Occurrence of lead in drinking water fountains at California State University, Sacramento:

A health risk assessment

Amaryl Griggs

ENVS 190A

December 15, 2016
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Abstract

Lead is a harmful contaminant that can be leached into drinking water from lead pipes, and other components in water systems. Low levels of lead exposure cause adverse health effects in humans, especially young children. Lead concentrations were measured at drinking fountains from several buildings on the California State University, Sacramento campus. This was a joint project between the Environmental Studies and Chemistry Departments to assess lead concentrations in drinking water fountains on the CSUS campus. Lead was detected in four buildings in at least one sampling day throughout the study. Lead was consistently detected in one building on each sampling day, ranging from 1.3 ug/L to 8.86 ug/L. Concentrations from fountains were converted to blood lead levels of both children and adults to determine risk using The EPA’s Integrated Exposure, Uptake and Biokinetic (IEUB) as well as an EPA model using biokinetic slope factor from a compilation of past lead data.

Introduction

The chemical element, lead, is a naturally occurring, soft heavy metal that exists in a solid state. Its molecular weight is 207.2 g/mol and its boiling and melting points are 3164°F and 621°F respectively. Elemental lead is insoluble in water but can react with air or water to form lead sulfates, oxides and, carbonates (U.S. NLM, 2007). These reactions help to form a protective layer around the metal, which allows it to resist environmental corrosion. Lead reacts with oxidizing compounds such as sodium carbide and chlorine trifluoride. Due to its malleability and resistance to corrosion, it has been widely used in water service pipes.

In the U.S., the use of lead pipes for water distribution began in the 1800s, and by the 1900s over half of the major cities had lead water systems (Rabin, 2008). Today, lead is still commonly found in pipes, car batteries, ammunition, glassware, imported foods, and toys. Its use in ammunition has been targeted for reduction due to its harmful effects in the environment.
Lead-based paints were widely used in the United States until they were banned in 1978 and can still be found in many pre-1978 built houses. Lead was also used as an additive in gasoline, but was eventually phased out through the 1980s until it was banned in 1996 (ASTDR, 2007). Though lead in gasoline has been largely phased out, lead can still exist in the environment in soil and water. Lead is readily adsorbed to soil particles and can remain in the top layers of soil, so it is not likely transported to groundwater. It can then be transported to rivers and lakes when rainwater washes away soil particles. Terrestrial plants can absorb lead from soil, which can then be deposited in the plant tissue, but this can vary by the concentration of lead in the soil and the species of plant (Eisler, 1988). Bioaccumulation of lead occurs in both terrestrial and aquatic plants and animals, meaning that lead ingested or inhaled can be stored in their tissues over time. Biomagnification, however, does not occur through the food chain.

*Exposure*

Ingestion of lead-based paint is still one of the major exposure routes for children in the United States (ASTDR, 2007). Lead-based paint was banned in 1978, but millions of homes built before then may have old and weathered paint that can be ingested by children and hand to mouth behavior. Ingestion of contaminated water another one route of exposure for lead. Generally, low amounts of lead are found in lakes or rivers as well as publicly supplied drinking water. Drinking water is the source of nearly 20% of human lead exposure in the United States and 50% of lead exposure for formula fed infants (Triantafyllidou and Edwards, 2012). Lead in drinking water most often occurs at the tap when lead piping and lead-containing components leach lead into the water. Lead poisoning in children living in North Carolina and Maine was traced to lead particles from solder that entered the drinking water, which can happen as the solder becomes corroded (Triantafyllidou, 2007). Water that sits stagnant in pipes may have
higher concentrations of leached lead in a first draw as corrosion of the pipes can release lead in the form of larger particulates or dissolved lead (Triantafyllidou and Edwards, 2012). Acidic water can also affect the amount of corrosion of lead components, and most public water treatment systems take this into account when treating water (ASTDR, 2007). In 2014, Flint, Michigan’s water distribution systems did not properly treat water for the city, and lead was leached into the water supply due to the lack of corrosion inhibitors (Hanna-Attisha et al., 2015). During the disinfection process at water distribution systems, increasing the amount of chloramine to reduce disinfection by-products has also been seen to elevate lead at the customer taps. Nationally, less than 1% of water systems produce water with lead levels of about 5μg/L (ASTDR, 2007). Currently, the EPA has set an action level under the Lead and Copper Rule for lead at 15μg/L, whereby public distribution systems must take steps to reduce the corrosivity of water if more than 10% of water sampled at taps exceed this value. Unfortunately, this approach does not protect all consumers, since out of the 100 homes that must be sampled per city, only 90 of them must fall below the 15μg/L threshold to be in compliance. This means that a small percentage of home water systems may contain lead at well over 15μg/L, and the city would still be in compliance. This action level is not to be confused with a maximum contaminant level, as it only serves as a guideline to economically manage water systems. The EPA recognizes that there is no safe level of lead and its stated maximum contaminant level goal is 0. The Lead and Copper Rule does not apply to schools and child care facilities that source water from public water systems, which account for more than 90% of schools in the U.S (EPA, 2006). These facilities are under the Lead and Contamination Control Act which only provides lead guidelines that are unenforceable. Schools under this act are not held to the action level of 15μg/L or any national standard, but rather they are recommended to not exceed 20μg/L in the first 250mL.
draw of water samples. In California, a Public Health Goal for lead has been lowered to 0.2 μg/L from its original value of 2 μg/L due to the growing evidence of adverse effects of lead at lower levels (OEHHA, 2009).

Buildings that were built before 1986 are more likely to contain lead pipes or plumbing fixtures because it was not until 1986 that an amendment to the Safe Drinking Water Act banned the use of lead pipes or solder. This amendment requires that all new plumbing be “lead free” by which lead can only account for 0.25% of pipe surfaces that have contact with water and 0.2% lead content for solder (EPA, 2016). Though this amendment has reduced the amount of new lead pipes to be used in plumbing, many old neighborhoods and buildings still have lead piping and connections. Many water systems use brass or bronze fittings, which are alloys that contain lead. These fittings can still be considered lead free even if they contain up to 8% lead by weight, though new brass components containing only up to 0.25% lead by weight are now required for use in California (Sandvig et al., 2008). Even new plumbing materials that comply with the “lead free” regulation can still leach some lead into water depending on the water chemistry or the amount of time the water has sat stagnant near the tap before use. New drinking fountains and water sources with new end components in Seattle Public schools were monitored and shown to have elevated lead levels in the first draw of water, indicating that the new plumbing components were contributing to lead contamination (Boyd, 2008). These elevated lead levels occurred right after installation of new drinking fountain components, but after many repeated uses and exposure to water there was a decline in the lead levels. It is not enough to just replace an old drinking water source that has lead contamination; after any replacement, the water should be tested again for lead.
Often, full replacement of lead water lines is difficult due to ownership of the piping by both the city’s utilities as well as the homeowner. Some cities have opted to partially replace lead water lines, while leaving behind the portion of lead lines that are owned by homeowners. In the long term, this has shown to reduce lead in water but lead concentrations may increase in the first few days or weeks due to the release of lead scale that has accumulated over time (Sandvig et al., 2008).

Inhalation of airborne lead particulates is the second route of lead exposure. Lead contamination in the air can come from industrial pollution involved with metal production such as iron, steel, brass, and lead-acid battery production (ASTDR, 2007). Lead may also become airborne from lead-based paint dust, cigarette smoke, or even from burning material containing lead. Leaded gasoline was once one of the major contributors of lead air pollution, but since its gradual elimination lead air concentrations have dropped nationally by over 90% between the 1980s and 2002 (EPA, 2006). Unfortunately, because lead does not degrade in the environment, lead particles that have adsorbed to soil can be suspended in the air as dust by wind. Particles can then be transported through the atmosphere and be deposited far from their original source. Since lead has been deemed a criteria air pollutant, the EPA has set its National Ambient Air Quality Standard through the Clean Air act at 0.15μg/m³ for an averaging time of three months (EPA, 2016). This standard level was set as both a primary standard to protect sensitive populations including the elderly and children as well as a secondary standard to protect the public welfare and damage to animals, crops or vegetation. Between 2000 and 2004, the average lead concentration measured at monitoring sites across the U.S. ranged from 0.10 to 0.22μg/m³ (EPA, 2006).
Populations at Risk

Due to its prevalence and persistence in the environment, nearly all of the general population is at risk for lead exposure. Those that work in industries involving lead may be at greater risk of higher lead exposure compared to the general population and children and fetuses of pregnant women may be at greater risk for the adverse effects of lead exposure. People who work in construction, manufacturing and demolition may be exposed to lead by breathing in lead particles. This is especially true for those that work in industries that involve lead refining, brass and solder production and battery manufacturing. Over 200,000 workers in California are exposed in these types of industries, and this can be harmful to their families and children as lead dust can be transferred through work clothing (ASTDR, 2007). To protect these employees, the Occupational Safety and Health Administration (OSHA) has set a permissible exposure limit (PEL) of 50 μg/m³ of lead per eight-hour work time (OSHA, 2012). Though this limit is in place, some occupations such as those that involve waste incineration expose workers to lead levels that can be as high as over 2,000 μg/m³ (ASTDR, 2007).

Children are particularly susceptible to adverse effects caused by lead exposure. Younger children are prone to ingest lead contaminated dust, soil or paint as they play or crawl on the ground (ASTDR, 2007). Infants are also likely to be exposed to lead through drinking water if they are primarily fed formula. Lead can also be present in breast milk and can influence an infant’s blood lead level (Levin et al., 2008). Children eating imported candy, usually from Mexico, are also potentially exposed to lead through both the candy itself and the food wrapper. Imported toys may also be lead contaminated and in 2007 over 2 million lead-contaminated toys originating from China were recalled. A child’s blood lead level (BLL) is usually highest at 15-24 months, which could be due to the fact that children often put their
hands or objects in their mouth as well as because their bodies retain and absorb lead better than adults (Tong et al., 1996). Children whose parents work in fields that involve lead exposure may also be exposed to higher levels of lead from dust on clothing. A study of children with parents in lead-related occupations found that 31% had BLLs that were over 10 μg/dL of blood compared to only 11% in the control group (Dilshad Ahmed et al., 2010). The National health and Nutrition Examination Surveys determined that between 1999 and 2002, nearly 2% of children nationally between 1 and 5 years old had BLLs over 10 μg/dL (ASTDR, 2007). As of 2012, the Center for Disease Control (CDC) has an action level for elevated child blood levels at 5 μg/dL. As newer evidence suggests that the effects of lead can occur at even lower values, this new level replaces the “level of concern” of 10 μg/dL, which did not encompass the number of children that had BLLs below that value.

Effects of Exposure

Lead has toxic effects on many systems of the body including the nervous, reproductive, cardiovascular, and renal systems. According to the EPA, there is sufficient animal evidence to assign inorganic lead as a probable human carcinogen, but since there is little human evidence available, lead does not have a cancer slope factor. The EPA also has not determined a reference dose for lead since it essentially has no threshold and can cause adverse health effects at very low levels. At BLLs less than 10 μg/dL, lead can inhibit heme biosynthesis in blood by disrupting the δ-aminolevulinic acid dehydratase (ALAD) enzyme, leading to anemia by shortening the lifespan of red blood cells as well as increase hormones that allow blood cells to mature too rapidly (ASTDR, 2007). Elevated blood pressure in adults exposed to low levels of lead can occur, which can increase their susceptibility to glomerular disease, which is a decrease in kidney function. In a study of 840 adults ranging in age from 21 to 80, increasing lead
concentration in the bones, but not the blood, was associated with increasing blood pressure and risk of hypertension (Cheng et al., 2001). This might suggest that long-term, cumulative lead exposure, which is associated with bone deposition and not BLL, is correlated with increased blood pressure. No threshold for the disruption of ALAD by lead has been determined, with effects correlated with BLLs of 3-34 μg/dL in people living in urban conditions (Hernberg and Nikkanen, 1970). Lead is also thought to cause hypertension by reacting with nitric oxide, which is crucial in blood pressure regulation. A symptom of acute lead poisoning with BLLs of 100-200 μg/dL in those exposed to lead through occupation is colic, with symptoms including cramps, nausea, weight loss and vomiting (ASTDR, 2007). For children, colic symptoms can begin between 60-100 μg/dL. BLLs of 33-120 μg/dL in children are associated with inhibition of vitamin D conversion in the body, but this effect is easily reversed by chelation therapy (Rosen et al., 1980). High levels of lead in both adults in children can lead to encephalopathy, or decreased brain function (ASTDR, 2007). These effects can include memory loss, hallucinations, coma, and finally death. These effects have been found in several studies to start in adults anywhere between 50 and over 300 μg/dL due to the variability in factors such as age and genetics. Symptoms of acute lead poisoning in children can appear between 60-450 μg/dL and encephalopathy associated with convulsions and coma between 80 and 800 μg/dL. Cases of encephalopathy have been reported in infants of about 4 months of age at BLLs of about 50-331 μg/dL (ASTDR, 2007). Neurological effects in children may even start at lead levels below 10 μg/dL, but clinical detection is much more difficult. Children with elevated BLLs are also seen to score much lower on IQ tests and have lower cognitive ability and information retention when compared to other children. As BLL increases by every 10 μg/dL in children, IQ is lowered by up to seven points (ASTDR 2007). Fertility in men can also be reduced as BLL increases. For
women who are pregnant, there is a greater chance for miscarriages if exposed to high levels of lead. Pregnant women working in a Swedish smelter industry were seen to have a greater frequency of miscarriage if they worked or lived near the plant during their pregnancy (Nordstrom et al., 1979). It is clear that high, occupational lead exposure has an adverse effect on pregnancy outcomes. Pregnant women with BLLs of 5-9 μg/dL are also more likely to have a spontaneous abortion and this risk nearly doubles for each 5 μg/dL increase in lead.

*Transport and Metabolism*

Lead enters the body through inhalation or oral exposure, but in its inorganic form it is not readily absorbed through the skin. During oral exposure, absorption occurs in the gastrointestinal tract where rates are influenced by factors such as age, presence of food, and particle size (ASTDR, 2007). For children up to 8 years of age, absorption is about 50% while for adults, absorption of ingested lead is only about 10%. Lead in water is seen to be absorbed by up to 63% in adults when taken without a meal, while absorption is only 3% when a meal is consumed (James et al., 1985). In rats, tissue absorption of lead was higher when the dosage contained particles with diameter less than 38 μm compared to rats that ingested particles that were up to 250 μm in diameter (Barltrop and Meek, 1979).

Once in the body, lead is transferred to the blood where it is primarily carried within red blood cells, and for adults the blood half-life is about 30 days (ASTDR, 2007). Autopsies of adults revealed that over 90% of the lead in the body is stored in the bones, whereas in children just over 70% of lead is stored in bone tissue (Barry, 1975). By forming complexes, lead can be stored in the bone by replacing calcium. Because of this sequestering of lead, it can still be detected throughout a lifetime in blood even if the main sources of exposure are reduced. This is especially concerning for the fetuses of pregnant women because bone mass is lost to the blood.
during pregnancy to aid the growth of the fetus. Though not as much as in the bones, some lead is also stored in the soft tissues, primarily in the liver and skeletal muscle (ASTDR, 2007).

Lead in its inorganic form is metabolized by forming complexes with ligands, including δ-aminolevulinic acid dehydratase (ALAD), which becomes inhibited during the beginning of the heme biosynthesis pathway. Inorganic lead, which is usually only found in occupational settings, is metabolized in the liver by cytochrome (CYP) P450 (ASTDR, 2007). Metabolites of inorganic lead such as triethyl and trimethyl lead, are neurotoxic and can then end up in the kidney, liver, and brain tissue. Excretion occurs through almost every bodily route including urine, feces, sweat, and breast milk, with feces being the primary excretion route.

*Dose-Response Relationship*

Lead is a unique toxic substance in that there is growing evidence to suggest that there is not any level of lead that would produce a no-observable-adverse-effect-level (NOAEL). It is also difficult to directly link the dose of lead from the environment that causes an adverse effect because lead effects are usually studied in terms of lead concentrations in either the blood or bones, though pharmacokinetic models have been developed to assess this. The Scientific Committee on Health and Environmental Risks (SCHER) in Europe has suggested NOAEL levels based on available data for the purpose of risk characterization (SCHER, 2009). For the purpose of this study and risk characterization, suggested levels will be considered lowest observed adverse effect levels (LOAEL). A LOAEL for child IQ development is at about 5 μg/dL of blood, which is also the level that the CDC considers a child’s BLL to be elevated. The SCHER has suggested a NOAEL of 45 μg/dL for reduced male fertility and a NOAEL of 10 μg/dL for developmental neurotoxicity for pregnant women. In animal studies, the CDC has
reported an LD\textsubscript{10} for a lead compound of 1,500 mg/kg body weight in pigs (CDC, 2014). An LD\textsubscript{10} is the lowest lethal dose given to the test subjects that caused death.

**Materials and Methods**

Eight buildings built before and after 1986 were chosen for lead sampling on the CSUS campus. In total, 80 samples were collected from 31 different fountains. Sampling was conducted on five separate days between March and June of 2016. Collection occurred in the early morning to ensure there was no student or faculty use in order to capture the maximum exposure amount of the fountain’s first flush. About 100mL of water was collected in 250mL polypropylene bottles which were washed with 5% nitric acid. Before final sample collection, about 25mL was first collected and poured out to condition the bottle. One mL of nitric acid was added to each sample after collection and each was stored at 4\textdegree C for preservation before analysis. Replicates and blanks were also collected in each building for quality control and quality assurance. Analysis was done by the CSUS Chemistry department using atomic absorption spectroscopy. The detection limit for analysis done in March was 0.3 μg/L and the limit of quantification was 1 μg/L. For all other analyses the detection limit and limit of quantification was 0.17 μg/L and 0.57 μg/L respectively. A batch of samples was also sent to the contract lab, BSK associates, and was found to not be statistically different than our results.

Exposure was calculated for the highest detection in each building of our study, and calculations are done as if these levels remain constant. The EPA’s Integrated Exposure, Uptake and Biokinetic (IEUB) model for lead in children is used to determine what BLL might be predicted due to the levels of lead in CSUS drinking fountains. The model offers a way to convert between environmental lead levels to levels in blood. For adults, the EPA has used a biokinetic slope factor to relate BLLs and lead uptake in water from past data. From this model
they were able to estimate that BLL increases by 0.4 μg/dL per μg/day of lead ingested (EPA, 2003).

**Results**

Drinking water fountains from eight buildings on the CSUS campus were sampled and analyzed for lead using atomic absorption spectroscopy. Lead concentrations that were detected are only from the first draw of water from each fountain. Of 31 fountains, four were found to have quantifiable lead levels. All other samples were either below detection limit or below the limit of quantification. Of the 8 sampled buildings, Alpine had detectable levels ranging from 1.30-8.86 μg/L. Amador had one fountain with a level of 1.82 μg/L. Mariposa had detections ranging from 0.51-1.36 μg/L and Sequoia had detections ranging from 0.97-1.19 μg/L (Figure 1). The fountain in Alpine had consistent detections for each day that it was sampled.

Using the IEUB model, children ages 6 months to seven years getting their total daily water from Alpine would experience a BLL increase of 0.5-0.9 μg/dL. Children sourcing their drinking water from Amador would have a BLL increase of 0.1-0.2 μg/dL. Finally, children drinking water from Sequoia and Mariposa would experience a BLL increase of 0.1 μg/dL. The average adult ingests 1043 mL/day of water. If an adult were to ingest all of his or her water for the day from Alpine, then the total lead ingested would be 9.24 μg/day and their BLL would increase by 3.70 μg/dL. If an adult were to source all of his or her drinking water from Amador, then the total lead ingested would be 1.90 μg/day and the BLL would increase by 0.76 μg/dL. As for Mariposa and Sequoia, the total lead ingested would be 1.42 and 1.24 μg/day and the BLL would increase by 0.57 μg/dL and 0.50 μg/dL respectively.
Discussion

There is no set reference dose for lead that a person can be exposed to each day in his or her lifetime. This is because the amount of lead in the body varies and can increase over time due to lead being stored in the tissues and bones. The highest BLL concentration calculated for both adults and children was from the fountain in Alpine. The BLL calculated for adults in Alpine was 3.70 µg/dL which is below the LOAEL for both effects on male fertility and developmental effects for fetuses in pregnant women. This does not mean that drinking from this water source has absolutely no effect on health due to lead because this BLL only adds to any other sources that the person may be getting lead contamination from, including other water sources, food and air. The calculation used to convert between environmental lead and blood lead also did not take women or pregnant women into consideration as the study was only based on adult men. This could mean that the actual BLL for women or pregnant women may be higher than calculated here.

The increase in BLL that children may experience from drinking from the Alpine fountain was found to be 0.5-0.9 µg/dL, which is below the CDCs value of 5 µg/dL blood for elevated blood lead. Again, the amount of lead children would ingest from the Alpine fountain is only one source out of many that they can be exposed to on any given day. Drinking from this fountain alone will not be the sole cause for health effects, but the amount of lead from this fountain adds to the child’s total BLL from past exposures. Taking this into consideration, even if the amount of lead from the fountain is lower than EPAs action value of 15 µg/L, there is still some risk to develop adverse health effects from drinking the fountain’s water.
Figure 1. Quantifiable results for lead sampling across five days. Amador Fl 3 was not resampled on 13, 16 or 23 of June. Mariposa was not resampled on 13 or 16 of June.
References Cited


CDC, 2014, Lead compounds (as Pb), http://www.cdc.gov/niosh/idlh/7439921.html (October 2, 2016)


Environmental Protection Agency, 2016, NAAQS Table. https://www.epa.gov/criteria-air-pollutants/naaqs-table#1 (September 3, 2016)


