLLs on a school-by-school basis.¹⁹ This model incorporates lead exposure input from multiple exposure pathways—air, soil, water, and food consumption—to estimate total lead exposure. Geometric mean BLLs are predicted on the bases of total lead intake, and the biokinetic properties of growth, digestion, and metabolism for different age groups; it assumes 3 months of consistent exposure through these pathways.¹⁹ The

3-month period of exposure is based on the premise that if a child is exposed to the same environment over time, he or she will have a steady-state BLL. We estimated geometric mean BLLs for 5–6-year-old children.

We used a background soil lead content of 24 ppm in the model. In Washington State, recent sampling of soil to determine background levels found soil lead concentrations ranging from 11 to 24 ppm, with the highest levels found in the Puget Sound region, which includes Seattle.²⁰ The lead concentration in air was input as 0.1 $\mu\text{g}/\text{m}^3$, the default value for the IEUBK model. This value derives from nationwide monitoring for background concentrations. Since closure of a lead smelter in Seattle in 1998 (maximum concentration that year was 2.03 $\mu\text{g}/\text{m}^3$ measured in close proximity to the smelter), air concentrations of lead in the Puget Sound have been less than 0.1 $\mu\text{g}/\text{m}^3$.²¹

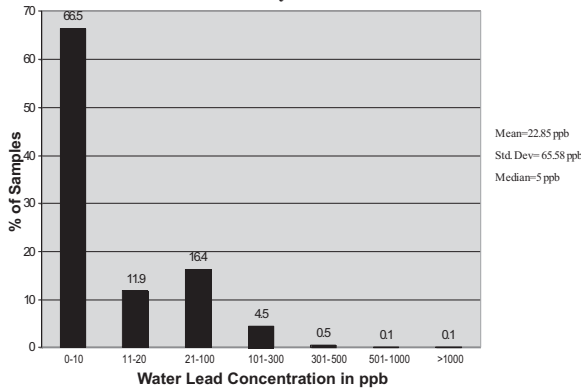
For drinking water exposure characterization, we selected the IEUBK model default for total daily water intake for a 5–6-year-old (0.58 L). This is based on EPA data on age-based consumption patterns.^{19,22} We estimated that a child drank half of his or her total daily water at home and half at school, or about 0.29 L (1 cup) at home and the same amount at school. We input home drinking water lead concentration as 10.3 ppb, the 90th percentile measured in high risk homes per datum available from the municipal water supplier.²³ We estimated that 25% of school water consumed came from standing samples and 75% came from running samples. We modeled 2 scenarios for each school to represent a “worst-case exposure” and “typical-case exposure.” We used the 90th percentile measured lead concentrations of all standing and running samples at each school to represent the potential worst-case scenario for children's exposure at that school. Specifically, we input each individual school's 90th percentile standing water lead concentration and 90th percentile standing water concentration for the worst-case scenario prediction. For the typical exposure scenario, we used the median lead concentration of all measured standing and running samples from a school to model a typical exposure situation.

RESULTS

Data were available for 1905 standing drinking fountain samples and 1850 running drinking fountain samples from the 71 elementary schools evaluated. The lead concentrations ranged from below the level of detection (<1 ppb) to 1600 ppb in standing samples and from below the level of detection (<1 ppb) to 370 ppb in running samples. The 90th percentile for all standing samples combined was 49 ppb, and the 90th percentile for all running samples combined was 7 ppb. The median values were 5 ppb and 1 ppb for the standing and running samples, respectively.

Figures 1 and 2 show the distribution of water lead concentrations for standing and running water samples. The standing samples had lead concentrations ranging from below the level of detection to 20 ppb (Figure 1) with 412 samples (22 %) above the EPA recommended guideline for safe school drinking water. The running water samples also had lead concentrations ranging from below

**Water Lead Concentrations: Standing Samples* (N=1,905)
71 Elementary and K-8 Schools**



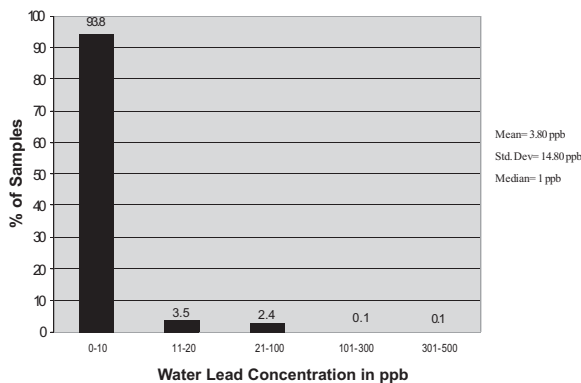
*Standing Samples – samples measured after non-use of the faucet or drinking fountain for approximately 8-18 hours

Figure 1. Bar graph illustrating water lead concentrations in 1905 standing samples in a total of 71 elementary and K-8 schools. Standing samples are samples measured after nonuse of the faucet or drinking fountain for approximately 8–18 hours.

the level of detection to 20 ppb (Figure 2), with 49 samples (2.6%) above the EPA recommended guideline for safe school drinking water. The nonnormal distribution was weighted toward lower concentrations with geometric mean concentrations of 6.1 ppb for standing water samples and 1.9 ppb for running water samples.

Predicted worst-case scenario geometric mean BLLs for 5–6-year-old children in these schools ranged from 1.7 to 5.0 $\mu\text{g}/\text{dL}$ (Figure 3). By using the 90th percentile water lead concentrations at each school to represent school-based exposure, 100% of drinking water lead concentrations yielded predicted geometric mean BLLs that were less than or equal to 5 $\mu\text{g}/\text{dL}$. Predicted BLLs for children in 3 schools were 4–5 $\mu\text{g}/\text{dL}$, and 95.5% of all BLLs were below 4 $\mu\text{g}/\text{dL}$. The predicted geometric mean BLLs using the typical-case scenario assumptions (median concentration of drinking water sources at the school used to represent exposure) ranged 1.6–2.8 $\mu\text{g}/\text{dL}$, with a large majority (97%) $\leq 2.5 \mu\text{g}/\text{dL}$.

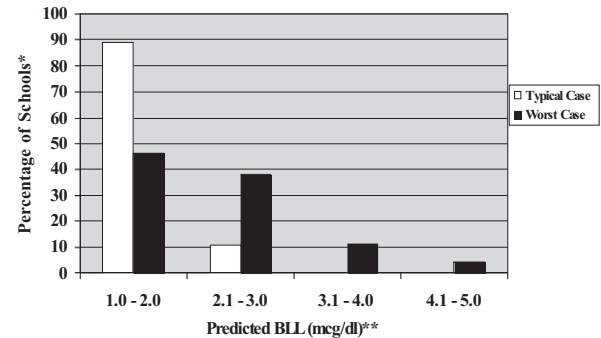
**Water Lead Concentrations: Running Samples* (N=1,850)
71 Elementary and K-8 schools**



*Running Samples – samples measured after faucet or drinking fountain had been running for approximately 30 seconds

Figure 2. Bar graph illustrating water lead concentrations in 1850 running samples in 71 elementary and K-8 schools. Running samples are samples measured after faucet or drinking fountain had been running for approximately 30 seconds.

Predicted Geometric Mean BLL for Worst Case and Typical Case Scenarios Using Observed Lead Concentrations in School Drinking Water



* Predicted BLLs for the population of children in each school
** Blood Lead Level is an estimated geometric mean from the EPAIEUBK model

Note: "Worst Case" assumes 90th percentile water lead concentrations from the running water sample and the standing water sample in each school; "Typical Case" assumes median water lead concentrations from the running water sample and the standing water sample in each school

Figure 3. Bar graph illustrating the predicted geometric mean potential blood lead levels (BLLs) for worst-case and typical-case scenarios using observed lead concentrations in school drinking water. "Worst case" assumes 90th percentile water lead concentrations from the running water sample and the standing water sample in each school; "typical case" assumes median water lead concentrations from the running water sample and the standing water sample in each school.

DISCUSSION

We estimated BLLs in school-age children on the basis of elevated concentrations of lead in school water to provide a public health risk assessment. Despite school water sources with lead concentrations exceeding guidelines, we found that consumption did not yield geometric mean BLLs exceeding the CDC's guideline of 10 $\mu\text{g}/\text{dL}$. This was based on modeling of BLLs using IEUBK. Our typical exposure scenario predicted geometric mean BLLs from 1.6 to 2.8 $\mu\text{g}/\text{dL}$. BLL measurements on children are usually conducted when a health care provider has a high index of clinical suspicion for potential lead poisoning. Although BLLs are not common in Washington State, they are reported to the State Department of Health. Our range of modeled BLLs is consistent with the State's reported BLLs for children aged 5–8 years of age on the basis of data from 1993 to 2002. These data show that 95% of reported BLLs were between 0 and 4 $\mu\text{g}/\text{dL}$.²⁴ These values are considered low BLLs by the state and do not warrant further follow-up.

In younger children (age 0–4 years), recent studies demonstrate adverse neurodevelopmental impacts associated with BLLs below 10 $\mu\text{g}/\text{dL}$.^{3,25,26} Several recent studies demonstrate that concurrent low-level lead exposures affect cognitive functioning in school-age children.^{3,18,27,28} In an analysis by Chen et al¹⁸ neurocognitive testing results in children ages 2–7 years were strongly associated with their concurrent BLLs independent of early childhood exposure, suggesting that ongoing lead exposure through childhood affects cognitive functioning. Schwartz²⁷ conducted a meta-analysis of lead exposure studies and found that concurrent low-level lead exposures were inversely associated with full scale IQ in school-age children. These data support the public health community consensus that reducing identified childhood lead exposures is critical to preventing long-term neurotoxicity.

Two instances of lead contaminated school drinking water are described in the scientific literature. In a Philadelphia, Pennsylvania, study, drinking water lead concentrations ranged from 20 to >100 ppb in 292 school buildings.¹⁰ The authors conclude that school drinking water may be a significant source of lead exposure for children depending on the volume of water consumed, but did not attempt to characterize the health impact and did not measure or model BLLs. In a Utah elementary school, 18 samples of standing and running drinking water were found to contain lead concentrations that ranged from below the level of detection to 84 ppb.⁹ A total of 116 of the 300 children attending the school had BLL testing within 16 days of the provision of an alternative drinking water source. One child had a BLL above 10 $\mu\text{g}/\text{dL}$. Three children had BLLs between 6 and 8 $\mu\text{g}/\text{dL}$, and the remaining 112 children had BLLs <6 $\mu\text{g}/\text{dL}$.⁹ Interestingly, these investigators also used the IEUBK model to predict BLLs and determined a predicted geometric mean of 17 $\mu\text{g}/\text{dL}$ in this population. The assumption and inputs used to arrive at these findings are not provided.

Childhood exposure to lead through drinking water is not limited to school drinking sources. In 2004, Washington, DC, found elevated lead concentrations in public water affecting hundreds of homes in the metropolitan area. Among a subset of the population that elected to have their BLL determined, higher BLLs were associated with living in a residence that had a lead pipe service line, demonstrating that water contributes to BLLs. However, overall BLLs were not high among the children tested.⁸ In a cross-sectional sample of homes with water lead concentrations >300 ppb, BLLs were reported for 13 school-age children as 1–4 $\mu\text{g}/\text{dL}$ with a median of 2 $\mu\text{g}/\text{dL}$. These values are consistent with the findings of our study and further support the conclusions that drinking water likely does not significantly contribute to high BLLs in children.

The Seattle Public School system is currently addressing exposure reduction with flexible, individualized school-based considerations of engineering. Challenges identified by the public schools include lead concentrations significantly exceeding EPA guidelines up to 200 hours after installation of new, certified low-lead brass bubblers and other plumbing components as a result of passivation (Seattle School District, personal communication, July 2005). The Seattle School Board has also adopted a more stringent action level of 10 ppb for the school district.¹⁵ Recommendations come from a newly formed water quality group with representative parent groups, the school board, local experts, and the district administration.

There were several limitations of our effort to characterize health risk using BLL predictions. We used background lead exposure levels in soil, dust, air, and home/school drinking water intake. Generally, we relied on relevant local data for nonwater sources, and when ranges were available, we erred on the conservative side by using higher-than-average exposure levels. For exposure to

drinking water, we may not capture important individual variation in drinking consumption patterns.

Gaps in knowledge allowing an adequate characterization of risk to school-age children from lead contaminated drinking water make this a difficult issue for school administrators and public health authorities. Assessment of childhood patterns of water consumption at school, coupled with techniques to capture temporal variability in lead concentrations at the faucet or fountain are lacking. However, our assessment suggests such efforts are needed only if drinking water lead concentrations far exceed the EPA recommendations (approximately 80–100 times the EPA's 15 ppb). These values are based on modeling that uses the extreme values in our data set. When we used the 1600 ppb value from standing water samples, we predicted a BLL of 16.7 $\mu\text{g}/\text{dL}$. We chose not to use these values for analysis because they did not represent a realistic chronic exposure scenario for the school-age children.

Importantly, data for drinking water sources at school were based on a single standing and running sample. Our exposure data showed a skewed distribution, with a few extreme values indicating very high lead concentrations. Repeat testing showed markedly decreased lead concentrations, and extreme values were not reproducible. These extreme values may be due to erratic spikes in lead concentration associated with flaking off of lead-containing particles as a result of mechanical disturbances of piping materials such as vibration, thermal expansion and contraction, and water flow.^{29,30} Laboratory and sampling error were investigated and ruled out as possible sources of lead variation on the basis of laboratory accreditation and standardized sampling methods (Seattle Public Schools, personal communication, June 2006). This reinforces the greater relevance of the typical scenario and worst-case scenario used in our study. In order to capture true exposures, a more sophisticated understanding of variation of water lead concentrations and chronic exposures of children is needed.

School drinking water is not likely to contribute to increased BLLs in school-age children. Given the known health impacts of low-level lead exposure on neurocognitive development in young children, primary prevention efforts should continue to focus on traditionally recognized exposure pathways. Gaps still exist in source identification of school water lead contamination and its variability, characterization of children's school drinking water consumption patterns, and assessment of health impacts of ongoing low-level lead exposures among school-age children. Further investigation of these issues and potential increased BLLs in school-age children is warranted if drinking water lead concentrations far exceed those in this study.

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