Critical Reviews in Environmental Science and Technology, 42:1297–1352, 2012
Copyright © Taylor & Francis Group, LLC
ISSN: 1064-3389 print / 1547-6537 online
DOI: 10.1080/10643389.2011.556556

Lead (Pb) in Tap Water and in Blood: Implications for Lead Exposure in the United States

SIMONI TRIANTAFYLLIDOU and MARC EDWARDS
Department of Civil and Environmental Engineering, Virginia Tech, Durham 418, Blacksburg, VA, USA

Lead is widely recognized as one of the most pervasive environmental health threats in the United States, 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 United States 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 presently believed. The authors describe sources of lead in tap water, chemical forms of the lead, and relevant U.S. 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 United States is to achieve its goal of eliminating elevated blood lead levels in children by 2020.

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

I. INTRODUCTION

Lead (Pb) is widely recognized as one of the most pervasive environmental health threats in the United States. Dramatic progress has been made over

Address correspondence to Marc Edwards, Department of Civil and Environmental Engineering, Virginia Tech, Durham 418, Blacksburg, VA 24061, USA. E-mail: edwardsm@vt.edu

EPA-R5-2018-0081360000188

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). 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 United States to achieve its goal of eliminating all instances of EBL in children by 2020 (U.S. 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 (U.S. Environmental Protection Agency [US EPA], 1993). 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 following:

- 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 U.S. schools and day care 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;

EPA-R5-2018-0081360000188

- 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.

II. 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). But as it enters building plumbing through service line connections, it may come into contact with lead-containing plumbing materials (Figure 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). 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). 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).

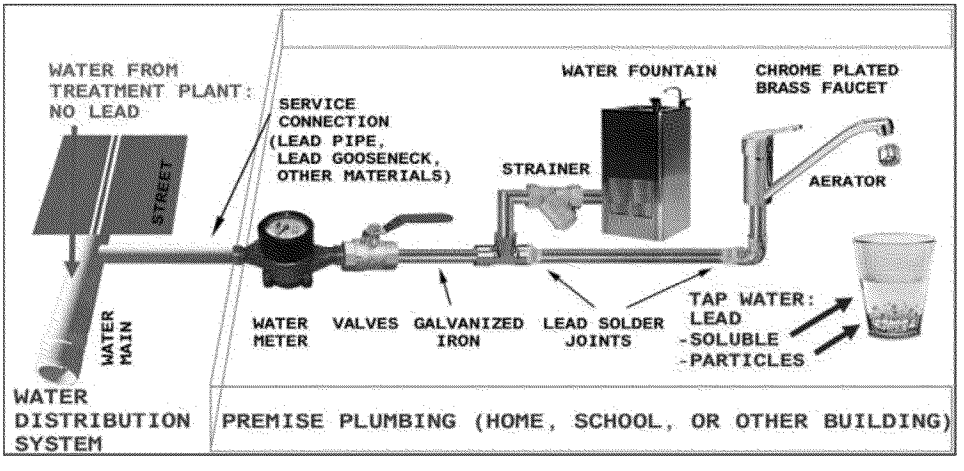

FIGURE 1. Potential sources of lead contamination in tap water of homes, schools, and other buildings.

EPA-R5-2018-0081360000188

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 (Anonymous, 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 U.S. cities through the 1950s, 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 U.S. history (Troesken, 2006). Even though the use of lead pipe in service lines or premise plumbing was prohibited in the United States by the Safe Drinking Water Act (SDWA) amendment of 1986 (US EPA, 2006a), 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 and 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 United States, 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 purported 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, 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 with homes with full lead pipes (Frumkin, 2010).

EPA-R5-2018-0081360000188

Lead Solder

Solder is a fusible metal alloy that is melted to join metallic plumbing materials together in a strong and water-tight seal (Figure 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 U.S. 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 lead solder in some new buildings in the United States (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, water coolers) used in modern homes can legally contain up to 8% lead by weight (Figure 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 to 8% by weight), the volume of water in contact with the faucet, the physical configuration of the faucet and how it was manufactured, and the 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

EPA-R5-2018-0081360000188

California and several other U.S. states are beginning to require their use in new construction (Sandvig et al., 2008).

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 (Schock 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 U.S. Households at Potential Risk

While poor record keeping makes it practically impossible to determine the exact type of plumbing materials at individual U.S. households, without exhuming and forensically evaluating plumbing materials underground and in walls, consideration of rough estimates is useful. Weston and Economic and Engineering Services (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 United States, corresponding to about 3% and 6% of total U.S. housing units, respectively (Table 1). For solder, it is estimated that the 81 million U.S. housing units (77% of total U.S. housing units) constructed prior to the federal ban of lead pipe and lead solder in 1986 (U.S. Census Bureau, 2000) are virtually certain to contain lead solder joints (Table 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). Only new housing units that incorporate nonleaded brass faucets and other nonleaded 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 nonleaded forms. It should be noted that the rough estimates presented (Table 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, or leaded brass is virtually below detection, due to formation of protective surface coatings.

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). 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 g is sufficient to contaminate every drop of water used by a U.S. family of three for three 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

TABLE 1. Estimated number of U.S. homes at potential risk from tap water lead contamination, depending on presence of lead-bearing plumbing materials

Lead-bearing plumbing material	Age of U.S. homes at potential Risk	Estimated number of US homes at potential risk (% of total)	Estimated mass of lead per home at potential risk (kg)
Brass plumbing components			
If 2% lead by weight	All	All	0.1[b]
If 8% lead by weight	All	All	0.3[b]
> 8% lead by weight	Pre-1986	81 million (77%)[a]	0.4[b]
Lead pipes, lead service lines, and lead goosenecks (100% lead by weight)	Pre-1986	3.3–6.4 million[c] (3–6%)[a]	19.1[d]
Lead solder (40–50% lead by weight)	Pre-1986	81 million (77%)[a]	Highly variable, but believed to be very significant[e]
Lead joints in water mains (100% lead by weight)	Pre-1986	All homes served by water mains installed pre-1986	Unknown but believed inconsequential[f]

Note. 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.

[a]Estimation based on year of home built from U.S. Census Bureau (2000). [b]Assumed one residential brass water meter of body weight 5 lbs (2.3 kg) and eight brass devices similar to brass ball valves of individual body weight 0.5 lbs (0.2 kg). [c]Estimation by Weston and Economic and Engineering Services (1990). More recent estimations have not been conducted. [d]Calculation for typical lead service line of 25 ft (7.6 m) length, internal diameter of $\frac{3}{4}$ inch, external diameter of 1 inch, and lead density of 11.3 g/cm^3. [e]Depends on workmanship of the soldering process at joints and resulting mass of solder in contact with water, believed one of the major sources of tap water lead contamination. [f]Currently believed that lead in these lead joints will not contact the water.

EPA-R5-2018-0081360000188

EPA-R5-2018-0081360000188

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 U.S. 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, the United Kingdom, and Germany as of 1999 was estimated at 40–50% (Hayes and Skubala, 2009). As of 1999, premise (building) plumbing in Portugal, France, and the United Kingdom also contained 30–40% lead pipes (Hayes and Skubala, 2009). In Japan, as of 2002, a total of 667 km lead pipe was found below roads and 3,248 km of lead pipe was 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 1950s and then again in the 1970s (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 and in infant and even adult mortality were attributable to this source (Renner, 2007; Troesken, 2008, 2006;). 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 1990s 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 (Edwards, 2010; Miller, 2010). On occasion, the lead paint water risk reward analysis has

EPA-R5-2018-0081360000188

been invoked to justify diverting a portion of funding originally intended for reducing the public's exposure to lead in water, toward the 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 U.S. children (Jacobs 1995; Levin et al., 2008). Although the conventional wisdom in the United States is that lead-based paint is the predominant source of lead poisoning in children, and all other lead sources are a distant second, a few potential weaknesses in this argument and alternative perspectives have 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, other sources), can be an equally important exposure pathway, compared with 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 United States 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; Agency for Toxic Substances and Disease Registry, 2007). The US EPA's Office of Chemical Safety and Pollution Prevention also recently issued the lead Renovation, Repair, and Painting 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 United States 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 has set an enforceable permissible exposure limit for lead in workplace air (Agency for Toxic Substances and Disease Registry, 2007). Lead is also present in consumer products. Dietary supplements, crystal glassware and ceramic pottery, polyvinyl chloride 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 U.S. standards (Levin et al., 2008).

While the conventional wisdom is that lead in paint and in dust account for a majority of EBLs in U.S. children, the CDC estimated that 30% or more of present EBL cases do not have an immediate lead paint source identified (Levin et al., 2008). The US EPA (2010) recently expressed an opinion, shared by many others (Levin et al., 2008; Scott, 2009), that as other agencies and

EPA-R5-2018-0081360000188

EPA offices focus primarily on other sources of lead exposure (e.g., lead-based paint, lead in dust and soil) lead in drinking water as an exposure path is becoming a bigger percentage of a smaller number.

III. U.S. REGULATIONS/GUIDELINES FOR LEAD IN TAP WATER, AND OTHER PUBLIC HEALTH GUIDANCE

Lead and Copper Rule of 1991

The US 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 (Table 2). The US 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 U.S. cities. The review showed that 96% of U.S. 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 (Figure 1). The LCR requires sampling at homes known to have plumbing with highest potential for lead contamination, and after a minimum of 6 hr 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. The US EPA (2006b) 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.

To illustrate, consider actual lead-in-water data from volunteers in a large U.S. 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 2). One percent of this population is exposed to over 70 μg/L lead, and 0.1% of the population is exposed to lead

TABLE 2. U.S. federal regulations and guidelines for lead in drinking water of homes and schools

Federal statute	Lead and Copper Rule (LCR) of 1991 for homes served by public water systems	Lead Contamination Control Act (LCCA) of 1988	No regulation
Applies to	- Homes and other buildings served by a public water system (~85% of U.S. homes) - Schools/day cares regulated as public water systems[a] (~10% of U.S. schools)	Schools/day cares served by a public water system (~90% of U.S. schools)	Homes with private water system (~15% of U.S. homes)
Enforceable?	Yes, federal regulation	No, voluntary guidance	Not applicable
Required sample number	5–100 taps, depending on the size of the population served (reduced to 5–50 taps, for utilities previously compliant with the rule)	Each school water outlet used for drinking and cooking	None
Sampling Frequency	every 6 months (reduced to as little as once every 3 years for utilities previously compliant with the rule)	Not specified	None
Sampling requirements	1 L cold water samples after at least 6 hr of stagnation	250 mL cold water samples after 8–18 hours of stagnation	None
Lead Limit	15 μg/L, termed "Action level" (AL)	20 μg/L	None
Failure criterion	Over 10% of samples exceeding AL of 15 μg/L lead (or else 90th percentile lead > AL)	Any water sample exceeding 20 μg/L lead	None
Remediation measures	Corrosion control optimization,lead service line replacement,public education	Flushing, point-of-use filters, remove plumbing, bottled water, public education	None
Reference	US EPA, 1991	US EPA, 2006b	US EPA, 2006c

[a]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.

EPA-R5-2018-0081360000188

EPA-R5-2018-0081360000188

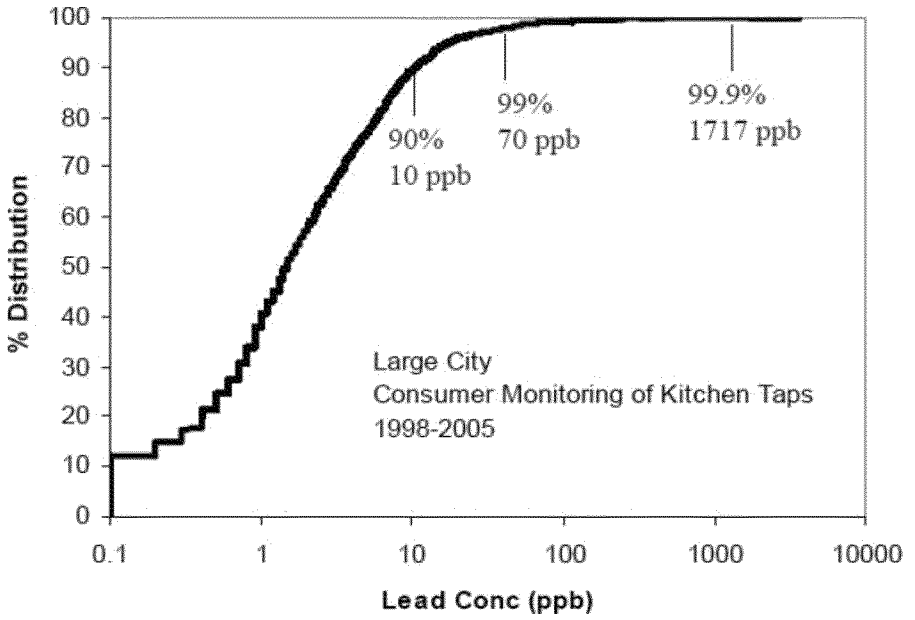

FIGURE 2. Cumulative distribution of lead-in-water levels (in logarithmic scale) at consumers' home taps in a large U.S. city from 1998 to 2005. Compiled from monitoring data of city residents, who voluntarily collected tap water samples and submitted them for lab analysis (S. Patch, personal communication, November 28, 2006).

over 1717 μg/L. If the U.S. 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 previous 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 (US EPA, 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

EPA-R5-2018-0081360000188

employed in major U.S. cities (Leonnig, 2004), although the majority of U.S. water utilities sample tap water and report monitoring data with the safety of their consumers in mind.

Lead Contamination Control Act of 1988

The LCR also applies to the 10% of U.S. schools that have their own water supply (Table 2). However, it does not extend to the majority of U.S. schools and day care facilities, which rely on public water systems for their water supply (Table 2). Instead, the Lead Contamination Control Act (LCCA) provides nonenforceable guidelines for these schools and day care facilities, recommending that drinking water should not exceed 20 μg/L lead in any 250 mL first-draw sample (US EPA, 2006; Table 2). In other words, aside from the 10% of U.S. 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 U.S. schools and day care facilities are not subject to any enforceable national lead-in-water standard (Table 2).

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 those 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 (Freking, 2009). Although much less information is available for the 90% of schools not subject to any sampling requirements, case studies in Baltimore, Maryland; Seattle, Washington; Philadelphia, Pennsylvania; Washington, DC; Maryland suburbs; and Los Angeles, California, revealed serious problems with lead contamination of school water in recent years (Table 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 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%)

TABLE 3. Representative case studies on lead-in-water problems at U.S. schools

School system location	Year school knew of problem	Year public informed	How discovered	Average % taps above LCCA guidance of 20 μg/L[1]	Highest reported Pb in water (μg/L)	References
Baltimore, MD	1992	2003	Parent inquired as to why water fountains had been turned off and a teacher turned whistleblower.	20% of fountains	655	City of Baltimore, 2007 J. Williams, personal communication, July 23, 2008
Seattle, WA	1990	2003	A parent was concerned due to discolored water, collected and analyzed sample finding high lead.	1990: 33–40% 2004: 25%	1,600	Odell, 1991 M. Cooper, personal communication, July 13, 2008 Boyd et al., 2008a
Philadelphia, PA	1993	1998	A source unofficially provided lead-in-water test results to EPA, after EPA had been told to get a search warrant when requested to sample water.	2000: 38% of fountains 48% of faucets	not available but 17% of schools > 100 μg/L	Fitzgerald, 2000 Bryant, 2004
Washington, DC	1987	2007	Freedom of Information Act (FOIA) requests by Virginia Tech; more than 80% of taps in some schools exceeded 15 μg/L lead[a]	2004: 4% 2006: 29% 2007: 13% 2008: 26%	2004: ***7,300*** 2006: 4,936 2007: ***20,000*** 2008: 1,987	P. Taylor, personal communication, November 2, 2007 Lambrinidou and Edwards, 2008 Lambrinidou et al., 2010
Washington Suburban, MD	2004	2004	School system voluntarily collected samples to participate in LCCA after problems were revealed in Washington, DC.	2004: 18%	***36,372***	Gerwin, 2004 Montgomery County Public Schools, 2004
Los Angeles, CA	1998	2008	Local news station; school personnel falsified daily reports regarding remedial flushing to reduce lead.	2008: 30%	Not available	Lambrinidou et al., 2010 Grover, 2008a

Note. 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).
[a]All data from Washington DC schools in this table use 15 μg/L as a failure criterion.

EPA-R5-2018-0081360000188

EPA-R5-2018-0081360000188

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 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 (Boyd et al., 2008b; Greenwire, 2004; Grover, 2008b; Montgomery County Public Schools, 2007). 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). Similar to 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 3 represent the good news, as most other school systems in urban areas have not systematically tested their water for lead in nearly three decades. Not shown in Table 3 are other case studies from (a) Davidson, North Carolina, 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); (b) Durham, North Carolina, where sampling revealed hazardous lead levels in some water fountains at 8 schools (Biesecker, 2006); and (c) 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 (2010) announced that it is developing a draft entitled "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.

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 and worst-case scenarios of drinking water consumption at

EPA-R5-2018-0081360000188

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 biokinetic 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, North Carolina, was exposed in a day care center (E. Robertson, personal communication, March 24, 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 (Y. Lambrinidou, personal communication, December 10, 2008). Reports of harmful exposure are more consistent with common sense expectations, considering that the higher levels of lead detected in some schools (Table 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 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 4) indicates that the US EPA MCLG for lead in water is equal to zero and that the state of

EPA-R5-2018-0081360000188

TABLE 4. Public health guidance regarding various levels of lead in tap water

Agency	Lead threshold (μg/L)	Health guidance and/ or warning	Reference
U.S. 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)
U.S. Environmental Protection Agency	40	Imminent and substantial endangerment to children (warning removed in 2004)	Renner (2010)
U.S. Consumer Product Safety Commission	700[a]	Acute health risk to children	CPSC (2005)
U.S. Environmental Protection Agency	5000[b]	Hazardous waste classification	US EPA (2009)

[a]Lead dose of 175 μg translated to lead exposure through water consumption of 250 mL (one glass).
[b]Based on the Toxicity Characteristic Leaching Procedure (TCLP) test for waste.

California has developed its own public health goal 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 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) advised children and pregnant women to not consume water that contains more than 15 μg/L lead (Table 4). As a further point of reference, the CPSC (2005) classified a lead dose of 175 μg as an acute health risk to children. 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 4). Finally, water containing more than 5,000 μg/L lead exceeds hazardous waste criteria (US EPA, 2009).

EPA-R5-2018-0081360000188

IV. 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 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 (McNeill 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 5). At the upper end of particulate lead sizes, these particles are big enough to be seen by the naked eye.

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., cerrusite and hydrocerrusite; see Table 5) are

TABLE 5. Classification of lead species in tap water and distinction between dissolved lead and particulate lead (adapted from De Rosa and Williams, 1992)

Operational definition	Approximate diameter size (μm-log scale)	Class	Example(s)
Dissolved lead		Free aquo ions	Pb^{+2}
	0.001	Organic chelates, other inorganic ions, ion pairs and complexes	Pb-EDTA $PbCO_3$
	0.01	Bound to macromolecules	Pb-fulvic acid complexes
	0.1	Highly dispersed colloidal material	Adsorbed on hydrous iron and manganese oxide colloids
0.45		Adsorbed on inorganic particulates	Adsorbed on hydrous iron and manganese oxides and clay minerals
Particulate lead	10+	Minerals and precipitates	$PbCO_3$(s)-Cerussite $Pb_3(CO_3)_2OH_2$(s)-Hydrocerussite

EPA-R5-2018-0081360000188

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 United States (HDR Engineering, 2009).

A small survey of lead in potable water from around the United States 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 U.S. locations with significant lead-in-water problems revealed that particulate lead release from the plumbing was often the cause (Figure 3; Table 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 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 AL) occasionally present in the water due to these particles, and their varying mineralogical content ranging from 3% to 100% lead (Table 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).

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

TABLE 6. Origin of representative lead particles identified in drinking water during field investigations, and level of resulting water contamination

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

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

EPA-R5-2018-0081360000188

EPA-R5-2018-0081360000188

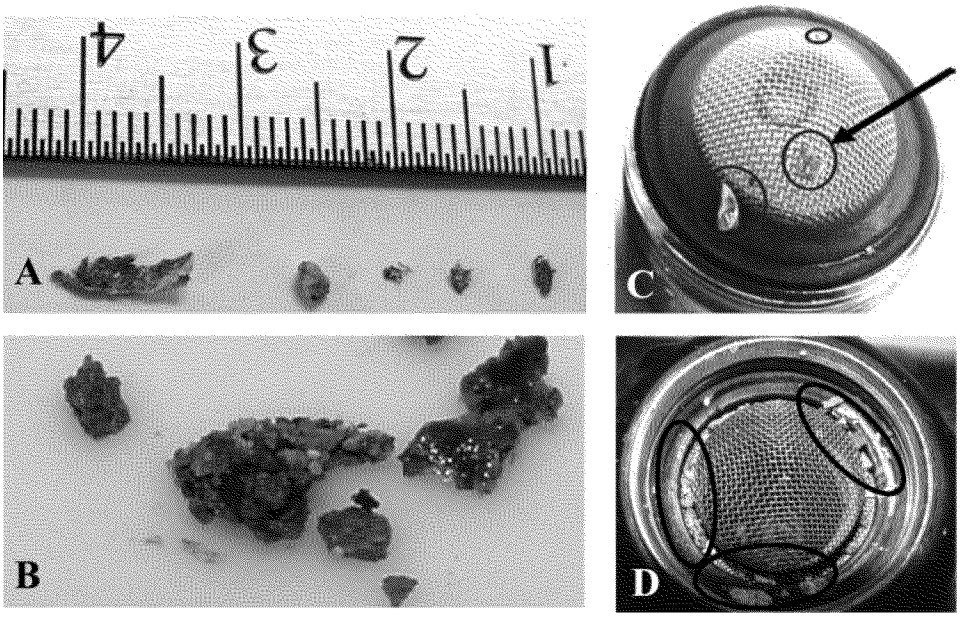

FIGURE 3. Lead-bearing particles were identified as the cause of severe tap water contamination during field investigations. (A and B) Brass particles trapped in two different strainers adjacent to two drinking water fountains at the University of North Carolina at Chapel Hill (Elfland et al., 2010). (C) Lead solder particles trapped in home faucet aerator screen in Washington, DC (Edwards, 2005). (D) Lead solder particles trapped in home faucet aerator screen in Greenville, North Carolina (Triantafyllidou et al., 2007).

been recognized that particulate lead can occasionally be the dominant form of lead in drinking water (Triantafyllidou et al., 2007). A preliminary synthesis (Table 6) indicates that such problems may not be an isolated occurrence, especially given the rarity of such measurements.

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

EPA-R5-2018-0081360000188

cause severe lead poisoning as they slowly dissolve. Bioavailability tests on lead solder particles collected from homes of lead-poisoned children in Greenville and Durham, North Carolina, 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 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 bioavailable once ingested.

In order to protect consumers from such exposures, corrosion control programs need to account for and 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 presently lacks the tools or knowledge to completely prevent or control particulate lead release.

V. BLOOD LEAD LEVEL AND MAJOR LEAD TOXICITY MECHANISMS

Potential harm from exposure to lead is typically tracked by measurements of the BLL. BLLs above 10 μg/dL are considered elevated (EBLLs) for infants and children, as they exceed the CDC threshold at which detectable mental impairment and behavioral changes have been documented (CDC, 2005). Cases in which blood lead exceeds 10 or 20 μg/dL are also termed *lead poisoning*, dependent on the specific U.S. jurisdiction. CDC surveillance for the year 2007 corresponded to only 13% (or else 3,136,843) of U.S. 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 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 of children (National Research Council Board on Environmental Studies and Toxicology, 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

EPA-R5-2018-0081360000188

TABLE 7. Blood lead level (BLL) and adverse health effects in children and in adults

	Age group	
	Children	Adults
BLL (μg/dL)		
<10	IQ (−), hearing (−), growth (−)	Uncertain
>10	Erythrocyte protoporphyrin (+)	Hypertension
>20	Nerve conduction (−)	Erythrocyte protoporphyrin (+)
>30	Vitamin D metabolism (−)	Systolic blood pressure (+) Hearing (−)
>40	Hemoglobin synthesis (−)	Nerve conduction (−), infertility (men), kidney failure
>50	Colic, frank anemia, kidney failure, brain disorders	Hemoglobin synthesis (−) frank anemia, brain disorders
>100	Death	Death

Note. The BLL of concern is presently set at 10 μg/dL. Adapted from Troesken (2006) and National Research Council Board on Environmental Studies and Toxicology (1993). Hemoglobin is the molecule which carries oxygen throughout the body. Nerve conduction is ability to send the impulse from the nerve to the muscle. Vitamin D is necessary for the absorption of calcium and phosphorus, and for bone growth. Erythrocyte protoporphyrin is the intermediate in heme biosynthesis. (−) = decreased function; (+) = increased function.

(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, and 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 (Mahaffey, 1985; Troesken, 2006, 2008).

Recent studies suggest that decreased IQ and cognition occur in children even at BLLs as low as 3.0 μg/dL (Bellinger and Needleman, 2003; Jusko et al., 2008), 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 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 U.S. Department of Health and Human Services (2000) established the ambitious goal of eliminating EBLLs in U.S. children by 2010. 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

EPA-R5-2018-0081360000188

eliminate EBLLs (i.e., BLLs ≥ 10 μg/dL) in children was not achieved, and the United States is extending this goal to 2020 (U.S. Department of Health and Human Services, 2010).

VI. 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 WLLs to 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 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 8) described subsequently, such data are often unavailable.

Lack of BLL Data for Sensitive Subpopulations

In the United States 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 1–6 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 (Edwards et al., 2009; Shannon and Graef, 1992).

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

EPA-R5-2018-0081360000188

TABLE 8. Potential difficulties in associating lead in water to lead in blood in population studies or in case studies

Issue	Illustrative reference(s)
BLLs and WLLs are not always available	
General lack of BLL measurements for sensitive sub-populations	Binder et al., 1996 Shannon et al., 1992
General lack of WLL measurements in schools/daycares under the LCCA	Edwards et al., 2009 Lambrinidou et al., 2010
Relatively small number of WLL measurements under the LCR	Renner, 2009
Relative exclusion of water lead measurements during home assessments of lead-poisoned children	Renner, 2009 Scott, 2009
WLL measurements do not always reflect actual lead in water	
Improper water sampling/preservation methods at "high-risk" taps under the LCR - Flow rate - Cold versus hot water - Sample preservation	Triantafyllidou et al., 2007 Triantafyllidou et al., 2009
Inherent variability in lead release from plumbing - Spatial (fluctuations within a city, a neighborhood, or even a single home) - Temporal (fluctuations in a single tap depending on season, or even time of day)	Levin, 2008 Schock, 1990 Matthew, 1981
Individual water consumption patterns affect individual exposure	
Variability in individual water consumption patterns - Amount of water consumed in/outside of home - Use of tap/filtered tap/bottled water	Troesken, 2006 Matthew, 1981
Underestimated indirect contribution of water to the total dietary lead intake - Preparation of foods and beverages	Triantafyllidou et al., 2007 Mesch et al., 1996 Moore, 1983
Individual risk factors affect individual response to a fixed lead dose	
Bioavailability of lead varies between individuals, depending on - Age - Diet - Genetics	Troesken, 2006 Lanphear et al., 2002 Matthew, 1981

analysis by the U.S. Government Accountability Office 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 U.S. 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.

EPA-R5-2018-0081360000188

Relative Exclusion of Water Lead Measurements During Assessments of Lead-Poisoned Children

Management strategies for childhood lead poisoning in the United States 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. Present 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 17 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, and 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 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 15 lead grantee municipalities routinely collected water samples during home inspections, and that 16 sometimes sampled drinking water (if lead was not found in paint or dust, or if drinking water was provided by a private well or 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 completely miss lead spikes (Table 8).

EPA-R5-2018-0081360000188

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 (US EPA, 2006b) 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 everyday 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 and 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 US EPA-recommended low flow rate missed 90% of the particulate lead present (Edwards, 2005), during a home investigation in Washington, DC, in 2006 (Figure 4).

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 USA EPA (2006b) 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

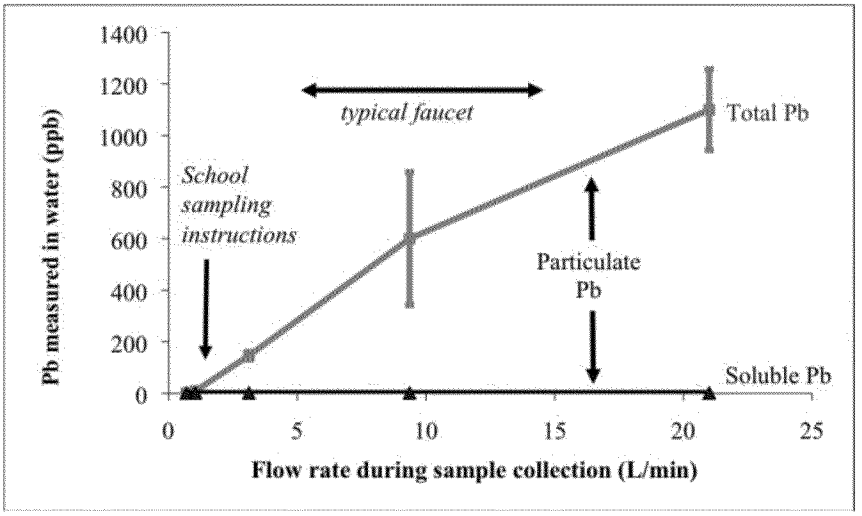

FIGURE 4. Lead measurement in flushed tap water samples versus flow rate in a home with lead pipe. Error bars represent 95% confidence intervals over triplicate samples collected on subsequent days at each indicated flow rate. Sample collection at the kitchen tap was timed to collect water derived from the lead pipe (Edwards, 2005).

EPA-R5-2018-0081360000188

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 and 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 (US EPA, 1994). Edwards and Dudi (2004) first showed that the standard US 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, 1981; Pocock, 1980). Yet present 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 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 et al., 2008). This translates to more variability in the lead concentration, compared with homes where water consumption is much more frequent and regular. In addition, pH or other chemical fluctuations, depending on how far from

EPA-R5-2018-0081360000188

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 that 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, tends 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 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. The Freedom of Information Act (Edwards, 2005) requests revealed that in four other flushed samples collected by the local water utility, lead in water ranged between 19 and 583 μg/L (Table 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.

Individual Water Consumption Patterns Affect Individual Exposure

Variability in Individual Water Consumption Patterns

In oversimplified terms the individual risk from lead-contaminated drinking water, or any other hazard, is also a function of exposure to that hazard.

TABLE 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).

Date	Lead determination (μg/L)	Sampling conducted by
7/26/2003	75	Water utility
3/23/2004	19	Water utility
3/23/2004	11	Department of Health
10/8/2004	21	Water utility
11/2/2004	583	Water utility

EPA-R5-2018-0081360000188

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 with 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 very 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 1 year old consumed on average 122 mL/kg of water a day if they were formula fed. This amount is about 3 times higher than the 44 mL/kg a day intake proposed by US 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 (CDC, 2010b; Edwards et al., 2009; Edwards, 2010). The key takeaway 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 (Baxter et al., 1992; Little et al., 1981; Moore, 1983). Specifically, vegetables can absorb 90% or more of the lead from the water they are cooked in (Moore, 1983). Smart et al. (1981)

EPA-R5-2018-0081360000188

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 (30–50%) than lead from food (10–15%; 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 gastrointestinal absorption rate of ingested lead is inversely related to age. The typical lead absorption rate for infants is 50%, compared with just 10% in adults (World Health Organization, 2000).

Dietary Habits. Diets low in calcium 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 23 adult volunteers, the lead retention from consumption of lead acetate was controlled by the type and 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 (US 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).

EPA-R5-2018-0081360000188

VII. 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 WLLs to BLLs (Table 10). These include, but are not limited to, the following:

- Focus on the most sensitive age groups (e.g., formula-fed infants, young children, 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 nonparametric correlations;
- Linear regression models versus curvilinear 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; and
- Association between WLL and BLL versus association between WLL and percentage of study population with EBLL.

Few studies are directly comparable, but nonetheless critically evaluating the available literature provides useful insights.

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 (World Health Organization, 2000). In fact, for bottle-fed infants using reconstituted formula with tap water, about 90% of their diet by weight is actually tap water, as 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 (World Health Organization, 2000), elevated lead in water is

TABLE 10. Representative population studies on the association between lead in water and lead in blood (in chronological order)

Sample population	Independent variable(s)	Dependent variable	Measure of association	Model	Reference
Different sectors of Scottish population (n = 949)	First-draw water lead (μg/L)	Blood lead level (μg/dL)	R = 0.52	BLL = 11.0 + 2.36(WLL)$^{1/3}$ (units adjusted)	Moore et al., 1977
Individuals in greater Boston (n = 524)	First-draw water lead (mg/L), other variables such as age, sex, education, dust lead	Blood lead level (μg/dL)	Model explains 19% of variance	Ln(BLL) = 2.73WLL − 4.70WLL^2 + 2.17WLL^3 + other terms for age, sex, education, dust [WLL was best predictor]	Worth et al., 1981
Mothers in Ayr, Scotland (n = 114)	Kettle water lead (μg/L)	Blood lead level (μg/dL)	R^2 = 0.56	BLL = 4.7 + 2.78(WLL)$^{1/3}$	Sherlock et al., 1984
Mothers in Ayr, Scotland (n = 114 from 1980–81, n = 116 from 1982–83)	Kettle water lead (μg/L)	Blood lead level (μg/dL)	R^2 = 0.65	BLL = 5.6 + 2.62(WLL)$^{1/3}$	Sherlock et al., 1984 Moore et al., 1985
Women in Wales (n = 192)	Kettle water lead (μg/L) Air lead (μg/m^{-3}) Dust lead (μg/g)	Blood lead level (μg/dL)	Model explains 38% of variance	Log(BLL) = 1.06 + 0.62(WLL)$^{1/3}$ + 0.18Log (ALL) − 0.02Log(DLL)	Elwood et al., 1984
Bottle-fed infants in Scotland (n = 93)	Composite kettle water lead (μg/L)	Blood lead level (μg/dL)	R = 0.57	BLL = 14 + 0.062WLL BLL = 15.6 + 0.052WLL BLL = 14.7 + 0.054WLL BLL = 15.4 + 0.052WLL	Lacey at al., 1985 World Health Organization, 2000
Adults in Vosgian Mountains, France (n = 155 men, n = 166 women)	Tap water lead after 5 s of flushing (mg/L)	Blood lead level (μg/dL)	Spearman's ρ = 0.30 for men and 0.47 for women	Not determined	Bonnefoy, et al., 1985
Children in Edinburgh, Scotland (n = 397)	Tap water lead (μg/L), dust lead (μg/g)	Blood lead level (μg/dL)	Model explains 43% of variance	Log(BLL) = 0.5Log(5326 + 103WLL + 3.81DLL) [WLL was best predictor]	Raab et al., 1987
Different sectors of population in Hawaii, with rain catchment systems (n = 384)	Tap water lead (μg/L), other water-related terms, other terms for soil and demographics	Blood lead level (μg/dL)	linear model explains 77% of variance	Linear model: BLL = 5.62 + 0.025WLL + 0.0008(GLASSES WLL) − 0.017(FILTER WLL) + other terms related to water, soil, age, sex, ethnicity etc.	Maes at al., 1991

(Continued on next page)

EPA-R5-2018-0081360000188

TABLE 10. Representative population studies on the association between lead in water and lead in blood (in chronological order) *(Continued)*

Sample population	Independent variable(s)	Dependent variable	Measure of association	Model	Reference
Citizens of Sante-Agathe-des-Monts, Québec, Canada (n = 72)	Average water lead from 6 samples (mg/L) and estimated daily water consumption (L/day)	Blood lead level (μg/dL)	R^2 = 0.25	BLL = 10 + 7 × WLL × water consumption (units adjusted)	Savard, 1992
School children in southern Saxonia, Germany (n = 69 for location A, n = 44 for location B)	Composite tap water lead (μg/L)	Blood lead level (μg/dL)	Location A: R^2 = 0.34 Location B: R^2 = 0.41	Location A: Log(BLL) = 0.74 + 0.14Log(WLL) Location B: Log(BLL) = 0.81 + 0.14Log(WLL)	Englert et al., 1994
Mothers in Glasgow, Scotland (n = 342)	Water lead (μg/L)	Blood lead level (μg/dL)	Spearman's ρ = 0.39	Not determined	Watt et al., 1996
Women in Hamburg, Germany (n = 142 for subsample with detectable water lead)	Average water lead (μg/L) from 3 specimens	Blood lead level (μg/dL)	Spearman's ρ = 0.43	Not determined	Fertmann et al., 2004
Children in Washington, DC (n = 2698 in high risk; n = 4791 in moderate risk; n = 2621 in low risk)	90th percentile water lead (μg/L)	% Increase in children with EBLL compared with U.S. average	R^2 = 0.83 in high risk; R^2 = 0.71 in moderate risk; R^2 = 0.50 in low Risk	Not determined	Edwards et al., 2009

Note. ALL = air lead level; BLL = blood lead level; DLL = dust lead level; WLL = water lead level.

EPA-R5-2018-0081360000188

EPA-R5-2018-0081360000188

a very significant concern for this population group. Infants typically consume 500–1000 mL of formula per day (World Health Organization, 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:

$$\frac{90\,\mu\text{g Pb}}{\text{L water}} \cdot \frac{0.75\,\text{L formula}}{\text{day}} \cdot 90\%\,\text{water in formula} = 61\,\mu\text{g Pb/day} \qquad (1)$$

Ryu et al. (1983) found that 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 5). Another group of infants, exposed to only 16 μg Pb/day through their diet, did not develop elevated blood lead (Figure 5). On this basis Ryu 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 of 25 μg/kg/week (or else 3.5 μg/kg/day) set by the World Health Organization (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.

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

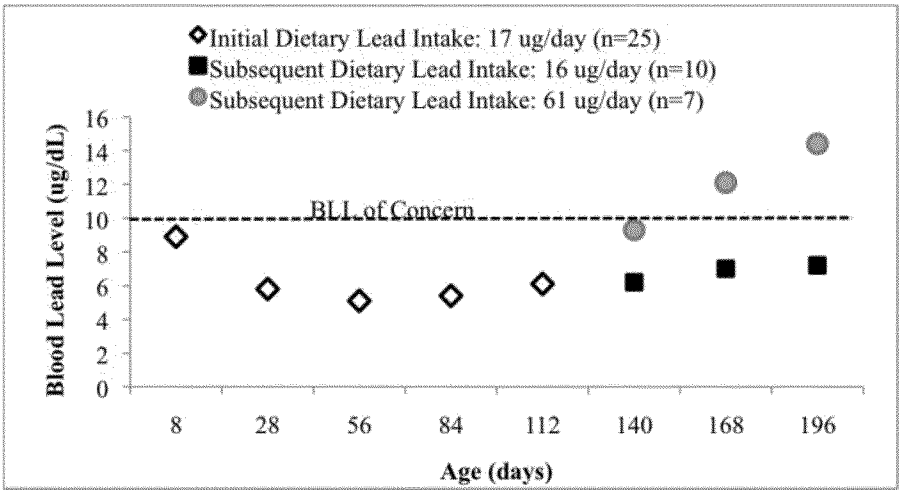

FIGURE 5. Average blood lead level (BLL) versus age for two groups of formula-fed infants, at two levels of dietary lead intake. Adapted from data in Ryu et al. (1983).

EPA-R5-2018-0081360000188

quantified. A simple linear relationship between lead in water collected from kettles and infant blood lead level was derived, with a correlation coefficient (R^2) of 0.32 (Table 10). This work demonstrates that due to genetic and other factors mentioned previously, perfect correlations are not to be expected between lead in water and lead in blood, even for the most susceptible subpopulation to lead exposure from water.

An investigation by Shannon and Graef (1992) revealed nine cases where lead poisoning occurred in Boston infants after consuming instant formula reconstituted with lead-contaminated water. 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 and Graef, 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 curvilinear relationship between water lead and blood lead was that of Moore et al. (1977), which yielded a correlation coefficient (R) of 0.52 by analyzing data from different sectors of the Scottish population (Table 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 with 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 curvilinear relationship (Table 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 (R^2) of 0.56 between kettle water lead and blood lead level (Table 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 (R^2) of 0.65 between kettle water lead and blood lead (Table 10). After increasing the pH of the water supply, water lead levels significantly dropped, and median blood lead levels also dropped from 21 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 10). For water lead levels up to 20 μg/L, the BLLs of men and

EPA-R5-2018-0081360000188

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, Scotland, 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 10). Coefficients for water and dust were significant in their model (Table 10), and the authors concluded that water lead was more important than dust in this population. An 8-year follow-up study of the same individuals in central Edinburgh, showed a dramatic decrease in their water lead and blood lead levels, which was attributed to improved corrosion control and removal of lead pipes from plumbing (Macintyre 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 with 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 the US EPA to influence formulation of the 1991 US EPA LCR, which in turn introduced modern corrosion control strategies for lead in U.S. 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

EPA-R5-2018-0081360000188

average lead concentration in tap water and lead concentration in blood for 142 young women (Spearman's ρ = 0.43, p < .0001; Table 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 ($p \leq .001$). 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). Fertmann et al. (2004) concluded that lead in tap water stands for an avoidable surplus exposure.

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, respectively (Englert and Horing, 1994). Lead pipes were used in about 50% of their houses. After log-transformation of their blood lead levels and their drinking water lead levels, 34% of the variation in blood lead levels was explained by log-WLL 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 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 that 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 newborn babies in various regions of southern Saxonia (Zietz et al., 2001). Overall, 3.1% of the 1,434 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 et al., 2001).

Following a case of lead intoxication by drinking water in Sainte-Agathe-des-Monts, Canada, a 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's $\chi^2(\ ) = 5.85$, $p = .02$ (Savard, 1992). A mathematical model was developed for the 72 citizens for which WLLs were measured (Table 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 (R^2) of 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

EPA-R5-2018-0081360000188

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 (Gwiazda et al., 2005; Rust et al., 1999).

Watt et al. (1996) assessed the relationship between tap water lead and maternal blood lead concentrations in Glasgow, Scotland, 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 World Health Organization 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 to 24 months old were monitored in Rochester, New York, a community not considered to have lead-in-water problems according to the US 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 ($p < .001$). 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 suboptimal 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 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 90th percentile lead in water concentration. Earlier studies on Washington, DC (Guidotti et al., 2007; Stokes et al., 2004), 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

EPA-R5-2018-0081360000188

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 biokinetic 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 United States (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 that 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 noncorrosive (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, Florida (Gasana et al., 2006), also found no association of BLLs to WLLs (Spearman's ρ = 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 three homes. However, correlations between BLL and floor dust (ρ = 0.27) and windowsill (ρ = 0.28) were statistically significant (p < .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 ρ = 0.4, p < .0001), with lead in soil (Spearman's ρ = 0.3, p < .001), and with lead in paint (Spearman's ρ = 0.2, p < .01), but not with lead in water (Spearman's ρ = 0.14, p = ns). 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 voltammetry. This analytical technique has recently been shown to accurately measure dissolved Pb^{+2},

EPA-R5-2018-0081360000188

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 et al. (1985) study. Moreover, samples were allowed to sit unacidified before analysis, which is now recognized to potentially miss some of the lead present in water (M. Rabinowitz, personal communication, December 10, 2006). Perhaps, partly because of these issues, only very low levels of lead (3.7–7.3 μg/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 μg/L during the same time period. Boston water, which was linked to lead poisoning via infant formula, was reported by Shannon and Graef (1989) to contain 132 μg/L. Even as late as 1996–2000, lead levels in first-draw tap water samples from Boston were 159 μg/L on average, and as high as 311 μg/L in the worst case for children with elevated blood lead (State of Massachusetts, Water Quality Assurance Section, Drinking Water Program, personal communication, November 2006). Even flushed water samples for lead poisoned children in the 2009 data from Massachusetts contained as high as 146 μg/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 10). For example, Costa et al. (1997) reported that very high water lead levels in a public school in rural Utah (up to 840 μg/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 (World Health Organization, 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 μg/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 appeared 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 that masked disparities among different neighborhoods (Edwards et al., 2009). Finally, as mentioned previously, virtually

EPA-R5-2018-0081360000188

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 (CDC, 2010b; Edwards et. al., 2009). The no-harm conclusion of Guidotti et al. (2007) has since been removed (Guidotti et al., 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 vs. filtered or bottled water) 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 that 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), 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 with those consuming bottled water (Leroyer et al., 2000). 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.

EPA-R5-2018-0081360000188

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., >10 μg/dL) of exposed populations. As would be expected based on present understanding of dietary intake and hand-to-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) exemplifies rigorously controlled studies that are unlikely to be improved on 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 U.S. studies (Bornschein et al., 1985; Rabinowitz et al., 1985), did not find any association between lead in water and in blood for children in Cincinnati and Boston, respectively. 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 United Kingdom, and the United States 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 reanalyzed, corrected, or clarified (CDC, 2010b; Edwards et al., 2009; Edwards, 2010; Guidotti et al., 2009; Miller, 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 (Englert et al., 1994; Fertmann et al., 2004; Lanphear et al., 2002).

VIII. 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 United States and other countries have

EPA-R5-2018-0081360000188

been attributed to lead-contaminated drinking water. Existing U.S. regulations and guidelines have not eliminated lead in water hazards in systems served by public water supplies, schools, day cares, 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 U.S. 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 U.S. 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 United States, the United Kingdom, 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 subpopulations, is timely considering forthcoming revisions to the LCR and acknowledged deficiencies in addressing lead in school drinking water.

IX. 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 U.S. 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-water at schools 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 vs. 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

EPA-R5-2018-0081360000188

either detect or completely miss lead spikes, especially when problems with particulate lead in water are important. The occurrence of particulate lead spikes in U.S. 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 and 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, were 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.

ACKNOWLEDGMENTS

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. The authors would also like to thank Dr. Yanna Lambrinidou for her useful suggestions, as well as the anonymous reviewers for their valuable comments. Portions of sections VI and VII were presented at the Yale University Drinking Water Symposium in April 2009 and appeared as a supplemental paper of that symposium.

REFERENCES

Agency for Toxic Substances and Disease Registry. 2007. *Lead toxicity: What are the U.S. standards for lead levels?* Retrieved from http://www.atsdr.cdc.gov/csem/lead/pb_standards2.html

Anonymous. (1923). Lead helps to guard your health (advertisement of the National Lead Company). *National Geographic* 44.

EPA-R5-2018-0081360000188

Bach D. (2005). Schools' drinking water still fails lead test. *Seattle Post-Intelligencer*, Wednesday July 20. Accessed on 6/9/08 at http://seattlepi.nwsource.com/local/233287_lead20.html

Baxter, M. J., Burrell, J. A., Crews, H. M., Smith A., and Massey R. C. (1992). Lead contamination during domestic preparation and cooking of potatoes and leaching of bone-derived lead on roasting, marinading and boiling beef. *Food Additives and Contaminants* 9, 225–235.

Biesecker, M. (2006, August 25). Lead found in schools' water. *The News and Observer*. Retrieved from at http://www.newsobserver.com/2006/08/25/53632/lead-found-in-schools-water.html#storylink=misearch

Binder, S., Matte, T. D., Kresnow, M., Houston, B., Sacks, J. J. (1996). Lead testing of children and homes: results of a national telephone survey. *Public Health Rep.* 111, 342–346.

Bisogni, J. J. Jr., Nassar, I. S., and Menegaux, A. M. (2000). Effect of calcium on lead in soft-water distribution systems. *ASCE Journal of Environmental Engineering* 126, 475–478.

Blette, V. (2008). Drinking water public right-to-know requirements in the United States. *Journal of Water and Health* 6, s43–s51.

Bolduc, G. 2006, September 20. Lakehurst Acres residents' tests indicate lead in blood. *Kennebec Journal*.

Bolduc, G. (2010, June 6). Lead levels watched. *Kennebec Journal*.

Bonnefoy, X., Huel, G., and Gueguen, R. (1985). Variation of the blood lead level as a result of lead contamination of the subjects drinking water. *Water Res.* 19, 1299–1303.

Bornschein, R. L., Hammond, P. B., Dietrich, K. N., Succop, P., Krafft, K., Clark S., Berger, O., Pearson, D., and Que Hee, S. (1985). The Cincinnati prospective study of low-level lead exposure and its effects on child development: Protocol and status report. *Environmental Research* 38, 4–18.

Boyd, G. R., Pierson, G. L., Kirmeyer, G. J., Britton, M. D., and English, R. J. (2008a). Lead release from new end-use plumbing components in Seattle public schools. *Journal of the American Water Works Association* 100(3), 105–114.

Boyd, G. R., Pierson, G. L., Kirmeyer, G. J., and English, R. J. (2008b). Lead variability testing in Seattle public schools. *Journal AWWA* 100(2), 53–64.

Britton, A. and Richards, W.N. (1981). Factors influencing plumbosolvency in Scotland. *J. Inst.Water Eng. Scient.* 35, 349–364.

Bryant, S. D. (2004). Lead-contaminated drinking water in the public schools of Philadelphia. *Journal of Toxicology Clinical Toxicology* 42, 287–294.

Burke, G. (2009, September 25). School drinking water contains toxins. *Associated Press*. Retrieved from http://www.usatoday.com/news/topstories/2009-09-25-3560128572_x.htm

Burney, M., and Dwight, O. (2003, March 13). Schools must surrender lead reports. *The Philadelphia Inquirer* B01.

California Environmental Protection Agency. (1997). *Public health goal for lead in drinking water*. Retrieved from http://oehha.ca.gov/water/phg/pdf/lead_c.pdf

Cartier, C., Prevost, M., Shokoufeh, N., and Pirog, M. (2009). *A rapid method for detection of lead service lines*. Paper presented at the Water Quality Technology Conference, Seattle, Washington.

EPA-R5-2018-0081360000188

Centers for Disease Control and Prevention. (1994). Lead-contaminated drinking water in bulk-water storage tanks—Arizona and California, 1993. Retrieved from http://www.cdc.gov/mmwr/preview/mmwrhtml/00032974.htm

Centers for Disease Control and Prevention. (2002). Managing elevated blood lead levels among young children: Recommendations from the Advisory Committee on Childhood Lead Poisoning Prevention. Atlanta, GA: U.S. Department of Health and Human Services. Retrieved from http://www.cdc.gov/nceh/lead/CaseManagement/caseManage_main.htm

Centers for Disease Control and Prevention. (2004a). Blood lead levels in residents of homes with elevated lead in tap water-District of Columbia. *Morb. Mort. Weekly Rep.* 53, 268–270.

Centers for Disease Control and Prevention. (2004b, June 18). *Brief report: Lead poisoning from ingestion of a toy necklace—Oregon, 2003*. Retrieved from http://www.cdc.gov/mmwr/preview/mmwrhtml/mm5323a5.htm

Centers for Disease Control and Prevention. (2005). *Preventing lead poisoning in young children*. Atlanta, GA: Author.

Centers for Disease Control and Prevention. (2006, March 23). *Death of a child after ingestion of a metallic charm—Minnesota, 2006*. Retrieved from http://www.cdc.gov/mmwr/preview/mmwrhtml/mm5512a4.htm

Centers for Disease Control and Prevention. (2009). Lead—national surveillance data (1997–2007). Retrieved from http://www.cdc.gov/nceh/lead/data/national.htm

Centers for Disease Control and Prevention. (2010a). *Lead tips: Sources of lead in water*. Retrieved from http://www.cdc.gov/nceh/lead/tips/water.htm

Centers for Disease Control and Prevention. (2010b). *Notice to readers: Limitations inherent to a cross-sectional assessment of blood lead levels among persons living in homes with high levels of lead in drinking water*. Retrieved from http://www.cdc.gov/mmwr/preview/mmwrhtml/mm5924a6.htm

Chambers, V. K., and Hitchmough, S. M. (1992). *Economics of lead pipe replacement* (TMU. 9030). Final report to the Department of the Environment, DOE 2956/1. Swindon, England: Water Research Center.

City of Baltimore. (2007, November 7). *Baltimore city public schools' CEO announces system-wide shift to bottled drinking water*. Press release.

Clark, C., Bornschein, R., Succop, P., Que Hee, S., Hammond, P., and Peace, B. (1985). Condition and type of housing as an indicator of potential environmental lead exposure and pediatric blood lead levels, *Environmental Research* 38(1), 46–53.

Consumer Product Safety Commission (CPSC), Office of Information and Public Affairs (2005, February 3). *CPSC Announces New Policy Addressing Lead in Children's Metal Jewelry*. Retrieved from http://www.cpsc.gov/cpscpub/prerel/prhtml05/05097.html

Copeland, L. (2004, March 3). Blood and water: The long search for the source of a baby's lead poisoning. *Washington Post*. Retrieved from http://washingtonpost.com/wp-dyn/articles/A28828-2004Mar3.html

Cosgrove, E. V., Brown, M. J., Madigan, P., McNulty, P., Okonski, L., and Schmidt, J. (1989). Childhood lead poisoning: Case study traces source to drinking water. *J. Environ. Health.* 52, 346–349.

EPA-R5-2018-0081360000188

Costa, R. A., Nuttall, K. L., Shaffer, J. B., Peterson, D. L., and Ash, K. O. (1997). Suspected lead poisoning in a public school. *Ann. Clin. Lab. Sci.* 27, 413–417.

De Mora, S. J., Harrison, R. M., and Wilson, S. J. (1987). The effect of water treatment on the speciation and concentration of lead in domestic tap water derived from a soft upland source. *Water Res.* 21, 83–94.

De Rosa, S., and Williams, S. M. (1992). *Particulate lead in water supplies* (TMU 9024). Final Report to the Department of the Environment. Swindon, England: Water Research Center.

DeMarco, J. (2004). *A partial lead service line replacement program! Does it make sense?* Paper presented at the U.S. EPA Workshop on *Lead Service Replacement*, October 26–27.

Edwards, D. (2007, March 31). Teacher discovers water tainted by lead at West Davidson high school. *The Dispatch*. Retrieved from http://www.the-dispatch.com/apps/pbcs.dll/article?AID=/20070331/NEWS/703310341/1005/news

Edwards, M. (2005, October 31). *Lead copper rule sampling and public health goals for lead*. Presented at the AEESP Scientists Luncheon. Washington, DC.

Edwards, M. (2010). *Preventing harm—protecting health: Reforming CDC's environmental public health practices*. Testimony before the U.S. House of Representatives Committee on Science and Technology, 111th Congress.

Edwards, M., and Dudi, A. (2004). Role of chlorine and chloramine in corrosion of lead-bearing plumbing materials. *Journal of the American Water Works Association* 96(10), 69–81.

Edwards, M., Scardina, P., Loganathan, G. V., Bosch, D., and Dwyer, S. (2007). *Non-uniform corrosion in copper piping—assessment*. AwwaRF Report #91217.

Edwards, M., Scardina, P., and Parks, J. (2006). Lead in Durham water: Site visit report, Durham, NC.

Edwards, M., Triantafyllidou, S., and Best, D. (2009). Elevated blood lead in young children due to lead-contaminated drinking water: Washington, DC, 2001–2004. *Environmental Science and Technology* 43, 1618–1623.

Elfland, C., Scardina, P., and Edwards, M. (2010). Lead contaminated water from brass plumbing devices in new buildings. *Journal AWWA* 102(11), 66–76.

Elwood, P. C., Gallacher, J. E. J., Phillips, K. M., Davies, B. E., and Toothill, C. (1984). Greater contribution to blood lead from water than from air. *Nature* 310, 138–140.

Englert, H., and Horing, H. (1994). Lead concentration in tap water and in blood of selected schoolchildren in southern Saxonia. *Toxicol. Lett.* 72, 325–331.

Fadrowski, J. J., Navas-Acien, A., Tellez-Plaza, M., Guallar, E., Weaver, V. M., Furth, S. L. (2010). Blood lead level and kidney function in U.S. adolescents: The third national health and nutrition examination survey. *Arch. Intern. Med.* 170(1), 75–82.

Fertmann, R., Hentschel, S., Dengler, D., Janssen, U., and Lommel, A. (2004). Lead exposure by drinking water: An epidemiological study in Hamburg, Germany. *International Journal of Hygiene and Environmental Health* 207, 235–244.

Fitzgerald, S. (2000, November 21). District: Unsafe lead levels found in 20% of water outlets in testing water in 289 older school buildings. The district knew of the problem years ago, an EPA official testified. *The Philadelphia Inquirer* B01.

EPA-R5-2018-0081360000188

Freking, K. (2009, December 9). EPA unveils plan for safe drinking water. *The Daily Reporter*. Retrieved from http://dailyreporter.com/blog/2009/12/09/epa-unveils-plan-for-safe-drinking-water/

Frumkin, H. (2010, January 12). *Important update: Lead-based water lines*. CDC's Healthy Homes and Lead Poisoning Prevention Branch. Retrieved from http://www.cdc.gov/nceh/lead/waterlines.htm

Galke, W., Dixon, S., McLaine, P., Wilson, J., and Ashley, P. (2006). *Study of HUDs risk assessment methodology in three U.S. communities*. Report prepared for the U.S. Department of Housing and Urban Development. Retrieved from http://www.nchh.org/LinkClick.aspx?fileticket=HZUenslvU/0=&tabid=217

Gasana, J., Hlaing, W. M., Siegel, K. A., Chamorro, A., and Niyonsenga, T. (2006). Blood lead levels in children and environmental lead contamination in Miami inner city, Florida. *Int. J. Environ. Res. Public Health* 3, 228–234.

Gerwin, S. (2004). *Lead in drinking water in schools and child care facilities meeting summary*. Retrieved from http://www.epa.gov/ogwdw/lcrmr/pdfs/summary_lcmr_review_school__12–07–04.pdf

Goss, C. (2008). *Lead ban surveillance project*. Pennsylvania Department of Environmental Protection, Bureau of Water Standards and Facility Regulation Safe Drinking Water Program. Retrieved from http://www.healthyhomestraining.org/codes/State_Codes/PA_Products_Lead_Plumbing_8–14-09.pdf

Greenwire. (2004). Lead: Schools adopt varying guidelines to reduce levels in drinking water. Section Wastes & Hazardous Substances. Vol. 10 No. 9, July 27, 2004.

Grover, J. (2008a, April 27). School water. *KNBC Channel 4 News Extra*.

Grover, J. (2008b, May 22). LAUSD outlines plan. Contaminated water: Expensive fix. *KNBC Channel 4*.

Guidotti, T. L., Calhoun, T., Davies-Cole, J. O., Knuckles, M. E., Stokes, L., Glymph, C., Lum, G., Moses, M. S., Goldsmith, D. F., and Ragain, L. (2007). Elevated lead in drinking water in Washington, DC, 2003–2004: The public health response. *Environ. Health Perspect.* 115, 695–701.

Guidotti, T. L., Calhoun, T., Davies-Cole, J. O., Knuckles, M. E., Stokes, L., Glymph, C., Lum, G., Moses, M. S., Goldsmith, D. F., and Ragain, L. (2009). Errata: Elevated lead in drinking water in Washington, DC, 2003–2004: The public health response. *Environ. Health Perspect.* 117, A342.

Gulson, B. L., James, M., Giblin, A. M., Sheehan, A., and Mitchell, P. (1997). Maintenance of elevated lead levels in drinking water from occasional use and potential impact on blood leads in children. *Science of the Total Environment* 205, 271–275.

Gwiazda, R., Campbell, C., and Smith, D. (2005). A noninvasive isotopic approach to estimate the bone lead contribution to blood in children: Implications for assessing the efficacy of lead abatement. *Environ. Health Perspect.* 113, 104–110.

Hayes, C. R., and Skubala, N. D. (2009). Is there still a problem with lead in drinking water in the European Union? *Journal of Water and Health* 7, 569–580.

HDR Engineering. (2009). An analysis of the correlation between lead released from galvanized iron piping and the contents of lead in drinking water. Retrieved from http://www.dcwasa.com/waterquality/plumbing/galvanized.cfm

EPA-R5-2018-0081360000188

Health Canada. (1992). *Guidelines for Canadian drinking water quality*. Retrieved from at http://www.hc-sc.gc.ca/ewh-semt/alt_formats/hecs-sesc/pdf/pubs/water-eau/lead/lead-plomb-eng.pdf

Health Canada. (2009). *Guidance on controlling corrosion in drinking water distribution systems*. Ottawa, Ontario, Canada: Water, Air and Climate Change Bureau, Healthy Environments and Consumer Safety Branch, Health Canada.

Heard, M.J., Chamberlain, A.C., and Sherlock, J.C. (1983). Uptake of lead by humans and effect of minerals and food. *Science of the Total Environment* 30, 245–253.

Hulsmann, A. D. (1990). Particulate lead in water supplies. *Journal of the Institute of Water and Environmental Management* 4(1), 19–25.

Jacobs, D. E. (1995). Lead-based paint as a major source of childhood lead poisoning: a review of the evidence. In Beard, M. E., and Iske, S. D. A. (Eds.), *Lead in paint, soil and dust: Health risks, exposure studies, control measures and quality assurance* (pp. 175–187). Philadelphia, PA: American Society for Testing and Materials.

James, H. M., Hilburn, M. E., and Blair, J. A. (1985). Effects of meals and meal times on Uptake of lead from the gastrointestinal tract in humans. *Human & Experimental Toxicology* 4, 401–407.

Jusko, T. A., Henderson, C. R., Lanphear, B. P., Cory-Slechta, D. A., Parsons, P. J., and Canfield, R. L. (2008). Blood lead concentrations <10 μg/dL and child intelligence at 6 years of age. *Environ. Health Perspect.* 116, 243–248.

Kalra, V., Gulati, S., Chitralekha, K. T., Pande, P., Makhijani, S. D., and Sharma, C. S. (2000). Plumbism: A mimicker of common childhood symptoms. *Indian J. Pediatr.* 67, 81–86.

Karalekas, P. C. Jr., Craun, G. F., Hammonds, A. F., Ryan, C. R., and Dorth, D. J. (1976). Lead and other trace metals in drinking water in the Boston Metropolitan area. *Journal AWWA* 90, 150–172.

Lacey, R., Moore, M., and Richards, W. (1985). *Lead in water, infant diet and blood*: The Glasgow duplicate *diet* study 1979–1980. *Sci. Total. Environ.* 41, 235–257.

Lambrinidou, Y., and Edwards, M. (2008). *Lead in DC public school drinking water: Filter monitoring and follow up testing*. Preliminary report prepared for the District of Columbia Public Schools.

Lambrinidou, Y., Triantafyllidou, S., and Edwards, M. (2010). Failing our children: Lead in U.S. school drinking water. *New Solutions* 20(1), 25–47.

Lanphear, B. P., Hornung, R., Ho, M., Howard, C. R., Eberley, S., and Knauf, K. (2002). Environmental lead exposure during early childhood. *Journal of Pediatrics* 140, 40–47.

Leonnig, C., Becker, J. and Nakamura, D. (2004, October 5). Lead levels in water misrepresented across U.S. Utilities manipulate or withhold test results to ward off regulators. *Washington Post* A01. Retrieved from http://www.washingtonpost.com/wp-dyn/articles/A7094-2004Oct4.html

Leonnig, C. (2009, August 4). D.C., U.S. underreported number of kids with high lead levels by more than half. *Washington Post*. Retrieved from http://www.washingtonpost.com/wp-dyn/content/article/2009/08/03/AR2009080303003.html

EPA-R5-2018-0081360000188

Leroyer, A., Nisse, C., Hemon, D., Gruchociak, A., Salomez, J.-L., and Haguenoer, J.-M. (2000). Environmental lead exposure in a population of children in northern France: Factors affecting lead burden. *American Journal of Industrial Medicine* 38, 281–289.

Levallois, P., Gingras, S., Caron, M., and Phaneuf, D. (2008). Drinking water intake by infants living in rural Quebec (Canada). *Science of the Total Environment* 397, 82–85.

Levin, R., Brown, M. J., Kashtock, M. E., Jacobs, D. E., Whelan, E. A., Rodman, J., Schock, M. R., Padilla, A., Sinks, T. (2008). Lead exposures in U.S. children, 2008: Implications for prevention. *Environmental Health Perspectives* 116, 1285–1293.

Linakis, J. G., Anderson, A. C., and Pueschel, S. M. (1996). Screening for lead poisoning. In Siegfried, M., Pueschel, J. G., and Linakis, A. C. (Eds.), *Lead poisoning in childhood* pp. 59–73. Baltimore, MD: Brookes.

Little, P., Fleming, R. G., and Heard, M. J. (1981). Uptake of lead by vegetable foodstuffs during cooking. *Sci. Tot. Environ.* 17, 111–131.

Lockitch, G., Berry, B., Roland, E., Wadsworth, L., Kaikov, Y., and Mirhady, F. (1991). Seizures in a 10-week-old infant: Lead poisoning from an unexpected source. *Canadian Medical Association Journal* 145, 1465–1468.

Loghman-Adham, M. (1997). Renal effects of environmental and occupational lead exposure. *Environ. Health Perspect.* 105, 928–938.

Lubin, A. H., Signs, S. A., and Caffo, A. (1984). Tapwater lead in homes of children with undue body lead burden. *The Ohio State Medical Journal* 80, 547–551.

Macintyre, C., Fulton, M., Hepburn, W., Yang, S., Raab, G., Davis, S., Heap, M., Halls, D., and Fell, G. (1998). Changes in blood lead and water lead in Edinburgh. An eight year follow-up to the Edinburgh lead study. *Environmental Geochemistry and Health* 20, 157–167.

Maes, E. F., Swygert, L. A., Paschal, D. C., and Anderson, B. S. (1991). The contribution of lead in drinking water to levels of blood lead. I. A cross-sectional study.

Mahaffey, K. R. (1977). Quantities of lead producing health effects in humans: Sources and bioavailability. *Environ. Health Persp.* 19, 285–295.

Mahaffey, K. R. (Ed.). (1985). *Dietary and environmental lead: Human health effects.* Amsterdam, the Netherlands: Elsevier.

Marcus, W. L. (1986). Lead health effects in drinking water. *Toxicol. Ind. Health* 2, 363–407.

Matthew, G. K. (1981). Lead in drinking water and health. *The Science of the Total Environment* 18, 61–75.

McCafferty, P., Schulz, R., and D'Ercole R. (1995). Lead contamination in Perth drinking water. *Chemistry in Australia* 62, 21–22.

McNeill, L. S., and Edwards, M. (2004). Importance of Pb and Cu particulate species for corrosion control. *Journal of Environmental Engineering-ASCE* 130, 136–144.

Mesch, U., Lowenthal, R. M., and Coleman, D. (1996). Lead poisoning masquerading as chronic fatigue syndrome. *The Lancet* 347(9009), 1193.

Meyer, I., Heinrich, J., Trepka, M. J., Krause, C., Schulz, C., Meyer, E., and Lippold, U. (1998). The effect of lead in tap water on blood lead in children in a smelter town. *Sci. Total Environ.* 209, 255–271.

EPA-R5-2018-0081360000188

Mielke, H. W., and Reagan, P. L. (1998). Soil is an important pathway of human lead exposure. *Environmental Health Perspectives* 106, 217–229.

Miller, B. (2010). *A public health tragedy: How flawed CDC data and faulty assumptions endangered children's health*. Retrieved from http://www.dcwatch.com/wasa/100520.pdf

Montgomery County Public Schools. (2004). *Updated analysis of drinking water samples*. Retrieved from http://www.montgomeryschoolsmd.org/departments/maintenance/leadinformation/docs/WaterAnalysisList10-11.pdf

Montgomery County Public Schools. (2007). *Schools remediation status*. Retrieved from http://www.montgomeryschoolsmd.org/departments/maintenance/leadinformation/remediationschedule.pdf

Moore, M. (1983). Lead exposure and water plumbosolvency. In Rutter, M., and Jones, R. R. (Eds.), *Lead versus health: Sources and effects of low level lead exposure* (pp. 79–106). Hoboken, NJ: Wiley.

Moore, M. R., Meredith, P. A., Campbell, B. C., Goldberg, A., and Pocock, S. J. (1977). *Contribution of lead in drinking water to blood lead. Lancet* 2, 661–662.

Moore, M. R., Richards, W. N., and Sherlock, J. G. (1985). Successful abatement of lead exposure from water supplies in the west of Scotland. *Environ. Res.* 38, 67–76.

Moralez, L. S., Gutierrez, P., and Escarce, J. J. (2005). Demographic and socioeconomic factors associated with blood lead levels among Mexican-American children and adolescents in the United States. *Public Health Rep.* 120, 448–454.

National Research Council Board on Environmental Studies and Toxicology. (1993). *Measuring lead exposure in infants, children, and other sensitive populations*. Washington, DC: National Academy Press.

Needleman, H. (2004). Lead poisoning. *Annu. Rev. Med.* 55, 209–222.

O'Flaherty, E. J. (1998). A physiologically based kinetic model for lead in children and adults. *Environ. Health Perspect.* 106, 1495–1503.

Odell, L. (1991). Reducing lead in school drinking water: A case study. *School Business Affairs* 12, 14–18.

Osawa, T. (2002). Treatment of lead in water pipe in Osaka city. *Osaka and Its Technology Magazine* 42, 28–37.

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

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

Pocock, S.J. (1980). Factors influencing household water lead: a British national survey. *Arch. Environ. Health* 35, 45–51.

Potula, V., Serrano, J., Sparrow, D., and Hu, H. (1999). Relationship of lead in drinking water to bone lead levels twenty years later in Boston men: The normative aging study. *Journal of Occupational & Environmental Medicine* 41, 349–355.

Powell, M. (1999). *Science at EPA: Information in the regulatory process*. Washington, DC: Resources for the Future.

Raab, G. M., Laxen, D. P. H., and Fulton, M. (1987). Lead from dust and water as exposure sources for children. *Environ. Geochem. Health* 9(3–4), 80–85.

EPA-R5-2018-0081360000188

Rabinowitz, M., Leviton, A., Needleman, H., Bellinger, D., and Waternaux, C. (1985). Environmental correlates of infant blood lead levels in Boston. *Environ. Res.* 38, 96–107.

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

Ramsay, C. (2003). *Scottish new homes lead survey. Stage 2.* Glasgow, Scotland: Scottish Centre for Infection and Environmental Health. Retrieved from http://www.documents.hps.scot.nhs.uk/environmental/scottish-new-homes-lead-survey/scottish-new-homes-stage-2.pdf

Renner, R. (2007). Public health: Plumb crazy. *Science* 315(5819), 1669.

Renner, R. (2009). Out of plumb: When water treatment causes lead contamination. *Environ. Health Perspect.* 117, A543–A547.

Renner, R. (2010). Exposure on tap: Drinking water as an overlooked source of lead. *Environ. Health Perspect.* 118, a68–a72.

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

Rust, S. W., Kumar, P., Burgoon, D. A., Niemuth, N. A., and Schultz, B. D. (1999). Influence of bone-lead stores on the observed effectiveness of lead hazard intervention. *Environ Res* 81, 175–184.

Ryu, J. E., Ziegler, E. E., Nelson, S. E., and Fomon, S. J. (1983). Dietary intake of lead and blood lead concentration in early infancy. *Am. J. Dis. Child* 137, 886–891.

Sandvig, A., Kwan, P., Kirmeyer, G., Maynard, B., Mast, D., Rhodes, R. T., Trussell, S., Cantor, A., and Prescott, A. (2008). *Contribution of service line and plumbing fixtures to lead and copper rule compliance issues.* AwwaRF 91229.

Sathyanarayana, S., Beaudet, N., Omri, K., and Karr, C. (2006). Predicting children's blood lead levels from exposure to school drinking water in Seattle, Washington, U.S.A. *Ambulatory Pediatrics* 6, 288–292.

Savard, M. (1992, August). *Rapport d'intervention en sante publique pour le plomb d'origine hydrique a Ste-Agathe-de Monts.* Departement de sante communautaire de Laurentides. Report in French.

Schock, M. R. (1990). Causes of temporal variability of lead in domestic plumbing systems. *Environ. Monit. Assess.* 15, 59–82.

Schock, M. R., Hyland, R. N., and Welch, M. M. (2008). Occurrence of contaminant accumulation in lead pipe scales from domestic drinking-water distribution systems. *Environ. Sci. Technol.* 42, 4285–4291.

Schock, M. R., Wagner, I., and Oliphant, R. J. (1996). *Corrosion and solubility of lead in drinking water in internal corrosion of water distribution systems.* In Denver, CO: AWWA Research Foundation.

Schwartz, B. S., Lee, B.-K., Lee, G.-S., Stewart, W. F., Simon, D., Kelsey, K., and Todd, A. C. (2000). Associations of blood lead, dimercaptosuccinic acid-chelatable lead, and tibia lead with polymorphisms in the vitamin D receptor and δ-aminolevulinic acid dehydratase genes. *Environ. Health Perspect.* 108, 949–954.

Scott, R. (2009). *Public health agency response to lead-contaminated drinking water.* Paper presented at the American Public Health Association Annual Conference and Exposition, Philadelphia, Pennsylvania.

Shannon, M., and Graef, J. W. (1989). *Lead intoxication from lead-contaminated* water used to reconstitute infant *formula. Clin. Pediatr.* 28, 380–382.

EPA-R5-2018-0081360000188

Shannon, M., and Graef, J. (1992). *Hazard of lead in infant formula* (letter). *New England Journal of Medicine* 326, 137–138.

Shannon, M. W. (1996). Etiology of childhood lead poisoning. In Siegfried, M., Pueschel, J. G., and Linakis, A. C. (Eds.), *Lead poisoning in childhood* (pp. 37–57). Baltimore, MD: Brookes.

Sherlock, J. C., Ashby, D., Delves, H. T., Forbes, G. I., Moore, M. R., Patterson, W. J., Pocock, S. J., Quinn, M. J., Richards, W. N., and Wilson, T. S. (1984). Reduction in exposure to lead from drinking water and its effect on blood lead concentrations. *Human Toxicol.* 3, 383–392.

Sherlock, J. C., and Quinn, M. J. (1986). Relationship between blood lead concentrations and dietary lead intake in infants: The Glasgow duplicate diet study 1979–1980. *Food Addit. Contam.* 3, 167–176.

Showman, R. (1994). *Experiences with lead-free brass in the manufacture of cast valves and fittings.* Paper presented at the American Water Works Association Annual Conference, New York.

Singhal, R. A., and Thomas, J. A. (Eds.). (1980). *Lead toxicity*. Baltimore, MD: Urban & Schwarzenberg.

Slaats, N., Blocker, M., and Versteegh, A. (2007). *Sampling of metals at the tap: Analyses of Dutch data over the period 2004–2006*. Paper presented at Meteau Conference, Turkey.

Smart, G. A., Warrington, M., Dellar, D., and Sherlock, J. C. (1983). Specific factors affecting lead uptake by food from cooking water. *J. Sci. Food Agric.* 34, 627–637.

Smart, G., Warrington, M., and Evans, W. H. (1981). The contribution of lead in water to dietary lead intakes. *J. Sci. Food Agric.* 32, 129–133.

State of Massachusetts, Water Quality Assurance Section, Drinking Water Program. (2006). Personal Communication on 11/2006.

Stokes, L., Onwuche, N. C., Thomas, P., Davies-Cole, J. O., Calhoun, T., Glymph, A. C., Knuckles, M. E., Lucey, D., Cote, T., Audain-Norwood, G., Britt, M., Lowe, M. L., Malek, M. A., Szeto, A., Tan, R. L., Yu, C., Eberhart, M., Brown, M. J., Blanton, C., Curtis, G. B., and Homa, D. M. (2004). Blood lead levels in residents of homes with elevated lead in tap water—District of Columbia, 2004. *MMWR Morbidity and Mortality Weekly Report* 53, 268–270.

Taylor, P. Deputy Director of Facilities Management. District of Columbia Public Schools. Personal Communication, November 2, 2007.

Triantafyllidou, S., Nguyen, C., and Edwards, M. (2009a). *Contribution of galvanic corrosion to lead (Pb) in water after partial lead service line replacements.* Paper presented at the Water Quality Technology Conference, Seattle, Washington.

Triantafyllidou, S., Parks, J., and Edwards, M. (2007). Lead particles in potable water. *Journal AWWA* 99, 107–117.

Triantafyllidou, S., Raetz, M., and Edwards, M. (2009b). *Lead in drinking water and associated public health risk.* Paper presented at the AWWA Annual Conference, San Diego, California.

Troesken, W. (2006). *The great lead water pipe disaster*. Cambridge, MA: MIT Press.

Troesken, W. (2008). Lead water pipes and infant mortality at the turn of the twentieth century. *Journal of Human Resources* 43, 553–575.

EPA-R5-2018-0081360000188

U.S. Census Bureau. (2000). *Year structure built and year household moved into unit* [Table QT-H7]. Washington, DC: Author. Retrieved from http://factfinder.census.gov/servlet/QTTable?_bm=y&-geo_id=01000US&-qr_name=DEC_2000_SF3_U_QTH7&-ds_name=DEC_2000_SF3_U

U.S. Department of Health and Human Services. (2000). Healthy people 2010: Understanding and improving health. Washington, DC.

U.S. Department of Health and Human Services. (2010). Proposed healthy people 2020 objectives. Washington, DC. http://healthypeople.gov/2020/topicsobjectives2020/pdfs/EnvironmentalHealth.pdf

U.S. Environmental Protection Agency. (1991). Safe drinking water act lead and copper rule (LCR). *Federal Register* 56, 26460–26564.

U.S. Environmental Protection Agency. (1993). *Lead in your drinking water*. Factsheet. EPA 810-F-93–001.

U.S. Environmental Protection Agency. (1994a). *Method 200.8: Determination of trace elements in waters and wastes by ICP-MS*. Revision 5.4. Cincinnati, OH: Environmental Monitoring Systems Laboratory, Office of Research and Development, U.S. Environmental Protection Agency.

U.S. Environmental Protection Agency. (1994b). *Lead in drinking water in schools and non-residential buildings*. EPA 812-B-94–002. Retrieved from http://www.epa.gov/ogwdw/consumer/pdf/leadinschools.pdf

U.S. Environmental Protection Agency. (2000). Rules and Regulations. *Federal Register* 65, 1965.

U.S. Environmental Protection Agency. (2002). *Users guide for the integrated exposure uptake biokinetic model for lead in children (IEUBK)*. Washington, DC: Office of Solid Waste and Emergency Response.

U.S. Environmental Protection Agency. (2004). *Controlling lead in drinking water for schools and day care facilities: A summary of state programs*. Retrieved from http://www.epa.gov/safewater/lcrmr/pdfs/report_lcmr_schoolssummary.pdf

U.S. Environmental Protection Agency. (2006a). *Air quality criteria for lead, Volume I*. EPA/600/R-5/144aF.

U.S. Environmental Protection Agency. (2006b). *3Ts for reducing lead in drinking water in schools*. Revised Technical Guidance, EPA 816-B-05–008. Retrieved from http://www.epa.gov/OGWDW/schools/pdfs/lead/toolkit_leadschools_guide_3ts_leadschools.pdf

U.S. Environmental Protection Agency. (2006c). *Private drinking water wells*. Retrieved from http://www.epa.gov/safewater/privatewells/index2.html

U.S. Environmental Protection Agency. (2010, June 4). Strict lead air, dust rules spur EPA to focus on drinking water risks. *Inside EPA Newsletter*.

U.S. Environmental Protection Agency. (2011). *Questions about the disposal of lead-contaminated items*. Retrieved from http://www.epa.gov/osw/hazard/tsd/lead/faq.htm

Watt, G. C. M., Britton, A., Gilmour, W. H., Moore, M. R., Murray, G. D., Robertson, S. J., and Womersley, J. (1996). Is lead in tap water still a public health problem? An observational study in Glasgow. *British Medical Journal* 313, 979–981.

Weston, R. F., and Economic and Engineering Services. (1990). *Lead service line replacement, a benefit-to-cost ratio analysis*. Denver, CO: American Water Works Association and Water Industry Technical Action Fund.

EPA-R5-2018-0081360000188

Wetmur, J. G., Lehnert, G., and Desnick, R. J. (1991). The delta-aminolevulinate dehydratase polymorphism: Higher blood lead levels in lead workers and environmentally exposed children with the 1–2 and 2–2 isozymes. *Environ. Res.* 56, 109–119.

World Health Organization. (1993). *Guidelines for drinking-water quality* (2nd ed.). Retrieved from http://www.who.int/water_sanitation_health/dwq/2edvol1c.pdf

World Health Organization. (2000). *Safety evaluation of certain food additives and contaminants*. WHO Food Additives Series, No. 44, 2000.

Worth, D., Matranga, A., Lieberman, M., De Vos, E., Karelekas, P., Ryan, C., and Craun, G. (1981). Lead in drinking water: The contribution of household tap water to blood lead levels. In Lynam, D. R., Piantanida, L. G., and Cole, J. F. (Eds.), *Environmental lead* (pp. 199–225). New York, NY: Academic Press.

Zietz, B., Dassel de Vergara, J., Kevekordes, S., and Dunkelberg, H. (2001). Lead contamination in tap water of households with children in Lower Saxony, Germany. *Sci. Total Environ*. 275, 19–26.