

**Lead (Pb) in U.S. Drinking Water:
School Case Studies, Detection Challenges and Public Health Considerations**

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Abstract

School case studies in Seattle WA, Baltimore MD, Philadelphia PA, Los Angeles CA, Washington DC and Maryland suburbs highlight the potential exposure of sensitive populations to very high lead doses through consumption of contaminated drinking water. Lead levels greater than the EPA guideline of 20 ppb were measured at 18-48% of school taps, while in some schools drinking water outlets released concentrations of lead sufficient to classify the water as “hazardous waste” (>5,000 ppb). Controlling lead hazards from drinking water, at schools and elsewhere, requires improved sampling protocols that can capture the inherent variability of lead release from plumbing, and that can quantify both the particulate and soluble lead fraction present in water. Depending on the type of lead-in-water problem(s) that they encounter, concise remediation guidance is needed for schools to follow. Most importantly, the potential public health implications need to be re-evaluated. Specifically, while the association between lead in water and lead in blood has been documented through decades of prior scientific research, the possible contribution of drinking water lead to the body’s lead burden is not a current focus of public health agencies. Accounting for this mis-understood source is necessary if the US is to achieve its goal of eliminating elevated blood lead levels in children by 2010.

Introduction

Lead (Pb) is widely recognized as one of the most pervasive environmental health threats in the United States. Dramatic progress has been made in the last few decades to reduce lead exposure from gasoline, paint, dust, food/drink cans and drinking water [1-3]. Despite such improvements, clinical evidence has recently demonstrated adverse health impacts at blood lead levels once considered safe [4-7]. Although effects are expected to vary from person to person due to individual and genetic risk factors [8, 9], drinking water consumption is believed to account for only 10-20% of total lead exposure nationally [10]. However, it was recently discovered that standard analytical methods used to assess lead exposure from water dramatically underestimated this source in at least some instances [11, 12], and other circumstantial evidence has accumulated suggesting that lead from water can occasionally be the dominant source of exposure [9, 13-15].

These new insights prompted a comprehensive review of the lead-in-water issue, with emphasis on problems:

- in US schools’ drinking water which have only recently been publicized
- in lead detection due to inadequate standard sampling protocols, and
- with understanding of public health implications for sensitive population groups

The Special Issue of Lead in Water of Schools and Day Care Facilities

The US Environmental Protection Agency (EPA) regulates public water supplies under the Lead and Copper Rule (LCR) through an “action level” for lead at home taps of 15 parts per billion (ppb) [16]. If lead concentrations exceed this action level in more than 10% of customer taps sampled, the water utility must take measures to control plumbing corrosion and inform the public about steps they should take to protect their health. When current management strategies were developed for childhood lead poisoning after implementation of the LCR in 1991, US health agencies largely assumed that lead in public drinking water supplies had been eliminated as a significant source of lead. However, even in cities complying with the LCR, up to 10% of water samples can exceed 15 ppb of lead (there is no upper bound or Maximum Contaminant Level established for lead in water) without any public notification. At the same time, high levels of lead can be present in drinking water of schools without any notification of students, parents or teachers because the LCR does not extend to drinking water in schools and daycare facilities [16, 17]. Instead, the Lead Contamination Control Act (LCCA) provides non-enforceable guidelines for schools and daycare facilities, recommending that drinking water should not exceed 20 ppb Pb in a 250 mL first draw sample [18]. There is no enforceable national, state or local lead standard for water of US schools and daycare facilities.

Controlling lead hazards from drinking water in schools and daycare facilities is an important, yet intractable task, for several reasons. First, many schools are unaware that they have complete responsibility for identifying and remediating this significant environmental hazard, and they often lack funding to do so. Second, school buildings have intricate plumbing systems, sometimes very old, containing multiple potential sources of water lead contamination. These include lead pipe, galvanized iron pipe, lead-containing solder joints, and system components (e.g. water meters, valves, faucets) made of lead-containing brass (Figure 1). All types of leaded brass legally contain up to 8% lead by weight [12]. Even though lead solder at joints, lead pipe, and lead goosenecks have been banned in the US since 1986 [12], these materials are present in older buildings, while illegal use of lead solder still occurs even in new buildings [19]. Third, the intermittent pattern of water consumption, with periods of little or no water use during weekends, holidays and summer break, produces very long stagnation periods for water inside the piping. This water use pattern is considered “worst case” for releasing hazardous levels of lead from the plumbing to the water [14, 20, 21]. Finally, school children, especially those attending elementary school or daycare centers, are much more vulnerable to adverse health effects from lead exposure relative to adults. This is because they can absorb up to five times more of the ingested lead, which exhibits its toxic effects in their blood and soft tissue for a longer biological half-life [9].

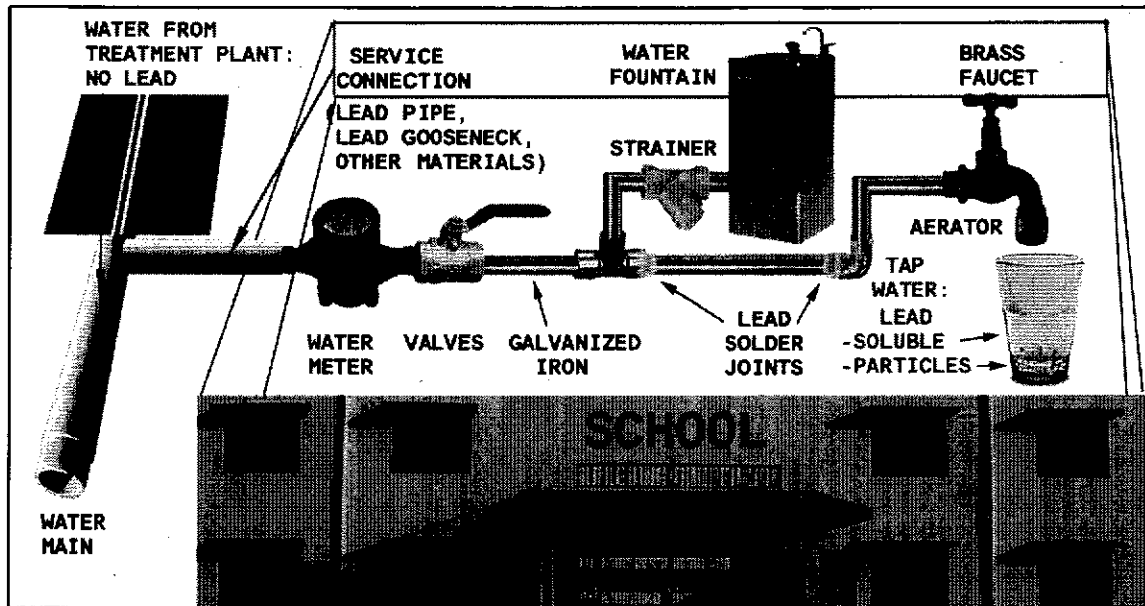


Figure 1: Potential sources of lead contamination in school drinking water.

Selected school case studies across the US (Table 1) illustrate prolonged exposure of vulnerable populations to high levels of water lead in the absence of a protective legal framework. With the exception of Washington DC Suburban Area, problems in schools of Baltimore MD, Seattle WA, Philadelphia PA, Washington DC, and Los Angeles CA were not revealed by the schools under the LCCA, but were revealed by parents, investigative reporters or students (Table 1). With the same exception, at least three years elapsed from the time the schools recognized a problem to the time the public was informed. A large percentage of taps in the school systems (18-80%) had lead in water above the current LCCA guideline, and several schools had taps with levels of lead sufficient to classify the water as “hazardous waste” (>5,000 ppb) (Table 1).

Not shown in Table 1, are other case studies from: 1) Davidson, NC (2007), where a problem was discovered after a high school chemistry experiment failed and the teacher eventually traced it to high lead [22], 2) Durham, NC (2005) where sampling for taste and odor problems revealed hazardous lead levels in an elementary school [23]; and 3) cases in New Jersey [24] and Australia [25] that have only recently been publicized. It is worth noting that several other school systems in US urban and suburban areas have not systematically tested their water for lead in nearly three decades. In all of the publicized cases, gaps in fundamental scientific and engineering knowledge contributed to insufficient or improper testing of water and inadequate analysis of the health threat.

Table 1: Representative Case Studies on Lead-in-Water Problems at US Schools.

School System	Year School Knew of Problem	Year Public Informed	How Discovered	% Taps Above EPA Guideline	Highest Pb Detected (ppb)	Remedial Measures	Follow up testing after remediation
Baltimore, MD [26]	1991	2003	Parent inquired as to why water fountains had been turned off and a teacher turned whistleblower.	20% of fountains	N/A	After several failed efforts, water fountains turned off and schools switch to bottled water	Unnecessary since bottled water is now used
Seattle, WA [27-31]	1990	2003	A parent was concerned due to discolored water, collected and analyzed sample finding high lead.	1990: 33-40% 2004: 25%	1,600	Some schools switched to bottled water, replaced bubbler heads, installed filters, turned off fountains, totally or partly replaced piping	Yes, 30% of taps exceeded US EPA guideline
Phili, PA [32-35]	1993	1998	A source "unofficially" provided lead-in-water test results to EPA, after EPA had been told to get a search warrant when requested to sample water.	2000: 38% of fountains 48% of faucets	N/A but 17% of schools > 100 ppb	shut off outlets with high lead levels, provided bottled water, flushing	No
Wash. DC [36, 37]	2004	2007	Freedom of Information Act (FOIA) requests by Virginia Tech. More than 80% of taps in some schools exceeded 15 ppb lead. All results before 2008 used steps in sampling protocol that hide lead.*	2/2004: 1% 3/2004: 4% 2006: 29% 2007: 13% 2008: 26%	2004: 7,300 2006: 4,936 2007: 20,000 2008: 1,987	Installed new coolers, fountains, and filters	Yes, some lead in water problems still persist
Wash. Suburban, MD [38-40]	2004	2004	School system voluntarily collected samples to participate in LCCA after problems revealed in Wash. DC	2004: 18%	36,372	Tested fixtures in school buildings, flushing for all fountains	Yes
Los Angeles, CA [41-44]	1990	2008	Local news station. School personnel falsified daily reports regarding remedial flushing to reduce lead.	2008: 30%	N/A	Filters, flushing	Not Yet

N/A: Not Available

Water lead levels in bold exceed 5,000 ppb, classifying the water as "hazardous waste"

*Data from Washington DC schools use 15 ppb as a failure criterion, instead of the 20 ppb school guideline.

Challenges in Assessing the True Extent of Water Lead Contamination

In order to assess the public health risk from elevated lead in school drinking water, it is obviously necessary to first measure the actual lead that is present in water. It was recently suggested that standard sampling protocols can sometimes “miss” lead hazards. This is because of inadequate recovery of lead particles in water samples, failure to collect lead particles in samples, and inherent variability in lead release.

Inadequate recovery of lead particles in water samples. Lead in water can be present in either a soluble or particulate form [12, 45, 46]. Lead particles in water originate from lead-bearing scale or rusts, as well as physically degraded pieces of leaded brass, lead solder or lead pipe (Figure 1). A small survey of lead in potable water from around the US revealed numerous instances in which lead was present as particulates, sometimes at concentrations greater than 1,000 ppb [47]. It has only recently been recognized that particulate lead can occasionally be the dominant form of lead in drinking water, and it is believed to be responsible for much of the lead in samples testing over 100 ppb lead in schools (Table 1).

The issue of whether lead is soluble or particulate is critical, because existing analytical methods are based on the assumption that lead is soluble, and that standard “preservation” of water samples at $\text{pH} \leq 2.0$ with addition of nitric acid is adequate for detecting all the lead that is present in the water. Digestion of samples with heat or stronger acid is not required unless turbidity exceeds certain thresholds [48]. Our own studies demonstrate that the standard EPA protocol can “miss” much of the lead that is actually present in water. For instance, water samples actually containing 508 ppb lead in Washington D.C., only measured as 102 ppb using the standard EPA protocol [49]. More recently, we collected a sample that actually contained more than 1500 ppb lead, but it only measured as 3 ppb using the standard EPA protocol (99.8% of the lead was missed). The reason for the large discrepancy is that particulate lead can settle or adhere to the plastic sampling containers. It therefore does not contribute to measured turbidity when aliquots are taken for measurement, so no additional acid digestion is required. Under the standard preservation procedure these particles do not dissolve in the weak acid specified by the EPA ($\text{pH} \leq 2.0$), and as a result they may not be introduced into the analytical detector when an aliquot is taken for quantification [11, 12].

Preliminary results further demonstrate that in the aggressive environment inside the human stomach (pH as low as 1.0, warm temperature, hydrochloric acid instead of nitric acid, mild agitation via churning) a large fraction of lead particles from brass, solder, lead pipe and lead rust can be dissolved and therefore can be bioavailable. Hence, lead in water that is “missed” by standard sampling procedures can be bioavailable once ingested [11, 12]. Our recent research on cases of childhood lead poisoning in Greenville NC [50, 51], Washington DC [52, 53], and Durham NC [54], demonstrated that a key source of lead exposure was lead solder particles of about 1 to 100 μm diameter in the water. It is our belief that the “worst case” lead levels detected in school water (Table 1) are also due to particulates, and that the actual lead concentrations in the school samples might be higher than those reported. This is alarming, since the higher concentrations of lead in water are already above “hazardous waste” levels of lead (some samples had lead levels more than 1000 times higher than the LCCA guideline of 20 ppb).

Failure to collect lead particles in samples. Problems with particulate lead in water can also be missed by the way sampling is conducted. Simply put, over the years, researchers have noticed that lead “spikes” often occur in samples from time to time [45, 46], and that one contributing factor is “scouring of lead particles” [55]. Unfortunately, to avoid the lead spikes, it has become commonplace to collect water samples at atypically low flow rates. This is accomplished by mandating sample collection by “gently and slowly turning on the tap,” or by providing sample bottles that have very small openings which cannot be filled by running taps with high flow. Even the most recent US EPA school sampling instructions recommend that bottles be filled by inducing a steady flow of water from the outlet as small as the width “of a pencil” [17]. Results from our testing illustrate that in some circumstances particulate lead in samples is a strong function of flow rate (Figure 2). As a result, existing protocols stipulating low flow rates are missing many of the lead hazards relative to those which children are exposed to during normal water use.

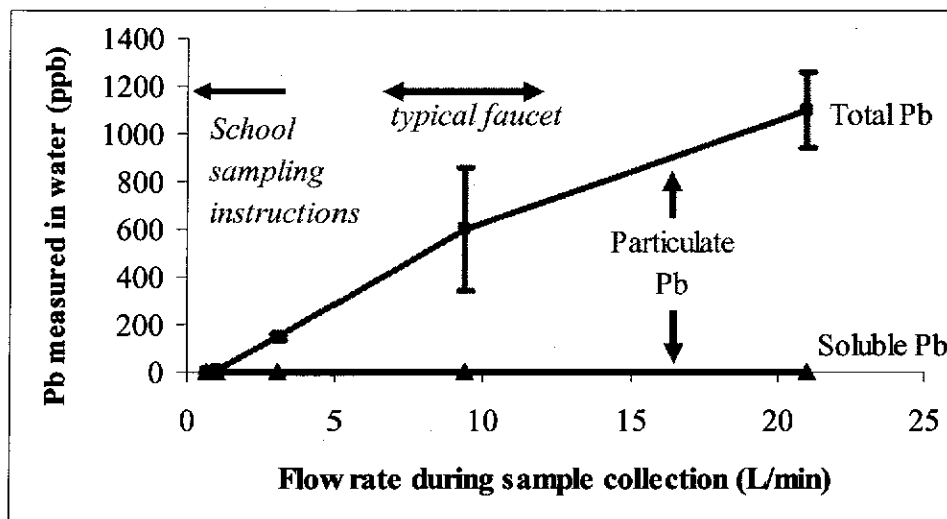


Figure 2: Lead measurement in water samples versus flow rate at a high-risk Washington DC home tap. Error bars represent 95% confidence intervals for triplicate samples. Sampling at low flow rates reduces the concentration of particulate lead collected.

In addition, existing sampling protocols do not: 1) sample hot water, which is believed to contain much higher lead, and in response EPA instructs consumers to never consume hot water [17], and 2) sample at taps which are not intended for water consumption, such as metered faucets and hose bibs. Children nonetheless occasionally drink from such taps, or consume hot water either purposefully or inadvertently (many single handle faucets leak hot water at a certain percentage even when adjusted to cold water only).

Inherent variability in particulate lead release. Unlike the case with soluble lead in water, which is not controlled by nuances of hydraulic flow disturbances from the tap, the mobilization of particulate lead from plumbing during sample collection can be random. That is, repeated testing of some taps can produce multiple samples with low lead, and yet with some finite frequency, lead particles occasionally detach revealing a clear lead hazard (Figure 3). This is a concern because current guidance will “clear” a school tap as safe based on collection of a single sample below 20 ppb. In some cases (Figure 3), collection of multiple samples will be necessary to prove a tap is safe.

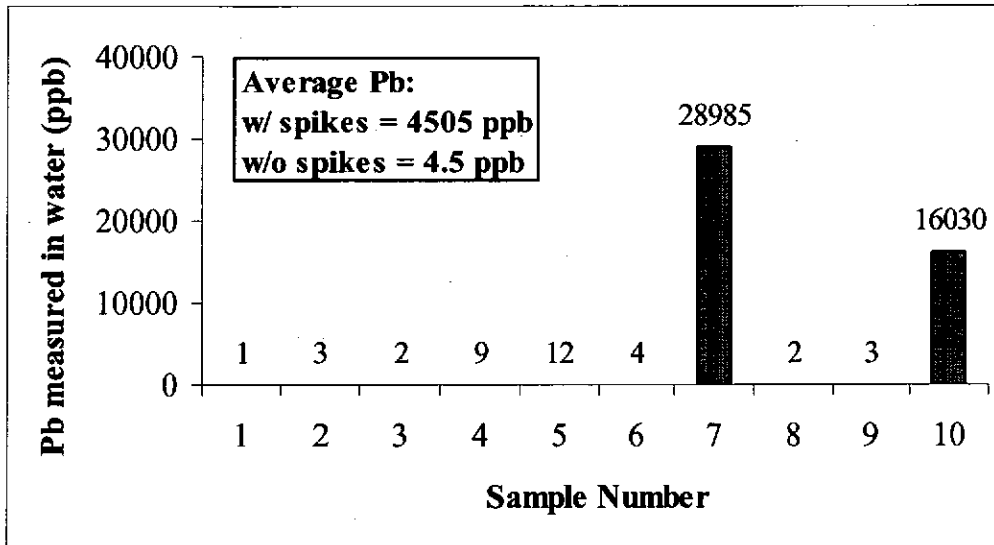


Figure 3: Lead measurement in repeated water samples collected from the same tap. Clearing a tap based on a single sample may be problematic.

Drinking Water Lead in Relation to Public Health Risks

Potential harm from exposure to lead is typically tracked by measurements of the blood lead level (BLL). Levels above 10 ug/dL are considered elevated (EBLLs) for infants and children, since they exceed the Centers for Disease Control and Prevention (CDC) threshold at which detectable mental impairment and behavioral changes have been documented [56]. Cases in which blood lead exceeds 10 ug/dL are also termed “lead poisoning” in many jurisdictions. Recent studies suggest that decreased IQ and cognition occur even at BLLs as low as 3 ug/dL, reinforcing the notion that no safe level of lead exposure exists [4-7]. The US has adopted the goal of eliminating EBLLs in children by 2010 [57].

The harmful health effects from lead exposure through drinking water have been recognized in the US since the 1850’s. In that era, before leaded gasoline and the widespread use of leaded paint, drinking water contamination by lead pipes was the main source of human-ingested lead, causing infant mortality, spontaneous abortion, neurological effects, and digestive problems [58]. One author suggested that use of lead pipes in major cities produced “one of the most serious environmental disasters” in US history [58]. In homes that have lead pipe, about 50-75% of the lead in water is attributed to this source, while the remainder is attributed to leaded solder and leaded brass plumbing materials [59]. Leaded brass plumbing sometimes poses significant problems even in brand new buildings [60].

The contribution of waterborne lead to EBLLs does not appear to be an existing focus, or a significant concern, of any US public health agency. That is, the Centers for Disease Control and Prevention (CDC) simply assumed that lead would never again exceed 15 ppb in water after implementation of the LCR, by essentially misinterpreting the LCR lead action level for a maximum contaminant level. The sampling data we have collected to date demonstrate that not only is this assumption erroneous, but actual water lead levels can be more than 1,000 times higher than the LCR action level of 15 ppb. But as a result of this misconception, water is rarely considered as a possible lead source when cases of childhood lead poisoning are investigated.

Recently, in North Carolina, a child's blood lead rose to dangerously high levels over the course of a year, during which health agencies tested in vain for traditional lead sources. It was only when the parent requested that the water be tested, that a lead hazard was discovered in the child's environment (water lead levels were up to 500 ppb) [54]. This discovery prompted North Carolina to pass the first guidance in the US that required testing of lead in water for all lead poisoned children, and its implementation immediately revealed other cases in which water was the only identified lead hazard [54]. At the national level, 30% of EBLL cases do not have an immediate lead-paint source identified [14].

Bioavailability Considerations. It has been suggested that hazardous levels of lead detected in drinking water might not cause harm, because that form of lead is not as readily bioavailable as is lead in paint [61, 62]. To test that hypothesis, we compared the potential bioavailability of representative lead paint to a similar mass of particles that detached from lead pipe using simulated gastric fluid (Table 2). The results demonstrate that five different types of paint chips, collected from residences of Washington DC, would leach variable but significant amounts of lead inside a child's stomach, depending on their lead content (Figure 4). At the same time however, an equal mass of lead particles consumed through drinking water would result in 2.3 times more lead exposure than the worst paint chip (Figure 4). Similar results regarding potential bioavailability were reported for lead particles derived from solder, brass and pipe scale [12].

Table 2: Experimental Conditions to simulate the human stomach.

	Simulated Gastric Fluid (SGF)
Constituents	Nanopure Water with 0.2% v/v NaCl, 0.32% pepsin, 0.7% v/v HCl
Typical pH	1.2
Temperature (°C)	37 (Body Temperature)
Mixing Pattern	Gentle Motion
Holding Time	3 hours (typical for solid food)

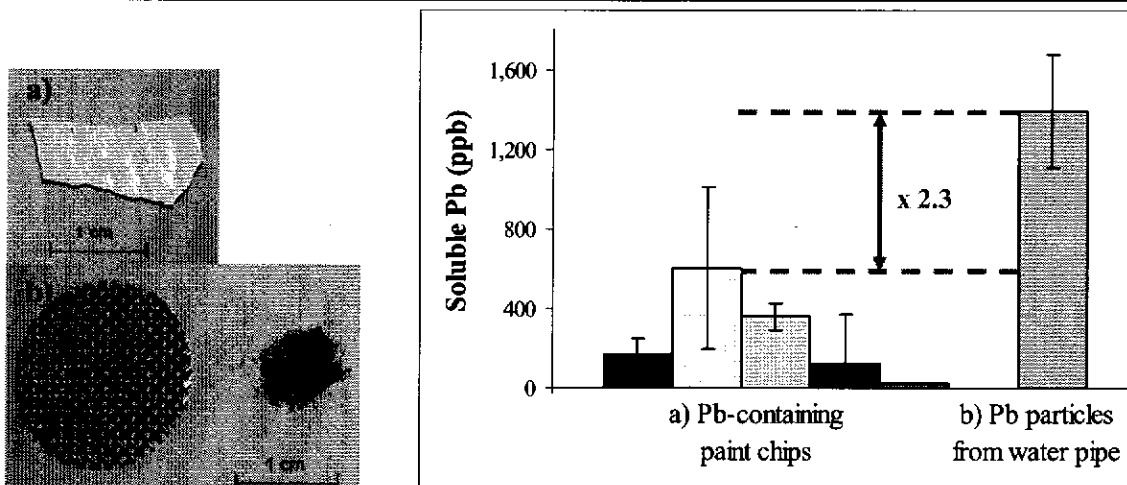


Figure 4: Lead dissolution in simulated gastric fluid of a) five different lead paint chips versus b) dissolution of equal mass of lead particles originating from a lead water pipe. The lead particles were small enough to pass through the openings of a medium faucet aerator screen. Soluble lead, which was quantified by Induced Coupled Plasma Mass Spectrometry after 3 hours of holding time, is assumed to be the bioavailable fraction. The error bars denote 95% confidence intervals for triplicate samples.

Improper Medical Diagnosis in Adults and Children. Lead toxicity can be difficult to diagnose. This is due to a wide range of common symptoms, which may appear to be unrelated and/or which are easily overlooked. To illustrate, an adult woman in Australia was mistakenly diagnosed with chronic fatigue syndrome for over 10 years, before she was found to have lead poisoning [63]. Throughout that time period, the woman suffered from debility, weight loss, constipation, difficulty with abstract reasoning and anemia. Eventually, her extremely high BLL was attributed to domestic hot water, which had 1.12-1.5 mg/L lead. The hot water cylinder contained leaded solder, which leached in the bottom and carried contaminated water throughout the pipes. Even though the cold water tap was cleared as safe (<0.005 mg/L lead), all family members used hot water to prepare instant coffee and cook meals, and all were eventually found to suffer from lead poisoning [63].

Even more worrisome are instances of lead poisoning in children that go undetected. Two recent cases of severe lead-poisoning in the US by accidental ingestion of lead-containing jewelry charms [64, 65], one of which was fatal, raise very important concerns. First, the child who died was initially misdiagnosed with a viral infection, and the heart-shaped object observed on his abdominal radiograph was mistaken for a temperature probe. By the time his condition was diagnosed as lead poisoning, the child died from respiratory arrest [64]. In the case of elevated blood lead through drinking water at school or at home, drawing the causal link is even more difficult. Common symptoms in children, such as fatigue, headaches, irritability and vomiting, might not ever be linked to high lead in water. Even if they are, actual measurement of blood lead usually takes place long after schools/parents are informed of high lead in the water, and have in the meantime taken steps to eliminate that exposure. Considering that the half-life of lead in blood is just one month, this type of data would mistakenly indicate lack of harm.

After the two documented cases of lead poisoning from ingested lead jewelry, decisive action was taken to protect children from both acute, as well as chronic lead hazards. The US Consumer Product Safety Commission (CPSC) established 175 ug of lead in jewelry as a dose triggering acute health concerns, product recalls and fines, which resulted in recalls of more than 150 million children's jewelry pieces in just 2004 [66]. This lead dose is equivalent to 700 ppb in the 250 mL water samples collected in schools. Our data illustrate that 10.3% of schools in Washington DC had at least one sample over the US CPSC acute health dose based on sampling in 2007. If a dose of 175 ug lead in a product not intended for human consumption will trigger fines and recalls due to concern over acute health threats from potential exposure, than a similar dose of lead in water (a product intended for human consumption) should also be a concern.

Acute Health Risk. Studies of lead-in-water hazards have not explicitly considered acute health risks. Our preliminary bio-kinetic modeling indicates that consuming a single 250 mL drink of water at the higher lead doses measured in schools, is predicted to cause a child's blood lead to spike above 50 ug/dL within one day (Figure 5). This level of lead can cause a variety of immediate common maladies including abdominal discomfort, nausea, headaches, and gastrointestinal upset (Figure 5). As noted above, the cause of such problems would rarely be traced back to lead in drinking water.

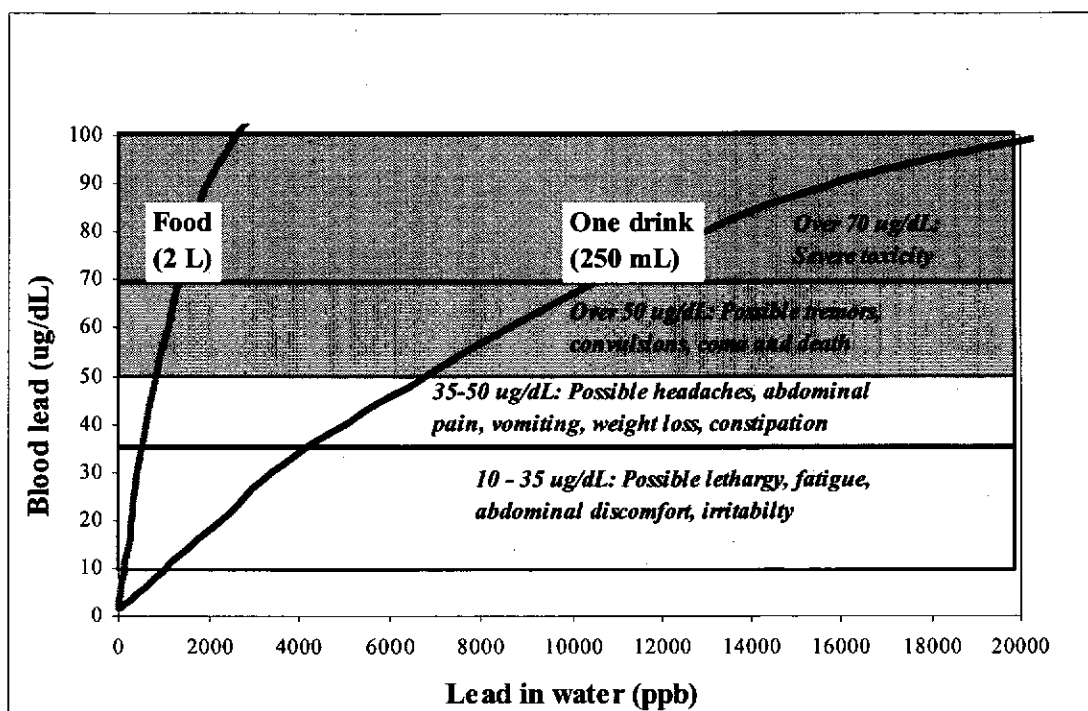


Figure 5: Predicted blood lead level of 2-year old child after direct consumption of lead-contaminated water, or consumption of food cooked with that water, and associated adverse health effects. The ICRP (International Commission for Radiation Protection) bio-kinetic model was employed.

Drinking water lead and blood lead level in children and adults. The contribution of drinking water lead to the body's total lead burden is a subject of contradictory literature. The population studies aiming to identify whether an association exists mainly focused on the most sensitive groups (Table 3). These include formula-fed infants, young children, and pregnant or breast-feeding women. Various approaches have been used throughout the years in an attempt to correlate water lead levels (WLLs) to blood lead levels (BLLs) (Table 3). These include, but are not limited to:

- different types of water sampling to capture actual lead intake through water consumption
- Parametric correlations (assuming normal distribution of water lead and blood lead) versus non-parametric correlations
- Linear regression models versus curve-linear models

The first study to show a curve-linear relationship between water lead and blood lead yielded a correlation coefficient of $R=0.52$, by analyzing data from different sectors of the Scottish population [67] (Table 3). That work concluded that *“Perhaps the most important aspect of this problem is the effect that high water lead has on the chances of a person having an unduly raised blood lead level”*. In that study for instance, 18% of people with first-flush water lead ≥ 1.44 $\mu\text{mol/L}$ (i.e. 298 $\mu\text{g/L}$ or else 298 ppb) had BLLs of 2 $\mu\text{mol/L}$ (i.e. 41 $\mu\text{g/L}$) or more, compared to only 0.3% of those with water lead < 0.24 $\mu\text{mol/L}$ (i.e. 50 $\mu\text{g/L}$). Additionally taking into account geographic risk factors,

recent work [68] found a strong correlation between the probability of children to have EBLs and the 90th percentile water-lead levels from 2000-2007 in Washington DC ($R^2=0.83$ in “High Risk” Neighborhoods) (Table 3). An earlier study on the same topic [62] had found no correlation between drinking water lead and children’s blood lead levels in Washington DC ($R^2=-0.031$) (Table 3).

The first study to show a significant association between infants’ dietary lead (mainly consisting of drinking water used to reconstitute infant formula) and blood lead was the Glasgow duplicate diet study [69]. For 13 week-old infants, a duplicate of their formula was collected for a week, so that the total lead content could be estimated and related to water and blood lead concentrations. A simple linear relationship between composite kettle water lead concentration and infant blood lead level was derived, with a correlation coefficient of $R^2 = 0.32$ (Table 3). Other sources of environmental lead aside from water were not assessed in that work, a limitation which attributed to the unexplained variability in blood lead levels. In addition, the study reflected conditions of very high water lead levels due to a plumbosolvent water supply, and most participating infants had blood lead levels above 10 ug/dL, which is the CDC current level of concern. Even so, this work was the first population study to show a strong association between lead in water and lead in blood for formula-fed infants.

Keeping the same assumption of a linear relationship, the World Health Organization (WHO) subsequently fit the same data into three models with different population distributions (normal, log-normal and normal fractional) [70]. All three models in the WHO analysis yielded an intercept of 14.8-15.6 ug/dL, and a slope of 0.052-0.054 (Table 3). These authors speculated that the high intercept may reflect exposure from dust/air or maternal exposure before birth, in an era prior to the banning of lead in gasoline [70].

More recent studies, which reflect reduced water lead levels, still suggest cause for concern. An epidemiological study in Hamburg, Germany [71] found a statistically significant correlation between average lead concentration in tap water and lead concentration in blood for 142 young women (Spearman’s rho = 0.43, $p<0.0001$). For those women who were exposed to water lead >10 ug/L, an intervention program was followed, either excluding exposure (by consuming bottled water) or minimizing exposure (by flushing water prior to consumption). Overall, after about 10 weeks of intervention, the blood lead levels decreased significantly (median decrease of 11 ug/L, $p \leq 0.001$). “Minimizers” lowered their blood lead level by about 21%, whereas excluders lowered their blood lead level by 37%. This reduction is fairly consistent with expectations, taking into account that the half-life of lead in blood is 28-36 days [70]. Blood lead levels in this German study were below 10 ug/dL even before intervention, and the authors concluded that “*lead in tap water stands for an avoidable surplus exposure*”.

Even though such studies are highly informative and useful, the main concern is to determine whether lead in water will make a sufficient contribution to total intake in order to constitute a hazard, relative to other sources of lead. The relative contribution of water lead to blood lead has not been thoroughly studied, or has even been ignored. For example, a broad Cincinnati study aimed to investigate lead sources and factors which

result in excessive intake for children in urban settings [72, 73]. Blood lead levels were systematically monitored from birth through 5 years of age, while cognitive and behavioral development tests were also performed. A broad range of lead sources in the children's environment were accounted for, including painted surfaces and dust, soil samples in outside playing area, street dirt, and any suspicious items which the children were mouthing. Water samples were not collected in this otherwise very thorough and definitive study. Instead, the author's cite sampling data collected by the water utility from the distribution system (< 5 ug/L) before the service line [73]. This mistakenly implies that lead in drinking water for children in the study was low. Other samples collected from Cincinnati drinking water have shown much higher water lead levels.

Another widely accepted study by Rabinowitz et al. [74] attempted to correlate BLLs of infants in Boston with lead in dust, soil, indoor air, paint and tap water. The authors reported statistically significant correlations of children's BLL at age 24 months with lead in dust (Spearman's $\rho = 0.4$, $P < 0.0001$), with lead in soil (Spearman's $\rho = 0.3$, $P < 0.001$), and with lead in paint (Spearman's $\rho = 0.2$, $P < 0.01$), but not with lead in water (Spearman's $\rho = 0.14$, not statistically significant). The conclusions of that work regarding contribution of dust, soil and paint to BLL are consistent with expectations. However, for the case of drinking water lead, analytical limitations in quantification may have masked any potential contribution to BLL. Lead in water was quantified by anodic stripping voltametry. This method is now known to only measure soluble Pb^{+2} , and not particulate lead or Pb^{+4} . The latter species however may be problematic in drinking water under at least some circumstances [12]. This may explain the very low levels of lead (3.7 – 7.3 ug/L) that were detected in drinking water samples during the early 1980's in that work. As a point of comparison, water sampling data from ten more recent cases of childhood lead poisoning in Boston (1996-2000) indicated lead levels in first-draw tap water samples averaging 132 ug/L on average, with a high of 311 ug/L [75].

Table 3: Representative population studies on the association between lead in water and lead in blood (in chronological order).

Sample Population	Independent Variable	Dependent Variable	Measure of Association	Model
Different sectors of Scottish population (n=949) [67]	First-draw water lead ($\mu\text{mol/L}$)	Blood Lead Level ($\mu\text{mol/L}$)	R = 0.52	$(\text{BLL}/20.7) = 0.533 + 0.675(\text{WLL}/207)^{1/3} *$
Mothers in Ayr, Scotland (n=114) [76]	Kettle water lead ($\mu\text{g/L}$)	Blood Lead Level ($\mu\text{g/dL}$)	$R^2 = 0.56$	$\text{BLL} = 4.7 + 2.78(\text{WLL})^{1/3}$
Mothers in Ayr, Scotland (n=114 from 1980-81, and n=116 from 1982-83) [76, 77]	Kettle water lead ($\mu\text{g/L}$)	Blood Lead Level ($\mu\text{g/dL}$)	$R^2 = 0.65$	$\text{BLL} = 5.6 + 2.62(\text{WLL})^{1/3}$
Bottle-fed infants in Scotland (n=93) [69, 70]	Composite kettle water lead ($\mu\text{g/L}$)	Blood Lead Level ($\mu\text{g/dL}$)	R = 0.57	$\text{BLL} = 14 + 0.062\text{WLL}$ $\text{BLL} = 15.6 + 0.052\text{WLL}$ $\text{BLL} = 14.7 + 0.054\text{WLL}$ $\text{BLL} = 15.4 + 0.052\text{WLL}$
Infants in Boston (n=202) [74]	Tap water lead after 4-L flush ($\mu\text{g/L}$)	Blood lead level ($\mu\text{g/dL}$)	Spearman's rho = 0.00-0.19 (varied by age)	Not Determined
School children in Southern Saxonia, Germany (n = 69 for location A, n=44 for location B) [78]	Composite tap water lead ($\mu\text{g/L}$)	Blood Lead Level ($\mu\text{g/dL}$)	Location A: $R^2 = 0.34$ Location B: $R^2 = 0.41$	Location A: $\text{Log}(\text{BLL}) = 0.74 + 0.14\text{Log}(\text{WLL})$ Location B: $\text{Log}(\text{BLL}) = 0.81 + 0.14\text{Log}(\text{WLL})$
Mothers in Glasgow, Scotland (n=342) [79]	Water lead ($\mu\text{g/L}$)	Blood Lead Level ($\mu\text{g/dL}$)	Spearman's rho = 0.39	Not determined
Women in Hamburg, Germany (n=142 for sub-sample with detectable water lead) [71]	Average water lead ($\mu\text{g/L}$) from 3 specimens	Blood lead level ($\mu\text{g/dL}$)	Spearman's rho = 0.43	Not Determined
Children in Washington DC with paired water lead and blood lead data (n=107) [62]	Tap Water Lead ($\mu\text{g/L}$)	Blood Lead Level ($\mu\text{g/dL}$)	$R^2 = -0.03142$	Not Determined
Children in Washington DC (n=2698 in "High Risk" n=4791 in "Moderate Risk" n=2621 in "Low Risk") [68]	90 th Percentile Water Lead ($\mu\text{g/L}$)	% Increase in Children with EBLL Compared to US average	$R^2 = 0.83$ in "High Risk" $R^2 = 0.71$ in "Moderate Risk" $R^2 = 0.50$ in "Low Risk"	Not Determined

*Conversion factors applied to original equation in order to convert units based on: 1 $\mu\text{mol Pb/L}$ blood = 20.7 $\mu\text{g Pb/dL}$ blood
1 $\mu\text{mol Pb/L}$ water = 207 $\mu\text{g Pb/L}$ water

Conclusions and Recommendations

Selected school case studies across the US illustrated prolonged exposure of vulnerable populations to high levels of water lead. In all of these cases, regulatory, scientific and engineering knowledge gaps contributed to: 1) insufficient or improper testing of water, failing to identify the extent and nature of the lead problem, 2) non-existent or inadequate remediation measures and 3) inadequate understanding of the health threat.

Enforceable lead standards are needed, in order to protect children from this hazard, in water of US schools and daycare centers. New legislation has been recently implemented in the State of Washington [29, 30] and in Canada [80], mandating tougher standards for lead in school water. Such initiatives are crucial, in order to better address this otherwise “orphaned” public health problem. **Concise remediation guidance** should become available to schools that encounter problems in the future. Several remediation measures can be implemented, depending on the type of lead-in-water problem(s) encountered. These include (but are not limited to) flushing the taps manually or automatically, providing bottled water, installing filters, replacing problematic lead-containing outlets (fixtures, valves, fittings), and re-plumbing. Decision-trees need to be formulated in order to guide schools to the appropriate solution, depending on sampling results (e.g., is the lead soluble or particulate, is the lead coming from end-point devices or the water mains, etc.).

Improved sampling protocols are needed to capture “worst-case” levels of water lead in schools and elsewhere. While standard sampling methods for lead in water might be sufficient for the typical case, modifications are needed to capture the “worst-case”. In the latter situation:

- Single samples might not be adequate to “clear” a tap as safe. Due to the complexity and high variability of lead release, a valid number of water samples and frequency of sampling must be determined, to insure representative results. Such monitoring programs are currently lacking.
- “Stronger” than typical preservation of the water samples with nitric acid might be required to fully dissolve and quantify all the lead.
- Sample collection at a high water flow rate is desirable, to account for physical disturbances that might release particulate lead from plumbing during normal water usage.
- Sampling of hot tap water, which may contain higher levels of lead than cold water, should be conducted.

An obvious implication due to insufficient sampling at hazardous taps is that correlations of water lead with health risks are often missed. This, combined with underestimation of lead-in-water potential bioavailability and toxicity, as well as improper medical diagnosis and other factors, has created the notion that drinking water lead is at most a minor source of elevated blood lead in children. **Re-evaluation of the public health risk** from lead in water is imperative, considering discussion in US EPA for major revisions of the Lead and Copper Rule, discussion in CDC on lowering the blood lead level of concern, and the goal of eliminating all incidences of elevated blood lead in US children by 2010.

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