Lead (Pb) Contamination of Potable Water: 
Public Health Impacts, Galvanic Corrosion and Quantification Considerations

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ABSTRACT

The issue of lead exposure through drinking water was re-examined in light of modern public health goals, recent high-profile cases of elevated lead in water, and emerging concerns regarding the efficacy of legally mandated remedial strategies.

A critical literature review revealed that serious lead-in-water hazards are present at many US schools and homes, and that the threat to individuals is not eliminated by existing regulations. Health studies have provided strong links between lead in water and lead in blood of exposed populations, even at relatively low levels of exposure compared to reported lead occurrence in US tap water samples. As efforts shift from addressing pervasive lead sources that once elevated the blood lead of large percentages of the population, to more isolated individual cases requiring exceptional attention, the importance of carefully considering lead in water as a potential source for elevated blood lead increases.

Consistent with decades of prior research linking elevated water lead to elevated blood lead (EBL), lead-contaminated water in the high-profile case of Washington DC markedly increased the incidence of EBL for very young children. Specifically, incidence of EBL for children aged ≤ 1.3 years increased more than 4 times during 2001-2003 when lead in water was high, compared to 2000 when lead in water was low. The incidence of EBL for children aged ≤ 1.3 years was highly correlated ($R^2 = 0.81$) to 90th percentile lead-in-water levels from 2000-2007, and the risk of exposure to high water lead levels varied markedly in different neighborhoods of the city. Analysis conducted herein focused on identifying “worst-case” neighborhoods and populations. Specifically, this was the first study of the Washington DC case to focus on infants who are most vulnerable to harm from lead in water, and to perform smaller area analysis at the neighborhood (i.e., zip code) level in order to capture pockets of high risk among local communities.

Prior biokinetic modeling efforts, examining the potential adverse impacts of lead-in-water exposure, were re-examined to explicitly consider new public health goals. This included impacts on the most sensitive population groups (e.g., young children and particularly formula-fed infants), the potential variability in blood lead levels (BLLs) amongst exposed individuals within those groups (e.g., most sensitive children at the upper tail of the BLL distribution), more conservative BLL thresholds reflecting low-level adverse effects (e.g., 5, 2 and 1 µg/dL versus 10 µg/dL), and the possibility of acute health impacts. This re-evaluation creates a paradigm shift, in that levels of lead in water that were previously considered inconsequential are demonstrated to be of concern in specific circumstances.

The replacement of lead service lines in front of consumers' homes is a costly, federally mandated remedial action if a water utility exceeds the US EPA lead action level. Because utilities do not own the entire lead service line, they often only replace the portion of the service line up to the property line, typically with copper pipe. Experiences in Washington DC, as
revealed by Freedom of Information Act requests, indicated that partial pipe replacements were not decreasing lead in water, and were actually associated with relatively high incidence of childhood lead poisoning. This prompted the first comprehensive investigation of potential long-term problems arising from galvanic corrosion between the remaining lead pipe and the newly installed copper pipe. Bench-scale experiments demonstrated that galvanic connections between lead pipe (new or aged) and copper pipe increased lead release into the water by 1.1-16 times, when compared to a full length of lead pipe alone. The small area of lead pipe adjacent to the copper joint (<0.5 ft) was gravely affected by galvanic corrosion, and accumulated a thick lead-rust layer (1 inch wide) that constituted a reservoir for semi-random particulate lead detachment into the water.

The work on simulated partial pipe replacements revealed that under worst-case scenarios of highly contaminated water samples, most of the lead was not quantified if water samples were not mixed thoroughly after standard preservation (i.e., after addition of 0.15% v/v HNO₃), or if water samples were transferred from one bottle to another prior to preservation. While there is no reason to believe that sample handling and pre-treatment dramatically skew regulatory compliance with the US EPA lead action level, slight variations from one approved protocol to another may cause lead-in-water health risks to be dramatically underestimated. This is of special concern in unusual situations of "worst-case" individual exposures to highly contaminated water, associated with childhood lead poisoning.

This work provides the water industry and health agencies with important new insights and perspectives on an old problem. Results can improve strategies to detect and mitigate lead-in-water hazards for individuals or populations, and inform future revisions to the US EPA Lead and Copper Rule.
AUTHOR’S PREFACE

All five chapters of this dissertation are presented as separate manuscripts according to the specifications of Virginia Tech’s journal article formatting. All chapters were produced via collaboration between student and advisor, with or without the participation of other co-authors. The student is the primary contributor, and thus the primary author, in four out of five chapters of this dissertation. The student’s contribution to Chapter 2, where she is listed as second author, is explained herein.

Chapter 1 is a comprehensive and critical literature review that reassesses the public health risk from lead in drinking water, by evaluating its potential association to lead in blood of children and adults. As part of this literature review, serious difficulties were identified in linking problems of elevated blood lead to exposure from water, even when lead in water is the major or sole lead source. This is the first comprehensive review in a quarter of a century on lead health effects from drinking water, and reflects new understanding about problems in quantifying lead in water, low-level lead health effects, contamination of water even in modern buildings and in cities that might meet federal regulations, and extent of current problems at US schools and homes. Chapter 1 has been accepted for publication in *Critical Reviews in Environmental Science and Technology (CREST)* and is available at the following link: [http://www.tandfonline.com/doi/abs/10.1080/10643389.2011.556556](http://www.tandfonline.com/doi/abs/10.1080/10643389.2011.556556)

Chapter 2 examines health impacts from a historic water contamination event, referred to in the popular press as the “Washington, DC Lead-in-Drinking-Water Crisis”. This is the first paper to document adverse consequences in young children, in contradiction to prior studies which claimed that the three years of elevated lead in water did not raise blood lead above levels of concern. For the analysis, tens of thousands of children’s blood lead records were obtained from an independent source, and records of the city’s water lead data were obtained via Freedom of Information Act requests. Statistical analysis yielded findings that were highly consistent with expectations based on biokinetic modeling predictions and with prior research presented in the literature review. The author of this dissertation assisted in the analysis of trends between water lead levels and children’s blood lead levels, edited the manuscript and was responsible for all statistical testing. She is appropriately listed as second author of Chapter 2, which has been published in *Environmental Science and Technology (ES & T)*: [http://pubs.acs.org/doi/pdf/10.1021/es802789w](http://pubs.acs.org/doi/pdf/10.1021/es802789w). This chapter received the journal’s Best Paper Award in the Science Category in 2009 (Editor’s choice) and was the basis for a 2010 Congressional Investigation into the role of the Centers for Disease Control and Prevention.

Chapter 3 is a modeling paper, examining the response of children’s blood lead to different scenarios of exposure to water lead at home or at school. It expands upon the scope of traditional modeling approaches, which focused on a typical child under scenarios representative of chronic lead exposures, by proposing a paradigm shift to consider worst-case exposures as exemplified by modern public health goals. This re-evaluation radically changes prior conclusions, including the notion that school water lead levels 80-100 times higher than US EPA standards would be necessary to instigate parental health concerns. Indeed, results indicate that some instances of childhood elevated blood lead (i.e., lead poisoning) in schools are expected, and that acute health concerns from water can sometimes exceed those which have prompted recalls/fines for lead-
containing products not intended for human consumption. Chapter 3 is currently under preparation for submission in *Science of the Total Environment*.

Chapter 4 is a long-term bench-scale study, assessing the contribution of galvanic corrosion to lead contamination of drinking water after partial lead service line replacements. It was motivated by historic warnings in the literature, regarding erratic lead release after implementation of expensive partial replacements aimed to reduce lead in water, and a recent effort promoting partial pipe replacements as a costly solution to elevated lead in water in both the US and Canada. This work, initially unfunded, was supported in its latter stages by the Water Research Foundation and the National Science Foundation (the Water Research Foundation report is freely available on-line: [http://www.waterrf.org/ProjectsReports/PublicReportLibrary/4088b.pdf](http://www.waterrf.org/ProjectsReports/PublicReportLibrary/4088b.pdf)). The work was then upgraded for publication in *Journal American Water Works Association (JAWWA)*: [http://www.awwa.org/publications/AWWAJournalArticle.cfm?itemnumber=57364](http://www.awwa.org/publications/AWWAJournalArticle.cfm?itemnumber=57364). This chapter and paper informed the debate of the US EPA’s Scientific Advisory Board on this subject in 2011.

Chapter 5 addresses limitations in detection of elevated lead in drinking water samples. It demonstrates that certain routine methods can "miss" lead in collected water samples, with significant implications for regulatory compliance and human exposure assessment. This chapter is a logical extension of the authors’ M.S. work at Virginia Tech, related to particulate lead in drinking water in case studies of lead-poisoned children. Chapter 5 is currently under review for publication in *Environmental Monitoring and Assessment*.

Research presented herein has been discussed at the following illustrative locations:

- Draft Report of US EPA Scientific Advisory Board on the Effectiveness of Partial Lead Service Line Replacement (July 2011): [http://yosemite.epa.gov/sab/sabproduct.nsf/0/38b92187b29155dd852577f80050b4d9!OpenDocument&TableRow=2.2](http://yosemite.epa.gov/sab/sabproduct.nsf/0/38b92187b29155dd852577f80050b4d9!OpenDocument&TableRow=2.2)
- Three Articles in *Environmental Health Perspectives (EHP)*:
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CHAPTER 1. LEAD (PB) IN TAP WATER AND IN BLOOD: IMPLICATIONS FOR LEAD EXPOSURE IN THE UNITED STATES

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ABSTRACT

Lead is widely recognized as one of the most pervasive environmental health threats in the United States (US), and there is increased concern over adverse health impacts at levels of exposure once considered safe. Lead contamination of tap water was once a major cause of lead exposure in the US and, as other sources have been addressed, the relative contribution of lead in water to lead in blood is expected to become increasingly important. Moreover, prior research suggests that lead in water may be more important as a source than is currently believed. This review describes sources of lead in tap water, chemical forms of the lead, and relevant US regulations/guidelines, while considering their implications for human exposure. Research that examined associations between water lead levels and blood lead levels is critically reviewed, and some of the challenges in making such associations, even if lead in water is the dominant source of lead in blood, are highlighted. Better protecting populations at risk from this and from other lead sources is necessary, if the US is to achieve its goal of eliminating elevated blood lead levels in children by 2020.

KEYWORDS: plumbing, tap water, dissolved lead, particulate lead, regulations, health effects, blood lead level, correlation

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INTRODUCTION

Lead (Pb) is widely recognized as one of the most pervasive environmental health threats in the United States (US). Dramatic progress has been made over the last four decades to reduce lead exposure from gasoline, paint, dust, food/drink cans and drinking water (Shannon, 1996). However, despite reduced exposure from nearly all sources, clinical evidence has demonstrated adverse health impacts at blood lead levels once considered safe (Fadrowski et al., 2010; Jusko et al., 2008; Bellinger and Needleman, 2003). As a result, while the incidence of elevated blood lead (EBL) has markedly decreased, public sensitivity and medical concern about even low level lead exposure has increased. In order for the US to achieve its goal of eliminating all instances of EBL in children by 2020 (US Department of Health and Human Services, 2010), improved understanding of exposure to all lead sources is necessary.

Defining a "typical" case for childhood lead exposure can be misleading, because lead exposure affects individuals whose behavior and environments are infinitely variable. Nonetheless, it is often stated that in the typical case, drinking water consumption is believed to account for up to 20% of total lead exposure nationally (US EPA, 2006). But the US EPA also acknowledged that for infants consuming formula it may account for more than 50% of their total lead exposure, and further predicted that the relative importance of lead in water as a source would increase as other lead sources were being addressed (US EPA, 1991). Recent work has demonstrated that in some cases, lead from water can be the dominant source of exposure in children with EBL. For example, isolated cases of childhood lead poisoning in North Carolina and in Maine were tied to drinking water (Triantafyllidou et al., 2007). In addition, a 2009 study linked a period of very high lead-in-water contamination in Washington DC, with increased incidence of EBL for the youngest children tested (Edwards et al., 2009). Finally, the Centers for Disease Control and Prevention (CDC) publicized preliminary results of an epidemiological study, which demonstrated associations between children’s EBL and partially replaced lead water pipes (Frumkin, 2010).

The goal of this work is to conduct a critical review of the literature, with emphasis on:

- The release of hazardous levels of lead in tap water from old lead-bearing plumbing materials
- Lead contamination of tap water as a public health concern even in modern buildings, and in cities that might meet federal regulations for lead in tap water
- The absence of federal regulations for lead in drinking water of US schools and daycare facilities
- The difference between dissolved and particulate lead release into tap water, and the challenges in monitoring and exposure assessment associated with the particulate lead fraction
- Some of the challenges in associating Water Lead Levels (WLLs) to Blood Lead Levels (BLLs) in population studies or in case studies
- Important aspects of population studies that did, or did not, find associations between lead in water and lead in blood.
Sources and Potential Importance of Lead in Tap Water

Sources of Lead in Tap Water. Drinking water usually contains little or no lead when it leaves the water treatment plant and as it travels through water mains (Figure 1-1). But as it enters building plumbing through service line connections, it may come into contact with lead-containing plumbing materials (Figure 1-1). These materials include lead pipe, lead-containing solder used to join copper and other metallic pipes together, and plumbing devices made of lead-containing brass (e.g., water meters, valves, components in water fountains and in faucets) (Figure 1-1). As water flows through or sits stagnant in the pipes and in other plumbing devices, it can become contaminated with lead through a variety of complex electrochemical, geochemical and hydraulic mechanisms (Schock et al., 1996). Lead that is released from the plumbing can contaminate water at the tap in one of two forms: as particulate lead or as dissolved lead (Figure 1-1). Ingestion of lead-contaminated water is a direct pathway to lead exposure (bathing and showering with that water are not expected to cause health problems because human skin does not absorb lead in water) (CDC, 2010a).

Figure 1-1: Potential sources of lead contamination in tap water of homes, schools and other buildings.

Lead pipe. Lead pipe was used for the conveyance of drinking water, because it is easily formed, cut and jointed, and because its flexibility provides resistance to subsidence and frost (Schock et al., 1996). An advertisement by the National Lead Company in 1923 (National Geographic, 1923) illustrated that in many cities the law required that “lead pipe alone be used to bring water from street mains into the building”. Use of lead pipe in service lines was standard practice in many US cities through the 1950’s, and despite well-known health concerns was even occasional practice until the Congressional ban effective 1986. Considering health impacts from drinking water contamination, one historian characterized use of lead pipes in major cities as “one of the most serious environmental disasters” in US history (Troesken, 2006). Even though the use of lead
lead pipe in service lines or premise plumbing was prohibited in the US by the Safe Drinking Water Act (SDWA) amendment of 1986 (US EPA, 2006), older buildings may still be connected to lead service lines, lead goosenecks and other pure lead components. Depending on their length and diameter, water corrosivity, water use patterns as well as hydraulic patterns, lead service lines generally account for 50-75% of lead contamination at the tap in older homes where they are present (Sandvig et al., 2008).

**Partially Replaced Lead Pipe.** In the US, ownership of the lead pipe in service lines is typically shared between water utilities and homeowners. The controversial and expensive practice of replacing the utility’s portion of an old lead service line with copper, while leaving behind the customer’s portion, has been conducted in many cities with the goal of reducing lead in drinking water at the tap. Such replacements are termed "partial lead pipe replacements." This practice can actually increase water lead concentrations at least in the short-term (days to weeks), and for an undetermined duration beyond that time (Sandvig et al., 2008). The short-term effect is due to disturbance of the lead rust (i.e., corrosion scale) that has accumulated on the lead pipe over decades/centuries of use, and/or from creation of metallic lead particles when the lead pipe is cut. Recent research has also shown that in some situations, the creation of a galvanic cell (i.e., battery) between the lead pipe and the copper pipe may create serious water lead contamination in both the short-term and longer-term (Triantafyllidou et al., 2009a), confirming long-held concerns (Chambers and Hitchmough, 1992). This might explain the higher incidence of EBL in children living in homes with partially replaced lead pipe, when compared to homes with full lead pipes (Frumkin, 2010).

**Lead solder.** Solder is a fusible metal alloy which is melted to join metallic plumbing materials together in a strong and water-tight seal (Figure 1-1). An increased lead content in the alloy improves ease of use and reduces leaks, and solder containing 40-50% lead by weight was used in US buildings until banned in 1986. Thereafter, only “lead-free” solder, containing less than 0.2% lead by weight, was allowed in buildings. Lead solder is still available in hardware stores because it is legal for use in hobby electronics, and plumbers still illegally use leaded solder in some new buildings in the US (Goss, 2008) and in Scotland (Ramsay, 2003). In fact, a Scottish study found links between illegal use of leaded solder in new homes and blood lead of residents (Ramsay, 2003). The contribution of lead solder to lead in water at a given tap is extremely variable, and is dependent on the number of joints, their age, workmanship when the joint was created, surface area of the solder exposed to water at each joint and the water chemistry (Sandvig et al., 2008). Recent cases of childhood lead poisoning from drinking water in North Carolina and in Maine were tied to lead solder particles that corroded and detached into the water supply (Triantafyllidou et al., 2007).

**Brass (and bronze) plumbing components.** Brass and bronze are copper alloys that contain lead. Historically, lead was added to these alloys to reduce leaks (Showman 1994). According to congressional definition, “lead-free” brass components (e.g., strainers, check valves, water meters, couplings, fittings, faucets, drinking fountains, bubblers and water coolers) used in modern homes can legally contain up to 8% lead by weight (Figure 1-1). The contribution of a brass component (e.g., a faucet) to lead levels
measured at the tap depends on the lead content of the brass (typically ranging from 1.5-8% by weight), the volume of water in contact with the faucet, the physical configuration of the faucet and how it was manufactured, water corrosivity and water flow conditions (Sandvig et al., 2008).

Recent problems with persistent lead contamination of tap water (up to 300 µg/L lead) in new buildings at the University of North Carolina at Chapel Hill were attributed to “lead-free” brass/bronze ball valves, installed before drinking water fountains. Locating and removing these ball valves was necessary to eliminate the lead problems at the fountains (Elfland et al., 2010). There are also case studies, in which elevated lead in water from brass was suspected to be the primary contributor to cases of childhood lead poisoning (CDC, 1994). Sampling of homes in the Netherlands also revealed some severe cases of high lead release (up to 5030 µg/L) from brass faucets (Slaats et al., 2007). New brass alloys have been developed that contain very low lead (0.1-0.25% lead by weight) (Sandvig et al., 2008), and California and several other US states are beginning to require their use in new construction (Sandvig et al., 2007).

Other lead sources in tap water. Galvanized pipes are steel pipes coated with a protective layer of zinc, and high levels of lead can be present as impurities in the zinc coating (Shock et al. 1996). The iron rust in these pipes can also accumulate and store lead from other plumbing sources (HDR Engineering, 2009). Thus, even after lead pipe is replaced, lead accumulated in this iron rust can contribute elevated lead to tap water for years (HDR Engineering, 2009).

Rough estimation of US households at potential risk. While poor record keeping makes it practically impossible to determine the exact type of plumbing materials at individual US households, without exhuming and forensically evaluating plumbing materials underground and in walls, consideration of rough estimates is useful. Weston and EES (1990) determined through anonymous surveys of water utilities, that there were about 3.3 million lead service lines and 6.4 million lead pipe gooseneck connections in the US, corresponding to about 3% and 6% of total US housing units respectively (Table 1-1). For solder, it is estimated that the 81 million US housing units (77% of total US housing units) constructed prior to the federal ban of lead pipe and lead solder in 1986 (US Census, 2000) are virtually certain to contain lead solder joints (Table 1-1). In addition, all housing units built after 1986 are almost certain to have “lead free” brass plumbing devices that contain 1.5-8% lead by weight (Table 1-1). Only new housing units that incorporate non-leaded brass faucets and other non-leaded brass components (<0.1% lead by weight), can completely eliminate the presence of lead in plumbing, and it was only recently that such products could be purchased in non-leaded forms. It should be noted that the rough estimates presented (Table 1-1) refer to potential risk, and that like lead paint, degradation of leaded plumbing via corrosion and flaking of scale or rust to the water can dramatically increase the hazard to residents. In some situations lead in water for homes containing lead pipe, lead solder and/or leaded brass is virtually below detection, due to formation of protective surface coatings.
Table 1-1: Estimated number of US homes at potential risk from tap water lead contamination, depending on presence of lead-bearing plumbing materials.
The year 1986 marked the federal ban of lead pipe and lead solder, and established a maximum lead content of 8% by weight for “lead-free” brass plumbing components.

To offer an additional perspective, simple calculations suggest that the mass of lead present in a typical lead service line is about 19 kg (Table 1-1). If only 0.1% of this lead pipe is “eaten away” at the pipe wall due to corrosion and is released to the water, the released lead mass of 19 grams is sufficient to contaminate every drop of water used by a US family of three for 3 years over the federal action level of 15 µg/L (calculation based on 1135 L/day water usage for the whole family). Before half the pipe wall (i.e., 50% of the lead pipe) is eaten away, likely subjecting the lead pipe to leaks and mandatory replacement with unleaded materials, the potential lead release is sufficient to contaminate every drop of water used by a family for 1,500 years. Coupled with the direct path to possible human ingestion, this analysis puts the potential magnitude of the lead pipe problem into perspective, and highlights the importance of corrosion control and safe water use practices in avoiding potentially harmful exposure. In 1993, the US EPA estimated that more than 40 million US residents used water that can contain lead in excess of the federal action level of 15 µg/L (US EPA, 1993).
Lead pipes are more common in other countries. For example, the percentage of lead service lines in France, UK and Germany as of 1999 was estimated at 40-50% (Hayes and Skubala, 2009). As of 1999, premise (building) plumbing in Portugal, France and UK also contained 30-40% lead pipes (Hayes and Scubala, 2009). In Japan, as of 2002, a total of 667 km lead pipe were found below roads and 3,248 km of lead pipe were found in residential areas (Osawa, 2002).

Other sources of environmental lead exposure and perceptions regarding their relative importance. Lead products have been used in numerous other applications, all of which constitute potentially harmful exposure sources worthy of mitigation. Before improvements in corrosion control reduced lead in potable water in the 1950's and then again in the 1970's (Karalekas et al., 1976; Moore et al., 1985), it was widely accepted that lead in water was a dominant pathway of human exposure and that high incidence of miscarriages, infant and even adult mortality were attributable to this source (Troesken, 2008, 2006; Renner, 2007). While it is accepted that exposure to lead from any source is potentially harmful, maximizing public health gains with scarce available financial resources has necessitated creation of a modern hierarchy of perceived risk and reward for public health interventions. This, in turn, has occasionally put the different lead sources in competition with one another.

Some individuals in the lead poisoning prevention community have expressed a fear that “focus on lead in drinking water reduces attention on other and, potentially more important, sources of lead in the household environment (e.g., paint, dust)” (Blette, 2008). This mindset reinforces reports that in the early 1990’s the then CDC director of the former Center for Environmental Health “…railed against doing much in drinking water because he did not want to disarm lead in paint” (Powell, 1999). There has been some speculation that the scientific presentation of research results and public health messaging, in response to a well-publicized incident of elevated high lead in drinking water of Washington, DC, was affected by these concerns (US Congressional report, 2010; Edwards, 2010). On occasion, the lead paint: water risk: reward analysis has been invoked to justify diverting a portion of funding originally intended for reducing the public’s exposure to lead in water, towards creation of lead paint educational programs (Renner, 2010). It is important to acknowledge these issues, because neither scientists nor popular belief can be assumed to be completely immune from preconceptions, and continued debate about where to invest scarce resources will intensify with reduced availability of funding.

Clearly, peeling lead paint chips and associated dust pose a great health concern to US children (Levin et al., 2008; Jacobs 1995). Although the conventional wisdom in the US is that lead-based paint is the predominant source of lead poisoning in children, and all other lead sources are a distant second, an alternative perspective has been provided by authors such as Mielke and Reagan (1998). Based on their work, lead in soil and in dust, even when deteriorating lead paint is not a contributing factor (e.g., soil contamination attributable to smelter emissions, past use of leaded gasoline and other sources), can be an equally important exposure pathway, compared to lead paint that is deteriorating in place (Mielke and Reagan 1998). Much has been done to address all environmental lead sources, and much more needs to be done. Since 1977 the Consumer Product Safety...
Commission (CPSC) has limited the lead content of paint in the US to 600 parts per million (or else 0.06% by dry weight of the paint), but older residencies may have paint present with much higher lead content (up to 50% lead before 1955) (ATSDR, 2007). The US EPA's Office of Chemical Safety and Pollution Prevention also recently issued the lead Renovation, Repair and Painting (RRP) Rule to protect against exposure from renovations that disturb lead-based paint (US EPA, 2010).

After the landmark phase-out of commercial leaded gasoline, which was completed in 1995, 78% of air lead in the US is attributed to industrial emissions (Levin et al., 2008). The US EPA has set an enforceable national quality standard for lead in ambient air, while the Occupational Health and Safety Administration (OSHA) has set an enforceable permissible exposure limit for lead in workplace air (ATSDR, 2007). Lead is also present in consumer products. Dietary supplements, crystal glassware and ceramic pottery, polyvinyl chloride (PVC) miniblinds, synthetic turf, imported candy and foods, and imported children’s toys have been found to contain high levels of lead (Levin et al., 2008). The CPSC has recalled thousands of imported products, including children’s toys, which contained lead and did not meet US standards (Levin et al., 2008).

While the conventional wisdom is that lead in paint and in dust account for a majority of EBLs in US children, the CDC estimated that 30% or more of current EBL cases do not have an immediate lead paint source identified (Levin et al., 2008). The EPA (EPA, 2010) has recently expressed an opinion, shared by many others (Levin et al., 2008, Scott, 2009), that "as other agencies and EPA offices focus primarily on other sources of lead exposure (e.g. lead-based paint, lead in dust and soil, etc.) lead in drinking water as an exposure path is becoming a bigger percentage of a smaller number" (EPA, 2010).

US REGULATIONS/GUIDELINES FOR LEAD IN TAP WATER AND OTHER

PUBLIC HEALTH GUIDANCE

Lead and Copper Rule (LCR) of 1991. 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 (or else 15 µg/L) (US EPA, 1991). If lead concentrations exceed this action level (AL) 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. The EPA has also set a maximum contaminant level goal (MCLG) of zero for lead at the tap. As an MCLG, this guideline is not enforceable, but represents the optimal lead-in-water level below which there is “no known or expected risk to health”.

Implementation of the LCR in 1991 significantly controlled lead contamination at the tap, as evidenced by a recent review of monitoring data from homes in many large US cities. The review showed that 96% of US utilities were below the lead AL of 15 µg/L (US EPA, 2006a). The LCR replaced the previous standard of 50 µg/L, which was ineffective because it measured lead at the entry point to the distribution system and before contact with lead containing plumbing (see Figure 1-1). The LCR requires sampling at homes
known to have plumbing with highest potential for lead contamination, and after a minimum of 6 hours in which the sampled water has to contact the plumbing (US EPA, 1991).

Reliance on the 90th percentile lead level to determine compliance with the LCR means that there is no maximum contaminant level (MCL) for lead in consumers’ water to meet the Federal regulation. The US EPA explicitly acknowledged this in 1991, by stating that “the AL does not determine the compliance status of a system as does an MCL, but merely serves as a surrogate for a detailed optimization demonstration” (US EPA, 1991). The EPA further clarified that the LCR “is aimed at identifying system-wide problems rather than problems at outlets in individual buildings” and that “the 15 µg/L action level for public water systems is therefore a trigger for treatment rather than an exposure level” (US EPA, 2006).

To illustrate, consider actual lead-in-water data from volunteers in a large US city living in homes that are not necessarily at high risk, and which would be in compliance with the LCR (i.e., 90th percentile lead in water = 10 µg/L < 15 µg/L) (Figure 1-2). One percent of this population is exposed to over 70 µg/L lead, and 0.1% of the population is exposed to lead over 1,717 µg/L (Figure 1-2). If the US goal of eliminating EBL in all children by 2020 is to be achieved, the higher risk at the upper tail of the WLL distribution needs to be acknowledged and remediated. Consistent with the above points, it is not surprising that a recent case of lead poisoning was attributed to lead contaminated tap water in Durham, North Carolina, even though the city was compliant with the LCR (Triantafyllidou et al., 2007). In addition, because the LCR is designed to monitor effectiveness of corrosion control and does not protect individual consumers, only 100 homes must be tested in large cities (USEPA, 1991), which translates to far less than 1 out of 1,000 households. The key point of this discussion is that compliance with the LCR lead action level does not guarantee, or even imply, that all individuals in the city are protected from lead-in-water hazards.

Moreover, LCR testing loopholes may allow high lead levels to be “missed”, either accidentally or intentionally, in the relatively small number of homes that are sampled (Renner, 2009; Scott, 2009). For example, failure to pick the worst-case houses, not allowing water to stagnate long enough inside the plumbing before sampling, removing the faucet aerator screen before sampling, or sampling in cooler months, can allow compliance with the LCR AL for lead, and effectively hide serious water contamination (Renner, 2009). Sampling practices that can "miss" lead-in-water hazards have been employed in major US cities (Leonnig, 2004), although the majority of US water utilities sample tap water and report monitoring data with the safety of their consumers in mind.
Figure 1-2: Cumulative distribution of lead-in-water levels (in logarithmic scale) at consumers' home taps in a large US city from 1998 to 2005. Compiled from monitoring data of city residents, who voluntarily collected tap water samples and submitted them for lab analysis (Patch, 2006).

**Lead Contamination Control Act (LCCA) of 1988.** The LCR also applies to the 10% of US schools that have their own water supply (Table 1-2). However, it does not extend to the majority of US schools and daycare facilities, which rely on public water systems for their water supply (Table 1-2). Instead, the Lead Contamination Control Act (LCCA) provides non-enforceable guidelines for these schools and daycare facilities, recommending that drinking water should not exceed 20 µg/L lead in any 250 mL first-draw sample (US EPA, 2006) (Table 1-2). In other words, aside from the 10% of US schools that are regulated as public water systems under the LCR due to use of their own water supply or well, the remaining 90% of US schools and daycare facilities are not subject to any enforceable national lead-in-water standard (Table 1-2).
Table 1-2: US federal regulations and guidelines for lead in drinking water of homes and schools.

<table>
<thead>
<tr>
<th>Federal Statute</th>
<th>Lead and Copper Rule (LCR) of 1991 for homes served by public water systems</th>
<th>Lead Contamination Control Act (LCCA) of 1988</th>
<th>No regulation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Applies to</td>
<td>- Homes and other buildings served by a public water system (~85% of US homes)</td>
<td>Schools/daycares served by a public water system (~90% of US schools)</td>
<td>Homes with private water system (~15% of US homes)</td>
</tr>
<tr>
<td></td>
<td>- Schools/daycares regulated as “public water systems” * (~10% of US schools)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Enforceable?</td>
<td>Yes, federal regulation</td>
<td>No, voluntary guidance</td>
<td>Not Applicable</td>
</tr>
<tr>
<td>Required sample number</td>
<td>5-100 taps, depending on the size of the population served (reduced to 5-50 taps, for utilities previously compliant with the rule)</td>
<td>Each school water outlet used for drinking and cooking</td>
<td>None</td>
</tr>
<tr>
<td>Sampling Frequency</td>
<td>every 6 months (reduced to as little as once every 3 years for utilities previously compliant with the rule)</td>
<td>Not specified</td>
<td>None</td>
</tr>
<tr>
<td>Sampling Requirements</td>
<td>1 Liter cold water samples after at least 6 hours of stagnation</td>
<td>250 mL cold water samples after 8-18 hours of stagnation</td>
<td>None</td>
</tr>
<tr>
<td>Lead Limit</td>
<td>15 µg/L, termed “Action level” (AL)</td>
<td>20 µg/L</td>
<td>None</td>
</tr>
<tr>
<td>“Failure criterion”</td>
<td>Over 10% of samples exceeding AL of 15 µg/L lead (or else 90th percentile lead &gt; AL)</td>
<td>Any water sample exceeding 20 µg/L lead</td>
<td>None</td>
</tr>
<tr>
<td>Remediation Measures</td>
<td>Corrosion control optimization, lead service line replacement, public education</td>
<td>Flushing, point-of-use filters, remove plumbing, bottled water, public education</td>
<td>None</td>
</tr>
</tbody>
</table>

*Schools that regularly provide water to at least 25 individuals per day and use their own water source (e.g., private well), or treat, or sell their water, are regulated as “public water systems”.

The recommended guideline of 20 µg/L applied to lead in school water is considered more stringent than the 15 µg/L lead action level for homes, because a 250 mL water sample under the LCCA tends to concentrate the lead in collected samples, compared to the 1-L samples collected under the LCR (US EPA, 2010). Passage of the LCCA in 1988 prompted many schools to test for lead in drinking water, but state adoption and enforcement of the guideline was often weak and even nonexistent (Lambrinidou et al., 2010). By 1990 many schools had not repaired or removed lead-tainted coolers, used sampling protocols other than that recommended by EPA, carried out very limited or inappropriate sampling, or failed to conduct water testing at all (Lambrinidou et al., 2010).

A recent investigative report by the Associated Press (Burke, 2009) and subsequent congressional hearing (Freking, 2009) revealed problems with high lead in water of hundreds of schools regulated as public water systems under the LCR. In response, the EPA has stated it plans to better address and enforce lead standards in such situations.
Chapter 1. Lead (Pb) in Tap Water and in Blood: Implications for Lead Exposure in the US

(Freking, 2009). Although much less information is available for the 90% of schools not subject to any sampling requirements, case studies in Baltimore MD, Seattle WA, Philadelphia PA, Washington DC, Maryland suburbs and Los Angeles CA revealed serious problems with lead contamination of school water in recent years (Table 1-3). In the vast majority of these cases, lead-in-water hazards were not revealed by the schools under the LCCA, but by parents/students or investigative reporters (Table 1-3). With only one exception, at least three years elapsed from the time the schools recognized a problem to the time the public was informed. Another key point is that a large percentage of taps in some of the schools (up to 80%) had lead in water above the LCCA standard of 20 µg/L. In addition, some schools had taps dispensing water with lead-levels exceeding “hazardous waste” criteria (i.e., > 5,000 µg/L lead) (Table 1-3).

Remedial measures in these school systems varied from replacing bubbler heads or installing new fountains to installing filters, flushing, turning off fountains, and providing bottled water (EA Engineering, Science and Technology, 2007; Boyd et al., 2008b; Greenwire, 2004, Montgomery County Public Schools, 2007; Grover, 2008c). These remedial measures invariably relied on a “trial and error” approach. Thankfully, some of these school systems appear to have resolved the majority of lead-in-water problems, at least in the short-term. However, remediation sometimes involved millions of dollars to replace fixtures and fountains, only to have the problem return a few months later (Bach, 2005). Like lead paint, lead-in-water problems can never be considered fully resolved, until the lead-bearing materials have been completely removed. It is also worth noting that the schools described in Table 1-3 represent the “good news,” since most other school systems in urban areas have not systematically tested their water for lead in nearly three decades. Not shown in Table 1-3, are other case studies from: 1) Davidson, NC where a problem was discovered after a high school chemistry experiment failed and the teacher eventually traced it to high lead in water (Edwards, 2007), 2) Durham, NC where sampling revealed hazardous lead levels in some water fountains at 8 schools (Biesecker, 2006) and 3) cases in New Jersey (Burney and Dwight, 2003).

The limited attention on lead in drinking water of schools and day-care facilities is disconcerting, given the potential public health risk. First, school children are much more vulnerable to adverse health effects from lead exposure relative to adults (Needleman, 2004). Second, the intermittent pattern of water consumption, with periods of little or no water use on weekends, holidays and over summer break, produces very long stagnation periods of water inside the piping and can be worst case for releasing hazardous levels of lead from the plumbing into the water supply (Levin et al., 2008). Finally, school buildings have intricate plumbing systems, sometimes very old, containing multiple potential sources of water lead contamination. In 2004, the US EPA requested information and compiled a summary of state programs, regarding implementation of LCCA guidance (US EPA, 2004). More recently, acknowledging the lack of information on drinking water of schools, the US EPA announced that it is developing a draft “Charge on Safer Drinking Water in Schools and Child Care Facilities Initiative” that will seek input on how to assess the risks of lead in school drinking water (US EPA, 2010).
### Table 1-3: Representative case studies on lead-in-water problems at US schools.

<table>
<thead>
<tr>
<th>School System Location</th>
<th>Year School Knew of Problem</th>
<th>Year Public Informed</th>
<th>How Discovered</th>
<th>Average % taps above LCCA Guidance of 20 µg/L*</th>
<th>Highest Reported Pb in water (µg/L)</th>
<th>References</th>
</tr>
</thead>
<tbody>
<tr>
<td>Baltimore, MD</td>
<td>1992</td>
<td>2003</td>
<td>Parent inquired as to why water fountains had been turned off and a teacher turned whistleblower.</td>
<td>20% of fountains</td>
<td>655</td>
<td>City of Baltimore, 2007 Williams, 2008</td>
</tr>
<tr>
<td>Seattle, WA</td>
<td>1990</td>
<td>2003</td>
<td>A parent was concerned due to discolored water, collected and analyzed sample finding high lead.</td>
<td>1990: 33-40% &lt;br&gt;2004: 25%</td>
<td>1,600</td>
<td>Odell, 1991 &lt;br&gt;Cooper, 2008 &lt;br&gt;Boyd et al., 2008a</td>
</tr>
<tr>
<td>Philadelphia, PA</td>
<td>1993</td>
<td>1998</td>
<td>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.</td>
<td>2000: 38% of fountains&lt;br&gt;48% of faucets</td>
<td>N/A but 17% of schools&gt;100 µg/L</td>
<td>Fitzgerald, 2000 &lt;br&gt;Bryant, 2004</td>
</tr>
<tr>
<td>Washington Suburban, MA</td>
<td>2004</td>
<td>2004</td>
<td>School system voluntarily collected samples to participate in LCCA after problems were revealed in Washington DC</td>
<td>2004: 18%</td>
<td>36,372</td>
<td>Gerwin, 2004 &lt;br&gt;Montgomery County Public Schools, 2004</td>
</tr>
<tr>
<td>Los Angeles, CA</td>
<td>1998</td>
<td>2008</td>
<td>Local news station. School personnel falsified daily reports regarding remedial flushing to reduce lead.</td>
<td>2008: 30%</td>
<td>N/A</td>
<td>Lambrinidou et al., 2010 Grover, 2008a; 2008b</td>
</tr>
</tbody>
</table>

N/A: Not Available

*All data from Washington DC schools in this table use 15 µg/L as a failure criterion

Bold italics indicate lead-in-water levels that were high enough (i.e., > 5000 µg/L) to classify the drinking water as “hazardous waste”, based on the Toxicity Characteristic Leaching Procedure (TCLP) test, which regulates lead in waste at a level of 5 ppm or else 5,000 µg/L (US EPA, 2009)
Despite these recently acknowledged problems with elevated lead in school water, one analysis that was conducted to examine the health risks, suggested that there was little cause for concern. Sathyanarayana et al. (2006) simulated typical-case and worst-case scenarios of drinking water consumption at Seattle schools, and predicted reassuring blood lead levels for school children of below 5.0 µg/dL in all cases. However, these authors dismissed the highest detected lead-in-water measurements as unrepresentative, and only considered the geometric mean blood lead level of the student population using a bio-kinetic model. It is likely that explicit consideration of the highest measured lead-in-water samples, and resultant impacts on blood lead of more sensitive children as opposed to only the geometric mean (i.e., the 50th percentile of blood lead levels), would indicate a much more serious risk. In support of this hypothesis, it was recently revealed that a child with elevated blood lead from water in Greenville, NC was exposed in a daycare center (Robertson, 2006), and environmental assessments in Washington DC attributed a child's elevated blood lead to contaminated water (7,300 µg/L lead) at an elementary school (Lambrinidou et al., 2010). Concerns related to a case of adult lead exposure for a teacher in an Oregon school in 2008, gave impetus to testing of tap water for water fountains at work which revealed high lead in water (Lambrinidou, 2008). Reports of harmful exposure are more consistent with common sense expectations, considering that the higher levels of lead detected in some schools (Table 1-3) indicate that a single glass of water can contain up to 29 times more lead than that deemed to constitute an acute health risk according to the CPSC (i.e., 20,000 µg/L lead in a 250 mL sample constitutes a single dose of 5000 µg lead, while the CPSC criterion is set at 175 µg lead).

Unregulated drinking water systems. About 15% of Americans operate their own private drinking water supplies (e.g., private wells and cistern type systems) (US EPA, 2006c). These systems are not subject to federal standards for lead monitoring (and other contaminants), although the major lead sources are similar to those found in public water supplies (Table 1-2). As a result, the magnitude of lead-in-water problems at these homes and the potential public health risks have not been studied (Schock et al., 1996).

Other public health guidance as it relates to lead contamination of tap water. The LCR and LCCA lead limits were derived from an estimation of lead concentrations considered at the time economically and technologically feasible to achieve, and as such, are not entirely health-based (Lambrinidou et al., 2010). A compilation of other health-based thresholds (Table 1-4) indicates that the US EPA Maximum Contaminant Level Goal (MCLG) for lead in water is equal to zero and that the state of California has developed its own Public Health Goal (PHG) for lead in water at 2 µg/L. The US EPA at one time indicated that 40 µg/L lead in water poses an “imminent and substantial endangerment to children” (Table 1-4). Health Canada (1992) and the World Health Organization (1993) have both developed a health-based guideline of 10 µg/L lead for drinking water, while the CDC (2010) advises children and pregnant women to not consume water that contains more than 15 µg/L lead (Table 1-4). As a further point of reference, the US Consumer Product Safety Commission (CPSC) classified a lead dose of 175 µg as an “acute health risk” to children (CPSC, 2005). This CPSC standard was used as a trigger for recalling millions of children’s toy jewelry (CPSC, 2005). If this standard, which was applied to
children’s jewelry and toys (products not intended for human consumption), was applied to lead in water (a product intended for human consumption), the one-time ingestion of 250 mL of water at 700 µg/L lead (resulting in a lead dose of 175 µg) would also be classified as an “acute health risk” to children (Table 1-4). Finally, water containing more than 5,000 µg/L lead exceeds “hazardous waste” criteria (US EPA, 2009).

Table 1-4: Public health guidance regarding various levels of lead in tap water.

| Agency                              | Lead Threshold (µg/L) | Health Guidance and/or Warning                                      | Reference       |
|-------------------------------------|----------------------|===================================================================|-----------------|
| US Environmental Protection Agency  | 0                    | Maximum Contaminant Level Goal (MCLG), below which there is no known or expected risk to health | US EPA (1991)   |
| California Environmental Protection Agency | 2                    | Public Health Goal (PHG) for all age groups                      | Cal/EPA (1997)  |
| Health Canada                       | 10                   | Maximum Acceptable Concentration (MAC) based on chronic health effects, for all age groups | Health Canada (1992) |
| World Health organization           | 10                   | Health-based guideline for all age groups                        | WHO (1993)      |
| CDC                                 | 15                   | Children and pregnant women should not drink the water           | CDC (2010a)     |
| US Environmental Protection Agency  | 40                   | Imminent and substantial endangerment to children (warning removed in 2004) | Renner (2010)   |

*lead dose of 175 µg translated to lead exposure through water consumption of 250 mL (one glass)
#based on the Toxicity Characteristic Leaching Procedure (TCLP) test for waste

**FORMS OF LEAD IN TAP WATER AND IMPLICATIONS FOR MONITORING AND EXPOSURE**

**Dissolved versus particulate lead in tap water.** Lead that is released from plumbing into drinking water can be present in a variety of distinct physicochemical forms including free aqueous ions, inorganic complexes, organic complexes, associations with highly dispersed colloidal matter, suspended particles of insoluble salts, or adsorbed on inorganic particulates (De Rosa and Williams, 1992). In some practical tests, the total lead content of drinking water is often demarcated into two fractions: the “dissolved lead” fraction and the “particulate lead” fraction (Table 1-5). Dissolved lead is
operationally defined as the fraction of total lead in water that is small enough to pass through a filter of 0.45 µm pore size (Mc Neill and Edwards, 2004). Particulate lead is the fraction of total lead in drinking water that is retained by a filter of 0.45 µm pore size (Table 1-5). At the upper end of particulate lead sizes, these particles are big enough to be seen by the naked eye.

Table 1-5: Classification of lead species in tap water and distinction between dissolved lead and particulate lead. Adapted from De Rosa and Williams (1992).

<table>
<thead>
<tr>
<th>Operational Definition</th>
<th>Approximate Diameter Size (µm-log scale)</th>
<th>Class</th>
<th>Example(s)</th>
</tr>
</thead>
<tbody>
<tr>
<td>“Dissolved” Lead</td>
<td>0.001</td>
<td>Free aquo ions</td>
<td>Pb(^{2+})</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Organic chelates, other inorganic ions, ion pairs and complexes</td>
<td>Pb-EDTA PbCO(_3)</td>
</tr>
<tr>
<td></td>
<td>0.01</td>
<td>Bound to macromolecules</td>
<td>Pb-fulvic acid complexes</td>
</tr>
<tr>
<td></td>
<td>0.1</td>
<td>Highly dispersed colloidal material</td>
<td>Adsorbed on hydrous iron and manganese oxide colloids</td>
</tr>
<tr>
<td>“Particulate” Lead</td>
<td>0.45</td>
<td>Adsorbed on inorganic particulates</td>
<td>Adsorbed on hydrous iron and manganese oxides and clay minerals</td>
</tr>
<tr>
<td></td>
<td>10+</td>
<td>Minerals and precipitates</td>
<td>PbCO(_3)(s)-Cerussite Pb(_3)(CO(_3))(_2)OH(_2)(s)-Hydrocerussite</td>
</tr>
</tbody>
</table>

Lead particles in tap water can originate from detachment of lead-bearing scale or rusts from plumbing, or by scouring/sloughing-off during water flow (Schock, 1990). Lead corrosion rusts in water plumbing materials are analogs of peeling lead paint, in that degradation of the underlying plumbing material can dramatically increase the creation of these particles, their detachment, and resulting human exposure. Indeed, the mineralogical forms of many lead rusts (i.e., cerussite and hydrocerussite, see Table 1-5) are identical to those in lead paint. Lead particles in tap water may also originate from physically degraded pieces of leaded brass, lead solder or lead pipe (Triantafyllidou et al., 2007). Unlike the case of dissolved lead in water, which is not controlled by nuances of water flow from the tap, the mobilization of particulate lead from plumbing can be highly variable, depending on changes in pressure and water flow velocity/direction (Schock, 1990).

Numerous investigators have reported lead particles in water. Flaking lead particles larger than 12 µm in diameter were observed detaching from pipe, along with colloidal lead fractions associated with iron oxides and humic acids (De Mora et al. 1987; De Rosa and Williams, 1992). An extensive British survey reported that the flaking lead problems were caused by large black/brown particles visible to the consumer, whereas colloidal lead problems were caused by smaller particles that were not visible (De Rosa and Williams, 1992). The British report further concluded that problems with particulate lead
were often associated with the presence of iron particulates, and that these problems were exacerbated by high water flows, especially during periods of high water demand (i.e., in the summer), as was recently highlighted in the US (HDR Engineering, 2009).

A small survey of lead in potable water from around the US revealed numerous instances in which lead was also present as particulates, sometimes at concentrations greater than 1,000 µg/L (McNeill and Edwards, 2004). Particulate lead was also clearly demonstrated to detach from lead-tin solder joints (Bisogni et al., 2000) and from lead pipes (Triantafyllidou et al., 2009a) in laboratory test rigs. In these laboratory studies, particulate lead was the predominant form of lead, comprising up to 99% of the total lead concentration in water samples (Triantafyllidou et al., 2009a).

Field investigations at various US locations with significant lead-in-water problems revealed that particulate lead release from the plumbing was often the cause (Figure 1-3; Table 1-6), and in some cases the source of the lead problem could be forensically linked to either lead pipe, lead solder or leaded brass (Table 1-6). A key point is the extraordinarily high levels of lead (up to 190,000 µg/L, or else more than 12,000 times the EPA action level) occasionally present in the water due to these particles, and their varying mineralogical content ranging from 3% to 100% lead (Table 1-6). The massive lead contamination occasionally resulting from partial lead pipe replacements is especially noteworthy, in light of the CDC report of EBL in Washington DC children (Frumkin, 2010).

Figure 1-3: Lead-baring particles were identified as the cause of severe tap water contamination during field investigations. (A, B) Brass particles trapped in two different strainers adjacent to two drinking water fountains at UNC, Chapel Hill-Photos from Elfland et al., (2010). (C) Lead solder particles trapped in home faucet aerator screen in Washington, DC-Photo from Edwards (2005). (D) Lead solder particles trapped in home faucet aerator screen in Greenville, NC- Photo from Triantafyllidou et al. (2007).
### Table 1-6: Origin of representative lead particles identified in drinking water during field investigations, and level of resulting water contamination.

<table>
<thead>
<tr>
<th>Location of case study</th>
<th>Surface composition of Lead-Bearing Particle(s)</th>
<th>Origin of lead particle(s)</th>
<th>Total Pb Concentration in Water (federal standard is 15 µg/L)</th>
<th>Documented Lead Poisoning?</th>
<th>Reference(s)</th>
</tr>
</thead>
<tbody>
<tr>
<td>University of North Carolina at Chapel Hill, NC</td>
<td>3-22 %Pb, 26-66% %Cu 4-40 %Zn (3-19 %Fe, 0% Sn)</td>
<td>Leaded Brass</td>
<td>Up to 350 µg/L</td>
<td>No</td>
<td>Elfland et al. (2010)</td>
</tr>
<tr>
<td>Greenville, NC</td>
<td>4 – 51% Pb 1-70% Sn (0-6% Cu)</td>
<td>Lead Solder</td>
<td>Up to 10,500 µg/L</td>
<td>Yes</td>
<td>Triantafyllidou et al. (2007)</td>
</tr>
<tr>
<td>Durham, NC</td>
<td>17-52% Pb 37-66% Sn</td>
<td>Lead Solder</td>
<td>Up to 650 µg/L</td>
<td>Yes</td>
<td>Edwards et al. (2006)</td>
</tr>
<tr>
<td>Raleigh, NC</td>
<td>3% Pb 97% Sn</td>
<td>Lead Solder</td>
<td>2,413 µg/L</td>
<td>No</td>
<td>Parks and Edwards (2008)</td>
</tr>
<tr>
<td>Manchester, ME</td>
<td>Pb and Sn (levels not specified)</td>
<td>Lead Solder</td>
<td>Up to 3,200 µg/L</td>
<td>Yes</td>
<td>Unpublished (2006)</td>
</tr>
<tr>
<td>Washington DC (after partial lead service line replacement)</td>
<td>Unknown, but presumably metallic lead (i.e., 100% Pb) and lead rusts</td>
<td>Lead Service Line</td>
<td>Up to 190,000 µg/L</td>
<td>Yes</td>
<td>Frumkin (2010) DC WASA (2008)</td>
</tr>
<tr>
<td>Washington, DC</td>
<td>63% Pb 37% Sn</td>
<td>Lead Solder</td>
<td>Not available</td>
<td>No</td>
<td>Edwards (2005)</td>
</tr>
<tr>
<td>Washington DC</td>
<td>Not analyzed</td>
<td>Lead solder, leaded brass</td>
<td>Up to 974 µg/L*</td>
<td>Yes</td>
<td>Unpublished (2008)</td>
</tr>
<tr>
<td>Washington DC Suburban Area</td>
<td>1.6-9.9 % Pb, 60-79% Sn, 1.8-5.0% Cu</td>
<td>Lead Solder, leaded brass</td>
<td>Up to 1,403 µg/L*</td>
<td>No</td>
<td>Unpublished (2006)</td>
</tr>
<tr>
<td>Small Community, TN</td>
<td>Not Analyzed</td>
<td>Lead Solder, Confirmed onsite via Spot Test</td>
<td>Up to 2,886 µg/L</td>
<td>No</td>
<td>Edwards et al. (2007)</td>
</tr>
</tbody>
</table>

*Aside from lead and tin presence, high amounts of copper and zinc in water samples suggested that brass was also contributing to the problem.

**Implications of particulate lead in tap water for monitoring, exposure assessment and corrosion control.** Chemical lead solubility models, human exposure models, water sampling protocols, and analytical quantification methods are often based on the presumed dominance of dissolved lead in drinking water. It has only recently been recognized that particulate lead can occasionally be the dominant form of lead in drinking water (Triantafyllidou et al., 2007). A preliminary synthesis (Table 1-6) indicates that
such problems may not be an isolated occurrence, especially given the rarity of such investigations.

It is useful to highlight some of the challenges associated with the presence of particulate lead in tap water, in terms of environmental monitoring and exposure. All models predicting lead at the tap, do so by considering soluble lead (Schock, 1990). Because the release of particulate lead in drinking water is often caused by physical factors and is erratic, its contribution is impossible to predict (Schock, 1990). At the same time, capturing actual particulate lead spikes in tap water via field sampling is very challenging. Schock et al. (2008) warned that if lead (and other contaminants) were mobilized into solution or released as particulates, this would result in long-term intermittent exposures of unknown impact that can easily go undetected.

Particulate lead in water can be ingested, and subsequently be dissolved or mobilized by human stomach acid (Schock, 1990). Mahaffey (1977) reported that lead absorption from small lead particles is greater than lead absorption from large particles. However, she also reported that when large pieces of lead are ingested, they may lodge in the gastrointestinal tract, and cause severe lead poisoning as they slowly dissolve. Tests on potential bioavailability of lead solder particles collected from homes of lead-poisoned children in Greenville, NC and Durham NC, revealed that a significant fraction of the particulate lead from solder dissolved in simulated gastric fluid (Triantafyllidou et al., 2007). Additional case studies of childhood lead poisoning in Maine and in Washington DC (Table 1-6), which were attributed to lead-baring particles that detached from the plumbing and contaminated tap water, also provide unambiguous proof that these lead particles were indeed bio-available once ingested.

In order to protect consumers from such exposures, corrosion control programs need to account for/prevent particulate lead release into the water. Modern corrosion control strategies were designed to reduce leaching from lead pipe, solder and brass materials by encouraging formation of low solubility lead hydroxyl-carbonate and phosphate films on the plumbing material surface, which can limit contamination to flowing water. But control of particulate release is dependent on minimizing the destabilization of the protective rust layer from water quality changes or hydraulic disturbances, and this process is poorly studied. The drinking water industry currently lacks the tools or knowledge to completely prevent or control particulate lead release.

**BLOOD LEAD LEVEL AND MAJOR LEAD TOXICITY MECHANISMS**

Potential harm from exposure to lead is typically tracked by measurements of the blood lead level (BLL). BLLs above 10 µg/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 (CDC, 2005). Cases in which blood lead exceeds 10 µg/dL or 20 µg/dL are also termed “lead poisoning”, dependent on the specific US jurisdiction. CDC surveillance for the year 2007 corresponded to 13% (or else 3,136,843) of US children aged < 6 years, of which 31,524 were diagnosed with EBLL (CDC, 2010).
Depending on the extent of uptake by the blood stream (Table 1-7), lead disturbs the heme biosynthetic pathway and can lead to anemia (Singhal and Thomas, 1980), causes kidney malfunction or even kidney failure (Loghman-Adham, 1997), but most importantly generates brain disorders in children (Needleman, 2004). Lead is a neurotoxin, which has the capacity to enter the blood-brain barrier and affect the central nervous system (CNS) of children (National Research Council, 1993). Nerve signaling is highly regulated by movements of charged ions, such as calcium, across cell membranes. At picomolar concentrations lead (Pb\(^{2+}\)) outcompetes/inhibits calcium (Ca\(^{2+}\)) from entering cells, halts release of neurotransmitters from the cell, and thus disrupts nerve signaling (Needleman, 2004). Encephalopathy (i.e. brain disorder) due to elevated lead burden has been associated with lower intelligence scores (IQ), learning disabilities, hyperactivity, attention deficit disorders, hearing/speech impediments, seizures, behavioral impairments/aggression, while some ecological studies even support an association with crime (Needleman, 2004). In addition, lead is considered an embryo-fetal poison for pregnant women, which at high levels has been historically associated with instantaneous abortion, premature delivery, stillbirth, infant mortality, low birth weight, and compromised mental and physical development of infants (Troesken, 2008, 2006; Mahaffey, 1985).

Table 1-7: Blood lead level (BLL) and adverse health effects in children and in adults. The “BLL of Concern” is currently set at 10 \(\mu\)g/dL. Adapted from Troesken (2006) and National Research Council (1993).

<table>
<thead>
<tr>
<th>Age Group</th>
<th>Children</th>
<th>Adults</th>
</tr>
</thead>
<tbody>
<tr>
<td>BLL ((\mu)g/dL)</td>
<td>Adverse Health Effects</td>
<td></td>
</tr>
<tr>
<td>&lt;10</td>
<td>IQ (-), Hearing (-), Growth (-)</td>
<td>Uncertain</td>
</tr>
<tr>
<td>&gt;10</td>
<td>Erythrocyte protoporphyrin (+)</td>
<td>Hypertension</td>
</tr>
<tr>
<td>&gt;20</td>
<td>Nerve conduction (-)</td>
<td>Erythrocyte protoporphyrin (+)</td>
</tr>
<tr>
<td>&gt;30</td>
<td>Vitamin D metabolism (-)</td>
<td>Systolic blood pressure (+), Hearing (-)</td>
</tr>
<tr>
<td>&gt;40</td>
<td>Hemoglobin synthesis (-)</td>
<td>Nerve conduction (-), infertility (men), kidney failure</td>
</tr>
<tr>
<td>&gt;50</td>
<td>Colic, frank anemia, kidney failure, brain disorders</td>
<td>Hemoglobin synthesis (-), frank anemia, brain disorders</td>
</tr>
<tr>
<td>&gt;100</td>
<td>death</td>
<td>death</td>
</tr>
</tbody>
</table>

(-) Decreased function, (+) Increased function

Hemoglobin: the molecule which carries oxygen throughout the body
Nerve Conduction: ability to send the impulse from the nerve to the muscle.
Vitamin D: necessary for the absorption of calcium and phosphorus, and for bone growth
Erythrocyte protoporphyrin: Intermediate in heme biosynthesis
Recent studies suggest that decreased IQ and cognition occur in children even at BLLs as low as 3.0 μg/dL (Jusko et al., 2008; Bellinger and Needleman, 2003), and that impaired kidney function occurs in adolescents even at BLLs as low as 1.5 μg/dL (Fadrowski et al., 2010). Emerging clinical evidence is therefore strongly reinforcing the notion that no safe level of lead exposure exists. Lead toxicity (Table 1-7) is notoriously difficult to diagnose, and creates a wide range of symptoms which are easily overlooked (Kalra et al., 2000). In light of these and other evidence, the US Department of Health and Human Services had established the ambitious goal of eliminating EBLLs in US children by 2010 (US Department of Health and Human Services, 2000). This was a qualitatively different goal from earlier policy, which focused on reducing the BLL considered toxic by various target amounts (CDC, 2005). Meeting the Healthy People 2010 objective to eliminate EBLLs (i.e., BLLs ≥10 μg/dL) in children was not achieved, and the US is extending this goal to 2020 (US Department of Health and Human Services, 2010).

IMPORTANT CONSIDERATIONS IN ASSOCIATING LEAD IN WATER TO LEAD IN BLOOD

Troesken (2006) acknowledged that exposure to water lead is subject to “an error-in-variables problem”, which makes it challenging to find an association to health risks, and introduces a downward bias into commonly applied statistical techniques attempting to link Water Lead Levels (WLLs) to Blood Lead Levels (BLLs). In order to avoid such a bias, it is necessary to meet several preconditions when attempting to associate BLLs to WLLs in population studies or in case studies:

- Water lead measurements and blood lead measurements need to be available, and without significant sampling delays between the two,
- Water lead measurements need to quantify the actual lead content of the water,
- Individual water consumption patterns need to be accounted for, and
- Individual responses to the same lead dose need to be understood.

Some of the difficulties in meeting the above criteria (Table 1-8) are highlighted in this section.

*Paired BLLs and WLLs are not always available.* It is obviously necessary to obtain BLL and WLL data, in order to examine any potential association between the two. For a variety of reasons (Table 1-8) described below, such data are often unavailable.

Lack of BLL data for sensitive sub-populations. In the US children’s blood lead screening is targeted to children at highest risk for exposure to lead paint and lead dust hazards (CDC, 2002), typically aged 1 to 6 years with developed hand-to-mouth activity (Linakis et al., 1996). Relatively little data is available for children aged less than 9 months, who are most vulnerable to lead exposure through water, due to use of reconstituted milk formula (Shannon et al., 1992; Edwards et al., 2009).
Table 1-8: Potential difficulties in associating lead in water to lead in blood in population studies or in case studies.

<table>
<thead>
<tr>
<th>Issue</th>
<th>Illustrative Reference(s)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>BLLs and WLLs are not always available</strong></td>
<td></td>
</tr>
<tr>
<td>General lack of BLL measurements for sensitive sub-populations</td>
<td>Binder et al., 1996</td>
</tr>
<tr>
<td></td>
<td>Shannon et al., 1992</td>
</tr>
<tr>
<td></td>
<td>Edwards et al., 2009</td>
</tr>
<tr>
<td>General lack of WLL measurements in schools/daycares under the LCCA</td>
<td>Lambrinidou et al., 2010</td>
</tr>
<tr>
<td>Relatively small number of WLL measurements under the LCR</td>
<td>Renner, 2009</td>
</tr>
<tr>
<td>Relative exclusion of water lead measurements during home assessments of lead-poisoned children</td>
<td>Renner, 2009</td>
</tr>
<tr>
<td></td>
<td>Scott, 2009</td>
</tr>
<tr>
<td><strong>WLL measurements do not always reflect actual lead in water</strong></td>
<td></td>
</tr>
<tr>
<td>Improper water sampling/preservation methods at “high-risk” taps under the LCR:</td>
<td>Triantafyllidou et al., 2007</td>
</tr>
<tr>
<td>- Flow rate</td>
<td></td>
</tr>
<tr>
<td>- Cold versus hot water</td>
<td></td>
</tr>
<tr>
<td>- Sample preservation</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Triantafyllidou et al., 2009</td>
</tr>
<tr>
<td>Inherent variability in lead release from plumbing:</td>
<td></td>
</tr>
<tr>
<td>- Spatial (fluctuations within a city, a neighborhood, or even a single home)</td>
<td>Levin, 2008</td>
</tr>
<tr>
<td>- Temporal (fluctuations in a single tap depending on season, or even time of day)</td>
<td>Schock, 1990</td>
</tr>
<tr>
<td></td>
<td>Matthew, 1981</td>
</tr>
<tr>
<td><strong>Individual water consumption patterns affect individual exposure</strong></td>
<td></td>
</tr>
<tr>
<td>Variability in individual water consumption patterns:</td>
<td>Troesken, 2006</td>
</tr>
<tr>
<td>- Amount of water consumed in/outside of home</td>
<td>Matthew, 1981</td>
</tr>
<tr>
<td>- Use of tap/filtered tap/bottled water</td>
<td></td>
</tr>
<tr>
<td>Underestimated indirect contribution of water to the total dietary lead intake:</td>
<td>Triantafyllidou et al., 2007</td>
</tr>
<tr>
<td>- Preparation of foods and beverages</td>
<td>Mesch et al., 1996</td>
</tr>
<tr>
<td></td>
<td>Moore, 1983</td>
</tr>
<tr>
<td><strong>Individual risk factors affect individual response to a fixed lead dose</strong></td>
<td></td>
</tr>
<tr>
<td>Bioavailability of lead varies between individuals, depending on:</td>
<td>Troesken, 2006</td>
</tr>
<tr>
<td>- Age</td>
<td>Lanphear et al., 2002</td>
</tr>
<tr>
<td>- Diet</td>
<td>Matthew, 1981</td>
</tr>
<tr>
<td>- Genetics</td>
<td></td>
</tr>
</tbody>
</table>
General lack of WLL data at schools and day care facilities. As of 2006, a survey by the CDC found that nearly half of all schools nationwide do not test their water for lead (Lambrinidou et al., 2010). A 2006 analysis by the US Government Accountability Office (GAO) revealed that few states have developed voluntary comprehensive testing and remediation programs for lead in school drinking water, and that about half the states have not developed programs at all (Lambrinidou et al., 2010). A recent nationwide Associated Press survey on the 10% of US schools that are subject to the LCR, revealed that lead-contaminated drinking water affects schools in at least 27 states (Lambrinidou et al., 2010). There is no scientific or practical reason to believe that the problem does not extend to other schools and to other states, which are not being monitored for lead-in-water problems.

Relative exclusion of water lead measurements during assessments of lead-poisoned children. Management strategies for childhood lead poisoning in the US have been developed based on the assumption that the LCR eliminated elevated water lead and that other environmental sources (e.g., lead in paint, dust or soil) are the most likely culprit. Current CDC guidance states that “if prior testing of a public water system shows that lead contamination is not a problem in homes served by that system, no additional testing is necessary, unless no other source of a child’s EBLL can be found” (CDC, 2002). Public health agencies routinely misinterpret compliance with the LCR action level as eliminating the need for water sampling in homes, schools or day care facilities of lead-poisoned children.

A Virginia Tech survey in 2006 verified that drinking water sampling is not standard practice during home assessments of lead-poisoned children. From the seventeen states that responded to the survey, only two required water testing in all cases of EBLL. Three of the jurisdictions “often” tested the water, eight of the jurisdictions “sometimes” tested the water, while four said they “never” did. A follow-up survey by the Alliance of Healthy Homes (Scott, 2009) revealed that in a state with a severe lead-poisoning rate “water is tested when no lead paint violations are identified, but this is virtually never”. Another state with similar problems claimed to “occasionally do this [test the water] if it’s the only way to convince the parents that the real hazard is lead-based paint in their home” (Scott, 2009). A different survey by the CDC (Renner, 2009) showed that fifteen lead grantee municipalities “routinely” collected water samples during home inspections, and that sixteen “sometimes” sampled drinking water (if lead was not found in paint/dust, or if drinking water was provided by a private well/ unregulated water system), while seven “never” tested drinking water.

Even when sampling is conducted, the CDC does not provide specific guidance on when and how to test water for lead (Renner, 2009). If a water sample is taken at all, it is typically a flushed sample taken during the inspection. This means that in the few instances where health agencies do collect tap water at homes of lead poisoned children, they are usually not collecting “worst-case” samples, and are thus not capturing worst-case lead-in-water exposures (Renner, 2009).
**WLL measurements do not always reflect actual lead in water.** In order to assess the public health risk from elevated lead in tap water, it is obviously necessary to first measure the actual lead content of the water. But lead-in-water measurements can be controlled by the season, day, hour of measurement, and subtle differences in sample collection procedures can either detect or miss lead spikes (Table 1-8).

Improper water sampling/preservation methods at “high-risk” taps may miss some of the lead present in water. Standard sampling/analytical protocols are adequate in quantifying lead in water in the typical case. In exceptional cases, e.g. when childhood lead poisoning may be caused by water, the detection of lead hazards can be critically dependent on the specifics of sampling.

Flow rate during sample collection. The most recent guidance for schools (EPA, 2006) suggests to “induce a small (e.g., pencil-sized) steady flow of water from the outlet”. These instructions translate to an unrealistically low flow rate of less than 1 L/min. Yet every-day water consumption typically employs higher flow rates, at which it has been long known that the water may physically scour lead deposits from the pipe (Britton & Richards, 1981; Schock, 1990). Sampling at a higher flow rate would therefore more likely capture lead spikes due to particulate lead release, and would be more representative of typical water usage. Collecting water from a “high-risk” tap at the EPA-recommended low flow rate “missed” 90% of the particulate lead present (Edwards, 2005), during a home investigation in Washington DC in 2006 (Figure 1-4).

![Figure 1-4: Lead measurement in flushed tap water samples versus flow rate in a home with lead pipe. Error bars represent 95% confidence intervals over triplicate samples collected on subsequent days at each indicated flow rate. Sample collection at the kitchen tap was timed to collect water derived from the lead pipe. (Edwards, 2005)]
Sampling of hot versus cold water. Existing protocols under the LCR and lead poisoning case management only require sampling of cold tap water. Instead of sampling hot tap water, which is occasionally known to contain much higher lead, the EPA (2006) simply recommends that consumers never drink hot water or use it for cooking. A case study in Australia, where three individuals were diagnosed with lead poisoning, revealed that hot tap water contained 260 times more lead than did cold tap water (Mesch et al., 1996). The family members used hot water to prepare instant coffee and to cook. In another Australian study, water was collected from water boilers, coffee machines from restaurants, offices, workplaces and schools. Excessive levels of lead were found in 67% of the samples, probably due to the contact of brass components with the hot water (McCafferty et al., 1995). In Washington DC, review of environmental risk assessments in the homes of children with elevated BLLs during 2006-2007, revealed that more than 50% of caregivers who were asked, stated that they used unfiltered hot tap water to mix infant formula, powdered milk, and juice (Lambrinidou and Edwards, 2008). Clearly, individuals consume hot tap water even though advised not to, and this risk is not quantified.

Sample Preservation. Existing analytical methods are based on the assumption that lead in water is dissolved, and that standard preservation of water samples at pH \( \leq 2.0 \) with addition of 0.15% 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 (EPA, 1994). Edwards and Dudi (2004) first showed that the standard EPA preservation protocol can sometimes “miss” much of the lead that is actually present in water. For instance, water samples actually containing 508 µg/L lead in Washington DC, only measured as 102 µg/L, using the standard preservation protocol (Edwards and Dudi, 2004). The reason for the discrepancy is that particulate lead can settle or adhere to the plastic sampling containers, and is "missed" when aliquots are taken for that measurement (Triantafyllidou et al., 2007).

**Inherent variability in lead release from plumbing cannot be captured by “single sample” WLL measurements.** Due to spatial and temporal variability in lead release from plumbing, especially in the case of particulate lead, surveys based on a single water sample may be inadequate to characterize exposure (Matthew et al., 1981, Pocock, 1980). Yet current monitoring programs under the LCR or the voluntary LCCA are based on a single water sample from each outlet, due to practical and financial constraints. Schock (1990) warned that if water monitoring programs do not account for this inherent variability, then the measurements will be unrepresentative and irreproducible.

Spatial Variability. Lead-in-tap water fluctuations are possible within a city (see Figure 1-3), a neighborhood, or a single home, even if water is collected under a standard protocol. For example, infrequent water consumption in municipal buildings or in schools, with periods of little or no usage during weekends and breaks, results in long stagnation periods of the water inside the piping and causes it to undergo chemical changes (Levin, 2008). This translates to more variability in the lead concentration, compared to homes where water consumption is much more frequent and regular. In addition, pH or other chemical fluctuations, depending on how far from the treatment
plant water is transported in order to reach consumer’s taps, also affects its corrosivity to leaded plumbing. Physical factors, such as the several interconnecting lines within a household plumbing system which route water to exterior faucets/bathrooms/kitchens/utility rooms, and the presence and type of leaded plumbing (e.g. leaded solder, leaded brass faucets, lead pipe) greatly affect lead levels at the tap (Schock 1990).

Temporal Variability. Fluctuations in lead levels from a single tap, depending on season or even on time of day, are possible. Seasonal fluctuations in temperature and chemical constituents, as well as seasonal variations in chlorination practice by the water utility may cause variable corrosivity of the water entering a household plumbing system (Schock 1990). In the course of one day, first-draw water, drawn from a tap in the morning after overnight stagnation, is considered worst-case in terms of lead release from the plumbing. Flushed water, or water collected after short holding times, tend to contain lower lead levels. Pocock (1980) argued that whatever type of water sample is collected, a single sample cannot provide a reliable estimate of the resident’s exposure to water lead. To illustrate, during an environmental assessment of a lead-poisoned child in Washington DC in 2004, the DC Department of Health (DOH) concluded that drinking water was not a potential hazard, based on collection of a single flushed water sample which measured lead at a reassuring concentration of 11 µg/L. Freedom of information act (FOIA) requests revealed that in four other flushed samples collected by the local water utility, lead in water ranged between 19-583 µg/L (Table 1-9). The samples collected by the utility provided strong indication that elevated lead in water was a potentially serious hazard, but the health agency sampling failed to make that connection based on their collection of a single flushed sample.

Table 1-9: Repeated flushed tap water sampling results from home of lead-poisoned child in Washington, DC. Data obtained through freedom of information act requests (Edwards, 2005).

<table>
<thead>
<tr>
<th>Date</th>
<th>Lead Determination (µg/L)</th>
<th>Sampling Conducted by</th>
</tr>
</thead>
<tbody>
<tr>
<td>7/26/2003</td>
<td>75</td>
<td>Water utility</td>
</tr>
<tr>
<td>3/23/2004</td>
<td>19</td>
<td>Water utility</td>
</tr>
<tr>
<td>3/23/2004</td>
<td>11</td>
<td>Department of Health</td>
</tr>
<tr>
<td>10/8/2004</td>
<td>21</td>
<td>Water utility</td>
</tr>
<tr>
<td>11/2/2004</td>
<td>583</td>
<td>Water utility</td>
</tr>
</tbody>
</table>

Individual water consumption patterns affect individual exposure

Variability in individual water consumption patterns. In over-simplified terms the individual risk from lead-contaminated drinking water, or any other hazard, is also a function of exposure to that hazard. Prior research has demonstrated a strong dependence between the quantity of tap water consumed and overall exposure. For example, Potula
et al. (1999) found that Bostonians who consumed medium or high levels of tap-water (≥ 1 glass/day) that contained greater than 50 µg/L of lead, developed progressively higher patella lead levels later in life, compared to those Bostonians with low levels of ingestion of the contaminated water (< 1 glass/day). Similarly, Galke et al. (2006) determined that the more glasses of tap water consumed, the higher the chance of an elevated blood lead level for children in Milwaukee and in New York. Consumption of two glasses of tap water per day corresponded to a high (50%) probability of having elevated blood lead (Galke et al., 2006).

Individual water consumption patterns may vary markedly between different age groups, and should be taken into consideration when assessing potential exposure. For instance, a Canadian survey on drinking water intake showed that infants less than one year consumed on average 122 mL/kg-day of water if they were formula-fed. This amount is about three times higher than the 44 mL/kg-day intake proposed by EPA (Levallois et al., 2008). These authors concluded that due to their high water intake on a body weight basis, formula-fed infants may be particularly susceptible to water contaminants (Levallois et al., 2008).

The use of tap, filtered tap or bottled water, also has an obvious impact. During the Washington DC lead-in-water crisis, BLLs were measured in residents of homes with water lead levels greater than 300 µg/L. All residents had BLLs lower than the CDC levels of concern (10 µg/dL for children and 25 µg/dL for adults), which was at first interpreted as indicating that the high lead in water was not harmful (Stokes et al., 2004). However, later analysis revealed that only a few individuals (and no children) had been consuming tap water for months prior to having their blood lead collected, and that virtually all were using lead filters and bottled water (Edwards et al., 2009; Edwards, 2010; CDC, 2010b). The key take away message from the "300 µg/L" study is that use of water filters, bottled water or even flushing, can be very effective at mitigating risk. Another study found that tap water can remain a significant lead exposure source through adolescence, with teens consuming bottled water having lower blood lead levels (BLLs) than those served by well or public water systems (Moralez et al., 2005).

Underestimated indirect contribution of water to the total dietary lead intake. The potential for massive accumulation of lead in food during cooking is not commonly realized. Use of relatively large quantities of water to boil vegetables, pasta, or other food, and effective concentration of the lead into food via adsorption has been demonstrated (Moore, 1983; Little et al., 1981, Baxter et al., 1992). Specifically, vegetables can absorb 90% or more of the lead from the water they are cooked in (Moore, 1983). Smart et al. (1981) showed that lead-in-water concentrations of 100 µg/L could contribute 74 µg/day of lead to the total dietary lead intake from vegetables and beverages, and at a total lead-in-water concentration of 500 µg/L the contribution was 378 µg/day. Green vegetables, carrots, rice and spaghetti concentrated more lead than many other foods (Smart et al., 1983). While humans generally absorb lead from drinking water more readily (30% to 50%) than lead from food (10% to 15%) (US EPA, 1986), the concentration effect can outweigh the reduced absorption factor. In addition to the report by Mesch et al (1996), in which an Australian family was poisoned by use of
lead-contaminated hot tap water to prepare instant coffee and cook meals, two cases of childhood lead poisoning occurred from contaminated water, even when the children did not directly consume the water. In both cases cooking of pasta, rice or potatoes was implicated as the source of the children's lead poisoning (Copeland, 2004; Triantafyllidou et al., 2007).

**Individual risk factors affect individual response to a fixed lead dose.** Variations in age, diet and genetics will produce a range of health effects in a population, in response to a fixed lead dose from water (or other sources).

**Age.** The gastro-intestinal absorption rate of ingested lead is inversely related to age. The typical lead absorption rate for infants is 50%, compared to just 10% in adults (WHO, 2000).

**Dietary habits.** Diets low in calcium and/or in iron, inadequate total calories and infrequent meals are believed to be associated with enhanced absorption of ingested lead (Shannon, 1996). In dietary experiments with twenty three adult volunteers, the lead retention from consumption of lead acetate was controlled by the type/timing of meals and beverages (James et al., 1985). Another study determined that subjects absorbed up to 50% of the lead on an empty stomach, 14% of the lead was absorbed when taken with tea or coffee, and 19% of the lead when taken with beer (Heard et al., 1983). Much lower uptakes (≥ 7%) were reported when lead was ingested in the course of a meal or with large amounts of calcium or phosphate (Heard et al., 1983).

**Genetics.** Genetic differences may result in different individual patterns of lead uptake and biokinetics (EPA, 2002). An increasing body of evidence suggests that tiny differences in the DNA sequence can modify the uptake, distribution, and elimination of lead by the body. For example, a 1991 study of lead workers in Germany and of environmentally exposed children in New York showed that small differences in two genes affected the absorption and excretion of lead by the participants (Wetmur et al., 1991). Another 2000 study that was performed in the Republic of Korea, with the participation of lead workers as well as persons without occupational lead exposure, reached similar conclusions (Schwartz et al., 2000).

**SUMMARY OF STUDIES ON THE ASSOCIATION BETWEEN LEAD IN WATER AND LEAD IN BLOOD**

The contribution of drinking water lead to the body’s lead burden (i.e., blood lead) is a subject of an extensive body of literature, which at first glance can appear contradictory. Marcus (1986) synthesized relevant studies as part of a broader evaluation of lead health effects from drinking water, and an update of that synthesis is undertaken herein. Various approaches have been used throughout the years in population studies, in an attempt to correlate water lead levels (WLLs) to blood lead levels (BLLs) (Table 1-10). These include, but are not limited to:
Focus on the most sensitive age groups (e.g. formula-fed infants, young children, or pregnant/breast-feeding women), versus lumping different age groups together
- Different types of tap water sampling to capture actual lead intake through water consumption, versus utilization of available water lead data from other sources
- Parametric correlations (assuming normal distribution of WLL and BLL) versus non-parametric correlations
- Linear regression models versus curve-linear models to fit the original WLL and BLL data, or regression after logarithmic transformation of the original data
- Exclusive focus on the contribution of WLL to BLL, versus contribution of other environmental lead sources (e.g. lead in paint, dust, soil) to BLL as well
- Association between WLL and BLL, versus association between WLL and % of study population with EBLL

Few studies are directly comparable, but nonetheless, critically evaluating the available literature provides useful insights.
### Table 1-10: Representative population studies on the association between lead in water and lead in blood (in chronological order).

<table>
<thead>
<tr>
<th>Sample Population</th>
<th>Independent Variable(s)</th>
<th>Dependent Variable</th>
<th>Measure of Association</th>
<th>Model</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Different sectors of Scottish population (n=949)</td>
<td>First-draw water lead (µg/L)</td>
<td>Blood Lead Level (µg/dL)</td>
<td>$R = 0.52$</td>
<td>BLL=$11.0+2.36(WLL)^{1/3}$ (units adjusted)</td>
<td>Moore et al., 1977</td>
</tr>
<tr>
<td>Individuals in greater Boston (n=524)</td>
<td>First-draw water lead (mg/L), other variables such as age, sex, education, dust lead</td>
<td>Blood Lead Level (µg/dL)</td>
<td>Model explains 19% of variance</td>
<td>Ln(BLL)=$2.73WLL-4.70WLL^2+2.17WLL^3$+other terms for age, sex, education, dust [WLL was best predictor]</td>
<td>Worth et al., 1981</td>
</tr>
<tr>
<td>Mothers in Ayr, Scotland (n=114)</td>
<td>Kettle water lead (µg/L)</td>
<td>Blood Lead Level (µg/dL)</td>
<td>$R^2=0.56$</td>
<td>BLL=$4.7+2.78(WLL)^{1/3}$</td>
<td>Sherlock et al., 1984</td>
</tr>
<tr>
<td>Mothers in Ayr, Scotland (n=114 from 1980-81, and n=116 from 1982-83)</td>
<td>Kettle water lead (µg/L)</td>
<td>Blood Lead Level (µg/dL)</td>
<td>$R^2=0.65$</td>
<td>BLL=$5.6+2.62(WLL)^{1/3}$</td>
<td>Sherlock et al., 1984, Moore et al., 1985</td>
</tr>
<tr>
<td>Women in Wales (n=192)</td>
<td>Kettle water lead (µg/L) Air Lead (µg/m³) Dust Lead (µg/g)</td>
<td>Blood Lead Level (µg/dL)</td>
<td>Model explains 38% of variance</td>
<td>Log(BLL)=$1.06+0.62(WLL)^{1/3}+0.18\log(ALL)-0.02\log(DLL)$</td>
<td>Elwood et al., 1984</td>
</tr>
<tr>
<td>Bottle-fed infants in Scotland (n=93)</td>
<td>Composite kettle water lead (µg/L)</td>
<td>Blood Lead Level (µg/dL)</td>
<td>$R = 0.57$</td>
<td>BLL=$14+0.062WLL$ BLL=$15.6+0.052WLL$ BLL=$14.7+0.054WLL$ BLL=$15.4+0.052WLL$</td>
<td>Lacey et al., 1985, WHO, 2000</td>
</tr>
<tr>
<td>Adults in Vosgian Mountains, France (n=155 men, n=166 women)</td>
<td>Tap water lead after 5 seconds of flushing (mg/L)</td>
<td>Blood Lead Level (µg/dL)</td>
<td>Spearman’s $\rho$ =0.30 for men =0.47 for women</td>
<td>Not determined</td>
<td>Bonnefoy, et al., 1985</td>
</tr>
<tr>
<td>Children in Edinburgh (n=397)</td>
<td>Tap water lead (µg/L), dust lead (µg/g)</td>
<td>Blood Lead Level (µg/dL)</td>
<td>Model explains 43% of variance</td>
<td>Log(BLL)=$0.5\log(5326+103WLL+3.81DLL)$ [WLL was best predictor]</td>
<td>Raab et al., 1987</td>
</tr>
<tr>
<td>Different sectors of population in Hawaii, with rain catchment systems (n=384)</td>
<td>Tap water lead (µg/L), other water-related terms, other terms for soil and demographics</td>
<td>Blood Lead Level (µg/dL)</td>
<td>linear model explains 77% of variance</td>
<td>Linear model: BLL=$5.62+0.025WLL+0.0008(GLASSES\cdot WLL)-0.017(FILTER\cdot WLL)+terms related to water, soil, age, sex, ethnicity etc.</td>
<td>Maes at al., 1991</td>
</tr>
<tr>
<td>Study Location</td>
<td>Water Lead Concentration</td>
<td>BLL</td>
<td>Equation/Details</td>
<td>Authors, Year</td>
<td></td>
</tr>
<tr>
<td>------------------------------------</td>
<td>--------------------------</td>
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<td>----------------------------------------------------------------------------------</td>
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<td></td>
</tr>
<tr>
<td>Citizens of Sante-Agathe-des Monts, Quebec, Canada (n=72)</td>
<td>Average water lead from 6 samples (mg/L) and estimated daily water consumption (L/day)</td>
<td>Blood lead level (µg/dL)</td>
<td>$R^2=0.25$</td>
<td>$\text{BLL} = 10 + 7 \times \text{WLL} \times \text{Water Consumption}$ (units adjusted)</td>
<td>Savard, 1992</td>
</tr>
<tr>
<td>School children in Southern Saxonia, Germany (n = 69 for location A, n=44 for location B)</td>
<td>Composite tap water lead (µg/L)</td>
<td>Blood Lead Level (µg/dL)</td>
<td>Location A: $R^2=0.34$ Location B: $R^2=0.41$</td>
<td>Location A: $\log(\text{BLL})=0.74+0.14\log(\text{WLL})$ Location B: $\log(\text{BLL})=0.81+0.14\log(\text{WLL})$</td>
<td>Englert et al., 1994</td>
</tr>
<tr>
<td>Mothers in Glasgow, Scotland (n=342)</td>
<td>Water lead (µg/L)</td>
<td>Blood Lead Level (µg/dL)</td>
<td>Spearman’s rho=0.39</td>
<td>Not determined</td>
<td>Watt et al., 1996</td>
</tr>
<tr>
<td>Women in Hamburg, Germany (n=142 for subsample with detectable water lead)</td>
<td>Average water lead (µg/L) from 3 specimens</td>
<td>Blood lead level (µg/dL)</td>
<td>Spearman’s rho=0.43</td>
<td>Not Determined</td>
<td>Fertmann et al., 2004</td>
</tr>
<tr>
<td>Children in Washington DC (n=2698 in “High Risk” n=4791 in “Moderate Risk” n=2621 in “Low Risk”)</td>
<td>90th Percentile Water Lead (µg/L)</td>
<td>% Increase in Children with EBLL Compared to US average</td>
<td>$R^2 = 0.83$ in “High Risk” $R^2 = 0.71$ in “Moderate Risk” $R^2 =0.50$ in “Low Risk”</td>
<td>Not Determined</td>
<td>Edwards et al., 2009</td>
</tr>
</tbody>
</table>

WLL: Water Lead Level; BLL: Blood Lead Level; EBLL: Elevated BLL; DLL: Dust Lead Level; ALL: Air Lead Level
Studies that found an association between WLL and BLL

**Association between WLL and BLL in formula-fed infants.** For infants and young children up to 5 months of age, milk, formula, and drinking water are considered highly significant sources of exposure to lead (WHO, 2000). In fact, for bottle-fed infants using reconstituted formula with tap water, about 90% of their diet by weight is actually tap water, since formula is typically prepared by adding 8 parts of water to 1 part of powder (Sherlock and Quinn, 1985). Additionally taking into account that the typical lead absorption rate for infants is 50%, compared to just 10% in adults (WHO, 2000), elevated lead in water is a very significant concern for this population group. Infants typically consume 500-1000 mL of formula per day (WHO, 2000). If the water used to reconstitute formula contains 90 µg/L of lead, an infant receiving 750 mL of such formula daily would ingest 61 µg Pb/day, based on the illustrative calculation:

\[
\frac{90 \mu g Pb}{L \text{ water}} \cdot \frac{0.75 \text{ L formula}}{\text{day}} \cdot 90\% \text{ water in formula} = 61 \mu g \text{ Pb/day} \quad \text{Equation (1)}
\]

In a study by Ryu et al (1983), conducted when infant formula commonly had elevated lead derived from solder, infants consuming daily formula with 61 µg Pb from 3.7-6.5 months of age, had elevated blood lead levels by 5.6 months of age (Figure 1-5). Another group of infants, exposed to only 16 µg Pb/day through their diet did not develop elevated blood lead (Figure 1-5). On this basis Rye et al. (1983) concluded that a lead intake of 16 µg/day, or else 3-4 µg/kg/day, is not associated with elevations in blood lead level above 10 µg/dL. This roughly corresponds to the provisional tolerable weekly intake (PTWI) of 25 µg/kg/week (or else 3.5 µg/kg/day) set by the World Health Organization (WHO, 2000). The Ryu et al. (1983) study is unique, because it provides unambiguous results for infants whose dietary lead intake was completely controlled. Due to obvious modern ethical concerns, similar experimental studies with infants are unlikely to be repeated.

![Figure 1-5: Average blood lead level (BLL) versus age for two groups of formula-fed infants, at two levels of dietary lead intake. Adapted from data in Ryu et al. (1983).](image-url)
Later studies also derived strong associations between Glasgow infants’ dietary lead (mainly consisting of drinking water) and blood lead (Lacey et al., 1985). For 13 week-old infants, a duplicate of their formula was collected for a week so that their total lead intake could be unambiguously quantified. A simple linear relationship between lead in water collected from kettles and infant blood lead level was derived, with a correlation coefficient of $R^2 = 0.32$ (Table 1-10). This work demonstrates that due to genetic and other factors mentioned earlier, perfect correlations are not to be expected between lead in water and lead in blood, even for the most susceptible sub-population to lead exposure from water.

An investigation by Shannon and co-authors revealed nine cases where lead poisoning occurred in Boston infants, after consuming instant formula reconstituted with lead-contaminated water (Shannon et al., 1992). In one such case, the formula was prepared each morning with first-draw water from the kitchen tap, which contained 130 µg/L lead attributable to lead solder (Shannon et al., 1989). Other cases of elevated blood lead from consumption of formula, with no other source of lead in the child's environment, have been reported (Cosgrove et al., 1989; Lockitch et al., 1991).

**Association between WLL and BLL in young children and adults before implementation of modern corrosion control.** The first survey to show a curve-linear relationship between water lead and blood lead was that of Moore et al. (1977), which yielded a correlation coefficient of $R=0.52$ by analyzing data from different sectors of the Scottish population (Table 1-10). 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, 18% of people with first-flush water lead ≥298 µg/L had BLLs ≥ 41 µg/dL, compared to only 0.3% of those with water lead < 50 µg/L.

Sherlock et al (1984), who analyzed lead in water and lead in blood of mothers in Ayr, Scotland, reinforced Moore’s notion of a curve-linear relationship (Table 1-10). Initially, lead in water and in blood were measured for 114 mothers during 1980-1981, when the Ayr water supply was very corrosive and lead pipes were predominant. That analysis yielded a correlation coefficient of $R^2=0.56$ between kettle water lead and blood lead level (Table 1-10). After changes in water treatment were implemented by increasing the pH from 5.0 to 8.5, and after some of the lead pipes had been removed, the same analysis was repeated during 1982-1983. The sample of women in the subsequent analysis included many of the same women as the 1980-1981 analysis (Sherlock et al., 1984). Combination of both data sets yielded a correlation coefficient of $R^2=0.65$ between kettle water lead and blood lead (Table 1-10). After increasing the pH of the water supply, water lead levels significantly dropped, and median blood lead levels also dropped from 21 µg/dL to 13 µg/dL.

A study of 321 adults in an area of France with relatively corrosive water and high incidence of lead pipe (Bonnefoy et al., 1985) revealed that the concentration of lead in tap water was significantly correlated to the residents’ BLL (Table 1-10). For water lead levels up to 20 µg/L, the BLLs of both men and women remained relatively constant, but if lead in water exceeded 20 µg/L BLLs increased substantially. Elwood et al. (1984) assessed the relative contributions of water lead, dust lead and air lead to blood lead of 192 women in various areas of Wales. The regression model indicated that even in areas with relatively low water lead levels for that time
period, water was an important source of blood lead. An increase of lead in water from 0 to 60 µg/L, resulted in an increase of 5.5 µg/dL in blood lead level (Elwood et al., 1984).

Raab et al. (1987) assessed the relative contributions of water lead and dust lead to blood lead of 6-9 year-old children in a part of Edinburgh with a high incidence of lead pipes and corrosive water supply. Their resulting model, accounting for exposure to water and dust, explained 43% of the variation in blood lead levels (Table 1-10). Coefficients for both water and dust were significant in their model (Table 1-10), and the authors concluded that water lead was more important than dust in this population. An eight year follow-up study of the same individuals in central Edinburgh, showed a dramatic decrease in both their water lead and blood lead levels, which was attributed to improved corrosion control and removal of lead pipes from plumbing (Macyntire et al., 1998).

Maes et al. (1991) assessed the contribution of lead from drinking water, dust, soil, and paint to BLLs of 384 individuals of various ages in Hawaii. This study relied on measurements from exterior house faucets previously conducted on behalf of the Department of Health. Lead in paint, dust and soil was measured, and information on water consumption patterns and demographics was obtained through questionnaire responses of the participants. Because this population was exposed to relatively high levels of lead from water and low levels of lead from soil, dust, and paint, the authors found a stronger rank-based correlation of BLLs with WLLs (r = 0.53), compared to other environmental sources (r = 0.35 for soil, 0.30 for dust, and 0.14 for interior paint) (Maes et al., 1991). Blood samples in this study were collected more than 2 months after residents had been informed to avoid tap water, unless it tested below 20 µg/L, and virtually no vulnerable young children (< 1 year of age) were tested. Even though the work of Maes et al. (1991) was never published, it was submitted to EPA to influence formulation of the 1991 US EPA LCR, which in turn introduced modern corrosion control strategies for lead in US drinking water.

Association between WLL and BLL in young children and adults after implementation of modern corrosion control. More recent studies, conducted after the phase-out of lead in gasoline and other lead reduction strategies, and with much lower water lead levels due to modern corrosion control, still indicate strong relationships between lead in blood and lead in water. An epidemiological study in Hamburg, Germany (Fertmann et al., 2004) 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) (Table 1-10). For those women who were exposed to water lead >10 µg/L, an intervention program was tested, which either involved eliminating tap water lead exposure (by consuming bottled water) or minimizing exposure (by flushing water prior to consumption). Overall, after about 10 weeks of intervention, the median blood lead level decreased by 1.1 µg/dL (p ≤0.001). Individuals flushing the water lowered their blood level by 21% whereas those drinking bottled water reduced their blood lead level by 37% (Fertmann et al., 2004). The authors concluded that “lead in tap water stands for an avoidable surplus exposure” (Fertmann et al., 2004).

In another German study conducted in Southern Saxonia, lead in blood and lead in tap water were measured for school children from two locations, A and B (Englert et al., 1994). Lead pipes were used in about 50% of their houses. After log-transformation of both their blood lead

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levels and their drinking water lead levels, 34% of the variation in blood lead levels was explained by logWLL in location A (i.e. $R^2=0.34$ for location A), and 41% of the variation was explained in location B (i.e. $R^2=0.41$ for location B) (Table 1-10). These authors concluded that in this part of Germany, lead exposure through drinking water was a greater concern than lead paint and other sources, due to the lead pipes in the water supply which had not yet been removed. Seven years later, after many lead pipes had been replaced with alternative materials, another study quantified WLLs in homes of new-born babies in various regions of Southern Saxonia (Zietz, 2001). Overall, 3.1% of the 1434 stagnation samples had lead higher than 10 µg/L. But certain geographic regions were at higher risk (>5% above 10 µg/L), and these authors concluded that the exceptional cases were due to leaching of domestic plumbing and fittings containing lead (Zietz, 2001).

Following a case of lead intoxication by drinking water in Ste-Agathe-des Monts, a Canadian study demonstrated a link between EBLLs and WLLs, as well as presence of lead service lines (Savard, 1992). Canada did not provide guidance for national corrosion control programs until 2009 (Health Canada, 2009) and this town still distributed corrosive water. On the basis of field investigations and 383 blood lead analyses, BLLs higher than 20 µg/dL were associated with the presence of lead service lines (Yates’ Chi-square test: 5.85; p=0.02) (Savard, 1992). A mathematical model was developed for the 72 citizens for which WLLs were measured (Table 1-10). Lead concentrations in those samples were as high as 4200 µg/L. Water consumption was obtained on the basis of a questionnaire. Using a linear regression between BLL and the estimated lead daily intake, a correlation coefficient of $R^2=0.25$ was obtained (Savard, 1992). The water corrosivity was rapidly identified as the problem (pH as low as 4.8 measured in some houses) and corrective measures were taken by increasing the pH to 8.4. After less than a month, WLLs were reduced by more than 90%, and the measured BLLs were significantly reduced by 24% in less than a year. Work with lead paint or dust mitigation has also demonstrated that, in some cases, mitigation of the suspected lead hazard only slightly reduces blood lead, if high levels of lead have been stored in bone (Rust et al., 1999; Gwiazda et al., 2005).

Watt et al. (1996) assessed the relationship between tap water lead and maternal blood lead concentrations in Glasgow, after the water supply was subjected to maximal water treatment to reduce plumbosolvency. Tap water lead remained the main correlate of raised maternal blood lead concentrations, accounting for 76% of cases of maternal blood lead concentrations above 10 µg/dL. The authors concluded that although tap water lead and maternal blood lead concentrations had fallen substantially since the early 1980s, tap water lead was still a public health problem in that area, especially for the estimated 13% of infants who were exposed via bottle feeds to tap water lead concentrations exceeding the WHO guideline of 10 µg/L.

Lanphear et al. (2002) assessed the contribution of lead in water versus other sources to children’s blood lead levels during early childhood. Children from 6 until 24 months of age were monitored in Rochester New York, a community not considered to have lead-in-water problems according to the EPA LCR. Samples of tap water, house dust, soil and paint were quantified for lead, with house dust being determined as the main source of lead exposure. Even so, water lead concentration was also directly associated with blood lead levels (p<0.001). Children who lived in housing with water lead concentration greater than 5 µg/L had slightly higher (1.0 µg/dL)
blood lead levels than children who had home water lead levels below 5 µg/L (Lanphear et al. 2002).

Taking into account geographic risk factors during an incident of sub-optimal corrosion control, Edwards et al. (2009) found a strong correlation between the frequency of EBL and the 90th percentile lead in water concentration from 2000-2007 in Washington DC. In neighborhoods determined to have the greatest frequency of lead pipe and highest lead concentrations, a correlation was found for children less than 30 months of age (Table 1-10). Older children, children living in neighborhoods with relatively few lead pipes or measurements of elevated lead in water, showed lesser impacts. But the youngest children (< 1.3 years) showed very strong correlations between the incidence of EBL and the reported 90thile lead in water concentration. Earlier studies on Washington DC (Stokes et al., 2004; Guidotti et al., 2007) did not focus on the youngest children or geographical factors, and saw little or no increased incidence of EBL during the time of high lead in water.

**Studies that did not find an association between WLLs and BLLs**

Many other studies have found little or no relationship between lead in blood and lead in water. These studies are occasionally cited as if results are contradictory to those highlighted in the preceding section. That work is critically reviewed herein, in an attempt to reconcile results that are superficially in conflict, but which are consistent with bio-kinetic understanding of relationships between lead in water exposure and lead in blood.

**Lack of Association between WLL and BLL When Lead in Water was reportedly Low.**

There are many areas in the US (and other countries) in which water lead concentrations are very low. This can occur in situations with modern plumbing which has no lead pipe, lead solder or leaded brass, and with optimized corrosion control which can dramatically reduce lead leaching. Some older cities with high incidence of lead pipe and lead solder, have pipes that are virtually completely lined by scale such as calcium carbonate, which effectively eliminates contact between the lead-bearing plumbing and the water. In such circumstances lead in water will not be a dominant, or even a significant contributor, to overall lead exposure.

For instance, in a study by Lubin et al. (1984), where water samples were collected in the homes of 50 children with BLL > 30 µg/dL in Columbus, Ohio, lead in water was always low (< 10 µg/L). It is believed that the water supply in that study was atypically non-corrosive (high pH of 9.6 and high hardness of 101 mg/L). Not surprisingly, there was no correlation between lead in water and lead in blood, even in the presence of lead pipes at the children’s homes. Likewise, a study in Germany (Meyer et al., 1998) in a town where lead in tap water was extremely low (< 1 µg/L) found no significant association between lead in domestic water and in blood for children. Another study of children’s BLL in Miami Inner City, Florida (Gasana et al., 2006), also found no association of BLLs to WLLs (Spearman’s rho = 0.03 for flushed water samples and 0.005 for first draw water samples). Water lead measured in 120 homes was reportedly low (< 15 µg/L), with the exception of 3 homes. However, correlations between BLL and floor dust (rho = 0.27) and window sill (rho = 0.28) were statistically significant (p < 0.05) (Gasana et al., 2006).
Another important study by Rabinowitz et al. (1985) examined the association of BLLs of infants in Boston with lead in dust, soil, indoor air, paint and tap water. The authors found 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 important contributions of dust, soil and paint to BLL are consistent with expectations. However, analytical limitations in quantification may have masked any potential contribution of WLL to BLL, if it were present. Specifically, lead in water was quantified using anodic stripping voltametry. This analytical technique has recently been shown to accurately measure dissolved Pb\(^{2+}\), but to not measure particulate lead or Pb\(^{4+}\) levels in water (Cartier et al., 2009). The latter species have recently proved to be present in drinking water under at least some circumstances (Triantafyllidou et al., 2007), but were not understood at the time of the Rabinowitz study. Moreover, samples were allowed to sit unacidified before analysis, which is now recognized to potentially miss some of the lead present in water (Rabinowitz, 2006). Perhaps, partly because of these issues, only very low levels of lead (3.7 – 7.3 \(\mu g/L\)) were reported for Boston drinking water samples (Rabinowitz et al., 1985).

To provide a historical perspective for Boston, Potula et al. (1999) found lead in water of Boston homes as high as 169 \(\mu g/L\) during the same time period. Boston water, which was linked to lead poisoning via infant formula was reported by Shannon et al. (1982) to contain 132 \(\mu g/L\). Even as late as 1996-2000, lead levels in first-draw tap water samples from Boston were 159 \(\mu g/L\) on average, and as high as 311 \(\mu g/L\) in the worst case for children with elevated blood lead (State of Massachusetts, 2009). Even flushed water samples for lead poisoned children in the 2009 data from Massachusetts contained as high as 146 \(\mu g/L\) lead.

### Lack of Association between WLL and BLL When Lead in Water was reportedly High.

Some studies have found no association between elevated lead in water and elevated lead in blood. Key aspects of such studies are critically reviewed herein, especially as they relate to potential limitations described in preceding sections (see Table 1-10). For example, Costa et al. (1997) reported that very high water lead levels in a public school in rural Utah (up to 840 \(\mu g/L\)) did not cause EBLL. In that study, measurements of blood lead were undertaken for only 40% of students, more than 16 days after notification of the problem and advice to drink bottled water, during which time lead in blood could drop, considering its half life of around one month (WHO, 2000). Even though one case of elevated blood lead was identified, it was dismissed as unrelated to water lead (Costa et al. 1997).

A CDC study reported that in 201 cases where home tap water contained more than 300 \(\mu g/L\) of lead in Washington DC, none of the individuals were found to suffer from EBLL (Stokes et al., 2004). Another study on the same topic cited the same data, and did not find an association between elevated lead in water and lead in blood, concluding that “there appears to have been no identifiable public health impact from the elevation of lead in drinking water in Washington DC, in 2003 and 2004” (Guidotti et al., 2007). Neither study focused on infants, who are most vulnerable to harm from lead in water. In addition, both studies lumped all the blood lead data for Washington, DC together, an approach which masked disparities among different neighborhoods (Edwards et al., 2009). Finally, as mentioned earlier, virtually no residents had
been consuming tap water for months prior to having their blood lead drawn, rendering the data useless for assessing impacts of lead in water on lead in blood (Edwards et. al., 2009; CDC, 2010b). The “no-harm” conclusion of Guidotti et al. (2007) has since been removed (Errata in Environmental Health Perspectives, 2009).

**Studies that did not measure lead in water at homes**

Some researchers attempted to assess the contribution of lead in water to lead in blood, without measuring lead in tap water at homes. For example, studies occasionally relied on qualitative data obtained from questionnaires regarding consumers’ water consumption habits (tap water versus filtered or bottled water), and/or knowledge regarding the presence of lead pipes in consumers’ home plumbing. Other studies relied on lead-in-water measurements obtained from the distribution system and not home taps, which can result in overlooking tap water as a potentially important source.

For example, a broad Cincinnati study aimed to investigate different lead sources and factors which result in excessive intake for children in urban settings (Bornschein et al., 1985; Clark et al., 1985). Blood lead levels were systematically monitored from birth through 5 years of age and 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, sampling data collected by the water utility from the distribution system, before the water even enters the service line where lead hazards are introduced (see Figure 1-1), were cited as having lead concentrations < 6 µg/L (Clark et al., 1985). Exposure from water was thus deemed to be insignificant when in fact, samples were never collected in a manner that would allow risks to be quantified if they were present. Historical data from Greater Cincinnati Water Works suggest that even in recent years, with modern corrosion control, some Cincinnati schools had tap water lead levels above 15 µg/L, while some homes tested at 180 µg/L after partial lead pipe replacements (DeMarco, 2004).

A study in Northern France (Leroyer et al., 2000) showed that BLLs doubled for children who reported consuming tap water in homes with lead plumbing identified under the kitchen sink. In cases where lead pipes were not visible under the kitchen sink, children drinking tap water still had significantly higher BLLs compared to those consuming bottled water (Leroyer et al., 2000). The authors (Leroyer et al., 2000) qualified their conclusions by suggesting that water sampling should be conducted to more carefully assess their findings, which relied on visual identification of lead plumbing and qualitative answers to a questionnaire.

**SYNTHESIS OF STUDIES ON THE ASSOCIATION BETWEEN LEAD IN WATER AND IN BLOOD**

Rigorous scientific studies prior to implementation of modern corrosion control provided strong links between elevated lead in water and elevated blood lead (i.e., greater than 10 µg/dL) of exposed populations. As would be expected based on current understanding of dietary intake
and hand-mouth behavior relative to significance of lead sources, impacts of elevated lead in water on lead in blood become more significant the younger the child, with especially high risks for children consuming reconstituted infant formula. The work of Lacey et al. (1985) and Ryu et al. (1983) exemplify rigorously controlled studies that are unlikely to be improved upon in the near future, and which served as the basis for the US EPA LCR and models predicting BLL developed by the US EPA.

Two landmark multimedia US studies (Bornschein et al., 1985 and Rabinowitz et al., 1985), did not find any association between lead in water and in blood for children in Cincinnati and Boston. The strong relationships established in that research between lead in paint, dust and soil and children's blood are not disputed, but each study had limitations or gaps in quantifying lead-in-water risks.

More recent studies in Canada, Germany, the UK and the US, sometimes found strong associations between WLLs and BLLs, and sometimes not. These studies reflect marked differences in the extent of lead-in-water exposure based on plumbing materials, corrosivity of the water, and other nuances of exposure. Some recent work by the CDC and others that concluded very high lead in water (> 300 µg/L ) did not impact incidence of EBL in an exposed population has been re-analyzed, corrected or clarified (Edwards et al., 2009; Errata in Environmental Health Perspectives, 2009, CDC, 2010b; Edwards, 2010; US Congressional report, 2010). That work is no longer inconsistent with decades of prior research. Other work has demonstrated strong links between lead in water and lead in blood even at much lower levels of lead in water exposure, in systems conducting "optimized corrosion control" or its equivalent (Lanphear et al., 2002; Englert et al., 1994; Fertmann et al., 2004).

SUMMARY AND CONCLUSIONS

As efforts shift from addressing pervasive lead sources that elevate the blood lead of large percentages of the population, to more isolated individual cases requiring exceptional attention, it will be necessary to more carefully consider lead in water as a potential source.

Although routine blood lead monitoring and environmental assessments are not designed to detect lead-in-water hazards when present, several recent cases of elevated blood lead in the US and other countries have been attributed to lead-contaminated drinking water. Existing US regulations/guidelines have not eliminated lead in water hazards in systems served by public water supplies, schools, daycares and privately owned homes.

Lead in drinking water originates from lead-bearing plumbing materials, which undergo corrosion reactions, and may severely contaminate the water supply. Contrary to popular belief that lead in water problems invariably decrease as water systems age and rust/scale develops on pipes, problems with sporadic detachment of rust/scale on lead-bearing plumbing might create acute human health risks that are hard to detect and link to elevations of lead in blood. Up to 81 million US homes are estimated to be at potential risk due to the presence of lead pipe and lead solder, and even new homes can occasionally experience high lead from brass/bronze plumbing. The occurrence of particulate lead in US drinking water has not been adequately examined, but
case studies suggest that the highest doses of lead are associated with the presence of particulate (and not dissolved) lead in tap water.

When water lead measurements are not available at “high risk” taps, or when they fail to quantify the actual lead content of drinking water, correlations of water lead with health risks may be missed. A strong association between lead in water and lead in blood has been documented through decades of prior scientific research. Epidemiological studies in the US, the UK, Germany, France, and Canada indicate that elevated lead in water can occasionally be the dominant, or a major contributor, to elevated blood lead. Re-evaluation of the public health risk from lead in water, with emphasis on particulate lead and sensitive sub-populations, is timely considering forthcoming revisions to the LCR and acknowledged deficiencies in addressing lead in school drinking water.

**RESEARCH NEEDS**

This literature review highlighted the need for additional research on lead occurrence in tap water and associated public health risks. Specifically, the occurrence of lead in drinking water at US schools needs to be systematically monitored, using sampling protocols that will allow identification of the source(s) of potential problems and development of concise remedial actions. Detailed case studies on lead-in-school water could then be synthesized, and serve as a guide for schools that encounter similar problems in the future.

The effects of sampling protocol (e.g., flow rate, cold versus hot water) and sample handling (e.g. sample preservation and holding time) on lead detection need to be evaluated for all situations including schools, homes and other buildings. Subtle differences in sample collection procedures can either detect or miss lead spikes, especially when problems with particulate lead in water are important. The occurrence of particulate lead spikes in US drinking water needs to be better characterized, because it may result in intermittent exposures of acute health concern, which can easily go undetected. Acute health effects from lead in water, concentration of lead in food, and potential exposure to elevated lead from hot water deserve explicit consideration.

Old lead service lines are a major contributor to lead levels at the tap, when they are present. Partial replacements of lead service lines in response to provisions of the LCR, as a means of reducing lead-in-water exposure, require re-evaluation in light of preliminary data showing short- and long-term problems with lead spikes and increased risks of elevated blood lead in children. Laboratory studies quantifying the long-term impacts in a range of waters, as well as the cost: benefit of the procedure, are necessary. Likewise, evaluation of impacts from newly installed leaded-brass plumbing devices is also needed.

Past approaches in modeling health impacts from elevated lead in water, based on prediction of the geometric mean BLL, was useful when considering impacts on populations. But as society shifts its concern to tracking and addressing individual cases of childhood lead poisoning, modeling approaches need to consider and predict impacts on susceptible individuals exposed to the highest sampled lead-in-water concentrations.
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REFERENCES


Cooper, M. Parent who discovered high lead in water at Seattle school. Personal communication on 7/13, 2008.


DC WASA (District of Columbia Water and Sewer Authority). Response to questions from Carol Leonnig of the Washington Post, in her e-mail dated 3/17/08.


Elfland, C., Scardina, P., and M. Edwards. Lead Contaminated Water from Brass Plumbing Devices in New Buildings . Accepted for publication in JAWWA, 2010


Gardner, C. Compliance Section Manager, Maine Drinking Water Program, Personal Communication on 12/27/2006.


Lambrinidou, Y. Personal communication on 12/10, 2008.


Chapter 1. Lead (Pb) in Tap Water and in Blood: Implications for Lead Exposure in the US


National Geographic Magazine. Lead Helps to Guard Your Health (advertisement of the National Lead Company), Volume 44, 1923.


Patch, S. Personal communication with Marc Edwards on 11/28, 2006.

Parks, J., and Edwards, M. Report to the City of Raleigh (NC) Lions Recreation Center, 2008.


Rabinowitz, M., Personal communication on 12/10/2006.


Robertson, E. Pitt County Health Department. Personal Communication March 24, 2006.

Chapter 1. Lead (Pb) in Tap Water and in Blood: Implications for Lead Exposure in the US


US EPA. Air quality criteria for lead, Volume I, 2006a. EPA/600/R-5/144A.


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CHAPTER 2. ELEVATED BLOOD LEAD IN YOUNG CHILDREN DUE TO LEAD-
CONTAMINATED DRINKING WATER: WASHINGTON DC, 2001-2004

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ABSTRACT

Incidence of EBL (blood lead ≥ 10 ug/dL) for children aged ≤ 1.3 years 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 levels from 2000-2007 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 2000-2007 (R^2 = 0.83) in the high risk neighborhoods. These effects are consistent with predictions based on bio-kinetic models and prior research.

Brief. Exposure to lead-contaminated drinking water in Washington, DC from 2001-2003 markedly increased the incidence of elevated blood lead for very young children.
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 US 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 ug/dL (3), no evidence was found of increased incidence over the 10 ug/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 using EPA approved methodology by the water utility. A “first draw” sample refers to a 1-liter sample collected from a tap after greater than 6 hours holding time in the household plumbing. After first draw samples are collected, water is flushed for a short time period (typically 30 seconds to 5 minutes) and a 1-liter “second draw” sample is collected.

Two datasets 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 re-organized 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 Appendix A, 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 (Appendix A, 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 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) bio-kinetic model, to more precisely identify the population(s) most sensitive to lead in water. The ICRP bio-kinetic 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 (Appendix A, 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 dataset (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 $\leq 1.3$ years are examined. Thereafter, a neighborhood analysis is presented for children aged $\leq 2.5$ years.

Temporal Trends in WLLs in Washington, DC.

The 90th percentile WLLs (Figure 2-1) 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-December are termed 2001b). Other support for dividing 2001 in half is presented in Appendix A-Supporting Information 2. The 90th percentile WLLs remained higher than the EPA regulatory “action level” of 15 ppb from 2001-2004 (Figure 2-1) 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 onwards) to mitigate high WLLs (Figure 2-1).

Following a January 31, 2004 front page Washington Post article that revealed the widespread problem with elevated WLLs, the public was eventually instructed to flush their water lines $> 10$ minutes 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 Aged ≤ 1.3 Years.

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 2-1). 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 Appendix A-Supporting Information 1, especially considering that 90th percentile WLLs were higher in late 2001 than in 2003 (rough predictions in Appendix A, Table A-1 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 2000-2007 (See Appendix A, Supporting Information 5, Figure A-7) is very strong (R² = 0.81).

The CDC database (3) was analyzed for the same trends. The incidence of EBL for children aged 1-16 months showed similar trends to the CNMC data (lower Figure 2-1). The absolute values of the CNMC data and the CDC data 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 there were only 90 children identified as age 1-16 months in data 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 was 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 phenomenon” was identified that played a key role in the observed variability of water lead in homes throughout the city (Appendix A, 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 Appendix A, 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 2-1). 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 (Table 2-1), and 2.7 times higher than in moderate risk part of the city. 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 2-1).

### Table 2-1: Summary data for neighborhoods of high, moderate and low relative risk of exposure to high elevated WLLs.

<table>
<thead>
<tr>
<th>Relative Exposure Risk</th>
<th>Est. Lead Pipes</th>
<th>% of total pop. in city (1000)</th>
<th>% pop with lead pipe*</th>
<th>% 1st draw over 100 ppb</th>
<th>% pop above indicated WLL (ppb)</th>
<th>1st draw &gt; 100</th>
<th>2nd draw &gt; 100</th>
<th>1st draw &gt; 400</th>
</tr>
</thead>
<tbody>
<tr>
<td>High</td>
<td>10086</td>
<td>22</td>
<td>126.3</td>
<td>17.6</td>
<td>15.0</td>
<td>2.63</td>
<td>3.43</td>
<td>0.13</td>
</tr>
<tr>
<td>Moderate</td>
<td>14743</td>
<td>55</td>
<td>314.3</td>
<td>10.3</td>
<td>9.4</td>
<td>0.97</td>
<td>1.59</td>
<td>0.02</td>
</tr>
<tr>
<td>Low</td>
<td>1318</td>
<td>23</td>
<td>131.4</td>
<td>2.2</td>
<td>12.8</td>
<td>0.28</td>
<td>0.37</td>
<td>0.00</td>
</tr>
</tbody>
</table>

**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 2-2). 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 2000-2007.

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 ≤ 30 months of age in the neighborhoods with high WLLs.

**Comparison of EBL in Washington, DC to the US Trend in BLLs, 2000-2007.** National trends in EBL incidence from 2000-2006 (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 2-2), is the difference between the US 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.
Figure 2-2: 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 (above). Correlation between increased incidence of elevated blood lead in Washington DC children aged ≤ 30 months and 90th percentile lead (below).

**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 2-2). 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 2-1). The slope of the trend-line in the highest risk part of the city is approximately double that observed 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 (Appendix A, Supporting Information 5). The 2004 data also deviate significantly from the trendline (lower Figure 2-2), in that the high WLLs did not increase the percentage of children aged \( \leq 30 \) months with EBL to the same extent as it 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 (Appendix A, 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 2-2 and the population of children aged \( \leq 30 \) 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 \( \leq 30 \) months had EBL in 2003 due to high WLLs, and that 517 additional children aged \( \leq 30 \) 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 \( \leq 30 \) months) is for 170 cases in 2003 (Appendix A, Supporting Information 1). The discrepancy (342 estimated cases of EBL using the CNMC analysis vs. 170 predicted for children aged \( \leq 30 \) 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 ug/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 2-1, Figure 2-2, Appendix A, Supporting Information Figure A-13 and A-14), 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 Appendix A, Supporting Information 2 Figure A-6). 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 to 24 months (Appendix A, 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 data 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 ≤ 1 year of age. The data presented herein for children ≤ 1.3 years of age (which is 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 ≥ 30 months of age are likely to have EBL during 2000-2003 because of exposure to high WLLs (Appendix A, 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 ug/dL declined by 70% from 2000-2003 across the US, 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 ug/dL across the city was also reduced during the time period that WLLs were high. For example, the CDC study reported that from 2000-2003, the incidence of BLL measurements > 10 ug/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 2-1; Appendix A, 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 of 2001 (Appendix A, Supporting Information Figure A-4 and A-14).

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 2nd 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 Appendix A, Supporting Information 7]
2) WLLs showed an “abrupt rise” in 2003 [the WLLs had risen by the second half of 2001 as per Figure 2-1],
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 Appendix A, Supporting Information 7].

This may explain why 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(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 US 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 WLL risks are rarely the cause of EBL in children should be re-evaluated.

ACKNOWLEDGEMENTS

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Supporting Information Available
Seven supporting analyses and three reports (EPA, 2004) are available in Appendix A.

REFERENCES

CHAPTER 3. PREDICTIVE MODELING OF CHILDREN’S BLOOD LEAD LEVELS FROM EXPOSURE TO LEAD IN TAP WATER

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ABSTRACT

Prediction of children’s blood lead levels (BLLs) through biokinetic models often concludes that lead in tap water is not a primary health risk for the “average” child under “typical” scenarios of “chronic” exposure, when applying a “10 µg/dL” BLL threshold. Re-examining the use of IEUBK and ICRP biokinetic models to simulate exposure to water lead at home and at school, the scope of prior modeling efforts was expanded to consider new public health goals. Specifically, explicit consideration of the most sensitive population groups (e.g., young children and particularly formula-fed infants), the variability in BLLs amongst exposed individuals within those groups (e.g., most sensitive children at the upper tail of the BLL distribution), more conservative BLL thresholds (e.g., 5, 2 and 1 µg/dL versus 10 µg/dL) and concerns of acute exposure revealed situations where elevated lead from water could pose a significant human health concern. This paradigm shift from traditional modeling approaches supports the US goal of eliminating all instances of elevated blood lead in children by 2020 and is consistent with increasing concern about low-level lead exposure.

KEYWORDS: children, reconstituted formula, water lead level, blood lead level, model, distribution, acute
INTRODUCTION

Lead in tap water and lead in blood. Lead-containing plumbing materials (lead pipe, lead solder, brass and bronze plumbing components) may contaminate drinking water at the tap, for which an “action level” of 15 µg/L has been set in the United States (US EPA, 1991). In the typical case drinking water consumption is believed to account for up to 20% of total lead exposure nationally (US EPA, 2006), but a recent literature review highlighted several cases where contaminated tap water was the dominant contributor to elevated blood lead (EBL) in children (Triantafyllidou and Edwards, 2011). The US Centers for Disease Control and Prevention (CDC) consider 10 µg/dL as the blood lead level (BLL) “of concern” in children, elevations above which cause detectable mental impairment and behavioral changes, although no levels of blood lead are deemed "safe" (US CDC, 2005).

To explore relationships between environmental lead and blood lead of exposed children, biokinetic models are frequently used for supporting risk assessment decisions (Pounds and Leggett, 1998). Three models have been commonly used in prior research to predict BLLs from exposure to lead in water and other environmental media: the US EPA Integrated Exposure Uptake Biokinetic (IEUBK) model for lead in children, the International Commission on Radiological Protection (ICRP) model for lead in children and adults (also called the Leggett model), and the O’Flaherty model for lead in children and adults (US EPA, 2006).

Paradigm shift in children’s lead exposure as it affects predictive BLL modeling. Prior research has often concluded that lead in tap water is not an issue that should concern parents for an “average” child under “typical” scenarios of “chronic” exposure, when applying the “10 µg/dL” BLL threshold. For example, modeling of BLLs in Seattle children, exposed to high lead in school water, concluded that “…drinking water does not significantly contribute to high BLLs in children”, and that “further investigation is warranted if drinking water lead concentrations far exceed those in this study” (>> EPA’s 15 µg/L action level) (Sathyanarayana et al., 2006). A similar modeling approach, which assessed lead exposure from tap water at home, concluded that unless a typical child consumed “…water at lead concentrations of approximately 100 µg/L, the BLL would not exceed the <CDC’s 10 µg/dL> recommended level of concern” (Gulson et al., 1997). Occasionally, simple dose-response equations have been applied to predict children’s BLLs from water exposure at home, and that work was used to conclude that “water is not a major route of lead exposure” (Portland Bureau of Water Works, 1997). The latter study even supported an exemption from requirements of the EPA Lead and Copper Rule (LCR) relevant to reducing lead in potable water (Renner, 2010).

Over the last decade significant new research and new policies make it desirable to re-examine the results and conclusions of prior modeling efforts. Specifically, the following significant changes have occurred:

- **The US has set the goal of eliminating all cases of EBL in children.** Now that significant progress has been made in addressing cases of EBL for large percentages of the population, the US has set the goal of eliminating every single instance of EBL in children by 2020 (US DHHS, 2010). This commitment increases the importance of identifying and addressing all potential lead sources, including atypical sources such as contaminated drinking water. For example, infants consuming reconstituted formula are considered a “high-risk” group, because tap water may account for more than 85% of their total lead exposure (US EPA, 1991). In addition, it has long been understood that variations in genetics and diets produce a range of BLLs in a population in response to a fixed lead dose (US EPA, 2006). Most modeling efforts conventionally focus on the “average” child through prediction of the geometric mean BLL and do not explicitly consider
the response of more sensitive sub-populations (i.e., the 90, 95 or 99th percentile BLL) or formula-fed infants.

- **Medical concern and public sensitivity over lower-level lead exposure have increased.** In the most comprehensive study for developmental neurotoxicity, Lanphear and co-authors concluded that although BLLs below 10 µg/dL in children are often considered “normal,” they are nonetheless associated with intellectual deficits (Lanphear et al., 2005). In fact, a recent review of all the available medical literature summarized evidence of neurodegenerative, cardiovascular, renal, and reproductive effects at BLLs under 10 µg/dL, and as low as 1–2 µg/dL (Health Canada, 2011). Emerging clinical evidence is therefore reinforcing CDC's position that no safe level of lead exposure exists. The European Union Risk Assessment Report recently proposed 5 µg/dL as an epistemic BLL threshold for impacts of lead upon societal cognitive resources, and 1.2 µg/dL as a reference point for the risk characterization of lead when assessing intellectual deficits in children measured by the full scale IQ score (EU SCHER, 2011).

- **Acute health risk from lead exposure is now being considered.** Lead has been traditionally considered a chronic poison, demonstrating a range of health effects due to cumulative, long-term exposures (WHO, 2006). It was only recently that consideration was also given to acute lead exposure in children in the US. Specifically, after two cases of severe lead-poisoning by accidental ingestion of lead-containing jewelry charms, one of which was fatal (US CDC, 2006; US CDC, 2004), action was taken to protect children from both acute as well as chronic exposure to lead in jewelry. The US Consumer Product Safety Commission (CPSC) established 175 µg of lead in jewelry as a dose triggering acute health concerns, product recalls and fines, resulting in recalls of more than 150 million children’s jewelry pieces in just 2004 (US CPSC, 2005). In this work it is considered reasonable that if a dose of 175 µg lead in a product not intended for human consumption triggers fines and recalls due to acute health concerns, then a similar dose of lead in water (a product intended for human consumption) is also of concern. No prior studies of lead-in-water hazards have explicitly considered acute health risks in children, and the ICRP is currently the only model that incorporates a 1-day time step to simulate short-term exposures.

- **As other lead sources are addressed, the contribution of lead in water to lead in blood may become more significant.** The US EPA acknowledged, that "as other agencies and EPA offices focus primarily on other sources of lead exposure (e.g. lead-based paint, lead in dust and soil, etc.), lead in drinking water as an exposure path is becoming a bigger percentage of a smaller number" (US EPA, 2010). Moreover, there has recently been a public health shift potentially increasing exposure to tap water at US schools and daycares to combat obesity, as opposed to soda and other soft drinks. The American Academy of Pediatrics (AAP) now discourages sugar-sweetened beverages or beverages with added sugars (e.g., fruit-flavored drinks, flavored milks, sodas) (AAP, 2004). In fact, California is the first US state to remove vending machines in schools to encourage tap water intake, based on state regulations adopting the AAP recommendations (Kolb and Medlin, 2004).

**METHODOLOGY**

This paper revisited prior modeling efforts, by expanding their analysis to reflect the above new considerations (Table 3-1). Specifically, the IEUBK model was used to expand on the work of:

1) Gulson et al. (1997) on the modeling of children’s blood lead levels from water exposure at home,

2) Sathyanarayana et al. (2006) on the modeling of children’s blood lead levels from water exposure at schools, and
3) Portland Bureau of Water Works (1997) on the modeling of children’s blood lead levels from water as a tool for policy making.

Additionally to the IEUBK model, the ICRP model was also utilized to:

4) Explore hypothetical scenarios of acute lead exposure from tap water in children.

Details for each of the four analyses follow.

**Modeling of Children’s Blood Lead Levels from Water Exposure at Home**

The work of Gulson et al. (1997) was expanded herein (Table 3-1), by:

- Considering the whole distribution of predicted BLL with emphasis on the upper tail (75%ile, 90%ile, 95%ile and 99%ile of predicted BLL), and not just the geometric mean BLL (i.e., 50%ile of predicted BLL). While the geometric mean BLL reflects the potential health impact for “average” children, the upper tail of the BLL distribution reflects the “most sensitive” children within a given age group, due to individual genetic and dietary factors affecting lead uptake and biokinetics.

- Considering lower BLL thresholds, aside from the current BLL of concern, which is set at 10 µg/dL by the US CDC. Specifically, 5 µg/dL, 2 µg/dL, and 1 µg/L were examined.

- Considering scenarios of formula-fed infants, consuming much higher volumes of water through reconstituted formula. Specifically, water consumption of 800 mL/day for 0-1 year-old infants relying on baby formula is considered average (US EPA, 2004; EU SCHER, 2011), whereas 1200 mL/day is considered high (EU SCHER, 2011; Benelam and Wyness, 2010).
Chapter 3. Modeling of children's blood lead levels from lead in tap water

Table 3-1: Summary of prior work on the modeling of children’s blood lead levels due to lead-contaminated drinking water, and expanded modeling analyses undertaken herein.

<table>
<thead>
<tr>
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<tbody>
<tr>
<td><strong>Goal</strong></td>
<td>Health Risk Assessment from elevated lead in water at a “worst-case” Australian home</td>
<td>Health risk Assessment from elevated lead in water at 71 elementary schools in Seattle</td>
<td>Health benefit assessment from corrosion control in Portland drinking water supply</td>
</tr>
</tbody>
</table>
| **Focus Group(s)** | Children aged 0.5-7 years, consuming all their water at that home | Children aged 5-6 years, consuming half their water at a given school and half at home | • Children < 6 months  
• 6 months < Children < 6 years  
• Adults |
| **Approach** | Predict Geometric Mean BLL based on daily profile of WLL (default values for other background lead exposures) | Predict Geometric Mean BLLs based on 2004 water sampling data at 71 schools and other background lead exposures (lead in water at home was assumed constant and equal to 10.3 µg/L) | Predict reductions in BLLs based on reductions in WLLs due to corrosion control |
| **Model Employed** | EPA Integrated Exposure Uptake Biokinetic Model for Lead in Children (EPA IEUBK) version 0.9 | EPA Integrated Exposure Uptake Biokinetic Model for Lead in Children (EPA IEUBK) | Individual-Based Model: Piece-wise linear dose-response function from EPA fit to the Lacey et al., 1985 data  
Population-Based Model: Exercise using same EPA linear function |
| **Scenarios Considered** | Five WLLs at home:  
• 4 µg/L (model default value)  
• 46 µg/L, 50% first draw  
• 46 µg/L, 100% first draw  
• 100 µg/L, 50% first draw  
• 100 µg/L, 100% first draw | 50% of water consumed at a given school and 50% consumed at home:  
• Worst-case: Exposure to 90th percentile lead-in-water concentration at a given school  
• Typical Case: Exposure to median lead-in-water concentration at that school (i.e., 50th percentile) | 40% reduction in water lead (limited corrosion control)  
60% reduction (moderate)  
70% reduction (optimal), translating to decrease of 90%ile WLL from 50 µg/L to 15 µg/L |
| **Key Conclusion(s)** | Predicted geometric mean BLLs in children aged 1-2 years only exceeded the 10 µg/dL level of concern when 100% of the consumed water contained 100 µg/L lead  
If more than 0.5 L of first-flush water was consumed, by formula-fed infants or pregnant women, then the BLL would easily exceed the recommended level | Geometric mean BLLs < 10.0 µg/dL in all cases  
Drinking water exposures up to 10-15 times the EPA guideline are unlikely to result in EBL  
In Seattle, elevated school drinking water lead concentrations are not a significant source of lead exposure in school-age children  
Further analysis needed only if water lead concentrations far exceed the EPA recommendations by ~ 80-100 times | Even optimal corrosion control of the water supply, expected to reduce lead in water by 70%, would result in only 10% reduction in BLLs  
The predicted percentage of the population with EBL would essentially remain unchanged as a result of corrosion control treatment  
Some resources originally targeted to reduce water lead exposure via the US EPA Lead and Copper Rule should be invested elsewhere. |
| **Additional Analysis (this work)** | Predict whole distribution of BLL with emphasis on upper tail that reflects the most sensitive children, not just geometric mean (i.e., 50%ile BLL) that reflects the “typical” child  
Predict % of children with EBL as a risk assessment criterion, not just geometric mean  
Reproduce analysis for formula-fed children consuming larger water volumes | Reproduce prior analysis by including true “worst-case” exposure to 100% percentile lead-in-water concentration  
Consider whole distribution of BLL with emphasis on upper tail, not just geometric mean that reflects typical child  
Predict % of school children with EBL as a risk assessment criterion, not just geometric mean BLL | Assess appropriateness of using linear EPA fit of Lacey et al., 1985 data to represent modern ranges of BLL and WLL in Portland  
Reproduce BLL modeling of children <6 months using Lacey et al., 1985 EPA fit (prior work), and compare to IEUBK model in terms of predicted BLL (this work) |

BLL: Blood Lead Level; WLL: Water Lead Level; EBL: Elevated BLL
Chapter 3. Modeling of children’s blood lead levels from lead in tap water

Modeling of Children’s Blood Lead Levels from Water Exposure at Schools

The work of Sathyanarayana et al. (2006) was expanded herein (Table 3-1) by:

- Considering the actual worst case for a high-risk Seattle school. Specifically, 960 µg/L of lead in first draw water and 370 µg/L in second draw water corresponded to the 100%ile of water exposure for Wedgwood Elementary (Seattle Public Schools, 2007). The prior work considered impacts of the 50%ile (“typical case” scenario) and 90%ile of lead-in-water occurrence. The 90%ile was deemed “worst-case”, while the lead values of the 99 and 100%ile of the actual lead sampling data were considered unrepresentative of chronic exposures.

- Considering the whole distribution of predicted BLL with emphasis on the upper tail (75%ile, 90%ile, 95%ile and 99%ile of predicted BLL), at each level of lead-in-water exposure. This approach allows estimation of the percentage of the population that is predicted to have EBL (i.e., BLL > 10 µg/dL). Moore (1977) first noted that “perhaps the most important aspect of this problem is the effect that high water lead has on the chances of a person having elevated blood lead”, and the IEUBK model allows for such considerations.

Modeling of Children’s Blood Lead Levels from Water as a Tool for Policy Making

The modeling analysis of Portland Bureau of Water Works (1997) for infants was revisited herein (Table 3-1), by:

- Assessing the appropriateness of using the linear EPA fit of the Lacey et al. (1985) data in representing modern ranges of BLLs and water lead levels (WLLs) in Portland

- Comparing the linear EPA regression analysis (prior work) to IEUBK model predictions (this work) in terms of predicted BLL in formula-fed infants. IEUBK modeling represents the current understanding of the curve-linear nature of the dose-response function, in the presence of other modern background lead exposures (having default values in the model), as opposed to the background lead exposures during the Lacey study (expressed by the high intercept of the WLL-BLL regression analysis).

Modeling of Children’s Blood Lead Levels under Acute Exposure Scenarios

Prior modeling efforts that utilized the IEUBK model (Gulson et al., 1997; Sathyanarayana et al., 2006) assessed chronic scenarios of lead-in-water exposures in children, encompassing the model’s time step of 1-year. From the three available models for lead in children (IEUBK, ICRP, O’Flaherty), only the ICRP incorporates a daily time step and can thus allow exploration of short-term lead exposures. Preliminary ICRP modeling was undertaken herein, in order to explore hypothetical scenarios of acute lead exposure through drinking water in children aged 5 years, by:

- Predicting BLL from direct consumption of a single glass of water (250 mL) containing various levels of lead (0-20,000 µg/L). Aside from this hypothetical one-time ingestion of the contaminated water, all lead exposures (including water) were assumed to be equal to zero at all other times.

- Predicting BLL from indirect consumption of the contaminated water, through consumption of one portion of pasta cooked with the water (750 mL of water required for one portion) containing various levels of lead (0-20,000 µg/L). Aside from this
hypothetical one-time ingestion of the contaminated food, all lead exposures (including water) were assumed to be equal to zero at all other times. This scenario also recognizes the potential for large accumulation of lead in food during cooking with large volumes of contaminated volume, which was recently implicated as the source of two children's lead poisoning (Triantafyllidou et al., 2007).

RESULTS AND DISCUSSION

Modeling of Children’s Blood Lead Levels from Water Exposure at Home

Gulson et al (1997) predicted that constant exposure to a WLL of about 100 µg/L (corresponding to first-flush water), resulted in exceedance of the recommended BLL of concern for a typical child (i.e., predicted geometric mean BLL > 10 µg/dL) (see Table 3-1). That work concluded that unless a typical child consumed 100% of first flush water at lead concentrations of approximately 100 µg/L, the BLL would not exceed the recommended level of concern. The authors qualified this conclusion by stating that if more water was consumed in drinks and formula using first flush water, then the BLL could easily exceed the recommended level (Gulson et al, 1997). Reproducing the work of Gulson et al. (1997) for children aged 1-2 years yielded a water lead level of 100 µg/L, in order for the geometric mean BLL to exceed the 10 µg/dL threshold (Table 3-2, scenario 1), thereby confirming Gulson's main conclusion that 50% of the exposed children are predicted to have EBL (i.e., BLL > 10 µg/dL).

The extended analysis performed herein reveals that a lower WLL of 55 µg/L is sufficient to elevate lead in blood (> 10 µg/dL) in 25% of the exposed children, whereas a WLL of 19 µg/L would be sufficient to elevate lead in blood in 5% of those children (Table 3-2, scenario 1). If the BLL threshold was set at a more stringent level of 5 µg/dL (instead of 10 µg/dL), then 24 µg/L of lead in water is predicted to cause 50% of the population to exceed that threshold, 7 µg/L is predicted to cause 25% of the population to exceed the threshold, and no WLL is needed to cause exceedance for 10%, 5% and 1% of the children’s population due to other background lead exposures (Table 3-2, scenario 1). To put these percentages into perspective, 1% of the children’s population aged 1-2 years old in a large US metropolis like Washington, DC would translate to almost a hundred cases of elevated blood lead (Edwards et al., 2009; US Census, 2011a), whereas the same percentage in New York City would translate to more than two thousand cases (US Census, 2011b).

For infants aged 0-1 years, consuming the average dose of baby formula daily in the presence of other background lead exposures, 60 µg/L of lead in water would elevate the blood lead of 50% of the population, whereas 28 µg/L is predicted to elevate the blood lead of 10% of the population (Table 3-2, scenario 2). If the BLL threshold was set at 5 µg/dL, then much lower WLLs would achieve such percentage exceedances for exposed infants consuming formula (Table 3-2, scenario 2).

For infants aged 0-1 years old, consuming a high dose of baby formula in the absence of any other lead exposure source, even the smallest WLL is predicted to affect some percentage of the population (Table 3-2, Scenario 3). For example, 50 µg/L of lead in water is predicted to elevate lead in blood for 50% of that population (i.e. cause BLL > 10 µg/dL), but just 4 µg/L would be enough for 10% of the population to exceed a BLL threshold of 2 µg/dL (Table 3-2, scenario 3).
Table 3-2: Required level of lead in water for a given percentile of children to exceed certain BLL thresholds. Results obtained with EPA IEUBK model, under three exposure scenarios.

<table>
<thead>
<tr>
<th>BLL Threshold (µg/dL)</th>
<th>Predicted Percentile of BLL</th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
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<tr>
<td></td>
<td>[50% exceed threshold]</td>
<td>[75% exceed threshold]</td>
<td>[90% exceed threshold]</td>
<td>[95% exceed threshold]</td>
<td>[99% exceed threshold]</td>
</tr>
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<td>100 µg/L*</td>
<td>55 µg/L</td>
<td>30 µg/L</td>
<td>19 µg/L</td>
<td>3 µg/L</td>
</tr>
<tr>
<td>5</td>
<td>24 µg/L</td>
<td>7 µg/L</td>
<td>0 µg/L*</td>
<td>0 µg/L*</td>
<td>0 µg/L*</td>
</tr>
</tbody>
</table>

Scenario 1: 1-2 year old child drinking tap water
Assumptions: 500 mL/day (IEUBK default value)
GSD=1.6 (IEUBK default)
Background exposures from other lead sources (IEUBK default)

Scenario 2: 0-1 year old infant consuming reconstituted baby formula, Average consumption
Assumptions: 800 mL/day (EPA, 2004; Average Consumption in EU SCHER, 2011)
GSD = 1.45 (EPA 2004)
Exposure through diet set to 0
Background exposures from other lead sources (IEUBK default)

Scenario 3: 0-1 year old infant consuming reconstituted baby formula, High consumption
Assumptions: 1200 mL/day (High Consumption in EU SCHER, 2011)
GSD = 1.6 (IEUBK default)
Background exposures from other lead sources set to 0

#Reproduction of Gulson et al. (1997) work. All other results reflect additional analyses undertaken herein.
*Due to other background lead exposures (e.g. air and soil/dust), even 0 µg/L lead in water would still result in exceedance of a given BLL threshold
BLL: Blood Lead Level, GSD: Geometric Standard Deviation; IEUBK: Integrated Exposure Uptake Biokinetic Model for Lead in Children
While the three modeled scenarios are not directly comparable, due to the different assumed background lead exposures and geometric standard deviation in each (see Table 3-2), the main point of this analysis is that substantial numbers of the “most sensitive” children within the age group of 1-2 years (those in the upper tail of the predicted BLL distribution) may be adversely affected by WLLs much lower than the previously reported 100 µg/L (Table 3-2, scenario 1). In addition, consistent with the Gulson et al. (1997) assertion, formula-fed infants (Table 3-2, scenarios 2 and 3) are much more vulnerable to even low-level lead contamination from tap water, due to the large volumes of water required to reconstitute infant formula and due to the high bioavailability factors of infants compared to adults.

Modeling of Children’s Blood Lead Levels from Water Exposure at Schools

Sathyanarayana et al (2006) simulated “typical case” and “worst case” scenarios of exposure to water lead for 71 Seattle elementary schools, yielding predicted geometric mean BLLs below 5.0 µg/dL for all cases, and the authors concluded that “school drinking water is not likely to contribute to increased BLLs in children” (see Table 3-1). Reproducing this prior work for children aged 5-6 years, exposed to the 50%ile and 90%ile WLL at Wedgewood elementary school in Seattle (Figure 3-1), confirmed a predicted geometric mean BLL below 5.0 µg/dL. Specifically, the geometric mean BLL was 1.8 µg/dL for the prior work’s “typical” exposure scenario and 3.9 µg/dL for the prior work’s “worst-case” scenario (Figure 3-2, tabulated data).

![Percentile lead distribution in first and second draw water of “worst-case” elementary school in Seattle in 2004. The horizontal red lines correspond to the 50, 90 and 99%’ile of the measured lead-in-water concentration. The 90% ile was modeled in prior work as “worst-case”, while the true “worst-case” level was deemed unrealistic for a chronic exposure scenario. Source of lead-in-water data: Seattle Public Schools, 2007.](image)

However, if the IEUBK model is re-run by using the Sathyanarayana et al. (2006) approach but considering the actual worst case for the specific Seattle school (i.e., 960 µg/L first draw and 370 µg/L second draw for Wedgwood Elementary, corresponding to the 100%ile of
exposure), the calculated contribution to blood lead is 13.2 µg/dL higher than for the 90%’ile. Indeed, the predicted true worst case geometric mean BLL of 17.1 µg/dL for Seattle children in this school is well over the 10 µg/dL level of concern that is classified as “lead poisoning” in many States (Figure 3-2). Explicit consideration of the actual “worst-case” scenario reflects a child that constantly consumes water from the same high-risk fountain at a given school.

Predicting the percentage of the children’s population with EBL using all the available data and the general approach of Sathyanarayana et al. (2006) offers another important perspective. Specifically, if water was routinely consumed at the 50%ile of lead occurrence, a child’s predicted likelihood of having EBL is 0.0% (Figure 3-2, tabulated data). Likewise, exposure to the 95%ile WLL corresponds to an 8.4% likelihood of EBL, while exposure to the 99%ile WLL corresponds to almost 80% predicted likelihood of EBL (Figure 3-2, tabulated data). By integration over the entire distribution of WLLs, the overall predicted risk of EBL for a 5-6 year old child, drinking half of his daily water at the specific school and half at home, was calculated to be ~3%. While such a risk is not indicative of an epidemic, most parents and health experts would consider it unacceptable that 3 out of 100 students attending classes in that specific school are predicted to have EBL from drinking water consumption, as modeled by using the approach and assumptions of Sathyanarayana et al. (2006).

Figure 3-2: Predicted log-normal distribution of BLLs in a population of 5-6 year old students, exposed to the 90 and 99%ile of lead-in-water concentration in worst-case Seattle elementary school (results from 50% exposure to school water and 50% exposure to home water). The predicted geometric mean BLL and % EBL are presented in the table for other levels of lead in water exposure. Analyses highlighted in grey reflect reproduction of prior work for the specific school, which was further expanded herein using the IEUBK model.

We also note that the highest observed WLLs (> 90%ile) that were previously excluded from consideration in Seattle schools as unrepresentative (Sathyanarayana et al., 2006), are
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actually 10-20 times lower than the highest concentrations reported in other school systems across the US (Triantafyllidou and Edwards, 2011). For demonstration purposes, the IEUBK model was used herein to predict EBL in students attending classes at a “worst-case” elementary school in Durham, NC, for which first-draw WLLs were available (50%ile = 7 µg/L, 75%ile = 56 µg/L, 90%ile = 208 µg/L, 95%ile = 407 µg/L, 99%ile = 5953 µg/L, 100%ile = 8567 µg/L). By integration over this school’s whole WLL distribution, similarly to the approach that was used for the Seattle school (see Figure 3-2, tabulated data), the overall risk of EBL for children drinking half their water at this school is estimated at 10%. Clearly, if the totality of the WLL distribution and of the IEUBK model predictions is considered, then the conventional wisdom about lack of concern from elevated lead in school water becomes questionable, for “worst-case” schools such as those investigated herein.

**Modeling of Children’s Blood Lead Levels from Water as a Tool for Policy Making**

The Portland Bureau of Water Works (1997) utilized a piece-wise linear dose-response model, to predict BLL reduction in infants, due to reductions in WLL achieved by means of corrosion control (see Table 3-1). The US EPA fit to the Lacey et al. (1985) data for formula-fed infants was employed, having a slope of 0.26 µg/dL blood per µg/L water at WLLs ≤15 µg/L, and a slope of 0.04 µg/dL blood per µg/L water at WLLs >15 µg/L (Portland Bureau of Water Works, 1997). Based on predictions of minimal anticipated health benefit (Portland Bureau of Water Works, 1997), the city received an exemption from the optimal corrosion control requirements under the EPA Lead and Copper Rule, if they contributed $500,000 annually to a public education campaign and lead paint abatement program (Renner, 2010). This program earned Portland Water Bureau a 2005 Children’s Environmental Health Recognition Award by the US EPA (Portland Bureau of Water Works, 2005).

Using the EPA piece-wise fit of the Lacey et al. (1985) data does indeed predict a reduction in BLL of only 1.4 µg/dL, if WLL is reduced by 70% due to corrosion control (from about 50 µg/L down to 15 µg/L) (Figure 3-3). However, the Lacey study best fit reflected high BLLs presumably from numerous sources, resulting in an intercept of about 14 µg/dL for the original linear model (Lacey et al., 1985). That is, even with no contribution from lead in water, the average BLLs in infants in the dataset used by Portland were already higher than the 10 µg/dL BLL of concern. For such a dataset, slight reductions of lead in water from 50 to 15 µg/L are not expected to yield a marked reduction in blood lead, especially considering that WLLs for that population often exceeded 300 µg/L.

To illustrate this point using the more sophisticated IEUBK model for infants aged 0-1 years who consume reconstituted formula, a daily water intake of 800 mL and a geometric standard deviation on 1.45 were assumed for the IEUBK simulation, based on US EPA approach in similar analysis (US EPA, 2004). Confirming the basic validity of the Portland approach if it had been applied in an earlier era of high lead exposure from numerous sources, the IEUBK model predicts a relatively modest reduction of 2.6 µg/dL in geometric mean BLL versus the 1.4 µg/dL reduction cited above, if water lead is reduced from 50 to 15 µg/L (Figure 3-3).

But if the EPA stepwise linear model is shifted to reflect an intercept of 0, as was done in the Portland Bureau of Water Works (1997) analysis, it extends outside the range of BLLs in the Lacey dataset. For this case, based on IEUBK modeling, the health benefit from reducing WLL from 50 to 15 µg/L would be a 4.8 µg/dL reduction in geometric mean BLL of formula-fed infants (from 7.5 to 2.7 µg/dL), which is much higher than the Portland estimate of only a 1.4 µg/dL reduction based on the linear EPA model (from 5.3 µg/dL to 3.9 µg/dL) (Figure 3-3). In other words, in a modern era of much lower lead exposures for which the BLL intercept is closer
to zero, reducing lead in water by 70% is predicted to reduce lead in blood by 64% in formula-fed infants (this work). This result is also more in keeping with expectations, given the predominance of lead in water as a source of potential exposure for formula-fed infants (US EPA, 1991). In summary, the scientific basis for exempting Portland from EPA LCR requirements was at least partly due to misapplication of a linear model to conditions outside its original dataset range.

Figure 3-3: Change in blood lead level as a function of water lead level for infants, using a linear piece-wise EPA model to fit the data of Lacey et al. (1985), versus IEUBK modeling prediction. The linear model was used in recent analysis of corrosion control alternatives in the water supply of Portland, OR. Optimal corrosion control would be expected to reduce the 90%ile water lead level from 50 µg/L to 15 µg/L (70% reduction).

**Modeling of Children’s Blood Lead Levels under Acute Exposure Scenarios**

Preliminary modeling with the ICRP suggests that one time exposure of a 5- year old child to a single cup of water containing high levels of lead can markedly raise lead in blood (Figure 3-4). Such requisite levels of lead in water to elevate lead in blood from a one-time dose have been reported in numerous US field investigations (Triantafyllidou and Edwards, 2011). These high levels were typically associated with particulate (rather than soluble) lead in water.
and indeed caused elevated lead in blood of some exposed individuals (Triantafyllidou and Edwards, 2011). Food that requires large volumes of water for cooking can also concentrate lead from the water. This exposure pathway, which has never been assessed in existing lead models, has the potential to cause blood lead elevations through consumption of a single food portion (Figure 3-4). Such a pathway was involved in isolated cases of lead poisoning due to food consumption when tap water was never directly consumed (Triantafyllidou et al., 2007).

If the US CPSC dose of acute health concern in children’s lead jewelry was applied to some of the higher levels of lead detected in water of US schools or homes (Triantafyllidou and Edwards, 2011), the one-time ingestion of 250 mL of water containing 700 µg/L lead (resulting in a lead dose of 175 µg) must also be considered an acute health risk to children. Similarly, a one-time consumption of pasta cooked with 750 mL water containing 233 µg/L of lead, would result in a lead dose of 175 µg and pose an acute health concern if all the lead was captured in the food.

![Figure 3-4](image)

Figure 3-4: Predicted blood lead level of 5-year old child after direct consumption of lead-contaminated water, or consumption of food cooked with that water. The ICRP bio-kinetic model (version 3000, excel interface) was employed to simulate one-time exposures.

Limitations

Limitations of modeling efforts apply not only to the work presented herein, but also to the prior research. Specifically, potential limitations of the ICRP and IEUBK models have been reported elsewhere (Pounds and Leggett, 1998; US EPA, 2006). It is also recognized that the ability of the IEUBK model to predict the "tail" of the BLL distribution is limited, which is why prior research can be justified in restricting conclusions to the typical case for an era with different public health goals. In addition, the high WLLs used in ICRP simulations are typically
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associated with particulate lead presence in tap water, which may be less bioavailable (Triantafyllidou et al., 2007; Deshommes et al., 2011) compared to the ICRP model default bioavailability factor of 30% for oral ingestion of lead. Regardless of any potential limitations, the general trends reported herein reflect expectations based on available models and emphasize the need to be more conservative when considering the possible adverse health consequences in individuals from exposure to elevated lead in water.

CONCLUSIONS

Reproduction of prior modeling work is consistent with the expectation that lead in tap water is not a major risk for the average child under typical chronic exposure scenarios when applying a 10 µg/dL BLL threshold for health concerns.

Considering the whole predicted distribution of BLLs and not just the geometric mean BLL for a population exposed to a fixed WLL, reveals significant health impacts for the most sensitive children at the upper tail of the distribution, even at low levels of water lead. Explicit consideration of formula-fed infants, which are a high-risk group due to their small body weight and heavy reliance on water as a major component of their diet, also revealed significant health concerns at relatively low WLLs (< 50 µg/L).

Expanding the analysis to consider lower BLL thresholds (e.g., 5, 2 and 1 µg/dL versus 10 µg/dL) is consistent with the increased medical concern about low level lead exposure, and suggested that relatively low WLLs (< 24 µg/dL) would have an adverse impact for high-risk groups (i.e., very young children and formula-fed infants). Extending the analysis to consider acute exposures suggested that a single glass of water, or one portion of food cooked with contaminated water in the upper range of water lead levels reported in modern US sampling events, has the potential to cause blood lead elevations in children.

Although all models suffer from inherent limitations that restrict the certainty of the absolute predictions, the evaluation reported herein indicates cause for public health concern at levels of lead in water previously considered inconsequential, when applying new approaches to conventions of earlier eras.

ACKNOWLEDGEMENTS

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REFERENCES


Seattle Public Schools, Drinking Water Quality Program, Results of Water Quality Testing for Wedgwood elementary school, Updated January 29, 2007


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US CDC. Preventing Lead Poisoning in Young Children. Atlanta, 2005


CHAPTER 4. GALVANIC CORROSION AFTER SIMULATED SMALL-SCALE PARTIAL LEAD SERVICE LINE REPLACEMENTS

Simoni Triantafyllidou and Marc Edwards

ABSTRACT

Partial lead service line replacement with copper pipe creates a galvanic cell which can accelerate lead corrosion. Bench-scale experiments under stagnant water conditions of high chloride-to-sulfate mass ratio (CSMR) demonstrated that galvanic connections between lead pipe (new or aged) and copper pipe increased lead release into the water by 1.1-16 times, compared to a full length of lead pipe alone. The extent of galvanic attack was dependent on drinking water quality. Exposure to water of high CSMR increased lead release in the lead:copper rigs by 3-12 times, compared to a less aggressive low CSMR water. Galvanic current also increased by 1.5-2 times, when switching from low to high CSMR. The small area of lead pipe adjacent to the copper joint (<0.5 ft) dissipated 90-95% of the total galvanic current, and accumulated a thick lead-rust layer (1 inch wide), which constituted a reservoir for semi-random particulate lead detachment into the water.

INTRODUCTION

Harmful health effects from lead exposure through drinking water have been historically recognized in the US since the 1850s. In that era drinking water contamination by lead pipes was thought to be the main source of human-ingested lead, causing infant mortality, neurological effects, and digestive problems (Troesken, 2006). Lead service lines were the standard in many US cities through the 1950s, and were even occasionally installed up to the ban of lead pipe in 1986 (Renner, 2010). As of 1990, 3.3 million lead service lines were estimated to be present across the US, while 6.4 million lead connections (e.g., goosenecks) were also acknowledged (Weston and EES, 1990). The actual number of old lead service lines in the US today is unknown.

Old lead service lines can still be significant contributors to lead-in-water hazards. Depending on their length, diameter, water corrosivity, water use and flow rates, these old lead pipes can account for 50-75% of the lead mass measured in standing sequential samples collected at the tap (Sandvig et al, 2009). Lead in US drinking water is regulated under the Lead and Copper Rule (LCR), which may require replacement of utility-owned lead service lines, if the LCR lead action level of 15 ppb is exceeded (US EPA, 1991). Voluntary utility-owned lead service line replacement also routinely occurs during other events such as system maintenance or road repair (Renner, 2010).

Partial lead service line replacement and its implications. If the lead service line extends onto the homeowner’s property, the utility is only required to replace the portion of pipe that it owns, leaving behind the customer-owned portion of lead pipe (Figure 4-1, left). The practice of “partial lead service line replacement” was assumed to provide health benefits because there is a smaller volume of water in contact with the lead pipe (US EPA, 2000). Although numbers vary dramatically from city to city or even from home to home, a national survey (Sandvig et al.
2009) indicated that the typical service line length in the US averages 55-68 feet, with 25-27 feet (i.e., 40-45%) being under the utility’s jurisdiction. Partially replacing a single lead service line can cost from $1,000 to more than $3,000 for water utilities (AWWA, 2005). Few customers voluntarily pay to replace their portion of the lead service line (Swertfeger et al, 2006).

![Figure 4-1: Typical plumbing configuration after partial lead service line replacement, where copper pipe is connected to lead pipe (left). Conceptualization of galvanic corrosion due to direct electrical connection of copper pipe to lead pipe (right).](image)

The practice of only replacing the utility portion of lead pipe received newfound attention during implementation of the largest lead service line replacement program in US history in Washington DC (Renner, 2010; Leonnig, 2008; Edwards, 2004), and then again after the Centers for Disease Control and Prevention (CDC) linked partial replacement of lead pipes to increased incidence of high blood lead levels in DC children (Frumkin, 2010). The CDC epidemiological study found that “when lead service lines are partially replaced, children are more likely to have blood lead levels greater than or equal to 10 µg/dL, compared to children living in housing with either undisturbed lead service lines or service lines that are not made of lead” (Frumkin, 2010), or at least, that “partially replacing lead service lines may not decrease the risk of elevated blood lead levels associated with lead service line exposure” (Brown et al., 2011). Some in the water industry have called for a moratorium on partial replacements based on this new information (Renner, 2010).

The increased lead in water after partial replacements can arise from a variety of mechanisms and can possibly be relatively short-term (days to weeks) or longer-term (months to years) in duration (Chambers and Hitchmough, 1992). Short-term problems occur from disturbing the lead rust (i.e., scale) that has accumulated on the pipe over decades of use, and/or from creating metallic lead particles when the pipe is cut (Schock et al, 1996). In the US these short-term mechanisms from cutting and scale disturbance were definitively documented in laboratory experiments (Boyd et al, 2004).
Longer-term problems might arise from creation of a new electrochemical or galvanic cell, if part of the old lead service line is replaced with copper pipe (Figure 4-1). Although there are numerous warnings in the literature about the harmful impact of partial replacements due to galvanic effects, there is a general lack of unambiguous laboratory data demonstrating whether these effects are significant (Table 4-1). Utilities in the UK have warned about adverse consequences of galvanic corrosion. For example, Chambers and Hitchmough (1992) noted, without citing data, that in practice the galvanic effect between lead and copper may annul any beneficial effects of reducing the length of lead pipe in the system (Table 4-1). Breach et al (1991) also noted the dangers from inserting copper pipes upstream of and electrochemically linked to lead, while Britton and Richards (1981) documented increased and erratic lead levels at the tap, when copper-lead plumbing connections were present in front of homes (Table 4-1). The latter study cited extensive full-scale testing data from homes with and without partial replacements.

Recent research work on galvanic corrosion indicated that any impact on lead leaching would not be significant. Reiber and Dufresne (2006) concluded that galvanic impacts were short-lived on aged/passivated lead service lines, based on surface potential measurements (Table 4-1). In that work, lead release to the water or the magnitude of the galvanic current was never measured. Another study, which also utilized continuous flow, concluded that galvanic coupling would not have any long term impacts on lead release (HDR Engineering, 2010) (Table 4-1). In both of these studies water was never allowed to sit stagnant inside the pipe, as occurs regularly in practice and which is known to initiate the “worst case” galvanic attack due to creation of much lower pH near the lead surface (see Figure 4-1, right). Prior work demonstrated that stagnation is key to initiation of galvanic corrosion problems in practice (Nguyen et al, 2010a; Dudi, 2004; Breach, 1991). More recently, DeSantis et al (2009) provided clear visual and mineralogical documentation of lead galvanic corrosion in some harvested pipe connections which were excavated after 70-114 years in service (Table 4-1).
Table 4-1: Summary of studies on galvanic corrosion between lead and copper plumbing (in chronological order).

<table>
<thead>
<tr>
<th>Study</th>
<th>Type</th>
<th>Flow Rate/Frequency</th>
<th>Measurements</th>
<th>Relevant concluding quote</th>
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</thead>
<tbody>
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<td>Field sampling of houses with mixed lead-copper plumbing</td>
<td>Real world conditions, flow rate unknown</td>
<td>Lead in water, sampling details not provided</td>
<td>“Occasionally, the insertion of copper pipe &lt;in lead plumbing&gt; can produce particularly bad results, and despite pH control it might be impossible to obtain satisfactory samples”</td>
</tr>
<tr>
<td>Breach et al., 1991</td>
<td>Practical experiences of a big UK water company in minimizing lead at the tap</td>
<td>Real world conditions, flow rate unknown</td>
<td>Lead in water, sampling details not provided</td>
<td>“Another area of possible concern that should not be overlooked is the dangers arising from inserting copper pipes or other apparatus upstream of and electrochemically linked to lead”</td>
</tr>
<tr>
<td>Chambers and Hitchmough, 1992</td>
<td>Practical experiences of UK water companies on partial LSL replacements</td>
<td>Real world conditions, flow rate unknown</td>
<td>Lead in water, sampling details not provided</td>
<td>“The creation of a galvanic cell, giving rise to increased and erratic levels of lead at the tap, may well annul any benefit of reducing the length of lead pipe in the system”</td>
</tr>
<tr>
<td>Dudi, 2004</td>
<td>Laboratory examination of lead-copper pipe connections</td>
<td>No flow except during water changes</td>
<td>Galvanic current, Lead in water</td>
<td>“Galvanic connections between copper and lead can dramatically worsen lead leaching under a wide range of circumstances”</td>
</tr>
<tr>
<td>Reiber and Dufresne, 2006</td>
<td>Laboratory examination of polarization cells mounting harvested LSLs and new copper tubing</td>
<td>Continuous flow</td>
<td>Surface potential No measurements of lead in water</td>
<td>“Galvanic impacts on aged and passivated LSL surfaces are minimal, and in the long-term, likely inconsequential”</td>
</tr>
<tr>
<td>DeSantis et al., 2009</td>
<td>Examination of corrosion scales on harvested domestic lead-copper joints</td>
<td>Real world conditions, flow rate unknown (&gt;70 years in service)</td>
<td>Micro/macro characterization of scale solids, XRD</td>
<td>“Deep localized corrosion in the area adjacent to the pipe joints suggests a galvanic mechanism”</td>
</tr>
<tr>
<td>HDR Engineering, 2010</td>
<td>Laboratory examination of harvested LSLs, later connected to new copper pipe</td>
<td>Low flow rate of 1.3 LPM/continuous flow</td>
<td>Lead in water</td>
<td>“Galvanic coupling has little relevance to accelerating metal release on the LSL, and is an easily managed process”</td>
</tr>
<tr>
<td>Nguyen et al., 2010a</td>
<td>Continuation of HDR work using same apparatus</td>
<td>No flow except during water changes</td>
<td>Galvanic current, lead in water</td>
<td>“Clear conclusions about the effects of Pb:Cu galvanic connections on leaching of lead from lead pipe are not possible. Additional research is needed”</td>
</tr>
<tr>
<td>Nguyen et al., 2010b</td>
<td>Laboratory examination of macrocells connecting copper pipe and lead wire</td>
<td>No flow except during water changes</td>
<td>Galvanic current, lead in water, chloride, sulfate, pH at anode/cathode microlayers</td>
<td>“Microlayer effects can explain a subset of persistent lead corrosion problems in some buildings and from lead:copper service line joints at some water utilities”</td>
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LSL: Lead Service Line, XRD: X-ray Diffraction, LPM: Liters Per Minute, Pb: Lead, Cu: Copper
Galvanic corrosion of lead. The importance of this type of attack was first documented in the UK as early as 1859, when it was realized that “the solution of lead was assisted by contact with other metals”, and that galvanic corrosion was “a most powerful agent in promoting the corrosive action of certain waters upon lead” (Ingleson, 1934). When copper pipe is electrically connected to lead pipe, it can accelerate corrosion of the lead pipe by galvanic action, above and beyond corrosion that would normally occur for lead pipe alone. The drinking water in contact with these dissimilar metals serves as the electrolyte. Based on the galvanic series (Davis, 2000), metallic lead typically serves as the anode of this galvanic cell and is therefore oxidized (i.e., corroded) to form Pb\(^{+2}\) (Figure 4-2, A). The copper pipe typically serves as the cathode, where the cathodic reaction (such as dissolved oxygen reduction) occurs over its surface (Figure 4-2, A).

The production of Pb\(^{+2}\), which is a Lewis acid, can cause a local pH drop and draw chloride ions to the lead anode surface (Nguyen et al., 2010b; Dudi, 2004). Lead leaching to water can be increased indefinitely due to the higher corrosion rate, lower pH, and higher chloride, especially in waters with a relatively high chloride-to-sulfate mass ratio (CSMR) (Edwards and Triantafyllidou, 2007; Edwards et al, 1999; Oliphant, 1983; Gregory, 1985). Further mechanistic insights that explain CSMR impacts on galvanic corrosion of lead-bearing materials are presented elsewhere (Nguyen et al., 2010b).

The Pb\(^{+2}\) ions that are produced at the junction can be directly released into drinking water and contaminate the water (Figure 4-2, B), or, they may accumulate in a lead-rust layer on the interior surface of the lead pipe (Figure 4-2, C). Intuitively, in the latter situation, under high water flow or other disturbance, lead particles might eventually detach, and contaminate drinking water with erratic “spikes” of lead (Figure 4-2, C).

Deposition corrosion of lead. Another potentially important, yet unappreciated, micro galvanic phenomenon is deposition corrosion (Dudi, 2004). Deposition corrosion can occur when soluble ions from a more cathodic metal (e.g., copper) are present in the drinking water...
flowing through a lead pipe. Britton and Richards (1981) first noted that “each site of copper deposition <on the lead pipe> has potential to act as an individual galvanic cell” and raise the water lead concentration. Deposition corrosion can be viewed as a two-step micro galvanic process. To illustrate, cupric ions (i.e., Cu\(^{2+}\)) released from a copper pipe installed upstream of lead pipe, can be directly deposited and plated onto the lead surface (Figure 4-3, A). The newly plated copper metal then forms a micro galvanic cell, which can catalyze corrosion of the underlying lead indefinitely (Figure 4-3, B). Through this process, numerous micro galvanic cells, or else batteries, can be “turned on” at the surface of the lead pipe, accelerating corrosion of the lead pipe and contamination of the flowing drinking water (Dudi, 2004). This mechanism was recently proved to be significant for lead pipe corrosion in experiments, in the presence of trace levels of Cu\(^{2+}\) ions (Nguyen et al, 2009). In those experiments, lead release increased when more Cu\(^{2+}\) was dosed to the water, while less than 15-50% of the initial dosed Cu\(^{2+}\) was measured in the water after exposure to the lead pipes. This suggested that the missing copper fraction had deposited onto the lead pipe (Nguyen et al, 2009).

The US Environmental Protection Agency (EPA) has indicated a willingness to re-evaluate regulations that cover partial lead service line replacement, as part of the 2012 long-term revisions to the LCR (Renner, 2010). This work is designed to inform decision-making by conducting the first long-term study of galvanic impacts on lead release into drinking water, under well-defined laboratory conditions.

**MATERIALS AND METHODS**

The experimental apparatus was constructed to track lead leaching from simulated small-scale partial lead service line replacement with copper. The test rigs consisted of a copper pipe section (type M, 3/4 inch internal diameter, 7/8 inch outer diameter) that was electrically connected to lead pipe (3/4 inch internal diameter, 1 inch outer diameter), with a total rig length of three feet (Figure 4-4). The lead portion and the copper portion of each rig were separated by a ¼ inch
insulating spacer, and could be externally connected via grounding strap wires (Figure 4-4). If the wires were disconnected, direct galvanic corrosion between lead and copper did not occur, but if the wires were connected the galvanic current flowed as usual (Figure 4-4, top).

Figure 4-4: Generalized schematic of experimental setup, assessing the contribution of galvanic corrosion to lead in water, as it would occur after partial lead service line replacement (top). Photo of experimental setup, with varying extent of lead:copper pipe ratio (bottom).

The fraction of the pipe that is lead and the fraction of the pipe that is copper were systematically varied, as would occur in partial replacements with different percentage of consumer ownership of the service line. Specifically, 100% lead pipe (simulating a lead service line before replacement), 100% copper pipe (simulating full replacement), and four increments in
between (17% copper pipe, 50% copper pipe, 67% copper pipe, and 83% copper pipe to simulate partial replacements) were tested (Figure 4-4, bottom).

Four sets of rigs were constructed, utilizing four different types of lead pipe:

- **New Pb pipe**, i.e., lead pipe which had not yet been used in experiments
- **Aged Pb pipe A**, i.e., lead pipe which was initially new but had been previously used in other short-term experiments of four months,
- **Aged Pb pipe B**, i.e., lead pipe which was initially new but had been previously used in other longer-term experiments of one year. Each of these older lead pipes was 0.5 feet long. For the needs of this experiment, the third set of rigs was constructed by connecting in series as many short Pb pipes together as needed, in order to add up to the desired Pb length
- **Pb (IV) pipe**, i.e., lead pipe which was exposed to re-circulating water with excess chlorine, to form an interior brown corrosion layer of Pb (IV) solids, right before the beginning of the experiment

The first three sets of lead pipe (new Pb pipe, aged Pb pipe A, and aged Pb Pipe B) were pre-treated in the same way prior to construction of the rigs. They were exposed to re-circulating pH 2.0 water (de-ionized water with the addition of H_2SO_4) for three hours, in order to remove surface rust and deposits. They were subsequently rinsed with de-ionized water for fifteen minutes, exposed to pH 10.0 water (de-ionized water with the addition of NaOH) for another three hours, and finally rinsed with de-ionized water for another fifteen minutes.

Several phases of experimentation were undertaken, that lasted for more than one year:

- **Phase 1.** During weeks 1-11, all rigs were exposed to synthetic tap water with a low chloride-to-sulfate mass ratio (CSMR) of 0.2 (“low CSMR water”). This water also had an alkalinity of 15 mg/L as CaCO_3, monochloramine disinfectant dosed at 4.0 mg/L as Cl_2, ionic strength of 4.6 mmol/L (by addition of salts to mimic other tap water constituents) and pH of 8.0 (Table 4-2).

- **Phase 2.** After baseline results were established in the non-aggressive water, the test water was switched to an aggressive synthetic tap water with a high CSMR of 16 during weeks 12-25 (“high CSMR water”). All other water parameters such as alkalinity, monochloramine, ionic strength and pH were kept the same as in the “low CSMR water” of Phase 1 (Table 4-2).

- **Phase 3.** For weeks 26-31 the rigs continued to be exposed to the “high CSMR water” as in Phase 2, but without direct galvanic corrosion between the lead and copper pipe due to removal of the connecting strap wires.

- **Phase 4:** For weeks 32-37 the rigs were kept being exposed to “High CSMR water” as in Phase 3, but the connecting strap wires between lead and copper pipe were re-connected, thereby re-activating galvanic corrosion.

- After Phase 4, additional experiments were conducted. The rigs were exposed to modified high CSMR water, by gradually increasing the alkalinity level from 15 to 50 mg/L as CaCO_3 (weeks 38-41), and then from 50 to 100 mg/L as CaCO_3 (weeks 42-51). Alkalinity levels throughout the experiment were adjusted by adding the appropriate amount of sodium bicarbonate (NaHCO_3) from a fresh stock solution to the synthetic water.
Table 4-2: Key characteristics of the synthetic waters utilized in the experiment.

<table>
<thead>
<tr>
<th></th>
<th>Cl⁻ (mg/L)</th>
<th>SO₄²⁻ (mg/L)</th>
<th>CSMR (-)</th>
<th>Alkalinity (mg/L as CaCO₃)</th>
<th>Ionic Strength (mmol/L)</th>
<th>NH₂Cl (mg/L as Cl₂)</th>
<th>pH</th>
</tr>
</thead>
<tbody>
<tr>
<td>Low CSMR Water (Phase 1)</td>
<td>22</td>
<td>112</td>
<td>0.2</td>
<td>15</td>
<td>4.6</td>
<td>4.0</td>
<td>8.0</td>
</tr>
<tr>
<td>High CSMR Water (Phase 2,3,4)</td>
<td>129</td>
<td>8.0</td>
<td>16</td>
<td>15</td>
<td>4.4</td>
<td>4.0</td>
<td>8.0</td>
</tr>
<tr>
<td>High CSMR Water, Alk. 50 (subsequent tests)</td>
<td>129</td>
<td>8.0</td>
<td>16</td>
<td>50</td>
<td>5.1</td>
<td>4.0</td>
<td>8.0</td>
</tr>
<tr>
<td>High CSMR Water, Alk. 100 (subsequent tests)</td>
<td>129</td>
<td>8.0</td>
<td>16</td>
<td>100</td>
<td>6.1</td>
<td>4.0</td>
<td>8.0</td>
</tr>
</tbody>
</table>

Throughout the experiment (i.e., in all phases), water was completely changed inside the pipes three times per week, using a “dump and fill” protocol. Lead release from the rigs was therefore evaluated under “worst case” stagnation conditions, which are known to promote galvanic corrosion problems in practice (e.g., Dudi, 2004). These worst-case stagnation conditions represent extremes in water usage that may be encountered at schools/ municipal-type buildings during weekends/breaks and at homes during prolonged absences. The contribution of galvanic connection (or lack thereof) to lead release was assessed by measuring total lead concentration in water and galvanic current magnitude:

- One composite water sample was collected at the end of each week from each pipe, by pouring the three water samples of that week into the same container. Total lead was quantified in these unfiltered composite water samples using a Thermo Electron X-Series inductively coupled plasma mass spectrometer (ICP-MS) per Standard Method 3125-B (APHA, AWWA, and WEF, 1998). Water samples and instrument calibration standards were prepared in a matrix of 2% nitric acid by volume.
- Galvanic current between the dissimilar metals (for Phases 1, 2 and 4 when the external wires were connected) was measured with a digital multi-meter purchased at a retail electronics store (internal resistance determined at 100 Ω). The current flowing in each rig was measured by connecting the multi-meter in-line for 15 seconds after disconnecting the wire between the two metals.

RESULTS AND DISCUSSION

Detailed results are presented for new Pb pipe, followed by a summary of results for other lead pipes (aged Pb pipe and Pb (IV) pipe).

Temporal trends on lead release. After an initial stabilization period of three weeks, lead release under exposure to low CSMR water (weeks 4-11) became relatively low for each rig (Figure 4-5). The rig with pure lead pipe (i.e. 0% Cu) released 120-320 ppb of lead during that
time frame. As expected, the rig with no lead pipe (i.e. 100% Cu) released no lead, providing assurance that copper pipes would not release any lead and that sample contamination was not occurring. In rigs with copper and lead galvanic connections, lead leaching was always increased when compared to pure lead pipe during exposure to low CSMR water (Figure 4-5).

When the water was switched to high CSMR in Phase 2 (weeks 12-25), lead release from the galvanic connections dramatically increased, and remained elevated throughout that time period (Figure 4-5). High CSMR water also increased lead release from pure lead pipe (i.e. 0% Cu pipe) (Figure 4-5). During weeks 26-31 (Phase 3), when galvanic corrosion was inactivated by disconnecting the wires, lead levels plummeted even though the high CSMR water was still fed to the rigs (Figure 4-5). When galvanic corrosion reversed its action, during weeks 32-37 (Phase 4), lead release to the water immediately rose, to levels previously experienced during Phase 2 (Figure 4-5). This demonstrates the direct role of galvanic corrosion in sustaining high lead concentrations in water, from the lead-copper rigs of the study.

![Figure 4-5: Lead release versus time for connections of lead pipe to copper pipe.](image)

**Synthesis: effect of galvanic corrosion and CSMR on lead release.** With the exception of weeks 1-3, when lead release had not yet stabilized, results were synthesized by averaging the lead data for each experimental phase. All simulated partial replacements (17%, 50%, 67% and 83% of Pb replaced by Cu) released more lead to the water than did the rig consisting of pure lead (i.e. 0% Cu) (Figure 4-6). This was true for all three galvanic experimental phases, and results were statistically significant at the 95% confidence level (Figure 4-6, error bars plotted). Specifically, lead release from simulated partial replacements increased by 4-27 times (depending on the extent of replacement) during Phase 1, by 2-7 times during Phase 2, and by 7-20 times during Phase 4, compared to a full length of lead pipe alone (Figure 4-6). Therefore for
this set of pipes, not only did the galvanic effect “annul any benefit from reducing the length of lead pipe” (Chambers and Hitchmough, 1992) but it further “exacerbated lead release” (Britton and Richards, 1981).

Figure 4-6: Lead release versus extent of lead pipe replacement by copper. Data were averaged for each experimental phase. The error bars denote 95% confidence intervals.

High CSMR water released much more lead to the water compared to low CSMR, when the wires were connected. In fact, when comparing high CSMR water (Phase 2) to low CSMR water (Phase 1), lead release increased by 5 times (in the case of 17% Cu) to as much as 12 times (in the case of 83% Cu) (Figure 4-6). However, it is still important to note that the adverse impacts of galvanic corrosion on lead leaching were still very significant even in the water with low CSMR. Disconnecting the wires under high CSMR water (Phase 3) decreased lead release by 4-6 times in all Cu:Pb galvanic couples, while reconnecting the wires under high CSMR water (Phase 4) increased lead release by 4.5-8 times (Figure 4-6).

During Phase 3, lead release from all the de-activated galvanic rigs was not statistically higher, when compared to lead release from pure lead pipe (Figure 4-6). Lead release from those rigs was not lower either, as would be expected due to their smaller lead surface area, compared to the pure lead pipe. This could be because other additional corrosion mechanisms were present in the Pb:Cu rigs during Phase 3 compared to the pure Pb pipe, even in the absence of galvanic connection to copper, such as copper deposition corrosion onto the lead pipe. Alternatively, it is possible that the rigs had not yet “recovered” from the previous Phase (Phase 2) of galvanic connection.
The extent of lead contamination was initially somewhat dependent on the extent of partial replacement. For instance, when the rigs were exposed to low CSMR water with connected wires (Phase 1), the rig consisting of 17% Cu (with the remaining 83% being lead) released the highest lead. The 50% Cu and 67% Cu were not statistically different, and the 83% Cu rig released the lowest lead amongst the conditions representing partial pipe replacements. For the worst-case experimental condition of high CSMR and connected wires (Phase 2), the rig consisting of 17% Cu released the highest lead, followed by the 50% Cu, 67% Cu, and 83% Cu rigs (Figure 4-6). In other words, less copper relative to lead worsened lead corrosion at the initial experimental phases. When the rigs were subsequently exposed to high CSMR water with disconnected wires (Phase 3), there were not statistically significant differences in lead release between the 17%, 50%, 67%, and 83% Cu rigs (Figure 4-6). Similarly, during Phase 4 of exposure to high CSMR water with reconnected wires, there were not statistically significant differences in lead release between the 17%, 50%, and 67% Cu rigs (Figure 4-6).

**Deviation of experimental lead leaching results from conventional theory.** The experimental results provide a scientific framework for evaluating extremes in behavior that might be encountered in practice. At one extreme, it is hypothetically possible that in some waters there is negligible galvanic corrosion between Pb and Cu pipe. In such cases, lead release from partial replacement of lead pipe with copper will follow a linear model (Figure 4-7). If 100% of the lead pipe is replaced, there will be no lead in water. If 50% of the lead pipe is replaced with copper, there will be 50% less lead in the water volume held in the service line, compared to before partial replacement. In such a case, partial replacement will be beneficial after short-term problems with pipe cutting dissipate (Boyd et al, 2004), and the extent of the benefit will be proportional to the extent of replacement of the lead pipe with copper pipe. Laboratory results obtained herein deviate significantly from the linear model due to galvanic corrosion. For the low CSMR water (Phase 1), partial replacements released 18-38 times more lead to the water, than what the theoretical linear model suggests (Figure 4-7, top). For the high CSMR water (Phase 2), partial replacements released 8-14 times more lead compared to the relevant linear theoretical model (Figure 4-7, bottom).
Mechanistic insights via galvanic current measurements. Measurement of the galvanic current between the lead and copper portion of the rigs can provide mechanistic insights on the observed lead leaching trends. The galvanic current is a direct measure of the instantaneous rate of galvanic corrosion between the lead pipe and the copper pipe. A higher magnitude of current indicates a higher rate of galvanic corrosion. Galvanic current measurements were taken during Phase 1, Phase 2, and Phase 4 of the experiment, when galvanic corrosion was activated. Since the wires were disconnected during Phase 3, thereby blocking electron flow between the two dissimilar metals, no current measurement was obtained. Lead always behaved as the anode and copper as the cathode of each galvanic cell in all measurements undertaken herein.

Figure 4-7: Deviation of experimental lead release results from a linear theoretical model, which assumes that lead dissolution is proportional to the length of lead pipe. Error bars on experimental data points denote 95% confidence intervals.
Chapter 4. Galvanic Corrosion after Simulated Small-Scale Partial Lead Service Line Replacements

Higher currents were measured when high CSMR water was fed to the rigs (Phase 2), compared to when low CSMR water was fed to the rigs (Phase 1) (Figure 4-8). This general trend is consistent with the trend in lead leaching (Figures 4-5 and 4-6). Under the high CSMR water condition (Phase 2), the highest current of 87 µA was measured for the 17% Cu rig, followed by successively lower current readings for the 50% Cu, 67% Cu, and 83% Cu rigs (Figure 4-8). The ranking of the rigs with respect to the magnitude of the measured galvanic currents is consistent with that based on the lead leaching results. For instance, the 17% Cu rig had the highest measured current (Figure 4-8), and it also resulted in the highest lead-in-water concentrations (Figure 4-6). Under the low CSMR condition (Phase 2), the highest current of 52 µA was measured for the 17% Cu rig, followed by the 50% Cu, 67%, and 83% Cu rig (with the lowest current of 30 µA). Under the high CSMR water and reconnected wires (Phase 4), the highest current of 85 µA was measured for the 17% Cu and the 50% Cu rigs, followed by the 67%, and 83% Cu rig (with the lowest current of 57 µA).

![Figure 4-8: Galvanic current versus extent of lead pipe replacement by copper. Data were averaged for each experimental phase. The error bars denote 95% confidence intervals.](image)

Unlike Phase 2, the ranking in terms of galvanic current magnitude during Phase 1 and Phase 4 (Figure 4-8) did not always agree with the ranking based on the respective lead leaching results (Figure 4-6). Obviously, the galvanic current between lead and copper is a measure of galvanic corrosion only, and does not account for lead release due to normal lead dissolution or deposition corrosion. Moreover, the lead corroded due to galvanic currents could also form lead-rusts on the pipe surface, and only partly be released into the water (see Figure 4-2, B, and 4-2, C). Pearson’s correlation coefficient $R^2$ between galvanic current magnitude and lead-in-water
concentration was 0.41 under exposure to low CSMR water (Phase 1), and 0.71 under exposure to high CSMR water (Phase 2) (Figure 4-9).

**Effect of alkalinity on lead release and galvanic current.** Increasing the alkalinity was not able to reduce either lead leaching or galvanic current magnitude, for any of the partial replacements. Specifically, increasing the alkalinity from 15 to 50 and then to 100 mg/L as CaCO₃ resulted in statistically similar lead-in-water levels for all rigs (Figure 4-10), and galvanic current actually increased by up to 20% (Figure 4-11).

Throughout the first four phases of this study, the alkalinity of the water entering the rigs was maintained at 15 mg/L as CaCO₃ (see Table 4-2). Waters with this low alkalinity level have been described as alkalinity deficient in terms of lead corrosion control, based on practical experiences (Edwards et al, 1999). Edwards et al (1999) identified alkalinity of 100 mg/L as CaCO₃ as an approximate upper bound to obtaining substantial improvements in lead release for waters with a pH below 8.5, based on utility monitoring data (pH was maintained at 8.0 throughout this study, see Table 4-2). It is possible that the detriments of simultaneously increasing the conductivity for the galvanic corrosion cells (see Table 4-2), offset the benefits of higher dissolved inorganic carbon in reducing soluble lead (Schock et al, 1996). But on the other hand, increasing the alkalinity did not decrease lead release from pure lead pipe either (Figure 4-10).

In this experiment, particulate lead constituted the majority of total lead in collected water samples (70%-99% of total lead was particulate - results not presented herein). The ineffectiveness of alkalinity in reducing total lead leaching can therefore be explained by the abundance of particulate lead in the absence of significant soluble lead.
Figure 4-10: Effect of alkalinity on lead release, under exposure to high CSMR water. Phase 4 (High CSMR Water, Wires Re-connected) is compared to subsequent tests of increased alkalinity. The error bars denote 95% confidence intervals.

Figure 4-11: Effect of alkalinity on galvanic current magnitude, under exposure to high CSMR water. Phase 4 (High CSMR Water, Wires Re-connected) is compared to subsequent tests of increased alkalinity. The error bars denote 95% confidence intervals.
**Area of galvanic influence and potential practical implications.** One set of rigs (aged Pb pipe B) was constructed by connecting in series many short Pb pipes together, in order to add up to the desired Pb length. For example, the 17% Cu rig consisted of one 0.5ft Cu section, connected to five sequential 0.5 ft Pb sections (Figure 4-12, bottom). This setup allowed measuring the galvanic current between the Cu and each section of Pb pipe. The total current between the Cu and Pb pipe was the sum of the individual currents between the Cu and each Pb section.

Throughout phases of the experiment when the wires were connected between lead and copper, the anodic galvanic current was concentrated in the Pb segment that was closest to the galvanic junction. For example, during Phase 1 of low CSMR Water, the current flowing between the Cu and the first Pb segment constituted on average 90% of the total current between Pb and Cu in the rig (Figure 4-12, top). The second Pb segment received 7% of the total current. The remaining three Pb segments, those furthest away from the connection to Cu pipe, accounted for only 3% of the total current (Figure 4-12, top). Similarly, during Phase 2 of high CSMR water, the first Pb segment received 95% of the total galvanic current, the second Pb segment by 3% of the galvanic current, and the remaining three Pb segments by 2% of the current (Figure 4-12, top). Since galvanic current is a direct measure of galvanic corrosion, these data suggest that the Pb segment that is closest to the galvanic junction is by far the most impacted (i.e., corroded) due to galvanic connection to copper, consistent with experimental results of Reiber and Dufresne (2006) and mineralogical and visual observations of DeSantis et al (2009).

Translating galvanic effects to lead release and human exposure illustrates serious concern, regardless of area of galvanic influence. DeSantis et al (2009) identified some failures at joints (i.e., lead pipe wall completely eaten away), due to the depth of galvanic corrosion in the pipe wall. Simple calculations suggest that even if only 1 inch length of lead pipe is half-eaten away at the pipe wall due to galvanic corrosion, the released lead mass equals 25 grams (calculation based on lead density of 11.3 g/cm³, lead pipe internal diameter of ¾ inches, and lead pipe external diameter of 1 inch). This mass of lead is sufficient to contaminate every drop of water used by a family of three for 4 years, to a level of lead above the LCR action limit of 15 ppb (calculation based on 300 gal/day, or else 1135 L/day water usage for the whole family). Thus, the limited area of attack does not translate to limited impacts on public health, consistent with the extremely high levels of lead in water measured in this experiment.

The above calculation assumes that all the lead released due to galvanic corrosion would contaminate all water equally. Cases of semi-random particle lead release, observed mostly at the latter stages of this experimental study, highlight serious potential for exposure to much higher lead doses. Specifically, by the end of this 13-month study, the Pb pipe area adjacent to the junction had accumulated a thick lead-rust galvanic corrosion layer (Figure 4-12, A), whereas no such thick layer was visible elsewhere on the lead surface (Figure 4-12, B). Thick corrosion layers were visually observed at the joint in all Pb:Cu rigs tested, and had a width of approximately 1 inch. Toward the end of the experiment, white pieces of lead scale were occasionally detaching from the pipe galvanic corrosion layer into the collected water samples (Figure 4-12, C).

After standard acid preservation of one such 260 mL water sample (collected on week 46 from the 50% Cu rig), the lead concentration was quantified at 50,000 ppb. The resulting lead dose of 13,000 µg from hypothetical consumption of just one glass of contaminated water, is equivalent to that obtained from ingesting 14 lead paint chips (Figure 4-12, D) assuming they have a 2.6% lead content by weight (equivalent lead dose from paint chips was calculated based
on their measured weight on an analytical balance, and their assumed lead content by weight). Clearly, such release poses an acute health risk even from fairly rare exposure events, and also highlights obvious difficulties in detecting semi-random particulate detachment from the joint during conventional field sampling.

Summary of experimental results for other types of lead pipe tested. Overall, similar trends were observed for the other three types of lead pipe examined (aged Pb pipe A, aged Pb pipe B, Pb (IV) pipe). In summary, partial replacements under both low and high CSMR water increased lead release compared to a full length of lead pipe alone, with only two exceptions (67% Cu and 83% Cu rigs from aged Pb pipe A under low CSMR water) (Table 4-3). Lead release and galvanic current magnitude were higher under high CSMR water compared to low CSMR water, for these three types of lead pipe (Table 4-3). Disconnecting the wires between lead and copper decreased lead release, while reconnecting the wires increased lead release (Table 4-3). All galvanic rigs deviated from the linear theoretical model due to galvanic corrosion, and increasing the alkalinity was not able to reduce lead leaching or galvanic current magnitude (Table 4-3).
Considering a) the increase in lead release from partial replacements compared to pure lead pipe in the vast majority of cases, b) the direct impact of disconnecting/reconnecting the wires to lead release into the water, and c) deviation from the linear theoretical model, galvanic corrosion made lead leaching worse under the experimental conditions of this study. However, the correlation between galvanic current and lead release was generally low, especially for aged lead pipes (Table 4-3). Aside from reasons partly explained in a previous section, strong correlations cannot be obtained when semi-random release of particles is occurring. This was especially true for aged lead pipes, where particulate lead release was more erratic. Overall, the three earlier considerations indicate that galvanic corrosion made lead leaching worse under the experimental conditions of this study, and therefore lack of a statistical correlation does not imply lack of practical association.

In an extreme case for pipes with Pb (IV) scale, there was no correlation between galvanic current and lead in water ($R^2 = 0.04$) (Table 4-3). Since the pipes were exposed to chloraminated water, thereby causing Pb (IV) to be reduced to Pb (II) and be released into the water (Xie et al., 2010; Lin and Valentine, 2008; Switzer et al., 2006), white flakes were consistently observed in the water samples collected from the Pb (IV) galvanic pipes. Release in this set of rigs was therefore further complicated by destabilization of the internal Pb (IV) corrosion layer, consistent with theory and prior practical experience (Xie et al., 2010; Lin and Valentine, 2008; Switzer et al., 2006; Lytle and Schock, 2005; Schock and Giani, 2004; Renner, 2004). Even so, for this set of pipes a) lead release from simulated partial replacements increased by 10-20% compared to pure lead pipe, b) disconnecting the wires to copper decreased lead release by 5-8 times, c) reconnecting the wires to copper increased lead release by 3-4 times, and d) galvanic connections to copper increased lead release by 1.5-2.0 times compared to the linear theoretical model (Table 4-3), affirming that galvanic corrosion was still significant, despite the lack of correlation.

LIMITATIONS AND FUTURE RESEARCH

The work presented herein is an important first step in understanding the implications of galvanic corrosion to lead release during partial lead service line replacement. Due to the very long stagnation times and relatively short lengths of pipe used herein, this work can be considered "worst case," and illustrates a significant long-term concern that has been largely overlooked. More research is needed to quantify the relevant contribution of galvanic corrosion to lead release from lead service lines, compared to other mechanisms such as normal dissolution, deposition corrosion, particle detachment, and lead retention in pipe scale. Future work should examine galvanic corrosion under intermittent flow patterns, which are more representative of typical water usage at homes. The effect of couplings between lead and copper pipe on galvanic corrosion (e.g., brass corporation valve or brass compression fitting, which are typically used in the field) should also be evaluated. The pipes used in this study included aged pipes with a somewhat passivated internal surface, but may not reflect the behavior of pipes with decades of accumulated scale. It would be prudent to do some testing with lead service lines harvested from the field after decades of use, or conduct additional testing of partially replaced lead pipes in the field. Finally, the effects of other water qualities on galvanic corrosion of lead (e.g., corrosion inhibitor such as phosphate added to the water) should be evaluated.
Table 4-3: Summary of experimental results for all types of lead pipe examined in this study.
Ranges represent minimum and maximum results from four partial replacements of the lead pipes (with 17% Cu, 50% Cu, 67% Cu and 83% Cu).

<table>
<thead>
<tr>
<th>Lead Pipe Tested</th>
<th>Effect of simulated partial replacement on Pb release, compared to low CSMR water</th>
<th>Effect of high CSMR Water compared to Pb release, under low CSMR water</th>
<th>Effect of galvanic corrosion on Pb release, compared to linear theoretical model</th>
<th>Effect of galvanic corrosion on Pb release, compared to high CSMR water</th>
<th>Correlation R² between current and Pb release</th>
<th>Effect of increasing alkalinity, under high CSMR water</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Pb (IV) pipe</strong></td>
<td><em>Results presented in detail in the Results and Discussion section</em></td>
<td><em>Results presented in detail in the Results and Discussion section</em></td>
<td><em>Results presented in detail in the Results and Discussion section</em></td>
<td><em>Results presented in detail in the Results and Discussion section</em></td>
<td><em>Results presented in detail in the Results and Discussion section</em></td>
<td><em>Results presented in detail in the Results and Discussion section</em></td>
</tr>
</tbody>
</table>
CONCLUSIONS

Under controlled experiments of simulated small-scale partial lead service line replacements with copper that lasted for 13 months:

- Galvanic connections between copper pipe and lead pipe (either new or aged) increased lead release compared to a full length of lead pipe alone, under stagnant water conditions.
- Removal of the galvanic connection between copper pipe and lead pipe decreased lead release by 2-8 times (depending on the type of lead pipe), under a high CSMR water condition (i.e., CSMR of 16). Subsequently restarting the galvanic connection increased lead release by 2-8 times (depending on the type of lead pipe), to levels experienced prior to galvanic connection removal.
- Water with a high CSMR of 16 released 3-12 times more lead to the water than did low CSMR water of 0.2, for both new and aged lead pipes connected to copper.
- Due to galvanic (and deposition) corrosion, lead leaching from simulated partial replacements deviated from the linear theoretical model by 1.5-38 times in low CSMR water and by 1.5-75 times in High CSMR water.
- High sustained galvanic currents between copper and lead pipe (up to 87 µA for new lead pipe) were measured when the CSMR was high, resulting in galvanic corrosion of the lead. When the CSMR was low, galvanic currents were lower (up to 52 µA for new lead pipe), consistent with corresponding lower lead leaching results.
- Perfect correlations between galvanic current and lead release were neither expected nor obtained. While galvanic current cannot be relied upon to predict lead-in-water levels, it is a direct measure of galvanic corrosion of lead, when connected to copper pipe.
- Increasing the alkalinity from 15 mg/L to 50 and then to 100 mg/L as CaCO₃ was not able to alleviate galvanic lead corrosion, in terms of lead release into the water and galvanic current magnitude.
- Based on galvanic current measurements, the area of lead pipe adjacent to the copper joint (<0.5 ft) was most affected by galvanic corrosion. The lead pipe sections that were furthest away from the copper junction (>0.5 ft) were the least affected by the galvanic connection to the copper.
- Visual observations at the end of the experiment further defined the area of severe galvanic attack to about 1 inch of width of the lead pipe, adjacent to the junction. A distinct thick lead corrosion scale accumulated in that area, constituting a large reservoir for semi-random particulate lead detachment into the water.

Overall, these findings indicate that under worst case stagnation conditions, galvanic corrosion was a dominant mechanism of lead release to potable water, even after more than one year following simulated small-scale partial lead service line replacements. More research is needed in order to test intermittent flow patterns, the effect of brass connections between lead and copper pipes, lead pipes harvested from the field, and other water chemistries.

ACKNOWLEDGEMENTS

Part of this work was performed with financial support of the Water Research Foundation, under project continuation reserve fund 4088. The authors would like to thank project manager Traci Case, and project advisory committee members Christopher Hill, France Lemieux, Joseph
Marcinko, and Anne Spiesman. The latter phases of experimentation were supported by the National Science Foundation (NSF) under grant CBET-0933246. Opinions and findings expressed herein are those of the authors and do not necessarily reflect the views of the Water Research Foundation or the NSF. The authors would also like to thank the anonymous reviewers for their valuable comments.

REFERENCES


Reiber, S.; and Dufresne, L., 2006. Effects of External Currents and Dissimilar metal contact on Corrosion of Lead from Lead Service Lines. Final Report to USEPA region III.


CHAPTER 5. LEAD (PB) QUANTIFICATION IN POTABLE WATER SAMPLES: IMPLICATIONS FOR REGULATORY COMPLIANCE AND ASSESSMENT OF HUMAN EXPOSURE

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ABSTRACT. Assessing the health risk from lead (Pb) in potable water requires accurate quantification of the lead concentration. Under worst-case scenarios of highly contaminated water samples, representative of public health concerns, up to 71-98% of the total lead was not quantified if water samples were not mixed thoroughly after standard preservation (i.e., after addition of 0.15% v/v HNO₃). Thorough mixing after standard preservation greatly reduced but did not sufficiently minimize errors (i.e., 35-81% of the total lead in some samples was still un-quantified). Transfer of samples from one bottle to another also created high errors (40-100% of the total lead was un-quantified in transferred samples). Although the Environmental Protection Agency’s (EPA) standard protocol avoids most of these errors, certain methods considered EPA-equivalent allow these errors for regulatory compliance sampling. Moreover, routine monitoring for assessment of human exposure has no standardized protocols for water sample handling and pre-treatment. Overall, while there is no reason to believe that sample handling and pre-treatment dramatically skew regulatory compliance with the lead action limit, slight variations from one approved protocol to another may cause lead-in-water health risks to be dramatically underestimated, especially for unusual situations of "worst-case" individual exposure to highly contaminated water.

KEYWORDS. worst-case exposure, sample pre-treatment, standard acid preservation, mixing, transfer
INTRODUCTION

After implementation of modern corrosion control and an Action Level (AL) of 15 parts per billion (ppb) through the federally mandated EPA Lead and Copper Rule (US EPA, 1991), it was believed that lead in water had been addressed as a significant human health threat in the US (CDC, 2002). Assessments of a massive lead contamination incident in Washington, DC tap water during 2001-2004 initially created some speculation that lead-in-water levels much higher than the AL would be necessary to elevate lead in children’s blood over the 10 µg/dL level of concern (CDC 2004; Guidotti et al. 2007). However, later and more detailed analysis of the Washington, DC experience revealed strong correlations between incidence of elevated blood lead (EBL ≥10 µg/dL) and the higher lead in water (Edwards et al., 2009), or presence of lead service lines (Brown et al., 2011). Brown et al. (2011) also determined that the risk from lead service lines (and presumably lead in water) extended to time periods when Washington DC was meeting the 15 ppb lead AL. This reinforced concerns (Levin et al. 2008, Edwards et al. 2009) that there is still a significant risk of EBL from water sources.

A recent literature review of other epidemiological studies in the US, the UK, Germany, France, and Canada reaffirmed that lead in water can be a dominant, or at least a significant contributor, to lead in blood, especially for infants and children (Triantafyllidou and Edwards, 2011a). For example, a study by Galke et al. (2006) determined that the more glasses of tap water consumed by children, the greater the risk of elevated blood lead (EBL ≥10 µg/dL) and the higher lead in water (Edwards et al., 2009), or presence of lead service lines (Brown et al., 2011). Brown et al. (2011) also determined that the risk from lead service lines (and presumably lead in water) extended to time periods when Washington DC was meeting the 15 ppb lead AL. This reinforced concerns (Levin et al. 2008, Edwards et al. 2009) that there is still a significant risk of EBL from water sources.

There is no current accepted protocol for collection and preparation/pre-treatment of tap water samples for lead monitoring that is universally followed (Cartier et al. 2011). The interpretation of results when using different protocols for monitoring, coupled with the inherent variability of lead in water, remain big challenges (Schock and Lemieux 2010; Schock 1990). In general, three categories of protocols can be identified in the US, including: 1) EPA Method 200.8 and 200.9 as supplemented by details in the Federal Register (henceforth termed EPA Standard Protocol), 2) other published methods explicitly deemed acceptable by the EPA, including Palintest Ltd. Method 1001 and Standard Method 3113 B, as supplemented by the Federal Register, and 3) methods developed on an “ad hoc” basis for targeted monitoring or for environmental assessments in homes of at-risk children. Only protocols from categories 1 and 2 can be used for compliance sampling under the EPA Lead and Copper Rule, whereas any method can be followed for environmental assessments or targeted monitoring. For each protocol, there are three types of potential deficiencies which can “miss” lead in water, either alone or in combination:

1. **Sampling steps or instructions that fail to introduce the lead-in-water hazards into the sampling bottle.** Lead particulates present in water during normal household use, can be missed during sampling if low water flow rates are used to fill bottles (Triantafyllidou and Edwards 2011a), if plumbing lines have been flushed extensively before sampling (as sometimes instructed by utilities even though EPA instructions do not mention flushing), or if faucet
aerators have been cleaned/removed (as is sometimes done even though EPA has officially discouraged the practice) (US EPA 2006; Triantafyllidou and Edwards 2011a). Likewise, only cold water samples are generally collected and analyzed for compliance with the EPA LCR or during environmental monitoring, even though it is believed that hot water sometimes contains much higher lead and could contribute to EBL (Triantafyllidou and Edwards 2011a).

II. Even if a water sample with high lead is collected, specific steps of sample preparation/pre-treatment may fail to mobilize the lead into an aliquot that will then be introduced to an appropriate lead detector. The concentration of acid preservative added to the sample, the extent of sample mixing after preservation, and sample holding time after preservation and prior to analysis are all specified by the EPA, because they can affect lead quantification. The only demonstrated limitation in the EPA Standard Protocol is the effectiveness of acid preservative, which created errors of up to 80% if certain types of lead particulates were present, because the acid was not sufficient to dissolve all the lead and left particulates at the bottom or on the walls of sample containers (Triantafyllidou and Edwards 2007).

III. Even if a water sample with high lead is collected and pre-treated appropriately before it is introduced to an analytical detector, lead is in a form that cannot always be quantified by the specific detector. For example, this could be a serious concern for the newly approved analytical method of Differential Pulse Anodic Stripping Voltammetry (DPASV), which measures dissolved lead ions (i.e., Pb^{2+}) but was shown to significantly underestimate particulate lead (i.e., Pb^{4+}) and colloidal lead (Cartier et al. 2009).

In relation to Type II errors, water sample preparation for lead quantification under the federally mandated Lead and Copper Rule (LCR) (Figure 5-1) typically involves: 1) Sample shipment to a certified lab after collection at the tap, 2) Acidification of sample to pH < 2.0 typically by addition of 0.15% v/v concentrated nitric acid (HNO₃) which is termed “standard preservation” in this work, 3) Minimum holding time of 16 hours, 4) Turbidity measurement in aliquot, and additional heated acid digestion of an aliquot only if the turbidity is greater than 1 ntu (nephelometric turbidity unit), which is termed “aliquot digestion” henceforth in this work, and 5) Analysis using an approved analytical method (either inductively coupled plasma mass spectrometry/ICP-MS or flame atomic absorption/FAA). The EPA has also approved other analytical instruments which may use different sample pre-treatment methods for LCR compliance monitoring (Table 5-1).
Chapter 5. Lead (Pb) Quantification in Potable Water Samples

Figure 5-1: EPA Standard Protocol (i.e., sampling and analytical procedures) for total lead in drinking water samples, as described in EPA Methods 200.8 (ICP-MS) and 200.9 (STGFAA). A slightly different digestion (not shown) is recommended if the sample contains >1% undissolved solids. Potential problems are emphasized in blue. This figure is a revised version of that published by Triantafyllidou et al. (2007) in Journal AWWA 99:6.
Chapter 5. Lead (Pb) Quantification in Potable Water Samples

Table 5-1: EPA-approved analytical methods for LCR compliance monitoring of total lead in drinking water samples as listed in the Federal Register (US EPA, 2009), and specific sample pre-treatment requirements for each method (US EPA, 1994; 2003; Palintest Ltd, 1999; APHA, 1998).

<table>
<thead>
<tr>
<th>Method Approved for Lead Compliance Monitoring under the Lead and Copper Rule*</th>
<th>Instrumentation</th>
<th>Sample Pre-Treatment Requirement</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Standard Preservation</td>
</tr>
<tr>
<td>EPA 200.8 Rev 5.4 Inductively Coupled Plasma - Mass Spectrometry (ICP-MS)</td>
<td>Yes</td>
<td>No unless turbidity &gt; 1 ntu</td>
</tr>
<tr>
<td>EPA 200.9 Rev 2.2 Stabilized Temperature Graphite Furnace Atomic Absorption (STGFAA)</td>
<td>Yes</td>
<td>No unless turbidity &gt; 1 ntu</td>
</tr>
<tr>
<td>Palintest Ltd. or Hach Co. Method 1001 Differential Pulse Anodic Stripping Voltammetry (DPASV)</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>Standard Method 3113 B Electrothermal Atomic Absorption</td>
<td>Yes</td>
<td>No unless turbidity &gt; 1 ntu</td>
</tr>
<tr>
<td>EPA 200.5 Rev 4.2 Axially Viewed Inductively Coupled Plasma - Atomic Emission Spectrometry (AVICP-AES)</td>
<td>Yes</td>
<td>Yes</td>
</tr>
</tbody>
</table>

*Either an original EPA method or a third party method approved by EPA.

If the lead in water is dissolved, standard preservation adequately prohibits lead from adhering to the sampling container at any step of the process (Figure 5-1) so that it will be fully quantified. In addition to extensive testing by the authors which showed no problems in detecting dissolved lead using the standard preservation, Deshommes et al. (2010) found no difference in the measured lead concentration of water samples, between the standard preservation and a more rigorous heated digestion (1% v/v HNO₃ + 0.5% v/v HCl, 85 °C) in cases where lead was predominantly dissolved and did not exceed 32 ppb. Similarly, Lytle et al. (1993) found that standard preservation recovered 100% of a very fine lead solder powder which had been deliberately introduced to water samples.

However, if larger lead particles are present that can settle from solution or adhere to the walls of containers, such as those implicated in childhood lead poisoning from water and thought to be present after partial lead service line replacement (Triantafyllidou and Edwards 2011a) or presumably present in actual samples collected by Lytle et al. (1993) for which lead was noted to rise with increased holding time, several problems may arise in quantification and detection (Figure 5-1). These potential problems depend upon the ability of standard preservation to render all the lead soluble (Figure 5-1). It is hypothesized that such problems may be exacerbated when coupled with lack of mixing of samples after standard preservation, the need for which is not explicitly mentioned in some EPA-approved protocols (Table 5-2). This work aims to examine the role of specific sample preparation steps in the quantification of lead for worst-case situations; specifically, those in which highly contaminated water could pose a public health risk. Implications for regulatory compliance monitoring are considered as part of that evaluation.
Table 5-2: Comparison of two EPA-approved sample pre-treatment protocols for quantification of metals in drinking water, with emphasis on total lead.

Interpretation of the different methods by the authors revealed various potential issues. The focus of this study is on the extent of sample acidification during standard preservation, coupled with sample mixing after standard preservation. These two issues are emphasized in bold.

<table>
<thead>
<tr>
<th>Issue</th>
<th>EPA Pre-Treatment Protocol (i.e., EPA Standard Protocol)</th>
<th>EPA-Approved Pre-Treatment Protocol</th>
<th>Potential Problem/Clarification</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Extent of sample acidification with HNO₃ under standard preservation</strong></td>
<td>Acidify sample to pH&lt;2.0 (typically by adding 0.15% v/v concentrated HNO₃ - termed standard preservation in this work)</td>
<td>Acidify sample to pH&lt;2.0 (typically by adding 1.5 mL concentrated HNO₃/L sample - i.e., standard preservation)</td>
<td>“Weak” acid might not dissolve all the lead if particulates are present, resulting in underestimation of total lead concentration in the sample</td>
</tr>
<tr>
<td>Time of sample standard preservation relative to sample collection</td>
<td>Recommended within 2 weeks - Immediately after sampling</td>
<td></td>
<td>• Inconsistency between the two methods</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>• The Code of Federal Register(CFR), which has precedence, mandates acidification within 2 weeks under the LCR</td>
</tr>
<tr>
<td>Mixing of sample after standard preservation</td>
<td>Yes</td>
<td>Not stated</td>
<td>Lack of mixing might reduce recovery of lead, resulting in underestimation of total lead concentration in the sample</td>
</tr>
<tr>
<td>Sample holding time after standard preservation and prior to lead analysis</td>
<td>Minimum of 16 h</td>
<td>Not specified</td>
<td>The CFR mandates a minimum of 16 hours under the LCR</td>
</tr>
<tr>
<td>Digestion requirement</td>
<td>Only if original sample turbidity &gt;1ntu** at the time of analysis</td>
<td>Only if original sample turbidity &gt;1ntu at the time of analysis</td>
<td>Turbidity measurement in aliquot might be less than 1 ntu, even though lead particulates may be present</td>
</tr>
<tr>
<td>Aliquot volume undergoing digestion if sample turbidity &gt;1 ntu</td>
<td>100 mL aliquot, poured after mixing the original 1 L sample volume</td>
<td>10 mL aliquot, after mixing the original 1 L sample volume*</td>
<td>• Inconsistency between the two methods</td>
</tr>
<tr>
<td></td>
<td>Add 2 mL (1:1) HNO₃ and 1 mL (1:1) HCl, Heat to 85 °C until aliquot volume reduced to 20 mL (do not boil)</td>
<td>Add 0.5 mL HNO₃, Heat to 105 °C for a minimum of 2 h (do not boil), add more acid if necessary</td>
<td>• Lead particles that rapidly settle to the bottom or attached to sample bottle may not be poured off into the aliquot tube</td>
</tr>
<tr>
<td>Aliquot digestion procedure if sample turbidity &gt;1 ntu</td>
<td></td>
<td>Inconsistency between the two methods</td>
<td></td>
</tr>
</tbody>
</table>

*for trace level concentrations (<=0.1mg/L). For high level concentrations (>0.1 mg/L) a volume of 100 mL is treated.

**DPASV requires digestion regardless of turbidity (same digestion protocol as described herein), and subsequent neutralization before analysis.

** AVICP-AES requires digestion of 50 mL aliquot regardless of turbidity (different digestion protocol than described herein)
MATERIALS AND METHODS

Water Sample Sources. Potable water samples exposed to lead pipe, leaded brass, or leaded solder plumbing were collected from four bench-scale experiments (Zhang et al. 2009; Hu et al. 2010; Triantafyllidou and Edwards 2011b; Nguyen et al. 2010) to examine specific limitations of various sample preparation procedures (Table 5-3). Collectively, these four sets of water samples represented worst-case extremes with high particulate lead from all three major sources of lead contamination (i.e., lead pipe, lead solder, leaded brass) in potable water, and were used in four distinct tests in this work to evaluate specific sample preparation issues (Table 5-3).

Effect of sample mixing after standard preservation (Tests 1 and 2). For tests 1 and 2, unfiltered water samples were analyzed for total lead after initial acidification with 0.15% v/v concentrated nitric acid (HNO$_3$) for at least 16 hours at room temperature (~20 ºC), following instructions of standard preservation but without sample mixing (Table 5-3). For comparison, the samples were subsequently mixed and re-analyzed, as per the mixing specification of the EPA pre-treatment protocol (Table 5-3). As a final step, the samples were exposed to a very rigorous in-the-bottle digestion (addition of 2% v/v HNO$_3$ at room temperature) to potentially recover a greater fraction of the lead compared to the standard preservation (Table 5-3).

Effect of sample transfer between sample bottles before analysis (Tests 3 and 4). For tests 3 and 4, each unfiltered water sample was first divided into two portions after mixing. One portion remained in the original sampling container, while a second portion was poured into a new container. Each portion received either the same preservation treatment (Test 3) or different treatment in terms of acidification extent or temperature (Test 4), for the same amount of time prior to lead analysis (Table 5-3). Unless otherwise stated (e.g., Tests 1 and 2, see Table 5-3) the samples from Tests 3 and 4 were always vigorously mixed following addition of HNO$_3$.

Analytical Methods. Total lead after each step of each test was measured in 10 mL sample aliquots with an inductively coupled plasma mass spectrometer (ICP-MS). Prior to analysis for total lead, all sample aliquots were acidified, if needed, to reach 2% v/v concentrated HNO$_3$. In steps where the standard preservation protocol was followed, the pH of the water was confirmed to be < 2.0 with an electrode according to Standard Method 4500-H$^+$ (APHA, 1998).
Table 5-3: Summary of tests to evaluate the adequacy of water sample preservation and preparation on the measured total lead concentration.

<table>
<thead>
<tr>
<th>Objective</th>
<th>Test 1</th>
<th>Test 2</th>
<th>Test 3</th>
<th>Test 4</th>
</tr>
</thead>
<tbody>
<tr>
<td>Evaluate adequacy of sample standard preservation versus more rigorous preservation, and in the presence/absence of sample mixing</td>
<td>Evaluate effect of sample transfer after mixing</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Concern</th>
<th>Test 1</th>
<th>Test 2</th>
<th>Test 3</th>
<th>Test 4</th>
</tr>
</thead>
<tbody>
<tr>
<td>Standard preservation recovers dissolved lead but is often inadequate in recovering particulate lead</td>
<td>-EPA 200.8/200.9 specify sample mixing after acid preservative added, but SM 3113B does not</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>EPA 200.8/200.9 specify sample handling in original sample container for AL compliance monitoring, but some instructions in cases of childhood lead poisoning or customer monitoring by utilities suggest transferring only a portion from the original sample bottle for analysis</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Water sample source</th>
<th>Water sample volume</th>
<th>Sample Preparation</th>
<th>Sample preparation/preservation steps (total lead was quantified after each step)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Experiments with lead pipes described in Hu et al. (2010)</td>
<td>1000 mL</td>
<td>Testing of unfiltered water sample in original container at room temperature</td>
<td>1. 0.15% HNO₃ (standard preservation), 17 h, unmixed</td>
</tr>
<tr>
<td>Experiments with lead solder described in Nguyen et al. (2010)</td>
<td>900 mL</td>
<td>Testing of unfiltered water sample in original container at room temperature</td>
<td>2. 0.15% HNO₃ (standard preservation), 17 h, mixed</td>
</tr>
<tr>
<td>Experiments with lead pipes described in Triantafyllidou and Edwards (2011)</td>
<td>900 mL</td>
<td></td>
<td>3. 0.15% HNO₃ (standard preservation), 24 h, mixed</td>
</tr>
<tr>
<td>Experiments with brass coupons and lead pipes described in Zhang et al. (2009)</td>
<td>200 mL from lead pipes</td>
<td></td>
<td>4. 2% HNO₃ (rigorous leachable lead), 138 h, mixed</td>
</tr>
<tr>
<td></td>
<td>100 mL from brass coupons</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

RESULTS AND DISCUSSION

Effect of sample mixing after standard preservation (Tests 1 and 2). The recovery and quantification of lead in water was dependent on sample mixing. Specifically, if the sample was not completely mixed immediately after adding 0.15% acid per unit volume, four representative water samples from lead pipes (Test 1) were determined to contain 369 ppb, 539 ppb, 58 ppb, and 1903 ppb Pb after 17 h holding time (Figure 5-2, top). When these samples were mixed at 17 h, lead concentrations increased to 1336 ppb, 13680 ppb, 185 ppb, and 20300 ppb, respectively (Figure 5-2, top). Allowing an additional 7 hours of holding time did not markedly increase the level of lead, but when acid concentration was increased to 2% v/v, the resulting measurement of total leachable lead was much higher in 2 of the 4 samples (Figure 5-2, top). Practically, at least 71% (Pb pipe 3) to 97% (Pb pipe 2) of the total leachable lead was "missed" in the unmixed samples preserved with 0.15% v/v HNO₃, whereas 6% (Pb pipe 4) to 81% (Pb pipe 1) of the lead...
was missed if the samples were mixed (Figure 5-2, top). In the latter case (Pb pipe 1), standard preservation (0.15% v/v HNO₃) was typically not sufficient to dissolve all the lead even if mixing was undertaken, compared to the more rigorous final digestion (2% v/v HNO₃). While the improved performance of stronger acid preservative (i.e., 2% instead of 0.15% v/v HNO₃) needs to be further investigated, it was shown to recover more lead compared to the standard preservation in some prior work (Triantafyllidou and Edwards 2007).

In the case of water samples containing particulates derived from lead solder (Test 2), an even greater impact of sample mixing was apparent. The standard preservation without mixing after 17 h holding time missed 96% (Solder 5, 6) to 98% (Solder 1, 2, 3) of the lead, when compared to a more rigorous 2% acid exposure to recover total leachable lead from the same sample after 65 h holding time (Figure 5-2, bottom). In contrast, if standard preservation was conducted with mixing, only 1-5% of the lead would be missed, when compared to the total leachable lead in the same sample (Figure 5-2, bottom).

In summary, mixing a sample after adding 0.15% v/v acid preservative greatly reduced (but not completely eliminated) errors in quantifying lead in this work. Labs and utilities that do not mix samples immediately after standard preservation, as is allowed in some EPA-approved protocols (see Table 5-2), run the risk of "missing" serious lead-in-water hazards. This, in theory at least, could allow utilities to comply with the EPA lead AL when they otherwise would not, or result in under-estimation of human health risks from potable water consumption.

**Effect of sample transfer between sample bottles before analysis (Tests 3 and 4).** When following instructions for home water customer sampling issued by a US water utility (Portland Water Bureau 2010) and in some other situations for environmental assessments of lead-poisoned children, where samples are transferred from bottle to bottle before analysis (exemplar in Figure 5-3, top left), relatively large white-colored lead-containing particulates rapidly settled to the bottom of the original sampling container and were visually observed (Figure 5-3, top right). When pouring off a portion of the sample to a smaller bottle, which is typically used for mailing and subsequent analysis, the lead particles remained at the bottom of the original container and were not transferred. In the experiments with lead pipe (Test 3) when the two bottles were preserved and subjected to mixing, the water transferred to the smaller container only contained 57% (Pb pipe 4) to 97% (Pb pipe 5) of the lead detected in the original sampling container (Figure 5-3, bottom). For these samples, subsequent determination of total leachable lead even with very strong acid (2% v/v HNO₃ after 96 h) did not increase recovery of lead (data not presented herein).
Chapter 5. Lead (Pb) Quantification in Potable Water Samples

Figure 5-2: Total lead quantification in water samples collected from laboratory experiments utilizing lead pipes (Test 1, top) and leaded solder wire (Test 2, bottom). Lead concentration is presented on a logarithmic scale for each preservation/mixing step.

In a similar test with water samples collected from experiments of leaded brass (Test 4), a similar result was obtained, in that water samples in the original container always had more lead. Specifically, water remaining in the original container had 50% (Brass 2) to 100% (Brass 4) more lead if the transferred portion received a relatively less rigorous preservation treatment (Test 4.a, Figure 5-4, top). Water remaining in the original container had 42% (Brass 2) to 65% (Brass 1) more lead, even when the other transferred half received a more rigorous and heated preservation treatment (Test 4.b., Figure 5-4, bottom). Similar trends were observed in lead pipe samples from Test 4, although lead concentrations were much higher in the lead pipe water samples (up to 424 ppb for transferred samples and 2189 ppb for remaining samples), compared to those from brass materials (up to 30 ppb for transferred samples and up to 771 ppb for remaining samples) (Figure 5-4).
Overall, the act of transferring water from one bottle to another created large errors in quantification of lead, even if samples were transferred immediately upon collection. These errors potentially arose from very rapid sorption of soluble lead ions to the walls of the original sampling containers, or from rapid settling of particulate lead in the original containers (which was visually obvious in one instance, see Figure 5-3).

Figure 5-3: Illustrative tap water sampling instructions issued to customers of a large US city specify sample transfer to a smaller container for mailing and subsequent lead quantification, after thorough mixing of original sample (top left). Lead-containing particles from laboratory experiment, which settled at the bottom of original sampling container (Test 3-Pb pipe 5), did not transfer to the smaller container for quantification and would have been discarded under these instructions (top right). Total lead quantification in transferred samples (which would have been analyzed under the instructions) and in remaining water samples (which would have been discarded under the instructions), from a laboratory experiment utilizing lead pipes on a logarithmic scale (Test 3, bottom).
Implications for AL monitoring and compliance under the LCR. Limitations in lead recovery, due to insufficient acid preservation and/or mixing (Tests 1, 2), might be causing lead hazards in worst-case water samples to be underestimated, during routine utility monitoring under the LCR. Despite these noted limitations of the EPA standard protocol under the worst-case scenarios examined herein, the protocol is adequate for the majority of situations where lead-in-water is present at lower levels and in the dissolved/colloidal form (Lytle et al. 2003; Triantafyllidou and Edwards 2007). In addition, even for the worst cases of high particulate lead levels, potable water samples have yet to be collected that tested below the 15 ppb AL if bottles are mixed (Triantafyllidou and Edwards 2007). The AL therefore has usefulness in detecting hazardous taps, and compliance would only rarely be affected by the limitations discussed, as long as the bottles are completely mixed after adding the acid preservative. Furthermore, EPA instructions under the LCR mandate that samples are preserved inside the original sampling container, making any limitations by the act of transfer (Tests 3, 4) irrelevant to AL monitoring and compliance.
Implications for customer sampling, environmental assessments of lead poisoned children, monitoring at schools and day care centers. The above issues become much more important for customer or school/day care sampling, and for environmental assessments of lead-poisoned children, which do not fall under the LCR compliance sampling requirements. In those situations, often involving the most sensitive population groups at risk of worst-case exposure, application of the standard EPA protocol (or its modifications) after transfer, might miss the true extent of the lead-in-water hazard, if present. Depending on the form (dissolved or particulate) and level of lead contamination, sampling conducted with modified instructions involving transfer of part of the original water sample into a new container for quantification might "miss" a large percentage of the lead present (Tests 3, 4). Even though such protocols are not employed for regulatory compliance purposes, assessment of human exposure threats using these existing protocols is a significant and under-appreciated concern.

Even more important is the monitoring of lead in water at homes of lead-poisoned children. In these worst-case situations, where a lead hazard is likely present, rigorous sampling for lead in water at multiple taps may be required. Health agencies typically follow some of the LCR instructions to conduct water sampling at the affected children’s homes. But if water is the dominant, or a contributing factor, to the children’s lead exposure, standard sample preservation might not always be sufficient to quantify all the lead in water. Indeed, many recent cases of childhood lead poisoning in the US involved particulate lead in water, ranging from hundreds to thousands of ppb (Triantafyllidou and Edwards 2011a). Those concentrations are not much different from the ones examined in this study, for which standard preservation in combination with insufficient sample mixing (Tests 1, 2) or with sample transfer (Tests 3, 4) “missed” much of the lead present in water.

Other Considerations. Aside from the specific sample preparation techniques examined herein, other sample pre-treatment steps might also be contributing to underestimation of lead hazards in water (Table 5-2). Those issues also deserve investigation, and are further complicated by certain inconsistencies between approved protocols, some of which are resolved by the mandates of the Code of Federal Register that takes precedence (Table 5-2).

Another area of concern is analytical instrument limitations (i.e., Type III of potential deficiencies mentioned in the Introduction of this paper). For example, lead quantification in water of US schools/daycares is voluntary, and does not fall under the umbrella of the federally mandated LCR. In the absence of explicit analytical requirements, schools and daycares may employ anodic stripping voltammetry for lead quantification (Goebel et al., 2004) without performing sample acid digestion. The federally mandated LCR requires acid digestion (Table 5-1), acknowledging that DPASV only measures dissolved lead and not particulate lead, and that all lead in the sample has to be first rendered soluble so that it can be quantified by DPASV. But even if a sample is completely digested and all the lead solubilized, the EPA-approved DPASV method requires neutralization of the sample pH before analysis (Palintest Ltd, 1999). There are certain circumstances in potable water, where neutralization of the sample to high pH, may defeat the purpose of the original digestion, because certain lead solids might re-precipitate through reactions with compounds such as phosphate, or lead can sorb to the walls of the container. If precipitation and/or sorption were to occur, anodic stripping voltammetry is anticipated to be subject to a large error in quantifying lead under EPA-approved procedures. Overall, the practicality and logic behind usage of DPASV on-site analyzers for total lead determination (for compliance reasons or otherwise) warrants future investigation.
ACKNOWLEDGEMENTS

The authors acknowledge the financial support of the National Science Foundation under grant CBET-0933246. Opinions and findings expressed herein are those of the authors and do not necessarily reflect the views of the National Science Foundation. John Consolvo of the Philadelphia Water Department graciously answered questions relevant to lead-in-water quantification methods. Finally, the authors would like to thank the anonymous reviewers for their valuable comments.

REFERENCES


Chapter 5. Lead (Pb) Quantification in Potable Water Samples


APPENDIX A

Supporting Information for Chapter 2

Elevated Blood Lead Levels in Young Children Due to Contaminated Drinking Water:

Washington, DC 2001-2004

Marc Edwards,* Simoni Triantafyllidou, Dana Best†

*Corresponding author, 418 Durham Hall, Civil and Environmental Engineering Department, Virginia Tech, Blacksburg, VA 24061
†Dana Best, MD, MPH, Department of General and Community Pediatrics, Children’s National Medical Center, Washington, DC

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3 Reports Outlining Unpublished EPA Bio-Kinetic Model Results.

In the paper these 3 reports are cited as US EPA (2004). National Center for Environmental Risk Assessment. Risks of elevated blood lead for infants drinking formula prepared with tap water. Documents produced in response to EPA FOIA #HQ-RIN-00337-06.
Date: March 2, 2004

To: Edward V. Ohanian, Director  
Office of Science and Technology (MC: 4304T)  
Office of Water

From: Rob Elias  
National Center for Environmental Assessment - RTP (B243-01)

Thru: Les Grant, Director  
NCEA-RTP (B243-01)

Subject: Request for IEUBK Modeling of Pb in Drinking Water

This memorandum responds to a request from your memorandum dated February 20, 2004, regarding NCEA assistance in evaluating potential impacts on pediatric blood lead (Pb) levels of elevated Pb in tap water detected during recent years in Washington, DC. Three exposure scenarios were provided for use by NCEA staff as inputs to the EPA Integrated Exposure Uptake Biokinetic (IEUBK) model (US EPA, 1994). This model was developed in the 1980s¹ and refined and used extensively since then to model the impacts of multimedia Pb exposure (from air, water, food, soil, and dust) on blood lead (PbB) levels in infants and young children (< 7 years old).

Approach

Table 1 below shows the concentration levels over time for the requested three scenarios for drinking water exposure in Washington, DC during a seven-year period. In developing model estimates we treated each exposure scenario as describing a concentration profile over time with the first 4 years at a lower concentration and the most recent 3 years at a higher concentration. We developed risk estimates to predict current blood lead levels in children. These risk estimates are broken out for children who are currently 1 through 7 years old. As a specific example, a child who is currently 7 years old would have had been exposed to the lower concentrations for 4 years (birth up to age 4) and to the higher concentrations for the last 3 years (ages 4 up to 7). As a second example, a child who is 2 years old would have been exposed to the higher concentrations for the full 2 years of life, i.e., the most recent 2 years of the water concentration profile.

¹The IEUBK model was originally developed at ECAO-RTP, predecessor office to NCEA-RTP by Jeff Cohen, Allan Marcus, and Rob Elias in the 1980s, for the purpose of estimating lead exposure from multiple sources and exposure media, e.g. air, food, drinking water, soil and dust. It was further developed as a potential risk assessment tool by a committee of experts, now called the Technical Review Workgroup, who refined the model and brought it through the process of validation and SAB review. Rob Elias continues to serve as a member of the TRW, as does Paul White, who also contributed to this report. The website for the model is: http://www.epa.gov/superfund/programs/lead/trwhome.htm.
The concentration profiles used here have been described to us as representing specific percentiles of a distribution of water concentration in the District. (Scenario 1, 99th percentile drinking water exposure; Scenario 2, 90th percentile drinking water exposure; and Scenario 3, median drinking water exposure.) In each case an estimate of first draw and flushed drinking water concentration was provided. We have not reviewed these percentile calculations, and for the purposes of this work they are treated as three different scenarios, each of which may be applicable to some homes in the District.

The estimates of blood lead levels developed here are specific to the exposure scenarios. The blood lead levels are presented in Table 2 using the modeling assumptions described in the attached footnote. The calculated geometric mean blood lead level for a scenario represents a prediction of a central or typical blood lead level that would be predicted in children living in homes that had that concentration profile. The estimated percent of children above 10 µg/dL, represents the percent of children living in homes and having concentrations as described in the specific scenario who would have blood lead levels above 10 µg/dL. This may also be interpreted as the likelihood that a single child living in such a house would have an elevated blood lead level. Note that the estimates here are not “population based”, that is, they do not attempt to predict a number or fraction of children in the District who may have elevated blood lead levels. Development of such an estimate would require considerably more input data and modeling work.

Model parameters

With three exceptions, the model used the default settings found in the IEUBK Guidance Manual. These exceptions were: (a) the settings for first draw and flushed drinking water concentrations were set to the requested specifications as indicated in columns 2 and 3 of Table 1; (b) the value for the percentage of water consumed from drinking fountains, normally 15%, was set to zero to focus specifically on the requested scenario, i.e., all drinking water consumed by a child is assumed to come from the tap in the child’s home. With this input, the model defaults then split the amount of first draw and flushed drinking water consumed at 50% first draw and 50% flushed; (c) this estimate includes a revised estimate of food lead concentrations typical of current food lead exposure that was posted to the EPA website based on recent data published by FDA. Without this correction for food lead ingestion, the model would have used food lead inputs based on data from the 1980s, and would have predicted a slightly higher blood lead concentration.

While all other model settings were left in their default mode, it is important to know that certain default settings might impact the interpretation of the present assessment.

1. The default setting for lead concentration in soil and dust is set for 200 µg/g, which may be substantially lower than typical for the urban neighborhoods of the type of concern here. Many urban soil Pb levels reach 400-1000 µg/g, and those soils contaminated by Pb-based paint can be much higher. The predicted blood lead concentrations would have been substantially higher if soil or paint lead exposure was indeed present at levels above the default settings. Consequently, if the results of Table 2 are compared with blood lead concentrations observed in this community, the modeled predictions would possibly underestimate the expected blood lead concentrations of children in neighborhoods with lead-based paint and/or elevated soil lead concentrations.
2. The water intakes assumed in these calculations for Table 2 are central values for the different age groups. It is important to note that the model default tap water intake for 1 year olds is 0.2 L/day. This value will be low compared to the intake rates for infants drinking formula reconstituted formula. A tap water intake of 0.8 L/day may reasonably be assumed for this infants drinking reconstituted formula. We have also done IEUBK based risk calculations for infants drinking reconstituted formula, and as may be expected, this group of children have higher risks of elevated blood lead levels. We will provide these evaluations in a separate communication.

References

### TABLE 1. ESTIMATED DRINKING WATER LEAD CONCENTRATIONS FOR THREE EXPOSURE SCENARIOS

**SCENARIO 1: 99th Percentile Drinking Water**

<table>
<thead>
<tr>
<th>AGE</th>
<th>FIRST DRAW Pb CONCENTRATION (µg/L)</th>
<th>FLUSHED Pb CONCENTRATION (µg/L)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>120.0</td>
<td>30</td>
</tr>
<tr>
<td>2</td>
<td>120.0</td>
<td>30</td>
</tr>
<tr>
<td>3</td>
<td>120.0</td>
<td>30</td>
</tr>
<tr>
<td>4</td>
<td>120.0</td>
<td>30</td>
</tr>
<tr>
<td>5</td>
<td>270.0</td>
<td>70</td>
</tr>
<tr>
<td>6</td>
<td>270.0</td>
<td>70</td>
</tr>
<tr>
<td>7</td>
<td>270.0</td>
<td>70</td>
</tr>
</tbody>
</table>

**SCENARIO 2: 90th Percentile Drinking Water**

<table>
<thead>
<tr>
<th>AGE</th>
<th>FIRST DRAW Pb CONCENTRATION (µg/L)</th>
<th>FLUSHED Pb CONCENTRATION (µg/L)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>5.5</td>
<td>1.4</td>
</tr>
<tr>
<td>2</td>
<td>5.5</td>
<td>1.4</td>
</tr>
<tr>
<td>3</td>
<td>5.5</td>
<td>1.4</td>
</tr>
<tr>
<td>4</td>
<td>5.5</td>
<td>1.4</td>
</tr>
<tr>
<td>5</td>
<td>100</td>
<td>25</td>
</tr>
<tr>
<td>6</td>
<td>100</td>
<td>25</td>
</tr>
<tr>
<td>7</td>
<td>100</td>
<td>25</td>
</tr>
</tbody>
</table>

**SCENARIO 3: Median Percentile Drinking Water**

<table>
<thead>
<tr>
<th>AGE</th>
<th>FIRST DRAW Pb CONCENTRATION (µg/L)</th>
<th>FLUSHED Pb CONCENTRATION (µg/L)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>0.5</td>
<td>0.1</td>
</tr>
<tr>
<td>2</td>
<td>0.5</td>
<td>0.1</td>
</tr>
<tr>
<td>3</td>
<td>0.5</td>
<td>0.1</td>
</tr>
<tr>
<td>4</td>
<td>0.5</td>
<td>0.1</td>
</tr>
<tr>
<td>5</td>
<td>32</td>
<td>8</td>
</tr>
<tr>
<td>6</td>
<td>32</td>
<td>8</td>
</tr>
<tr>
<td>7</td>
<td>32</td>
<td>8</td>
</tr>
</tbody>
</table>

---

²Exposure scenarios provided by Ed Ohanian, February 20, 2004.
Table 2. IEUBK Estimated Blood Lead Levels by Drinking Water Exposure Scenario

<table>
<thead>
<tr>
<th>SCENARIO 1: 99TH PERCENTILE DRINKING WATER SCENARIO</th>
<th>AGE 1</th>
<th>AGE 2</th>
<th>AGE 3</th>
<th>AGE 4</th>
<th>AGE 5</th>
<th>AGE 6</th>
<th>AGE 7</th>
</tr>
</thead>
<tbody>
<tr>
<td>PREDICTED BLOOD Pb µg/dL</td>
<td>9.8</td>
<td>14.1</td>
<td>14.1</td>
<td>13.8</td>
<td>13.5</td>
<td>13.2</td>
<td>12.6</td>
</tr>
<tr>
<td>% above 10 µg/dL</td>
<td>48</td>
<td>77</td>
<td>77</td>
<td>76</td>
<td>74</td>
<td>72</td>
<td>69</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>SCENARIO 2: 90TH PERCENTILE DRINKING WATER SCENARIO</th>
<th>AGE 1</th>
<th>AGE 2</th>
<th>AGE 3</th>
<th>AGE 4</th>
<th>AGE 5</th>
<th>AGE 6</th>
<th>AGE 7</th>
</tr>
</thead>
<tbody>
<tr>
<td>PREDICTED BLOOD Pb µg/dL</td>
<td>5.8</td>
<td>8.0</td>
<td>7.8</td>
<td>7.5</td>
<td>7.0</td>
<td>6.6</td>
<td>6.2</td>
</tr>
<tr>
<td>% above 10 µg/dL</td>
<td>12</td>
<td>31</td>
<td>29</td>
<td>27</td>
<td>23</td>
<td>19</td>
<td>16</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>SCENARIO 3: MEDIAN PERCENTILE DRINKING WATER SCENARIO</th>
<th>AGE 1</th>
<th>AGE 2</th>
<th>AGE 3</th>
<th>AGE 4</th>
<th>AGE 5</th>
<th>AGE 6</th>
<th>AGE 7</th>
</tr>
</thead>
<tbody>
<tr>
<td>PREDICTED BLOOD Pb µg/dL</td>
<td>4.0</td>
<td>4.8</td>
<td>4.6</td>
<td>4.4</td>
<td>3.9</td>
<td>3.5</td>
<td>3.2</td>
</tr>
<tr>
<td>% above 10µg/dL</td>
<td>3</td>
<td>6</td>
<td>5</td>
<td>4</td>
<td>2</td>
<td>1</td>
<td>1</td>
</tr>
</tbody>
</table>

These IEUBK results represent model runs that assume default model parameters except for drinking water Pb concentrations and age of exposure, which were taken from the exposure data provided (see Table 1). First draw/flushed drinking water concentrations were adjusted according to the following scenarios: 99th percentile, 120 µg/L / 30 µg/L for years 1-4 and 270 µg/L / 70 µg/L for years 5-7; 90th percentile, 5.5 µg/L / 1.4 µg/L for years 1-4 and 100 µg/L / 25 µg/L for years 5-7; median percentile, 0.5 µg/L / 0.1 µg/L for years 1-4 and 32 µg/L / 8 µg/L for years 5-7. Age at time of exposure was calculated back through time, i.e., a one-year old child would have been exposed to the drinking water concentrations at year 7, a 2-year old at years 6-7, etc. In this assessment, the percent above the blood lead level of 10 µg/dL was calculated for the 12-month period of the age range.

Estimates of current (2004) blood lead levels as a function of age and lead exposure levels over time.
MEMORANDUM

TO: Edward V. Ohanian, Director
    Office of Science and Technology (MC: 4304T)
    Office of Water

FROM: David A. Bussard, Director
    Washington Division (8623D)
    National Center for Environmental Assessment
    Office of Research and Development

SUBJECT: Request for IEUBK Modeling of Pb in Drinking Water

In addition to the estimates NCEA provided you based on the assumption that infants were not drinking formula that is prepared using tap water, we also looked at the implications on blood lead levels in one-year olds of using tap water to prepare infant formula. Infant formula is a significant pathway of water intake, and infants drinking formula prepared with tap water will typically have substantially greater tap water intakes than others in this age group.

The results using the Integrated Exposure, Uptake, Biokinetic Model for Lead in Children (IEUBK model) are summarized in the attached table, which we will label as “Table 3” since it expands upon Tables 1 and 2 in the memo “Request for IEUBK Modeling of Pb in Drinking Water” sent to you by Rob Elias on 3/02/04. The additional model results in today’s memo are based on calculations described in detail in an attached memo by Paul White, and have been reviewed by Rob Elias in NCEA-RTP as well as by Karen Hogan of the NCEA IRIS staff who agree with them. We modeled the same scenarios as in the previous memo except for adding in the impact of using tap water to prepare infant formula. Note, however, as the estimates address current risks of elevated blood lead levels in one year olds, they are based on the current drinking water concentrations in the scenarios (and do not depend on the concentration estimates for previous years).
The concentration profiles used here have been described to us as representing specific percentiles of a distribution of water concentration in the District. (Scenario 1, 99th percentile drinking water exposure; Scenario 2, 90th percentile drinking water exposure; and Scenario 3, median drinking water exposure.) In each case an estimate of first draw and flushed drinking water concentration was provided. We have not reviewed these percentile calculations, and for the purposes of this work they are treated as three different scenarios, each of which may be applicable to some homes in the District. The values presented here on risk of exceeding a blood lead level of 10 µg/dL apply to children living in homes that have those particular drinking water concentrations. These are not estimates of the proportion of children in the District who would have elevated blood lead levels, any estimates of this population risk would require much more input data and analysis. In addition, as a point of reference we calculated results for the current lead drinking water action level to estimate what the model would predict for blood lead levels in one-year olds drinking formula if the average water concentration were 15 µg/L.

The specific IEUBK parameter values used in modeling the infant formula prepared with drinking water are described in the attached memo. Briefly, these include intake of 0.8 L/day of tap water including prepared formula and a scenario specific geometric standard deviation (GSD) estimate of 1.45. The GSD is a measure representing inter-individual variability of blood lead levels and the estimate here was developed from empirical data for infants consuming formula containing lead. As discussed in the attached memo, under most situations the model default GSD (1.6) is appropriate for environmental assessments; the infant formula scenario is a specialized situation. The use of a situation-specific variability measure does not affect the predicted mean levels but should improve the prediction of the risk of blood lead levels being above 10 µg/L. By comparison, the estimates for one year olds not assuming intake of formula prepared with tap water utilized model defaults for the intake of tap water (0.2 L/day for age 1) and the GSD (1.6). Both analyses assumed water intake proportions of 50% water intake from first draw and 50% from flushed tap water.

In addition to modeling runs using the IEUBK model, we examined two other sources of data, which had also been used in the development of the IEUBK model. We looked at key studies examining (1) measured blood lead levels for infants receiving canned formula (which in past years contained lead from soldered can joints); and (2) measured blood leads from a study of infants using formula prepared with tap water. These results yield supporting conclusions.

Attachments

cc: Paul White
    Rob Elias
    Karen Hogan
    Chon Shoaf
    Lester Grant
    John Vandenberg
    Peter Preuss
    Linda Tuxen
### Table 3. IEUBK ESTIMATED CURRENT BLOOD LEAD LEVELS BY DRINKING WATER EXPOSURE SCENARIO
With and Without Using Tap Water to Prepare Infant Formula

<table>
<thead>
<tr>
<th>SCENARIO 1: 99TH PERCENTILE DRINKING WATER SCENARIO</th>
<th>Lead concentrations 270 µg/L first draw ; 70 µg/L flushed</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>AGE 1 assuming tap water is not used to prepare formula</strong></td>
<td><strong>AGE 1 assuming tap water is used to prepare infant formula</strong></td>
</tr>
<tr>
<td>Predicted geometric mean blood Pb</td>
<td>9.8 µg/dL</td>
</tr>
<tr>
<td>Proportion above 10 µg/dL</td>
<td>48 %</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>SCENARIO 2: 90TH PERCENTILE DRINKING WATER SCENARIO</th>
<th>Lead concentrations 100 µg/L first draw ; 25 µg/L flushed</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>AGE 1 assuming tap water is not used to prepare formula</strong></td>
<td><strong>AGE 1 assuming tap water is used to prepare infant formula</strong></td>
</tr>
<tr>
<td>Predicted geometric mean blood Pb</td>
<td>5.8 µg/dL</td>
</tr>
<tr>
<td>Proportion above 10 µg/dL</td>
<td>12 %</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>SCENARIO 3: MEDIAN PERCENTILE DRINKING WATER SCENARIO</th>
<th>Lead concentrations 32 µg/L first draw ; 8 µg/L flushed</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>AGE 1 assuming tap water is not used to prepare formula</strong></td>
<td><strong>AGE 1 assuming tap water is used to prepare infant formula</strong></td>
</tr>
<tr>
<td>Predicted geometric mean blood Pb</td>
<td>4.0 µg/dL</td>
</tr>
<tr>
<td>Proportion above 10 µg/dL</td>
<td>3 %</td>
</tr>
</tbody>
</table>

**DRINKING WATER ACTION LEVEL 15 (µg/L)**
(Used as average intake)

<table>
<thead>
<tr>
<th><strong>AGE 1 assuming tap water is not used to prepare formula</strong></th>
<th><strong>AGE 1 assuming tap water is used to prepare infant formula</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td>Predicted geometric mean blood Pb</td>
<td>3.7 µg/dL</td>
</tr>
<tr>
<td>Proportion above 10 µg/dL</td>
<td>1.6 %</td>
</tr>
</tbody>
</table>

UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
MEMORANDUM

SUBJECT: Risks of elevated blood lead for infants drinking formula prepared with tap water

FROM: Paul White, Chief
Quantitative Risk Methods Group (8623D)
Washington Division
National Center for Environmental Assessment
Office of Research and Development

TO: David A. Bussard, Director
Washington Division (8623D)
National Center for Environmental Assessment
Office of Research and Development

The Office of Water has requested NCEA’s assistance in evaluating the effects of lead in drinking water on children’s blood lead levels. Infants drinking formula prepared with tap water are a subgroup warranting particular attention as they have a higher intake of tap water per unit body weight than do older children or adults. Several sources of information are available that provide insight on risks of elevated blood lead in infants consuming formula prepared with tap water containing lead:

- Estimates of risk of elevated blood lead levels using the Integrated Exposure, Uptake, Biokinetic Model for Lead in Children (IEUBK) Model;

- Empirical data on infants receiving canned formula (which in past years contained lead from soldered joints); and

- Measured blood leads from a study of infants using prepared formula.

Key studies in the second and third categories were evaluated and used during EPA’s development of the IEUBK model and also serve to provide key information on absorption and biokinetics of lead in young children. These three sources provide complementary information on the elevation of children’s blood lead levels resulting from consumption of formula containing lead.
The information developed here confirms that blood lead levels in infants are sensitive to drinking water lead concentration. Table 4 in this memo summarizes the analyses and shows that similar effects on blood lead would be predicted from the three approaches analyzed. As shown in the table, central estimates of children’s blood lead levels would be predicted to increase by approximately 6 μg/dL for an increase in water lead concentration of 50 μg/L; blood lead would be predicted to increase 11 μg/dL for a increase in water lead concentration of 100 μg/L. These are central estimates of effect of lead in drinking water for an average or typical child. Results provided in Table 1 provide IEUBK model risk estimates of the probability that a infant drinking reconstituted formula will have an elevated blood lead as a function of the lead concentration in tap water. Table 2 provides IEUBK based risk estimates for specific concentrations of lead in first draw and flushed tap water for three scenarios addressing recent drinking water concentrations in Washington, D. C., as provided by Dr. Edward Ohanian.

I appreciate the assistance of Karen Hogan and Rob Elias provided in reviewing this analysis.

**IEUBK Risk Estimates**

The Integrated Exposure, Uptake, Biokinetic Model for Lead in Children provides a tool to address lead risks to infants from reconstituted formula, but requires consideration of appropriate values for appropriate parameter values for this application (see http://www.epa.gov/superfund/programs/lead/trwhome.htm and the references provided there).

The IEUBK model was developed specifically for the purpose of estimating risks of elevated blood lead in children with multimedia exposure to environmental lead. The model has been frequently used for evaluating exposure to elevated concentrations of lead in soil, house dust and air, as well as drinking water. In particular, the model has been used by the EPA Air Office in considering the National Ambient Air Quality Standard for lead, has been used of the Office of Pesticides and Toxic Substances as a principal risk assessment tool for the Hazard Standards for Lead in Paint, Dust, and Soil, and is extensively used by Office of Solid Waste and Emergency Response (OSWER) in risk assessments to support site specific cleanup decisions.

EPA conducted substantial validation work to compare IEUBK model predictions and empirical observations in residential lead studies. The model performed adequately. When the IEUBK modeling focused on children with representative exposure measurements, there was reasonably close agreement between observed and predicted blood lead distributions. (Hogan et al, Integrated Exposure Uptake Biokinetic Model for Lead in Children: Empirical comparisons with epidemiologic data. Environ. Health Prospect. 106 (Supplement 6): 1557-1567 (1998) ) The model was validated using the standards presented and discussed at a model validation workshop in October, 1996 and published in December, 1998. Application of the model to drinking water exposures is consistent with the model development and these validation efforts by EPA. The model was peer-reviewed by the EPA Science Advisory Board in 1992. (EPA SAB IAQC-92). The SAB committee concluded that the model approach was sound and EPA implemented additional refinements in response to committee suggestions.

**Parameter values for IEUBK assessment of lead exposures in infant formula**
Parameter values are intended to represent estimates specific to this exposure scenario. The IEUBK model provides an estimate of a central estimate of blood lead based on these inputs and utilizes a lognormal probability model, with an appropriate geometric standard deviation (GSD) to represent variability in the population of children exposed under this scenario.

**Age range:** Modeling is conducted for the 0-12 month age range; model risk estimates will reflect a values averaged over this age range. This age range was used as a period of concentrated use of formula.

**Drinking water intake:** Infants receiving reconstituted formula are assumed to drink a total of 0.8 L/day of tap water. This included tap water consumed as part of formula as well as other sources of tap water intake. A statistical estimate of the quantity of water intake in formula for infants in the US was not available for this analysis. However, the value of 0.8 L/day would represent a reasonable input considering the following. The Ryu (1983) study discussed below reported average formula intake in infants < 6 months of age in the range of 0.7-0.8 L/day. This value represented volume of formula with would be somewhat greater that the volume of water in formula, however this study did not include the upper part of the age range (6 - 12 months) addressed here where formula intake may be greater, and did not include other intakes of water. The Child-Specific Exposure Factors Handbook (EPA, 2002) provides a distribution of tap water intake (direct and as added to drinks and food) for the < 1year age group. This distribution would encompass both children consuming reconstituted formula and those who do not (e.g., infants being breast fed or those receiving ready to eat formula), this is reflected in the average tap water intake of 0.34 L/day. The upper range of the distribution would be most likely to reflect infants on reconstituted formula: 75th percentile, 0.65 L/day; 90th percentile 0.88 L/day, 95th percentile 1.0 L/day, 99th percentile 1.4 L/day.

**Drinking water concentration:** The values input to the model for these runs represent daily average concentrations consumed by children. This daily value would normally be calculated as a weighted average of concentrations in first draw, flushed, and other tap water sources. If tap water sources outside the household are not considered, the IEUBK default assumptions would calculate the average concentration as a 50/50 average of first draw and flushed values.

**Dietary lead intake (sources other than drinking water):** A value of 3.16 μg/day, specific to the <1 year age group is used. This is an updated value provided in the EPA web site FAQ (frequently asked questions) for the IEUBK model (Source: http://www.epa.gov/superfund/programs/lead/ieubkfaq.htm ). Updated values on the web site were developed using dietary lead intake estimates for each of the 15 IEUBK food categories. The category specific lead estimates were generated using food residue data from the U.S. Food and Drug Administration Total Diet Study (TDS; FDA, 2001) and food consumption data from NHANES III (CDC, 1997). Note that while the TDA estimates do include infant formula, the formulas tested were commercially prepared - ready to eat, and were not found to contain detectable lead.

**GSD:** A geometric standard deviation of 1.45 is used in these analyses. EPA guidance for the IEUBK model generally encourages use of the model default GSD of 1.6 without modification for most environmental assessments. IEUBK assessments commonly address multiple sources...
of exposure including soil, dust, water, etc., and model risk for children ages up to age 7. Our experience has been that use of the default GSD is an appropriate approach for assessments involving various environmental media in numerous applications and sites. In this evaluation of infant formula, it is plausible that variability in intake rates could be lower than with some other pathways, additionally infants might be expected to have fewer concurrent sources of lead exposure. Given that data was available on variability of blood lead levels for infants drinking formula containing lead, it was felt valuable to have a scenario specific estimate of the GSD. An attachment to this document describes GSD calculations using data on the variance in blood leads of infants receiving formula in Ryu et al (1983) and Lacey (1985) data sets. Note that the use of this case specific GSD of 1.45 instead of the model default of 1.6 will lead to lower estimates of the probabilities of elevated blood lead levels in any situation where the geometric mean blood lead is less than the risk assessment reference level of 10 μg/dL.

Using these scenario specific estimates and baseline defaults for other IEUBK parameters, the following risk estimates for elevated blood lead levels are obtained.

Table 1

IEUBK estimated blood lead levels as a function of tap water lead concentration: Intake of reconstituted formula, Ages 0-12 months

<table>
<thead>
<tr>
<th>Drinking water concentration (μg/L)</th>
<th>Geometric Mean PbB (μg/dL)</th>
<th>Percent Prob PbB &gt;10 μg/dL</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>3.1</td>
<td>0.6</td>
</tr>
<tr>
<td>10</td>
<td>4.6</td>
<td>1.9</td>
</tr>
<tr>
<td>15</td>
<td>5.4</td>
<td>4.6</td>
</tr>
<tr>
<td>20</td>
<td>6.0</td>
<td>8.6</td>
</tr>
<tr>
<td>50</td>
<td>9.6</td>
<td>45</td>
</tr>
<tr>
<td>100</td>
<td>14.1</td>
<td>82</td>
</tr>
<tr>
<td>200</td>
<td>20.8</td>
<td>98</td>
</tr>
</tbody>
</table>

Table 2 provides IEUBK based risk estimates for specific concentrations of lead in first draw and flushed tap water for three scenarios addressing recent drinking water concentrations in Washington, D. C., as provided by Dr. Edward Ohanian.

Table 2

IEUBK estimated blood lead levels for three scenarios of water lead concentration: Intake of reconstituted formula, Ages 0-12 months
First draw drinking water concentration (μg/L) | Flushed drinking water concentration (μg/L) | Geometric Mean PbB (μg/dL) | Percent Prob PbB >10 μg/dL
---|---|---|---
32 | 8 | 6.2 | 8.6
100 | 25 | 10.8 | 59
270 | 70 | 19.0 | 96

Model inputs:

1. 0.8 L/day water intake including reconstituted infant formula
2. 50% water intake from first draw, 50% from flushed tap water
3. Dietary lead intake 3.16 μg/day
4. GSD 1.45, estimate specific to infant formula scenario

Study of Lead in commercial infant formulas

Ryu et al (1983) determined the impact of lead in commercial infant formula (as marketed in 1975-1976 prior to government efforts to control this lead source) on blood lead levels. (Ryu et al, Dietary intake of lead and blood lead concentration in early infancy, Am J Dis Child 137:886-891 (1983) ). All infants first received formula supplied in glass units that resulting in lower dietary intakes of lead (for ages 8 - 111 days). From ages 112 to 195 days, one subset of the infants were received milk from cartons (lower dietary intake of lead) while the other group of infants received commercial formula marketed in cans (higher lead intake). Samples of infants’ blood were collected periodically over the study period and total dietary intake of lead was also estimated based on weights of formula and other foods consumed.
Table 3
Effect of lead intake in infant formula on blood lead, Ryu et al (1983)

<table>
<thead>
<tr>
<th></th>
<th>Age 8-111 days All use formula from glass bottles (n=25)</th>
<th>Age 112-195 days Formula from cartons (lower lead group, n=10)</th>
<th>Age 112-195 days Formula from cans (high lead group, n=7)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean</td>
<td>SD</td>
<td>Mean</td>
</tr>
<tr>
<td>N</td>
<td>25</td>
<td></td>
<td>10</td>
</tr>
<tr>
<td>Volume formula consumed L/day</td>
<td>0.783</td>
<td>0.100</td>
<td>0.843</td>
</tr>
<tr>
<td>Lead Concentration in formula μg/L</td>
<td>20</td>
<td>- -</td>
<td>10</td>
</tr>
<tr>
<td>Lead intake from formula μg/day</td>
<td>16</td>
<td>12.5</td>
<td>8</td>
</tr>
<tr>
<td>Other Foods, lead intake μg/day</td>
<td>1</td>
<td>1.0</td>
<td>8</td>
</tr>
<tr>
<td>Total lead intake μg/day</td>
<td>17</td>
<td>2.5</td>
<td>16</td>
</tr>
<tr>
<td>Blood lead μg/dL</td>
<td>6.1 (day 112)</td>
<td>1.7</td>
<td>7.2 (day 196)</td>
</tr>
</tbody>
</table>

This study provides data that are directly useful in interpreting effects of lead concentration in formula on blood lead. Infants receiving formula with a lead concentration of 70 μg/L as compared with 10 μg/L had mean blood lead concentrations of 14.4 μg/dL as compared with 7.2 μg/dL at the end of the study. Thus an increase in the formula concentration by 60 μg/L was associated with an average increase in blood lead by approximately 7 μg/dL. Note also that the infants receiving the high lead formula consumed a somewhat lower volume of formula as compared with low lead group. The study data, not reproduced here, indicated that the divergence in blood lead levels between the two formula groups continued to increase over the duration of the formula intake (85 days).
Appendix A. Supporting Information for Chapter 2

Glasgow water lead study

Lacey et al (1985) reported on the effect of drinking water lead on infants blood lead in Glasgow Scotland. (Lacey et al, Lead in water, infant diet and blood: The Glasgow duplicate diet study, Science of the Total Environment, 41:235-257 (1985). A stratified group of 131 mothers and babies were followed from pre-birth registration until the children were three months of age. Considerable care was taken collecting representative samples of drinking water from homes and obtaining duplicate diet samples for the infants. Statistical analyses were presented using the concentration from a composite sample of drinking water (obtained as daily sub-samples of water from the kettle used to heat water for formula). An estimate of the overall slope of the relationship between water concentration and the infants blood lead at approximately 12 weeks of age presented. The estimated slope 0.62 mg/L blood lead per mg/L water lead is equivalent to a slope of 0.062 \( \mu \)g/dL blood lead per \( \mu \)g/L water lead. Equivalently, and increase in water lead of 100 \( \mu \)g/L would correspond to an increase in average blood lead by approximately 6 \( \mu \)g/dL.

Lacey et al (1985) stated that the linear relationship provided an adequate fit, but acknowledged that some nonlinearity may be present. Figure 4 in Lacey et al (1985) presents stratified estimates of mean infant blood lead level versus mean water lead concentration. This figure indicates that there is a steeper increase in blood lead for the lower water lead levels in this investigation (e.g., 100 \( \mu \)g/L) as compared with higher levels. Further analysis of this issue, supporting the presence of a nonlinear relationship was provided in paper Sherlock and Quinn (1986) and EPA (1994), which fit different nonlinear models to the data on estimated total dietary intake of lead versus blood lead this same study. (See: Sherlock, JC and MJ Quinn, Relationship between blood lead concentrations and dietary lead intake in infants: The Glasgow Duplicate Diet Study 1979 - 1980. Food Additives and Contam., 3: 167-176 (1986) and USEPA Guidance Manual for the Integrated Exposure Uptake Biokinetic Model for Lead in Children, EPA/540/R-93/081 (1994)).

For the current analysis regarding the effects of water lead concentration on blood lead, linear regression models were fit to the stratified data in Figure 4 of Lacey et al (1985). For the full data set, a slope of 0.060 \( \mu \)g/dL blood lead per \( \mu \)g/L water lead was obtained (closely similar to the Lacey value sited above), however when the data is restricted to concentrations < 150 \( \mu \)g/L (that is the first 5 group data points in the figure) a slope of 0.114 is obtained (p=.018). See attached graph (Figure 1) showing the both the full data set regression and the steeper line fit to the <150 \( \mu \)g/L data. For comparison, note that a closely similar result is obtained from a regression using the first four data points (concentrations up to 103 \( \mu \)g/L).

The slope at lower concentrations, 0.114 \( \mu \)g/dL blood lead per \( \mu \)g/L, would imply an increase of 11 \( \mu \)g/dL in blood lead from an increase of 100 \( \mu \)g/L in the concentration drinking water used in reconstituted formula.
Comparison of estimates

Table 4 provides a summary of the results from the three analyses presented here, indicating the consistency of the data on the effect of lead present in formula consumed by infants and increases in blood lead levels.

Table 4

Central estimates of increase in blood lead for infants drinking formula containing lead

<table>
<thead>
<tr>
<th>Study</th>
<th>Increase in blood lead (μg/dL) for 50 μg/L increase in water lead</th>
<th>Increase in blood lead (μg/dL) for 100 μg/L increase in water lead</th>
</tr>
</thead>
<tbody>
<tr>
<td>IEUBK modeling</td>
<td>6.5</td>
<td>11</td>
</tr>
<tr>
<td>Ryu et al (1983)</td>
<td>6</td>
<td>12</td>
</tr>
<tr>
<td>Lacey et al (1985)</td>
<td>5.5</td>
<td>11</td>
</tr>
</tbody>
</table>

Proportionality is assumed for the increases in the 50 - 100 μg/L concentration range for the Ryu et al (1983) and Lacey et al (1985) studies.

Attachment: GSD information pertinent to infants for IEUBK modeling of formula intake

Ryu et al (1983) reported group mean and standard deviation information for blood lead in infants drinking commercially marketed formulas.
The group of infants receiving canned formula, which had higher lead content had the following summary statistics (Ryu et al (1983), page 889). The higher lead group receiving formula with 70 μg/L was used here as the best indicator of variability among children having significant exposure to lead in formula.

<table>
<thead>
<tr>
<th>Days of age</th>
<th>Mean PbB</th>
<th>SD</th>
<th>Calculated CV</th>
</tr>
</thead>
<tbody>
<tr>
<td>140</td>
<td>9.3</td>
<td>4.0</td>
<td>.430</td>
</tr>
<tr>
<td>168</td>
<td>12.1</td>
<td>4.0</td>
<td>.333</td>
</tr>
<tr>
<td>196</td>
<td>14.4</td>
<td>4.4</td>
<td>.306</td>
</tr>
</tbody>
</table>

Using parameter relationships for lognormal distributions \([ CV = (\exp(\sigma^2)-1)^{.5} \) and, \(GSD=\exp(\log(\sigma))\)], where \(\sigma^2\) is the log scale variance of the lognormal distribution, estimates of the GSD can be obtained. For the three groups above, the calculated (approximate) GSDs are 1.51, 1.38, and 1.35. Combining these values to generate a pooled estimate of the log scale variance yields an equivalent GSD of 1.41. Given that each of be above data is based on only seven measurements, and that an approximate, rather than direct calculation of the GSD was made, this estimate has associated uncertainties.

The graphed data in Figure 3 in Lacey et al (1985) provides data on the variability of blood lead levels among individual children drinking formula reconstituted with lead containing tap water. The overall exposure range in this figure covers tap water concentrations up to 600 μg/L. For this analysis the data for concentrations up to 200 μg/L was divided into 8 equal intervals (of width 50 μg/L) and the points in each of these bins read from the graph. The range of data up to 200 μg/L was selected for analysis here as both more relevant to most concerns about drinking water contamination, and as that part of data set in which sampling points were more densely grouped to allow for stratification into concentration bins. Using graphed data for this purpose induces some approximations into the estimates, however the resolution of the figure was judged adequate for the analyses here. Log scale variances for the 8 concentration bins were calculated as...
Concentration range (μg/L) | Number of points | Log scale variance
--- | --- | ---
0 – 25 | 8 | 0.468
25-50 | 16 | 0.184
50-75 | 11 | 0.116
75-100 | 17 | 0.112
100-125 | 7 | 0.249
125-155 | 5 | 0.082
150-175 | 3 | 0.014
175-200 | 5 | 0.086

Using a pooled log scale variance estimate from these values yields an equivalent pooled GSD of 1.52.

In consideration of both data sets, a value of 1.45 was selected as a reasonably representative GSD estimate for the variability in blood lead levels among infants consuming formula containing lead.
Supporting Information 1.

Prediction of Elevated Blood Lead Incidence in 2003 using the NCEA predictions and reasonable assumptions.

In April 2004 the US EPA National Center for Environmental Assessment (NCEA) predicted the impact of the high WLLs in Washington DC on the BLLs of children in the city (See Reports 1-3). Predictions were made 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. The WLLs used by EPA to characterize the exposure to lead in water are perfectly consistent with the utility’s database on WLLs in homes with lead pipe during 2003 used in this research.

The highest risk of EBL due to high WLLs is for infants consuming formula. For this group, if formula was routinely made from water at the 99th percentile of lead occurrence in the city, the child’s predicted likelihood of having EBL is 96% (See EPA Report).

![Figure A-1: Predicted impact of WLLs on the likelihood of EBLs using bio-kinetic model for children consuming formula reconstituted with lead-contaminated water for the first year of life. Figure from model data in NCEA reports presented elsewhere in this packet.](image)

Consumption of formula prepared in the 20% of homes with lead pipe with the lowest WLLs is predicted to have a negligible impact on BLLs (Figure A-1). By integration, the overall predicted risk of EBL for a 1 year old child fed formula made from tap water in DC homes with lead pipe is estimated to be 21.1% (Table A-1).

Similar calculations were made using the NCEA predictions for children aged 1-6 years who consumed drinking water but did not consume infant formula (Table 1). The highest risk for children drinking tap water (and no formula) is for children at 2 years of age, who had an 11.2% overall likelihood of EBL if they consumed tap water and lived in homes with lead pipe.

Numerous assumptions are necessary to make rough projections of numbers of children with EBL in each age group across the city. For example, it is important to consider that...
approximately 44% of consumers do not drink tap water (EPA, 2008) and that up to 75% of infants are breastfed and are therefore somewhat protected from high water lead (CDC, 2008). Calculating an extreme potential impact of these factors, assuming that the 45,549 DC children under age 6 years are equally distributed amongst 282,894 DC housing units, leads to an estimate that 243 children under age 6 years living in Washington, DC homes with lead service line pipe would have EBL in 2003 due to the high WLL (Table A-1). The estimates presented here are probably biased low. More reasonable estimates might also consider exposure via food preparation for residents not drinking tap water, or instances in which infants are partly breastfed and partly formula fed. Considering both of these factors would lead to much higher overall incidence of EBL.

Table A-1: Predicted incidence of elevated blood lead (> 10 ug/dL) in Washington DC due to elevated lead in water in 2003.

<table>
<thead>
<tr>
<th>#Age, Years</th>
<th>1 w/F*</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
<th>Sum</th>
</tr>
</thead>
<tbody>
<tr>
<td>Predictions for children living in homes with lead service line pipe</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>% Children with EBL if 100% consume tap water #</td>
<td>21.1</td>
<td>5.7</td>
<td>11.2</td>
<td>10.3</td>
<td>8.0</td>
<td>6.8</td>
<td>6.2</td>
</tr>
<tr>
<td>% Children with EBL if 44% of do not consume any tap water</td>
<td>11.8</td>
<td>3.2</td>
<td>6.2</td>
<td>5.8</td>
<td>4.5</td>
<td>3.8</td>
<td>3.5</td>
</tr>
<tr>
<td># children living in homes with lead pipe</td>
<td>209</td>
<td>627</td>
<td>836</td>
<td>836</td>
<td>836</td>
<td>836</td>
<td>5,016</td>
</tr>
<tr>
<td># of children with EBL</td>
<td>25</td>
<td>20</td>
<td>52</td>
<td>48</td>
<td>37</td>
<td>32</td>
<td>29</td>
</tr>
<tr>
<td>Predictions for children living in residences without lead service line pipe</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td># children living in residences without lead pipe</td>
<td>1,689</td>
<td>5,067</td>
<td>6,755</td>
<td>6,755</td>
<td>6,775</td>
<td>6,775</td>
<td>40,533</td>
</tr>
<tr>
<td># of children with EBL</td>
<td>43</td>
<td>35</td>
<td>92</td>
<td>84</td>
<td>65</td>
<td>55</td>
<td>51</td>
</tr>
<tr>
<td>Overall predictions for the city</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Percentage of children with EBL Due to Lead Contaminated Water</td>
<td>3.6</td>
<td>1.0</td>
<td>1.9</td>
<td>1.7</td>
<td>1.4</td>
<td>1.1</td>
<td>1.0</td>
</tr>
</tbody>
</table>

*w/F = with Formula, or children exposed to infant formula made with reconstituted tap water.

#The indicated age in years, refers to blood lead predictions for children in the city on the indicated birthday. In other calculations, Age 1 year was applied to children 0-1 years of age, Age 2 to children 1-2 years of age, etc.


In order to make a similar prediction for children living in homes without lead pipe, it was determined based on 2004 utility monitoring data that the risk of very high WLLs (Pb > 150 ppb) was 4.6 times lower in DC homes without lead pipe than in homes with lead pipe (Figure A-2). Leaded solder and brass are the predominant sources of lead in the water supply of homes without lead pipes. Assuming this rough risk reduction factor applies to the incidence of EBL in DC homes without lead pipe (Table A-1), provides an overall estimate of 669 children (600-700 cases) under age 6 years in 2003 with EBL due to high WLLs.

The key point of this discussion is that the estimate of 600-700 cases represents only 0.1% of the total city population and only 1.5% of the population under age 6 years.

![DC WASA summary results](image.png)

Figure A-2: DC WASA summary results of sampling from homes with and without lead pipe service lines, produced via Freedom of Information Act. For homes with lead pipe service lines, 115 out of 6218 homes sampled (= 1.8%) had first draw lead in water over 150 ppb. For homes with copper service lines, 20 out of 5184 homes sampled (= 0.39%) had first draw lead in water above 150 ppb.
Figure A-3: Illustrative ICRP model predictions under various exposure scenarios with tap water lead of 60 ppb. Representative results graphed above assume exposure to 1 L/day for first year and 0.61 L/day thereafter to simulate expectations for use of reconstituted infant formula in year 1. For children not exposed to formula, the model assumes consumption of 0.61 L/day. If children are not exposed to contaminated tap water as infants (through year 1 of their life, magenta or cyan lines), levels of lead in tap water must generally exceed 60 ppb to elevate blood lead > 10 ug/dL. However, with or without formula, children exposed to 60 ppb lead in water from birth have a high likelihood of exceeding 10 or even 20 ug/dL blood lead. For both of these groups blood lead is predicted to peak at about 6-9 months of age. At about 30 months of age, only slight evidence remains of serious elevations to blood lead from prior exposure to lead contaminated tap water due to the exposure occurring at < 1 year of age.
Supporting Information 2.
Considerations in Splitting 2001 into Halves as a Transition Year

Splitting of Water Lead Data

DC WASA Monitoring Experience. The local water utility has extensive experience with lead in water testing. A Freedom of Information Act request revealed e-mails illustrating knowledge that lead in water was lower in the winter months of 2003 than in the summer. This is summarized in an investigation by Eric Holder accessed on January 10, 2009 at http://www.washingtonpost.com/wp-srv/metro/specials/water/wasa071604.pdf. Other e-mails indicated that this knowledge existed for data collected before 2003 as well.

Holder report, page 123

By November 2003, WASA’s Engineering & Technical Services Division had three days of meetings to develop a plan of action for the lead service replacement program for 2003-2004. The stated goal of these meetings was to learn from the details of the 2003 lead service replacement effort in developing a strategy for the 2004 program. (Tab 291). A subsequent e-mail from Roger Gans describing the 2004 lead service replacement plan and addressing the lessons learned from 2003 suggests that one of the lessons learned from the 2003 program was that “[s]ampling and testing should be done in the winter when the water temperature makes for lower lead concentrations.” (Tab 292).

Breakout of EPA First Draw Monitoring Data. There are no monitoring data reported to EPA before June 13, 2001 or after August 29, 2001. However, for data that are available, average and 90th percentile lead levels were relatively low in June/July and rose steadily thereafter.

![Figure A-4: Reported EPA first draw monitoring data broken into increments during 2001. Average lead rose steadily after the June-July time period. There are no data after August 29, 2001.](image)

n = 12
n = 23
n = 13

n = 158

n = 75
n = 47
**Washington Aqueduct Data.** Experiments with lead pipe from Washington DC water were conducted at the Washington Aqueduct. The test water had orthophosphate inhibitor, which is believed to reduce lead leaching relative to what occurred in 2001-2004. The results illustrate that monthly average first draw lead tends to be low from November to March, and begins to rise starting in April and May. After orthophosphate was added the lead tends to peak in June, July and August and remains high through October. This trend is consistent with the utility’s experiences for 2003 reported in the preceding section.

For the year starting May 05, average lead levels were about 50% high for months July to December, relative to months from January to June.

Relative to the transitional year of 2001, since chloramine was first dosed in November 2000, the data below would suggest that lead in water would remain relatively low until temperature rose about March of 2001. Because it can take several weeks or a month to have an effect on blood lead (see Ryu results later in this section), there is relatively little chance the high lead in water would immediately impact incidence of EBL for the population for the time period January to June of 2000. The serious effects would be observed in the second half of 2001 (see Figure 1 in paper).

![Figure A-5: Monitoring Data from Washington Aqueduct.](image)
**Lag Between Effects of High WLLs and EBL.** Ryu et al (1983) conducted a study of infants consuming formula which contained two levels of lead. At the start of the study (time 0 in Figure A-6) the infants were 112 days (about 3.6 months) of age. The average blood lead levels for a group of infants receiving formula with 70 ppb lead rose markedly in one month and became elevated (i.e., exceeded the 10 ug/dL CDC level of concern) in two months. The average blood lead of a group of infants fed formula with 10 ppb lead remained low. These results demonstrate that 1) exposure to formula has almost immediate impacts on blood lead of children, and 2) at levels of exposure that would be considered moderate in Washington DC during 2001-2003 (i.e., 60-70 ppb), it would take a month or two for blood lead to become elevated. Thus, for the typical case, high WLLs in May or June of 2001 would not immediately translate to EBL.

![Figure A-6: Blood lead data from Ryu et al. (1983).](image-url)
Appendix A. Supporting Information for Chapter 2

Supporting Information 3.

GIS Evidence of a Neighborhood Effect

In the beginning of August, Baker Killam discovered a solution to the problem of finding enough lead service lines to test below the LAL. In an August 7, 2003 e-mail, Mr. Gans informed Mr. Benson that Baker Killam had conducted a geographic analysis and found that the lead service lines with high levels of lead were geographically related. (Tab 260). Specifically, certain neighborhoods in Ward Four had much higher lead concentrations than other neighborhoods. (Id.). Consequently, Baker Killam began to avoid these neighborhoods in conducting WASA’s lead sampling for testing in lieu of replacement. (Id.). Baker Killam told Mr. Gans that it was confident that it could obtain 1,600 “passing” results and still maintain geographic diversity. (Id.)

Roger Gans
08/07/2003 03:43 PM

To: Leonard Benson/ENGINEER/DCA/WASA
cc: Jenny Russell/ENGINEER/DCA/WASA, Curtis Cochrane/ENGINEER/DCA/WASA, John Wujek/Contractor/ENGINEER/DCA/WASA

Subject: Lead Service Replacement Sampling Update

Len,

As you are aware, we were very concerned because the pass rate on the replacement sampling program had taken a nose dive recently such that the prior 75-80% pass rate had degenerated to a 25% pass rate. Our fall back plan of supplementing the physical replacement program with sample replacement to meet the 9/30 deadline was thus in jeopardy. In an update this afternoon, John Ricks informed me that a geographic analysis, using GIS, revealed that the low pass rate is geographically related; i.e., certain areas of Ward 4 have much higher lead concentrations than other areas of the city. There is some influence of temperature as well; i.e., higher summer temperatures are making concentrations city-wide higher. But the main impact seems to be this geographic phenomena.

With this insight, the accelerated sampling program will target areas other than those hot spots in Ward 4. Since a lot of prior sampling has been done in Ward 4, we can do this and still maintain sufficient geographic diversity. Baker is confident that we will now be able to achieve at least 1600 passed samples/replacements.

FYI, the split sampling has ruled out lab procedure as the cause of the high failure rate.

This certainly demonstrates the power of GIS analysis for this type of thing.

Roger
Supporting Information 4.

Demarcation of city into neighborhoods with relatively high and low risk of exposure to elevated WLL.

To calculate the relative risk of exposure to very high WLLs in the different neighborhoods, two criteria were combined. First, because high WLLs occurred at higher frequencies in homes with lead pipe, the percentage of homes in each zip code with lead service lines was calculated (see 4\textsuperscript{th} column of Table A-2 below). This percentage ranged from 0 to 22.3%. In addition, the percentage of homes in which first draw WLLs exceeded 100 ppb was calculated. The 100 ppb criterion was selected based on the Gulson et al. (1997) prediction that continuous exposure to tap water \(\geq\) 100 ppb could cause elevated BLLs. This percentage ranged from 0 to 29% (see 5\textsuperscript{th} column of Table A-2 below). The relative risk of exposure to high WLLs in first draw samples in each zip code is calculated by multiplying these two numbers (see 6\textsuperscript{th} column of Table A-2).

To illustrate use of Table A-2 and interpretation of the data, in zip code 20011, 3\% of the population lived in homes with lead pipe that had first draw WLLs \(\geq\) 100 ppb. In contrast, in zip codes 20005, 20032 and 20037, 0\% of the population lived in homes with lead pipe and with first draw WLLs > 100 ppb. In zip code 20024, the likelihood of detecting high WLLs in the first draw sample collected from a home with lead pipe (29.4\%), is balanced by the fact that only 1.1\% of households had lead pipe, producing a overall risk that is relatively low for residents in this zip code.

The zip codes were then sorted from highest to lowest risk (see Table A-3 below and Table 1 paper). To capture the risk of exposure to high WLLs for the different neighborhoods, while also maintaining sufficient statistical power, neighborhoods of highest relative risk were pooled (i.e., the 22\% of the population in zip codes 20011, 20010, 20018, 20003)). Neighborhoods of moderate risk were the 55\% of the population in zip codes 20002, 20012, 20007, 20017, 20015, 20001, 20016, 20008, 20020, 20009. Neighborhoods of lowest risk were the 23\% of the population in zip codes 20019, 20024, 20036, 20005, 20032, 200037, 20004, 20006, 20319, 20332, 20336). Bold lines in Table A-2 indicate the lines demarcating high, moderate and low risk neighborhoods on a relative basis.
Table A-2. Calculating Relative Risk of Exposure to High WLLs by Zip Code.

<table>
<thead>
<tr>
<th>Zip Code</th>
<th>Est. Lead Pipes</th>
<th>Pop. (1000)</th>
<th>% pop with lead pipe *</th>
<th>% 1&lt;sup&gt;st&lt;/sup&gt; draw over 100 ppb</th>
<th>% pop above indicated WLL (ppb)</th>
<th>1&lt;sup&gt;st&lt;/sup&gt; draw &gt; 100</th>
<th>2&lt;sup&gt;nd&lt;/sup&gt; draw &gt; 100</th>
<th>1&lt;sup&gt;st&lt;/sup&gt; draw &gt; 400</th>
</tr>
</thead>
<tbody>
<tr>
<td>20011</td>
<td>4822</td>
<td>57.4</td>
<td>18.5</td>
<td>17.0</td>
<td>3.13</td>
<td>4.10</td>
<td>0.07</td>
<td></td>
</tr>
<tr>
<td>20010</td>
<td>2033</td>
<td>28.8</td>
<td>15.5</td>
<td>16.7</td>
<td>2.59</td>
<td>3.11</td>
<td>0.27</td>
<td></td>
</tr>
<tr>
<td>20018</td>
<td>889</td>
<td>17.0</td>
<td>11.5</td>
<td>19.1</td>
<td>2.20</td>
<td>2.42</td>
<td>0.07</td>
<td></td>
</tr>
<tr>
<td>20003</td>
<td>2342</td>
<td>23.1</td>
<td>22.3</td>
<td>9.2</td>
<td>2.05</td>
<td>3.21</td>
<td>0.04</td>
<td></td>
</tr>
<tr>
<td>20002</td>
<td>4796</td>
<td>49.3</td>
<td>21.4</td>
<td>8.3</td>
<td>1.78</td>
<td>2.76</td>
<td>0.02</td>
<td></td>
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<tr>
<td>20012</td>
<td>576</td>
<td>13.6</td>
<td>9.3</td>
<td>18.8</td>
<td>1.75</td>
<td>2.24</td>
<td>0.00</td>
<td></td>
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<tr>
<td>20007</td>
<td>1546</td>
<td>28.8</td>
<td>11.8</td>
<td>14.4</td>
<td>1.70</td>
<td>2.71</td>
<td>0.00</td>
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<tr>
<td>20017</td>
<td>749</td>
<td>19.2</td>
<td>8.6</td>
<td>12.6</td>
<td>1.09</td>
<td>2.17</td>
<td>0.00</td>
<td></td>
</tr>
<tr>
<td>20015</td>
<td>793</td>
<td>15.8</td>
<td>11.0</td>
<td>8.6</td>
<td>0.95</td>
<td>2.38</td>
<td>0.00</td>
<td></td>
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<tr>
<td>20001</td>
<td>1821</td>
<td>33.6</td>
<td>11.9</td>
<td>7.5</td>
<td>0.90</td>
<td>1.46</td>
<td>0.03</td>
<td></td>
</tr>
<tr>
<td>20016</td>
<td>1146</td>
<td>31.4</td>
<td>8.0</td>
<td>9.6</td>
<td>0.77</td>
<td>1.45</td>
<td>0.00</td>
<td></td>
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<tr>
<td>20008</td>
<td>898</td>
<td>26.2</td>
<td>7.5</td>
<td>7.8</td>
<td>0.59</td>
<td>1.21</td>
<td>0.00</td>
<td></td>
</tr>
<tr>
<td>20020</td>
<td>769</td>
<td>49.9</td>
<td>3.4</td>
<td>16.7</td>
<td>0.57</td>
<td>0.54</td>
<td>0.04</td>
<td></td>
</tr>
<tr>
<td>20009</td>
<td>1649</td>
<td>46.6</td>
<td>7.8</td>
<td>6.3</td>
<td>0.49</td>
<td>0.90</td>
<td>0.02</td>
<td></td>
</tr>
<tr>
<td>20019</td>
<td>694</td>
<td>52.8</td>
<td>2.9</td>
<td>16.5</td>
<td>0.48</td>
<td>0.65</td>
<td>0.01</td>
<td></td>
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<tr>
<td>20024</td>
<td>58</td>
<td>11.8</td>
<td>1.1</td>
<td>29.2</td>
<td>0.32</td>
<td>0.32</td>
<td>0</td>
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</tr>
<tr>
<td>20036</td>
<td>98</td>
<td>3.8</td>
<td>5.7</td>
<td>3.9</td>
<td>0.22</td>
<td>0.22</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>20005</td>
<td>51</td>
<td>10.6</td>
<td>1.1</td>
<td>0.00</td>
<td>0.00</td>
<td>0.00</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>20032</td>
<td>280</td>
<td>31.7</td>
<td>1.9</td>
<td>0.90</td>
<td>0.00</td>
<td>0.06</td>
<td>0</td>
<td></td>
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<tr>
<td>20037</td>
<td>116</td>
<td>12.6</td>
<td>2.0</td>
<td>0.00</td>
<td>0.00</td>
<td>0</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>20004</td>
<td>8</td>
<td>0.90</td>
<td>2.0</td>
<td>na</td>
<td>na</td>
<td>na</td>
<td>na</td>
<td></td>
</tr>
<tr>
<td>20006</td>
<td>13</td>
<td>1.9</td>
<td>1.5</td>
<td>na</td>
<td>na</td>
<td>na</td>
<td>na</td>
<td></td>
</tr>
<tr>
<td>20319</td>
<td>0</td>
<td>0.05</td>
<td>0.0</td>
<td>na</td>
<td>na</td>
<td>na</td>
<td>na</td>
<td></td>
</tr>
<tr>
<td>20332</td>
<td>0</td>
<td>0.63</td>
<td>0.0</td>
<td>na</td>
<td>na</td>
<td>na</td>
<td>na</td>
<td></td>
</tr>
<tr>
<td>20336</td>
<td>0</td>
<td>4.6</td>
<td>0.0</td>
<td>na</td>
<td>na</td>
<td>na</td>
<td>na</td>
<td></td>
</tr>
</tbody>
</table>
Examining the pooled data (see Table A-3 above) demonstrates that other possible criteria used to rank neighborhood exposure risk gave similar trends. For example, the relative ranking of the three risk zones in the city is maintained if WLLs are based on second draw samples ≥100 ppb. Likewise, consumption of 0.44 liters from first draw samples with WLLs of 400 ppb or higher, represents an acute lead dose that exceeds the Consumer Product Safety Commission (CPSC) health risk triggering product recalls in toys and jewelry. The DC population living in the most severely impacted neighborhoods (high risk) had 6.5 times greater likelihood of exposure to levels of lead that exceed the CPSC acute health risk level via consumption of a single glass of water, relative to those living in moderate risk neighborhoods. Consumers living in the lowest risk neighborhoods had almost no likelihood of exposure to lead doses above the CPSC acute health criteria from a single glass of drinking water (Table A-3).


Illustrative Calculation:

WASA summary results of first draw lead sampling for different lead plumbing materials produced via Freedom of Information Act Request.

The following table summarizes the percent of first draw and second draw test results, which were above the lead action level of 15 ppb:

<table>
<thead>
<tr>
<th>Service Line Material</th>
<th>No. of residential addresses sampled</th>
<th>1st Draw &gt; 15 ppb</th>
<th>2nd Draw &gt;15 ppb</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lead</td>
<td>10,567</td>
<td>61.0%</td>
<td>60.6%</td>
</tr>
<tr>
<td>Copper</td>
<td>6,312</td>
<td>18.5%</td>
<td>17%</td>
</tr>
<tr>
<td>Brass</td>
<td>1,401</td>
<td>7.9%</td>
<td>6.4%</td>
</tr>
<tr>
<td>Unknown</td>
<td>5,202</td>
<td>27.1%</td>
<td>26.4%</td>
</tr>
</tbody>
</table>

Weighted average using the above data to estimate % homes without lead pipe and first draw lead > 15 ppb = (6312)(18.5%) + (1401)(7.9%) + (5202)(27.1%))/(12915) = 21%
Illustrative Calculation. Estimate the percentage of children in high risk part of city exposed to first draw WLLs below 15 ppb.

Raw Data and Assumptions:

Homes with lead pipe in High Risk Part of City = 10,086 (Table 1 in paper)

% First draw in homes with lead pipe and >15 ppb in High Risk Part of City = 77% (Figure A-1)
% First draw in homes with lead pipe and ≤15 ppb in High Risk Part of City = 23%
% Population not drinking tap water = 44% (See EPA reference in text)
% Population drinking tap water = (100-44)% = 56%
Est % Homes without Lead Pipe and with First Draw Over 15 ppb = 21% (Supporting Calculation S.1)

Fraction of Population in High Risk Part of City and with Lead Pipe Exposed to First Draw Water Lead Above 15 ppb:

77% x 56% = 43.1%

Fraction of Population in High Risk Part of City and without Lead Pipe Exposed to First Draw Water Lead Above 15 ppb:

21% x 56% = 11.8%

Weighted Average of Population in High Risk Part of City Exposed to First Draw Lead Above 15 ppb:

Total Households in High Risk Parts of City (From Table 1 of paper):
126,300 people ÷ 2.2 people/household = 57,409 households
10,086 homes with lead pipe/57,409 households = 17.6%
Fraction without lead pipe = (100-17.6)% = 82.4%
Weighted Average Population Exposed to First Draw Water Lead Above 15 ppb = (43.1% x 17.6%) + (11.8% x 82.4%) = 17%

Percentage of Population in High Risk Part of City Exposed to First Draw Water Lead Below 15 ppb:

100% – 17% = 83%
Figure A-7: Correlation between EBL incidence and 90th percentile Pb in water for 2000-2007.

Figure A-8: Replot of Upper Figure 2-2 in paper with 2001 CNMC blood lead data split into a first and second half.
Figure A-9: Correlation resulting from not splitting the data from transition year 2001 into a first and second half.

Figure A-10: Improved correlation resulting if transition year 2004 had been excluded.
Supporting Information 6.

EXAMINING BLOOD LEAD SPLIT IN 2001

DATA FROM CDC DATABASE FOR CHILDREN < 1.3 YEARS OF AGE WITH 1999, 2000 and 2001 SPLIT

Figure A-11: Incidence of EBL in CDC database comparing the first to second half of each year 1999-2001 for children age 1-16 months. While there is a trend towards increased incidence of EBL in the second half of the year versus the first half of the year in the CDC data in 1999 and 2000, after chloramine was added there is an anomalous spike in the second half of 2001 (above). The same trend is observed for the overall CDC data (all ages below).
Figure A-12: For the CNMC data in 2000, in the high and moderate risk parts of the city EBL incidence for children aged ≤ 30 months was higher in the first half of the year versus the second half of the year (above). After chloramine was added, in 2001 the opposite trend was observed (below). Thus, based on the data in 2000, splitting 2001 in half is not inherently biased towards higher EBL incidence in the second half of the year. Moreover, the extent of the impact in the second half of 2001 increases with neighborhood risk level. All of these factors point to the high WLLs as the likely cause for higher EBL incidence in the second half of 2001.
Figure A-13: In early 2001 the EBL ratio was below 1 before about August. An EBL ratio below 1 indicates that blood lead had continued its decades’ long decline for a given month in 2001 versus 2000. For instance, if EBL incidence was 5% in March 2000 and 2.5% in March 2001, the EBL ratio = 2.5/5 = 0.5. However, if an EBL ratio is above 1, EBL incidence was higher in 2001 versus 2000. On the basis of average and 90th percentile WLL data collected in June, July and August, average Pb and 90th percentile Pb in water rose markedly in between June and August 2001. Above data based on entire CDC database (3).
Supporting Information 7.

**Date of Chloramine Addition was November 2000**


- On November 1, 2000, WA converted the residual disinfectant from free chlorine to chloramines for the purpose of lowering disinfection byproducts to meet new regulatory requirements. This conversion facilitated a reduction in oxidation reduction potential (ORP) to a range that favors the predominance of Pb (II) scales, which are highly influenced by low and fluctuating pH levels. This conversion from free chlorine to chloramines likely changed the nature of the predominant scale from Pb (IV) to Pb (II) and thus facilitated an increase in the release of lead from the lead service lines into the water at consumers' taps.

**Date of Public Health Interventions**

Table A-4: The actual implementation date of public health interventions in Washington, DC versus the date reported by Guidotti et al. (4).

<table>
<thead>
<tr>
<th>WASA Implemented Public Health Intervention</th>
<th>Reported Date EHP</th>
<th>Actual Date</th>
</tr>
</thead>
<tbody>
<tr>
<td>10 minute flushing advisory</td>
<td>2003</td>
<td>February 2004</td>
</tr>
<tr>
<td>Multilingual advice sent to households with lead service lines</td>
<td>2003</td>
<td>March 2004</td>
</tr>
<tr>
<td>Lead filters distributed</td>
<td>2003</td>
<td>March 2004</td>
</tr>
<tr>
<td>DC WASA voluntarily accelerated lead service line program</td>
<td>2003</td>
<td>July 2004</td>
</tr>
<tr>
<td>Offer to replace owner’s lead service line at cost</td>
<td>2003</td>
<td>Required by Federal Law in the EPA LCR</td>
</tr>
<tr>
<td>Low cost financing</td>
<td>2003</td>
<td>November 2004</td>
</tr>
<tr>
<td>Free water testing offered to any customer</td>
<td>2003</td>
<td>February 2004</td>
</tr>
</tbody>
</table>