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A RETROSPECTIVE STUDY OF EPA’S RECORD OF DECISION REPORT ON THE WOOLFOLK ARSENIC AND LEAD CONTAMINATION IN FORT VALLEY, GEORGIA

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Abstract
Heavy metal contamination is a major anthropogenic challenge in communities with heavy presence of chemical industries. Most of the contamination is due to the improper disposal of chemical waste. The study involved the evaluation of the levels of arsenic and lead regarding remediation activities carried out by the Environmental Protection Agency (EPA). It compared the results of the EPA’s 1993 soil analysis of arsenic and lead, and a subsequent soil analysis of the same mapped areas. It also covered the evaluation of the level of arsenic dust in the attics of residential properties and evaluation of the EPA’s record of testing of arsenic-exposed residents of Fort Valley, Georgia. The results revealed that there is a high level of arsenic still present in the mapped areas cleaned by EPA. Therefore, there is a need for continuous monitoring of health status of the people who live in the affected area.

Keywords: Environmental Protection Agency, Arsenic Contamination, Lead Contamination, Woolfolk Chemical Company, Fort Valley, Georgia

Introduction
Arsenic exists naturally in the earth, largely distributed in the air, water, and land, and it occurs in many forms, including in sulfur and metals. Arsenic is primarily used in the form of metallic alloy combined with lead in the production of car batteries and ammunition. Arsenic combines with oxygen to form oxides. Such compounds, especially trioxide, are used in the production of pesticides such as herbicides and insecticides. Inorganic arsenic is toxic and cannot be destroyed but can only change its form. In its natural form, arsenic readily dissolves in water, when washed out of the air by rain before settling into the ground; this poses a toxic threat to the environment (Shrivastava, 2017; Agency for Toxic Substances and Disease Registry [ATSDR], 2015). The greatest toxic threat to public health from arsenic originates from contaminated groundwater (Quansah and Arman, 2015). Oxides of arsenic in soil are a contaminant when a concentration of arsenic in the soil exceeds 20ppm (Smedley and Kinniburgh, 2002). Much of the basis for the current understanding of the mechanism of bio-accumulation of arsenic in living systems comes from scientific work in animals. In the late 1800s and during the early1900s, arsenic drugs were produced for the treatment of diseases such as pellagra and malaria. However, arsenic have been shown to cause cytotoxic effects in single cells (Gorby, 1988).

The signs and symptoms of acute arsenical poisoning may manifest according to the type of arsenic encountered. Acute intoxication (i.e., quick onset) usually begins to manifest symptoms within 30 minutes of exposure. Initially, a patient may have a metallic taste or notice slight garlic odor on the breath, experience a dry mouth and difficulty or discomfort in swallowing (dysphagia), severe nausea and vomiting, abdominal pain, and profuse diarrhea. In acute arsenic poisoning involving massive amounts of arsenic, there are lesions on the gastrointestinal tract indicating that endothelial cellular toxicity remains the predominant clinical feature. Capillary damage leads to generalized vasodilation, transudation of plasma in which there are nucleated cell counts less than
500 to 1000/µL, which would likely result in shock to the human body. Arsenic’s effect on the mucosal vascular supply while not a direct corrosive action, does lead to transudation of fluid into the bowel lumen, mucosal vesical formation, and sloughing of tissue fragments leading to necrosis. Cyanosis, hypoxic encephalopathy, seizures, acute tubular necrosis, and death may occur due to associated hypovolemia if left untreated. Chronic poisoning results in long-term cutaneous complications, including the development of Bowen’s Disease and manifestation of malignant tumors of the cutaneous tissues. Other signs include plantar hyperkeratosis pigmentation, anemia, leukopenia, brittle nails, and facial edema. Peripheral neuropathy is the hallmark of chronic poisoning of both sensory and motor nerve fibers (Gorby, 1988; James and Byer, 2015).

While lead is a naturally occurring metal in the earth’s crust, the widespread exposure of lead has led to anthropogenic presence in nature. Human activity such as mining, fossil burning, use of both lead-based paint and gasoline as well as the use of lead-arsenate based pesticides in agriculture have been, and still, are contributors to environmental lead contamination. In the U.S, children have historically, been primarily exposed to lead poisoning through contact with lead-based paints. Children remain a targeted population for the prevention of lead poisoning in the 21st Century (Schaffer and Campbell, 2007). Symptoms of lead poisoning remain varied and do not readily manifest due to buildup of toxic levels over time. This is often the case when repeated exposures to small quantities of lead are experienced. Lead toxicity is considered rare due to single incidence of exposure or ingestion of lead, although repeated lead exposure may induce such symptoms as: abdominal pain, abdominal cramps, aggressive behavior, constipation, sleep problems, headaches, irritability, loss of developmental skills in children, high blood pressure, kidney dysfunction, anemia, and loss of memory. Since a child’s brain is still developing, lead toxicity can lead to intellectual disability causing symptoms such as behavioral problems, low IQ, poor grades, problems with hearing, short- and long-term learning difficulties and growth delay (Gorby, 1988).

**History of Contamination**

Production, formulation, and packaging of pesticides, herbicides, and insecticides took place at the Woolfolk Chemical Site located in Fort Valley, Georgia, from the 1910s until the 1980s. The pesticides formulated included arsenic and lead-based formulas. During World War II, the Woolfolk Facility served as the base of operation to produce arsenic trichloride for the US military. The operations expanded to include production of Dichloro-Diphenyl Trichloroethane (DDT), toxaphene and other pesticides. Contamination of the Woolfolk Chemical Facility and its surrounding environments mainly occurred due to careless handling of products during pesticide packaging and the use of an unlined disposal pit at the expound facility. Because of these actions the ground was contaminated with chemicals (Environmental Protection Agency [EPA], 1995a).

Most buildings on site were built primarily of wood and sheet metal. Absorption of chemicals via migration through the floors contributed to contamination of the underlying soil. Building W located on the Georgia Agricultural Chemical property to the west at the Facility was used for production of pesticides in powder form which led to the entire building being covered in layers of toxic dust. In addition, those working to process the chemicals, were also consistently covered with arsenic dust. Elevated levels of arsenic, lead, pentachlophenol, and dioxin were found in soil samples taken from the area beneath and adjacent to Building W. Building E was the site of repackaging chemicals. It has also been documented that routinely, 30 to 55-gallon drums containing Silvex, often had some of their contents spilled leading to further contamination. Silvex
contains 2, 3, 7, 8-tetrachlorodibenzo-p-dioxin (TCDD); when spilled, it results in localized contamination of floors and the underlying soil. This contamination had serious health implications for those living in the area (EPA, 1995a). The Woolfolk Chemical Company site became a superfund site in 1990 after determination of the spread of arsenic contamination to surrounding areas by the EPA. The EPA has mapped the residential areas of Fort Valley as a way of documenting contamination of residential properties and the surrounding environment with arsenic and lead over the past four decades, as well as evaluating the health assessment records of residents in these areas during the period of remediation. The objectives of this study were to (1) determine the effect of remediation conducted in Fort Valley, Georgia, by the EPA, (2) determine the amount of arsenic exposure to residents living within the Woolfolk Chemical Site, and (3) evaluate the potential of continuous arsenic contamination.

**Literature Review**

The presence of heavy metal deposits in surface and groundwater can cause contamination of all species living within the aquatic environment as well as those species relying on such environments for hydration. A study carried out in Utah by Giddings (2001) confirmed the ease with which surface and groundwater contamination can occur because of anthropogenic activities. According to Magellan and Barral-Fraga (2014), from 1800 to the 1970s, many mining activities took place near the creeks of Near Park City, Utah. They found that these activities led to the contamination of the creeks by heavy metals. While some remediation has been done, many creeks still register deposits of lead and silver ore. Concentrations of silver, cadmium, copper, lead, mercury, and zinc in the streambed sediment of creeks greatly exceed background concentrations of these metals; the levels of these elements exceed their respective aquatic life criteria values.

The U.S. Food and Drug Administration [FDA] in 1991 initiated testing of food for the presence of arsenic. In 2011, the FDA proposed a limit of 100 parts per billion (ppb) for inorganic arsenic in baby cereal (FDA, 2011). It is recognized that higher concentrations of arsenic in groundwater will lead to higher levels in agricultural products (Das and Chowdhury, 1996). For this reason, foodstuffs are a major source of exposure regarding arsenic intake (25-300 microgram/day). The concentration of arsenic in food is not mainly due to natural occurrence of arsenic in groundwater but also through man-made activities, including mining, industrial waste disposal, and insecticide application which can all serve as methods of contamination. Inorganic arsenic can persist in the soil for more than 45 years; therefore, even land that has not been in use since the 1980s could be dangerously affected by arsenic and contribute to toxic effects (ATSDR, 2007).

The mechanism of lead-induced toxicity is not completely understood; however, heme synthesis enzymes, thiol-containing antioxidants, glutathione peroxidase, antioxidant molecules such as glutathione (GSH) are some of the prime targets of lead toxicity in humans. The low blood-lead levels are enough to inhibit the activity of these enzymes and induce generation of reactive oxygen species such as superoxide, hydrogen peroxide, and hydroxyl radicals, all of which are a part of cell signaling processes (Medicinet, 2017). Oxidative stress is the pathogenesis of lead-induced toxicity and other associated diseases. Oxidative stress is essentially an imbalance between the production of free radicals and the ability of the body to counteract or detoxify their harmful effects through neutralization by antioxidants (Djordjevic and Cosacic, 2008).

Lead exposure continues to be a growing concern for humans, especially with growing children and pregnant women. In the U.S., it has been found that low levels of lead can produce harmful effects in children; however, there has also been a decrease in the mean levels of lead in blood in
children (ATSDR, 2017a). Previous studies have linked low IQs in children to suffering from lead poisoning (<10μg/dL) due to exposure during their developmental years. These studies found that cognitive functioning in children and infants was reduced as much as 7.4 points as average blood lead concentrations increased from 1μg/dL to 10 μg/dL and then reduced 4.6 points for every 10μg/dL increase after that (Canfield et al., 2003). Several studies have been conducted on the health impacts of living close to lead-contaminated areas. Brander et al. (2011) found an increase in pregnancy outcomes (birth defects, fetal death, low birth weight, preterm birth, and spontaneous abortion), childhood cancer, cardiovascular, respiratory illnesses, end stage renal diseases, and diabetes among residents who live close to lead contaminated areas.

**Methodology**

**Data Collection**
The study focused on the adverse effects of environmental pollution, specifically arsenic and lead, in residents and in the surrounding environment of Fort Valley, Georgia, during the four decades of the operation of the Woolfolk Chemical Company. This study was conducted as a retrospective case study with no control group. However, two soil samples taken in two separate years, 1993 and 2015, were compared in order to determine the effect of the remediation action by the EPA. Also, residents were tested for arsenic contamination. The participants for the arsenic testing were volunteers from Fort Valley, Georgia, who lived within EPA’s mapped area and workers of Woolfolk Chemical Company, who most likely would have been exposed to arsenic and other chemicals from the Woolfolk Facility. The 53 volunteers included adults and teenagers from ages 15-75 years. They comprised 31 females and 22 males; 49 lived within the EPA mapped site. Four worked at some point at the Woolfolk Chemical Site; 41 resided within three blocks of the Woolfolk Chemical Site. The participants were demographically listed as 91% African Americans and 9% Caucasians. There was no testing conducted for lead toxicity. Perhaps, this is due to the fact that the maximum concentration level of toxicity in 1995, was below EPA’s standard cleanup maximum concentration goal, and therefore, unlikely to cause any adverse exposure to residents.

Data were collected from the EPA’s Record of Decision Health Assessment results of the 53 subjects for arsenic contamination. The Record is based on the EPA’s 1993 soil sampling of areas within 10-mile radius of the Woolfolk Chemical Site, the level of arsenic dust found in residential properties, as well as Dr. Jacques Surrency’s (one of the authors) 2015 soil sampling of the same mapped areas of cleanup by EPA.

**Data Analysis**
The data were analyzed using Excel. Both numerical and categorical variables were generated using tables, pie charts, and bar charts. T-test was used to calculate the mean for arsenic and lead levels in 1993 and 2015.

**Results and Discussion**

**Soil Sampling of EPA’s Mapped Areas for Cleanup, 1993 and 2015**
The EPA’s standard acceptable level of arsenic in soil is 0.39ppm (Ricky, 2014). The arsenic levels in specific areas of Fort Valley, Georgia, were compared between the 1993 EPA’s soil sampling and Dr. Jacques Surrency’s 2015 sampling as shown in Figure 1. The average level of arsenic present in the soil in 1993 was 23.05ppm. When compared to 2015, there was a drastic reduction
to 9.21 ppm. The fact that there is still a measurable level is important as it has been demonstrated that low levels of these chemicals are hazardous to health. Roots of crops, especially vegetables, could be easily affected by inorganic arsenic through absorption of contaminated water from the soil (Upadhyay et al., 2018).

![Mean Residential Soils Levels of Arsenic](image)

**Figure 1. Mean Average of Levels of Arsenic in 1993 and 2015**

Similarly, the levels of lead present in the soil in Fort Valley, Georgia, in 1993 and 2015 were compared to EPA’s acceptable level of lead in soil as shown in Figure 2. The results revealed that the levels of lead had been greatly reduced by 2015. The EPA’s acceptable levels of lead in play and non-play areas are 400 ppm and 1,200 ppm, respectively (ATSDR, 2017b). Pre-remediation in 1993 showed levels of lead of approximately 147 ppm (EPA 1995b) is below the clean-up criteria for play and non-play areas, and did not require any form of remediation. However, the remediation of arsenic also helped to reduce lead further to a safer level (about 50 ppm) for children.

![Mean Residential Soil Level for Lead](image)
Figure 2. Mean Average of Levels of Lead in 1993 and 2015

![Graph Showing Correlation between 1993 and 2015 Data]

Figure 3. The Correlation between 1993 and 2015 Soil Sampling Data

The correlation of 1993 soil sampling and 2015 soil sampling (Figure 3) shows a strong relationship between both years. The EPA only conducted remediation in areas where the levels of arsenic were within the criteria for cleanup. This graph shows that the 2015 sampling was within the same sampling area examined by EPA in 1993.

**Arsenic in Residential Properties**

Incidence rate of heavy metal exposure is higher in people who live close to industrialized areas when compared with people who do not live near such areas (Branda et al., 2011). Figure 4 shows the level of arsenic residues in attics of residential properties in seven streets (Troutman, Preston, Oak, Morris, Pine, Elm, and O’Neal), located within 10 mile-radius from the Woolfolk Chemical facility. The results show that Oak Street recorded the highest concentration of approximately 3,238 ppm of total arsenic distributed in attics of different residential properties. Although there is no air ambient quality for arsenic, the EPA lists arsenic as an air pollutant, which has the potential of causing diseases and death (ATSDR, 2013). The EPA’s Final Record of Decision (ROD) of August 2004 revealed complete cleanup of all residential properties contaminated with arsenic dust (EPA, 2004).
Figure 4. Levels of Arsenic Dust in Attics of Residential Properties in 1993

**Evaluation of the 1993 Health Assessment Testing for Arsenic Poisoning Conducted by the Agency for Toxic Substances and Disease Registry (ATSDR)**

Table 1 shows that 53 subjects were present for medical testing. Eleven participants were biopsied for palmar-plantar keratosis, 18 had skin biopsies collected, 24 had urine, hair, or nail samples collected. It is unclear as to why certain participants were tested via biopsy and others by non-invasive tests such as urinalysis, hair or nail sampling. The testing was conducted in 1993 by the ATSDR (EPA, 2002).

Table 1. Participants Statistics of Arsenic Testing in Fort Valley, Georgia, 1993 (N=53)

<table>
<thead>
<tr>
<th>Summary of Testing</th>
<th>Number of Participants</th>
</tr>
</thead>
<tbody>
<tr>
<td>Palmar-plantar Biopsied</td>
<td>11</td>
</tr>
<tr>
<td>Skin Biopsies Collected</td>
<td>18</td>
</tr>
<tr>
<td>Urine Samples Collected</td>
<td>16</td>
</tr>
<tr>
<td>Hair Samples Collected</td>
<td>4</td>
</tr>
<tr>
<td>Nail Samples Collected</td>
<td>4</td>
</tr>
</tbody>
</table>

Source: EPA’s Superfund Record of Decision Declaration, 1994

Table 2 shows that 16 samples of urine were collected for analysis. It reports that urine arsenic and urine creatinine, were, respectively, <38ug/L and <50ug/L (lab ref. range) and 0ug/L and 0ug/L (# above ref. range). Also, four samples each were taken to test for nail arsenic and hair arsenic. It shows that nail arsenic was <1.0mg/g and hair arsenic was <1.0mg/g for lab ref. range and zero each for # above ref. range. The reason why only 16 samples were collected was not given. Perhaps, samples were not collected from the other 37 subjects because they did not meet the
inclusion criteria for analysis. The same conclusion could be drawn for nail and hair analysis. The report of the testing for arsenic exposure and toxicity showed no noticeable contamination in the 53 people tested by the ATSDR.

Table 2. Laboratory Results of Arsenic Testing in Fort Valley, Georgia, 1993

<table>
<thead>
<tr>
<th>Analysis</th>
<th>Number of Samples</th>
<th>Lab. Ref. Range</th>
<th>No. Above Ref</th>
</tr>
</thead>
<tbody>
<tr>
<td>Urine Arsenic</td>
<td>16</td>
<td>&lt;38µg/L</td>
<td>0</td>
</tr>
<tr>
<td>Urine Creatinine</td>
<td>16</td>
<td>&lt; 50µg/L</td>
<td>0</td>
</tr>
<tr>
<td>Nail Arsenic</td>
<td>4</td>
<td>&lt; 1.0µg/g</td>
<td>0</td>
</tr>
<tr>
<td>Hair Arsenic</td>
<td>4</td>
<td>&lt;1.0µg/g</td>
<td>0</td>
</tr>
</tbody>
</table>

Mcl of Arsenic in urine=50 µg/L
Mcl of Arsenic in Creatine>1000 µg/L
Mcl of Arsenic in hair<1000 µg/g

Figure 5. Gender of Participants

The number of females were more than males (58 vs. 42%) who participated in the 1993 testing conducted by ATSDR. This is quite possibly due to the fact that more women may have been available than men during the time due to less females working outside of the home at the time of the study, and therefore more accessible.
The majority of subjects tested during the arsenic testing were African Americans, 91% vs. 9% Whites. This may be attributed to the population of Fort Valley, Georgia, which is predominantly African American. Also, it may be due to the fact that more African Americans lived closer to the Woolfolk Chemical Site than Whites.

Figure 7 shows the age of the participants in the testing. The majority (48%) of the participants were between the ages of 45 and 59 years, followed by 22% (30-44 years), and 18% (60-74 years). The group that had the lowest percentage was the less than 15 years group, which was two percent.
Arsenic toxicity affects all age groups. However, the neural and cognitive function impairment is worse with younger people (Tyler and Allan, 2014). Based on the Tyler and Allan (2014) study, clinical assessment related to the Woolfolk Chemical arsenic exposure should have targeted younger people, particularly children. Perhaps this statistic informed the decision by ATSDR to suggest continuous health monitoring in Fort Valley, Georgia (EPA, 1995a).

**Conclusion**

Remediation exercise in Fort Valley, Georgia, greatly reduced the levels of arsenic and lead in the soil. However, the arsenic level is still high in the mapped areas where the cleanup activities took place. The current mean arsenic level still poses a potential risk to agriculture. The health assessment of people in the ATSDR (1995) Report did not detect any arsenic toxicity. Additionally, the health monitoring should be continuous to greatly minimize, or completely root out the impact of the Woolfolk Chemical arsenic exposure. Furthermore, continuous soil analysis should be the norm for residents who engage in backyard gardening to avoid consuming contaminated produce. The study makes three main recommendations. First, the presence of arsenic and lead in soil should be tested in every part of Fort Valley using the geographic Information System (GIS) every 2-3 years. This information would help in determining areas that have been contaminated. Second, a randomized prospective study should be conducted with clinical assessment of both arsenic and lead toxicity, particularly on younger people. Third, tests other than urinalysis should be used; for example, tissue testing such as liver function and bone marrow status should be done to test for chronic toxicity.

**References**


