Public Health Assessment

PRICE BATTERY SITE
HAMBURG BOROUGH, BERKS COUNTY, PENNSYLVANIA
EPA FACILITY ID: PAN000305679

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Pennsylvania Department of Health Disclaimer

The Pennsylvania Department of Health’s (PADOH’s) top priority related to the former Price Battery Superfund site is to ensure residents living near the site have the best information to safeguard their health. The Agency for Toxic Substances and Disease Registry (ATSDR) provides technical assistance and funding, through a cooperative agreement to PADOH, to help identify and evaluate environmental health threats to communities by using the best available science, taking responsive public health actions, and providing trusted health information. More information about ATSDR is available online at www.atsdr.cdc.gov. Although the Price Battery Site Public Health Assessment (PHA) was supported by this cooperative agreement, it was not published by ATSDR. The conclusions and recommendations presented in this PHA document are based on an analysis of the data and information made available to PADOH within a limited timeframe. The availability of additional sampling data, new information and/or changes in site conditions could affect the conclusions and recommendations.

On September 4, 2009, ATSDR and PADOH published the Price Battery Site PHA for public comment (a copy of this document may be requested online at http://www.atsdr.cdc.gov/hac/pha/publiccomments.asp). The original public comment period for the PHA ended Sept. 30, 2009; however, the public comment period was extended until after the ATSDR and PADOH public meeting was held in October 2009. The current PHA document serves as a finalization of the original Sept. 2009 public comment version. Therefore, site activities and data available after 2009 have not been added to this document. Instead, PADOH is preparing an updated Price Battery Site Health Consultation (HC). The HC will contain updated site information and data available since 2009.
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Summary

Introduction

PADOH’s top priority related to the former Price Battery Superfund site is to ensure that the community living and conducting activities near the site has the best information possible to safeguard its health. PADOH prepared this Price Battery Site PHA. While this PHA was supported by a cooperative agreement, it was not published by ATSDR. The Price Battery site was proposed to the Superfund National Priorities List (NPL) in September of 2004. The U.S. Environmental Protection Agency (EPA) determines whether a site will be proposed to the NPL. EPA formally added this site to the NPL in April 2005.

The Price Battery Superfund site (the site) is an inactive lead battery recycling and manufacturing operation located in a mixed commercial, industrial and mostly residential area of Hamburg Borough, Berks County, Pennsylvania. The site operated a secondary lead smelter at the facility from the 1940s through the 1970s. Smelting is a process in which lead is melted down. Former smelting operations, located at the Price Battery facility, have resulted in air deposition of lead particulates in the surface soil of residential areas at the site. Lead battery casings and wastes used as fill material have also contaminated the soil in additional locations in the community. This site initially comprised an approximately 25-block (about one-quarter square mile) area. EPA actions at the site have taken place since 2002. As a result of the ongoing remedial investigation activities by EPA Region 3, the target area and environmental sampling area had expanded by 2009 to encompass an approximately 35-block (about one-half square mile) area. In 2009, soil that had been contaminated with lead, arsenic and antimony was still being discovered in areas of the site. PADOH and ATSDR held their public meeting (public availability session) on Oct. 14, 2009. A PADOH and ATSDR PHA was released jointly for public comment on Sept. 4, 2009. No public comments were received by ATSDR or PADOH.

PADOH is preparing an additional Price Battery site HC for future publication. The HC, which will be published under cooperative agreement with ATSDR, will contain updated information and data available since 2009.

CONCLUSIONS

PADOH reached two important conclusions in the October 2009 public comment PHA and in this final Price Battery Site PHA. PADOH concluded that:
Conclusion 1  Consistently ingesting (accidently eating) the contaminated soil containing lead at this site for a year or more could harm people’s health. The site is a public health hazard for those residential properties that have not been fully remediated for lead.

Basis for conclusion  PADOH bases this conclusion on the following: The ongoing exposure to lead of children 6 years of age and younger in the Price Battery site study area is a public health concern. Review of environmental sampling data indicates that soil at or near the surface is at levels that could cause adverse health effects. The elevated levels of lead deposited on the surface of the soil warrants remediation to eliminate the exposure pathway or reduce the levels of lead in the surface soil to below those associated with adverse health effects. Prior to 2010, there was a collaborative goal from the Centers for Disease Control and Prevention (CDC) to eliminate childhood lead poisoning by the year 2010. Progress has been made, and a new goal has been put in place by CDC to eliminate elevated blood lead levels (BLLs) in children and to reduce BLLs in children by 2020.

Conclusion 2  Ingesting (accidently eating) the contaminated soil containing antimony and arsenic at the average levels found in the surface soil at this site is not expected to harm people’s health.

Basis for conclusion  Exposure to the average levels of antimony and arsenic in the surface soil at the site would not be expected to result in noncancerous (not cancer causing) adverse health effects in children and adults or to result in increased lifetime cancers. The arsenic levels in the soil are not high enough to cause an increased incidence of cancer risk. Antimony is not known to cause cancer.

RECOMMENDATIONS (NEXT STEPS)  PADOH recommended the following (in October 2009):

1. Public health actions should continue to focus on the main sources of lead at the Price Battery site: lead contaminated residential surface soil from former facility emissions and battery waste off-site.

2. Although not related to the former Price Battery facility, lead-based paint from pre-1978 constructed homes is a source of lead at the Site. PADOH should continue to make information available about health effects from lead exposures available to the homeowners and residents in this area.

3. EPA should continue to sample and remediate the lead-contaminated soil from properties, particularly homes of children with elevated BLLs, residential yards of children, childcare facilities, schools and homes with pregnant women.
4. PADOH, ATSDR and EPA should continue to collaborate and focus public health actions on testing the BLLs of as many children 6 years of age and younger as possible in and around the Price Battery Site and northern Berks County. This would help locate and ensure remediation of lead-contaminated soil or mitigate exposures due to lead-based paint at residences where children with elevated BLLs live. These ongoing actions should continue to focus on primary prevention. This makes homes lead safe through removal of lead-contaminated soil and/or reduction of the exposure to lead-based paint.

5. PADOH, ATSDR and EPA should continue to work together on increasing the knowledge in the Hamburg community regarding the identified lead hazards, encouraging primary prevention activities, and promoting and facilitating ongoing BLL testing for all children 6 years of age and younger.

6. Health education activities should continue to focus on residents living in the higher risk areas who have both soil contamination and older housing. These activities should continue and center around reducing health risk in the long-term (by implementing primary prevention methods) as well as the short-term (by increasing knowledge of interim strategies to reduce exposure to lead).

7. EPA, PADOH and ATSDR should continue having discussions with Hamburg Borough officials regarding call before digging projects.

FOR MORE INFORMATION: For concerns about health relating to this site, please contact your health care provider. You may also call PADOH at (717) 346-3285.
Purpose and Health Issues

PADOH prepared this Price Battery Site PHA. EPA proposed the Price Battery site to the NPL in September 2004; it was formally added to the NPL in April 2005. ATSDR is mandated to prepare a PHA for sites proposed to the NPL by EPA. A PADOH and ATSDR PHA was released jointly for public comment on Sept. 4, 2009. This PHA serves as the finalized document of the September 2009 PHA. While this PHA was supported by a cooperative agreement, it was not published by ATSDR.

The Price Battery site is an inactive lead battery recycling and manufacturing operation located in a mixed commercial, industrial and mostly residential area of Hamburg Borough, Berks County, Pennsylvania. The Price Battery facility operated a secondary lead smelter at the facility from approximately the 1940s to the early 1970s. The lead smelter was operated at the facility as part of a battery recycling process. Former smelting operations at the Price Battery facility have resulted in air deposition of lead particulates in the surface soil in residential areas surrounding the Price Battery facility. Lead battery casings and wastes used as fill material have also contaminated the soil with lead at additional locations in the community.

When PADOH and ATSDR held a public availability session (public meeting) on Oct. 14, 2009, EPA was continuing to investigate and remediate residential properties at the Price Battery site. In preparing for this PHA, PADOH reviewed environmental sampling data obtained from EPA, visited the site, reviewed health outcome data, and gathered community concerns during discussions with residents throughout Hamburg. PADOH is preparing an additional update to this document through a future Price Battery site HC. This HC will contain updated information and data collected since the PHA published on Sept. 4, 2009, and will be published under cooperative agreement with ATSDR.

Background

Site Location and Description

The former Price Battery facility is located near the center of a populated residential area in Hamburg Borough, Berks County, Pennsylvania (Appendix A: Figure 1 and Figure 2). The former facility is located at 246 Grand Street in Hamburg and is part of the Price Battery Superfund site. The former facility covered approximately eight acres within Hamburg. The site includes the portion of Kaercher Creek, which flows through Hamburg and the Price Battery facility before entering the Schuylkill River. In the early stages of investigation, EPA targeted an area southeast of the Price Battery facility; this area was in the expected prevailing downwind direction (Appendix A: Figure 2). In 2003, EPA had determined the northern site boundary through surface soil sampling investigations. The site border to the east was defined by the Hamburg Borough boundary line with Windsor Township and Sixth Street. To the south of the site, the boundary was defined by Hawk Ridge Drive. The Schuylkill River formed the boundary to the west. By 2009, the target area had expanded to encompass an approximately 35-block (about one-half square mile) area in Hamburg.
Site History

From the 1940s to approximately February 1966, the Price Battery facility operated as a lead battery recycling and manufacturing plant [1]. Operations at this facility included a secondary lead smelter and smokestack, as well as an oxide plant. During the facility operations, the lead smelting processes created emissions that exited the smokestack and contaminated soil in residential areas. Lead was the primary contaminant released by the plant. However, the plant also released arsenic and antimony during this period. Broken battery casing wastes, used as fill material throughout Hamburg, also contributed to contamination of the soil [1]. In 1971, the facility demolished the smelter and smokestack [2]. The plant at the Price Battery facility is no longer in operation.

In July 2002, an EPA contractor collected surface soil samples from 18 residential properties in the vicinity of the Price Battery facility. The results from the initial sampling event identified lead contamination in residential yards near the plant. The analysis of these samples showed concentrations of lead exceeding 10,000 parts of lead per million parts of soil (ppm) in four of the 18 yards sampled [3]. The maximum concentration of lead detected during the initial sampling was 173,000 ppm in a yard and 680,000 micrograms per square foot (µg/ft²) from a dust wipe sample from a home interior [4].

In October 2002, ATSDR published an initial HC that included an evaluation of the initial surface soil samples collected from all 18 residential locations. Based on the referenced information in that document, ATSDR concluded that lead contamination in the surface soil in yards near the Price Battery facility was present at levels that pose a public health hazard. ATSDR also indicated that the extent of lead contamination in the residential community around the Price Battery facility was not fully characterized and additional sampling should be conducted to further identify the areas with lead contamination in surface soil (see Appendix A: Figure 3) [3]. EPA is completing a Remedial Investigation/Feasibility Study (RI/FS) report (for more information and updates, see the EPA Webpage online at http://www.epa.gov/reg3hwmd/npl/PAN000305679.htm). EPA negotiated an agreement with Exide, the owner of the site facility, to remediate the facility. Since September 2008, Exide has been carrying out a separate RI/FS on the Price Battery plant property.

One focus of the RI/FS report is delineating the remaining surface soil contamination for future remedial actions. This ongoing RI/FS is not limited to examining lead in surface soils because of air deposition from lead smelting activities at the former Price Battery facility. To determine whether the lead contamination in the yards could have been tracked into homes by normal household activities, the dust in the interiors of the homes that have elevated lead concentrations in their surface soil are also sampled.

As of April 20, 2009, EPA continued to address the residential properties with the highest lead contamination and/or target populations as they were identified. Target properties included homes in which at least one child under 6 years of age and/or at least one pregnant woman resided. Prior to July 2005, EPA had sampled the dust of the interiors of 167 homes found to have elevated lead contamination in their yards. The results from the dust wipe samples collected from these interior surfaces revealed that 153 of these 167 properties had lead in dust
above the EPA action level (A.L.) of 40 µg/ft². The proposed health-based A.L. was based on a study that utilized the integrated exposure uptake biokinetic (IEUBK) model and estimated bioavailability of lead at the site based on in vitro analysis of swine [5]. As of June 2005, a total of 85 interiors had been remediated or cleaned to below the A.L. [6].

An EPA study indicated that there is a linear relationship between lead contamination and arsenic and antimony contamination in the surface soils [1]. Using this finding, EPA estimated that three properties located along Third Street would have significantly elevated arsenic levels. EPA further noted that chronic exposure to the predicted arsenic levels would theoretically result in an elevated cancer risk for one person out of 10,000 persons exposed (meaning a low excess lifetime cancer risk). However, most of the properties that were sampled had lower arsenic levels. Chronic exposure to the actual arsenic levels would theoretically result in an elevated cancer risk for one person out of 100,000 persons exposed (meaning a no apparent excess lifetime cancer risk). The remediation of arsenic and antimony was addressed when the primary contaminant of concern (lead) in the surface soils was remediated.

Site Demographics

The Price Battery site is located in the southern portion of Hamburg. Residential land makes up a majority of the targeted area. The site of the former Price Battery facility is zoned industrial. The homes subjected to elevated levels of lead in their surface soil from air deposition from past smelting activities at the Price Battery facility and evaluated in this PHA are zoned high density residential.

According to the 2000 census data, approximately 4,114 people live in Hamburg Borough. Among the total population, 7.2 percent are children under the age of 6 years. Hamburg consists of approximately 1,824 households, and 49.2 percent of these households have children under 18 years of age [7]. Approximately 56.6 percent of Hamburg’s homes were constructed prior to 1950. The median year of housing construction is 1941 [7].

Site Visits

From 2002 through October 2009, PADOH and ATSDR staff conducted active blood lead screenings and health education initiatives in the Hamburg community. These have involved numerous visits to the community, including participation in community events and visits to local health providers. The Community Health Concerns section of this document describes these efforts in more detail. During February 2004, PADOH staff visited the Price Battery site. ATSDR Region 3 and EPA Region 3 staff accompanied PADOH on the tour of the site. PADOH observed the topography of the site, the residential locations, a creek that flows beneath the Price Battery facility, and the areas surrounding the site (including a community park and other sites throughout the greater Hamburg area) with lead contamination due to battery casing waste. EPA was addressing battery casing waste located throughout the Hamburg area under various other removal actions. These sites included: the Hamburg Lead Site, Port Clinton Avenue Site, the Rail Cut Site, the Peach Alley Site, the Woodland Road Site and the Kaercher Creek Site.
PADOH and ATSDR partnered with Berks County Childhood Lead Poisoning Prevention Program staff in providing free blood lead screening events for Hamburg area children on May 21, 2007; July 21, 2007; November 5, 2007; and May 13, 2008. The purpose of lead screenings was to educate community residents in the region about the local sources of lead contamination, health effects from lead exposure and the resources available to prevent or reduce lead exposure. On Oct. 16, 2007, PADOH, ATSDR and EPA conducted a health professional update for two physicians and 10 nurses and other health care providers from a family practice office serving the Hamburg community. On Oct. 14, 2009, PADOH held a public availability session, another blood lead screening session, and conducted another site visit.

Discussion

Data Used
The data utilized in preparing this PHA included a PADOH database on child BLLs and EPA data sets containing the available soil lead data. From November 2002 to May 2003, EPA’s Superfund Technical Assistance and Response Team (START) collected surface soil samples (0 to 6 inches) from residential properties surrounding the Price Battery facility [1]. Because of this study, approximately 222 residential properties were documented to have lead-contaminated surface soil at levels more than three times the background concentration.

In 2003, EPA’s START collected approximately 960 surface soil samples (0 to 6 inches) from the target area within the residential area near the Site during a removal action [2]. In December 2003 and January 2004, additional surface soil samples were collected from approximately 40 properties in the target area for the analysis of lead, arsenic and antimony to help fill data gaps for EPA’s Risk Assessment. A smaller portion of these samples was also analyzed via EPA’s in vitro method for assessing bioavailability of arsenic and lead. A majority of these samples were analyzed for lead using handheld x-ray fluorescence (XRF) devices. The XRF allows samples to be analyzed in the field. Approximately 10 percent of these samples underwent further laboratory analysis using EPA methodology to validate the XRF results.

PADOH has also collected the results of blood lead testing for children 6 years and younger in Hamburg since 1998. As of 2008, approximately 318 children in the Hamburg ZIP code area have had their blood tested for lead. The blood lead results were reported by private physicians or obtained through PADOH/ATSDR/EPA blood lead screening efforts in Hamburg and other work of the PADOH Childhood Lead Poisoning Prevention Program. However, before 2002, laboratories and physicians were only required to report childhood BLLs above 10 µg /dL. After 2002, all childhood BLL results were required to be reported, regardless of the result. Most of the blood lead data came from voluntary participation in the testing. In the October 2002 HC, ATSDR recommended BLL screening for children (6 months to 6 years of age) and pregnant women residing on properties where lead contamination is greater than or equal to 500 ppm [3]. It is unknown how many of these children and pregnant women were actually screened.
Contaminants of Concern

The available data indicate that lead is the primary contaminant of concern at the Price Battery site. Therefore, this PHA focuses mainly on the potential health effects associated with lead exposure. As discussed earlier, data also revealed detections of arsenic and antimony at this site at levels of potential health concern. Through environmental laboratory analysis of approximately 40 composite surface soil samples collected from yards throughout the Price Battery Site target area, it has been determined that there appears to be an association with soil concentrations of lead, arsenic and antimony. Generally, the yards sampled with the highest lead concentration also had the highest arsenic and antimony concentrations in their surface soil. PADOH evaluated the exposures to antimony and arsenic in this PHA as secondary contaminants of concern.

**Lead**

As indicated in Figure 3 of Appendix A, lead is present in surface soil of residential properties primarily around the area of the Price Battery facility. During smelting operations at the Price Battery facility, lead was released from the stack into the air and settled onto the ground in the neighboring community. Lead particles from emissions became tightly bound to soil particles. For the most part, the particles remained in the upper portions of the surface soil after deposition. Since lead does not dissipate, biodegrade or decay, the risk of exposure is long-term.

Another source of lead in this location is lead battery waste, mainly casings, used as fill material throughout the greater Hamburg area. The use of leaded gasoline was phased out in the early 1980s, yet surface soil contamination from the leaded particulates in the automobile exhaust remains in the upper soils today. In addition, lead from interior and exterior lead-based paint may also be present in houses and soil surrounding homes constructed prior to 1978, when lead was banned from paints in the United States.

Individuals may be exposed to lead through incidental ingestion of soil during activities such as gardening and outdoor play [8]. People could also be exposed to lead from inhaling dust and drinking water contaminated with lead. The biological fate of lead is well known [8]. When ingested, 10 percent to 80 percent (depending on various factors) is absorbed and distributed throughout the body by the bloodstream. Lead is primarily distributed to the kidneys, bone marrow, liver, brain, bones and teeth. Bone and tissue have been found to contain 95 percent of the total amount of lead stored in the body. Therefore, collecting and analyzing blood samples for lead measures recent and ongoing exposures, but not the lead that is deposited other areas of the body. However, lead may be mobilized from bones (during pregnancy, menopause, etc.), and the blood sample measurements may reflect preexisting exposures.

**Health effects in Children from Lead**

Children 6 years of age and younger are considered to be at a greater risk for health effects from lead exposure than older children and adults [8]. Some of the reasons for children’s increased vulnerability to lead poisoning are the following:
1. Children have a developing central nervous system.
2. Hand-to-mouth behavior exhibited by children increases the ingestion rate for either contaminated soil or the ingestion of lead containing dust or paint chips.
3. Children's efficiency of lead absorption from the gastrointestinal tract is greater than adults.
4. Iron and calcium deficiencies that are prevalent in children may enhance the absorption and increase the toxic effects of lead [8].

Most children with lead poisoning have no obvious symptoms, thus their condition often remains undiagnosed and untreated [9].

Fetuses are even more vulnerable to the effects of lead than children [8,9]. Since lead crosses the placenta, a woman exposed to lead during her pregnancy can pass on lead to her developing fetus. Lead in bones of women who were exposed before pregnancy may be mobilized because of the physiological stresses of pregnancy resulting in exposure to the fetus as well.

Studies that involve exposure to lead in children and the developing fetus have demonstrated an association between lead and several health effects [8,9]. These health effects include both physical and mental impairments, hearing difficulties, impaired neurological development and reduced birth weights and gestational age [8,10]. Health effects can also include behavioral effects such as impulsivity, aggression and short attention span when exposure levels are high and distractibility, poor organization, a lack of persistence and daydreaming resulting from low-level exposures to lead [11]. The neurotoxicity of lead is a particular concern. Some health effects, such as impaired academic performance and motor skills, may become irreversible and persist as a result of lead exposure, even when BLLs return to lower levels [8,12].

Evaluation of Health Effects from Exposure to Lead

Generally, and for the evaluation of most chemicals, PADOH calculates an exposure dose and compares it to a health guideline. The exposure dose is a calculation that estimates the amount of a contaminant that may enter a person’s body for a given exposure scenario. The health guidelines typically used by PADOH for comparison purposes are ATSDR’s minimal risk levels (MRLs) or EPA’s reference doses (RfDs). ATSDR has developed MRLs for many contaminants commonly found at hazardous waste sites. MRLs are estimates of daily exposure to a contaminant below which noncarcinogenic (non-cancer) adverse health effects are unlikely to occur. In other words, adverse health effects would not be expected to occur at estimated exposure doses below the MRL. MRLs are developed for different routes of exposure, such as ingestion and inhalation. They are also developed for different lengths of exposure, such as acute (14 days or less), intermediate (15-364 days), and chronic (365 days or more). The EPA RfD is an estimate, with uncertainty or safety factors built in, of the daily lifetime dose of a substance that is unlikely to cause harm in humans.

However, ATSDR has not derived MRLs for lead exposure, nor has EPA developed a RfD for inorganic lead and lead compounds. The reason for this is because studies do not indicate clear dose-response relationships using environmental concentrations of lead [8,13]. No health guidelines or threshold values have been established for health effects resulting from exposure to lead in various environmental media. However, strong evidence from health studies indicates a
link of health effects to elevated BLLs [8,9]. Levels of 10 to 20 µg/dL, and maybe even less, in children’s blood have been associated with decreases in IQ and slightly impaired hearing and growth [8,9]. BLLs of 20 µg/dL and greater are associated with changes in nerve conduction velocity. Vitamin D metabolism, which is essential in bone development, can suffer at elevated BLLs of 30 µg/dL [9]. In children, lead begins to affect hemoglobin synthesis at 40 µg/dL. Colic, anemia, kidney disease and diseases of the brain occur at BLLs between 60 to 100 µg/dL. CDC considers BLLs greater than 10 µg/dL in children and adults to be elevated and levels at which public health actions should be taken.

There is some evidence, from human and animal studies of lead exposures, for the cancer-causing potential of lead [8,13,14,15]. An International Agency for Research in Cancer (IARC) working group reviewed six studies of workers exposed to elevated concentrations of lead and lead compounds and found limited evidence linking lead with stomach, kidney, lung and brain cancer [14].

For this PHA, PADOH used BLLs or a prediction of BLLs to evaluate the possible health effects of lead exposure. The following sections will discuss the relationship between lead levels in surface soil and BLLs and the impact of remediation of soil contaminated with lead and BLLs.

Impact of Soil Lead Concentrations on Children’s BLLs

There has been substantial variation in studies regarding the relationship of soil lead concentrations and BLLs. One 1991 study found lead soil concentrations greater than 500 ppm were associated with average blood lead concentrations greater than 10 µg/dL in children [16]. Another study in 1989 reported a correlation between a soil lead concentration of 250 ppm and an estimated BLL of 2 µg/dL [17]. In 1991, CDC reported that, in general, BLLs increase 3 to 7 µg/dL for every 1,000 ppm increase in the soil lead concentration [9]. The differences reported among studies are a reflection of the different exposure conditions (i.e., ground cover, seasonal variations), the different absorption rates of lead and lead-containing compounds, and different exposed populations. In addition, pre-existing health conditions, such as iron deficiencies, can enhance lead absorption and toxicity [8].

A number of studies report that the increases in BLLs as a function of the concentration of lead in soil is not linear. In other words, the rate of increase in BLLs is not directly linked with exposure to elevated lead concentrations in soil. According to one study, an increase in lead concentrations in soil from 100 ppm to 1,000 ppm was linked to a change of the predicted BLL of 7.3 µg/dL to 13.0 µg/dL, an increase of 5.7 µg/dL [16]. However in this same study, exposure to soil with an average lead concentration of 2,100 ppm was linked to an estimated BLL of 15.2 µg/dL, an increase of 2.2 µg/dL [16].

To deal with this nonlinear relationship, EPA developed the IEUBK [18]. The IEUBK model is used to predict the risk of elevated BLLs in children less than 7 years old that are exposed to lead from various sources. The model also predicts if a typical child exposed to media specific lead concentrations will have a BLL greater than or equal to the level associated with adverse health effects. The IEUBK Model is EPA’s primary tool for identifying clean-up levels for lead-
contaminated soil. This tool was used at the Price Battery Site for EPA’s Human Health Risk Assessment.

The following criteria are factored into the IEUBK Model [18]:

1. **Intake of lead in soil, house dust, air, water and food.** Sampling data on lead in these various media are used to identify site-specific intake rates. Media specific default intake rates are used in this model if the sampling data are not available. These default rates were carefully determined from available research data.

2. **Uptake of lead from the contaminated media into the bloodstream.** Only a fraction of the lead that an individual is exposed to is taken in and absorbed by the bloodstream. Typically, default uptake rates are used in the IEUBK model. However, the study conducted at Price Battery estimated the bioavailability of lead in soils through EPA’s in vitro studies.

3. **Biokinetics of lead within the body.** The biokinetics of lead, or where lead goes within the body and how fast it is eliminated, is also considered in the IEUBK Model through default values that are used to calculate a mean BLL.

4. **Distribution of BLLs within the population of concern.** The mean identified in the biokinetic component is then used to calculate the most probable distribution of BLLs within a population using default assumptions on the distribution. These assumptions include variability in physiology, behavior, sampling and analysis. These results are used to determine the probability that a child will have a BLL above a specific level.

The validity of the IEUBK Model was calibrated against two separate blood and soil lead community studies [18]. Subsequent comparisons involved well-conducted blood and environmental lead studies of children with adequate exposure characterizations. Those comparisons demonstrated reasonably close agreement between mean observed and predicted BLLs and between observed and predicted BLLs in excess of 10 µg/dL. Both of these studies focused on communities with at least 15 percent of the children having BLLs that exceeded 10 µg/dL.

**Impact of Lead-Contaminated Soil Abatement and Childhood BLLs**

Since children have multiple potential sources (i.e. paint, dust, water, soil) of lead exposure, there is variation among studies that measure the effectiveness of the abatement of lead contaminated soil and BLLs in children. In a 1993 study in urban Boston, Massachusetts, soil abatement containing an average of 1,790 ppm of lead was independently associated with an approximated reduction in BLL [19]. However, even though this study reported that lead-contaminated soil contributed to the overall lead burden in children, remediation of soil resulted in only modest declines in BLLs. Interior dust and loose paint abatement was also performed as part of the remediation in an attempt to significantly reduce the amount of lead-bearing dust in the treated homes.

In a 2003 study, the effect of soil abatement on BLLs in children living near a former smelting and milling operation demonstrated a more significant decline in children’s BLLs [20].
results of this study reported that soil removal in residential yards with lead greater than 500 ppm resulted in a 2.5 µg/dL reduction in BLLs among children ages 6 to 36 months. In addition, for every 1,000 ppm reduction in soil lead concentration, the estimated decline in BLLs was 3.5 µg/dL. This study also found a significant decline in measured indoor dust levels of lead and arsenic resulting from the removal of contaminated soil.

Another Boston study, which reported the impact of remediation of soil contaminated with lead on reduction of BLLs in children, was performed in two parts [21]. The first part of the study measured whether soil abatement reduced elevated BLLs (7 µg/dL to 24 µg/dL). A soil lead reduction of 2,060 ppm was associated with a 2.25 to 2.70 µg/dL reduction in the children’s “established” BLLs. The second part of the study assessed a child’s BLLs and household dust levels following lead-based paint hazard remediation alone and then in combination with soil abatement. The study concluded that by properly removing the source or sources of lead contamination, the exposure pathways may be reduced or eliminated [21].

The findings of these studies support the importance of primary prevention, particularly abatement of residences prior to their occupancy, and secondary prevention through remediation based on observed BLLs.

Lead Standards, Regulations, and Recommendations

The following section briefly describes some of the regulations and standards regarding exposure to lead.

EPA regulates lead under the Clean Air Act of 1977 and has designated lead as a hazardous air pollutant [8]. Prior to the Act, the amount of lead discharged from industrial sources was not restricted. Contaminants released to the air from the stacks at industrial facilities, like the Price Battery facility, settled out of the air on nearby soil and accumulated over time.

In the early 1970s, EPA began to phase-out the use of leaded gasoline because of its effects on the environment from automobile emissions [8]. By 1988, less than 1 percent of gasoline contained lead. Congress completely phased out leaded automotive gasoline after Dec. 31, 1995.

In 1988, the Lead-Based Paint Poisoning Prevention Act became law. It prohibits the use of lead-based paint in residential structures built or renovated by any federal agency [22]. The Act also gives the Department of Housing and Urban Development (HUD) authority to develop regulations on the removal of lead-based paint from housing constructed prior to 1978. In addition to HUD, EPA, the U.S. Department of Health and Human Services (DHHS) and the Department of Labor’s Occupational Safety and Health Administration (OSHA) are the primary federal agencies for promulgating regulations aimed at minimizing lead exposure.

In compliance with the Toxic Substance Control Act (TSCA) §403, in 2001, EPA published a final rule for dangerous levels of lead. That rule established a soil-lead hazard of 400 ppm for bare soil in play areas and 1,200 ppm for bare soil in non-play areas [23]. As stated in the TSCA §403 rule, lead contamination at levels equal to or exceeding the 400 ppm and 1,200 ppm standards may pose serious health risks. The potential risks are site-specific and may warrant timely response actions.
**Arsenic**

Arsenic occurs naturally in soils and rocks. Commercial products containing arsenic include, but are not limited to, wood preservatives, pesticides, paints and leaded gasoline. Other industrial processes that can release arsenic are de-sulfuring of gases and/or fossil fuels, burning preserved wood, and metal alloy production. Historically, arsenic was used in certain medicines and drugs. There are two forms of arsenic in the environment: organic and inorganic. The inorganic forms are usually more toxic than the organic ones. The standard analytical method to detect arsenic in varying environmental media does not distinguish the specific form of arsenic [24]. To ensure a conservative or protective toxicological/public health estimate, PADOH assumes that all the arsenic detected in this evaluation is inorganic arsenic.

**Health Effects from Exposure to Arsenic**

Health-based studies show that ingesting a small amount of inorganic arsenic may cause stomach irritation and a decreased blood cell count. Exposures to very high levels may cause fatigue, abnormal heart rhythm and blood vessel damage resulting in bruising due to the lack of blood cell production. Impaired nerve function causing a pins and needles sensation in the hands and feet may also occur at high exposure levels. Chronic exposures to very high levels (but not at the levels seen at this site) may lead to “Blackfoot Disease”, a condition in which blood circulation is lost, ultimately resulting in cell death in the hands and feet. At exposures to very high levels, the most characteristic effect of chronic oral exposure to arsenic is skin changes. These changes include darkening of the skin and appearance of warts on the palms, soles and upper-body. Some of the warts may also lead to skin cancer. EPA classifies inorganic arsenic as a known human carcinogen. Exposure to arsenic at high enough levels increases the risk of liver, bladder, kidney, prostate and lung cancers [24].

**Evaluation of Health Effects from Exposure to Arsenic**

Persons can be exposed to arsenic from the environment by ingesting contaminated food or soil, drinking contaminated water, or breathing contaminated air. Young children may be exposed to arsenic from eating dirt because of their tendency to place their hands in their mouths. Dermal (skin) contact with soil or water that contains inorganic arsenic is not considered a risk factor.

The human body absorbs various forms of arsenic differently, depending on the environmental media in which it is contained. Arsenic in soil is more difficult to absorb than the soluble arsenic forms found in groundwater at some sites. Health studies demonstrate that the bioavailability of arsenic in soil might be quite small in some soil types. These studies suggest that arsenic in soil may be imbedded in minerals or occur as insoluble compounds and, therefore, not be taken up by the body from the gastrointestinal tract [24]. This is important for estimating human doses. PADOH used the ATSDR comparison values (CVs) as a screening tool to identify contaminants of concern. The ATSDR cancer risk evaluation guide (CREG), for an adult, is a theoretical calculation that assumes a consumption of 100 milligrams of soil per day by a 70 kg person over a lifetime without consideration of absorption rate or the bioavailability of arsenic from soil. This assumption is very conservative.
By 2005, PADOH had received reports from EPA of arsenic detections in surface soil ranging from 6.9 ppm to 57.5 ppm at the Price Battery site. PADOH used this range of arsenic levels for the health assessment in this PHA. Also by 2005, arsenic was detected in all of the 40 yards sampled [2].

Antimony

Antimony is a silvery white metal of medium hardness that is very brittle. To make it stronger, antimony is usually blended with other metals such as lead to form mixtures of metals called alloys. These alloys were used in production of lead storage batteries [25]. Antimony, along with lead and arsenic, has entered the environment as a result of the smelting operations at the former Price Battery facility [2]. Antimony has contaminated surface soil in a nearby residential area through air deposition of the particulates that exited the stack from the facility. Once in the environment, antimony remains in the soil for a long time. Most antimony will end up in the soil or sediment, where it strongly attaches to some metals [25].

Health Effects from Exposure to Antimony

The extent of adverse health effects in people who ingest antimony has not been well studied. In one study, persons vomited after a one-time ingestion of water contaminated with 19 ppm antimony [25]. It is also uncertain whether exposure to antimony can cause cancer, birth defects or affect reproduction in humans. The DHHS, IARC and EPA have not classified antimony as a carcinogen.

Antimony has been used in the past for medicinal purposes to treat persons infected with parasites. Those who overdosed while taking antimony-containing medication or were sensitive to it when it was injected into their blood or muscle have experienced adverse health effects including diarrhea, joint and/or muscle pain, vomiting, anemia, and altered electrocardiograms (heart problems) [25].

Evaluation of Health Effects from Exposure to Antimony

Antimony can enter the body when a person drinks water or eats food, soil or other substances that are contaminated with antimony. Young children may be exposed to antimony from eating dirt because of their tendency to place their hands in their mouths. It is not clear whether antimony can enter the body when there is dermal exposure. The human body absorbs and metabolizes various forms of antimony differently, depending on the environmental media in which it is contained. Antimony in soil is difficult to absorb, and only a small amount of the antimony that is ingested enters the blood after a few hours of exposure. After antimony enters the blood, it goes to the liver, lungs, intestines and spleen. Antimony leaves the body over several weeks [25].

The antimony reference dose media evaluation guide (RMEG), derived from the EPA RfD, was used for the evaluation of exposures to the soil in this PHA. The RMEG is 20 ppm for children and 300 ppm for adults [11]. PADOH used this CV as a screening tool to identify contaminants of concern. At exposures greater than the RfD, the potential for adverse health effects increases.
Lifetime exposure above the RfD does not imply that an adverse health effect will necessarily occur.

**By 2005, PADOH had received EPA reports of antimony detections in surface soil ranging from less than 0.61 to 305 ppm at the Price Battery site. PADOH used this range of antimony levels for the health assessment in this PHA.** Also by 2005, approximately 16 residential locations of the 40 sampled had surface soil sample results that exceeded the child RMEG (20 ppm) for antimony.

### Exposure Pathway Analysis

PADOH defines human exposure pathways by examining environmental and human components that might lead to contact with contaminants of concern. A pathway analysis considers five main elements:

1. A source of contamination;
2. Transport through an environmental medium;
3. A point of exposure;
4. A route of human exposure; and
5. A receptor population.

Completed exposure pathways are those in which all five elements are present and exposure to a contaminant has occurred in the past, is currently occurring or will occur in the future. PADOH regards those people who contact contaminants as being exposed. The exposure can occur through breathing airborne contaminants, drinking water known to be contaminated or playing, digging in or accidently ingesting (eating) contaminated soil. The identification of an exposure pathway does not imply that health effects will occur. Exposure may or may not be substantive. Thus, even if exposure has occurred, human health effects may not necessarily result.

PADOH reviewed site history, information on site activities, and the available sampling data for the Price Battery Site. From this review, PADOH identified several exposure pathways that warranted consideration. The completed or potential exposure pathways are discussed in the following section. A discussion of pathways that have been eliminated for further evaluation follows.

### Completed or Potential Exposure Pathways

The completed or potential exposure pathways for the Price Battery Site are listed in Table 1 and are described on the following pages.
<table>
<thead>
<tr>
<th>Pathway</th>
<th>Media and Transport</th>
<th>Exposure Point</th>
<th>Exposure Route</th>
<th>Exposed Population</th>
<th>Period of Exposure</th>
<th>Exposure Status</th>
</tr>
</thead>
<tbody>
<tr>
<td>Soil</td>
<td>Lead, antimony and arsenic present in soil and house dust as a result of operations at the site</td>
<td>Surface soil at site and house dust with contaminated soil</td>
<td>Incidental ingestion (accidently eating), inhalation (breathing)</td>
<td>Residents (particularly children 6 years of age and younger)</td>
<td>Past</td>
<td>Completed</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Present</td>
<td>Completed</td>
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<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Future</td>
<td>Completed</td>
</tr>
<tr>
<td>Lead-based paint (not site related)</td>
<td>Lead present in house dust, soil and paint chips from use of lead-based paint</td>
<td>House dust, soil and paint chips in homes with deteriorating lead-based paint</td>
<td>Incidental ingestion (accidently eating)</td>
<td>Residents (particularly children 6 years of age and younger)</td>
<td>Past</td>
<td>Completed</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
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<td></td>
<td>Present</td>
<td>Completed</td>
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<td></td>
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<td></td>
<td></td>
<td>Future</td>
<td>Completed</td>
</tr>
<tr>
<td>Former Price Battery facility emissions</td>
<td>Airborne lead, antimony and arsenic emissions from former Price Battery facility</td>
<td>Likely the same areas with elevated soil lead levels</td>
<td>Inhalation (breathing)</td>
<td>Residents</td>
<td>Past</td>
<td>Completed</td>
</tr>
<tr>
<td>Ingesting (eating) home-grown produce</td>
<td>Uptake of contaminants from soil via fruits and vegetables grown in gardens</td>
<td>Produce consumption</td>
<td>Ingestion (eating)</td>
<td>Residents</td>
<td>Past</td>
<td>Potential</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Present</td>
<td>Potential</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Future</td>
<td>Potential</td>
</tr>
</tbody>
</table>
Soil

EPA identified the locations with soil lead levels above 400 ppm within the targeted area (the original boundaries of this area are shown in Figure 2 of Appendix A). Exposure to lead contaminated soil may have been occurring since the 1940s, when the Price Battery facility initiated its smelting operation and air deposition of lead contaminants occurred [1]. Lead-battery waste, used as fill material throughout Hamburg, also contributed to the lead contamination in the soil. The soil sampling results provided by EPA indicate that site-related lead contamination extends beyond the north of the site boundary identified by EPA when the Price Battery Site was proposed to the NPL. In addition, the laboratory analysis of surface soil samples collected from approximately 40 residential yards indicate the coexistence of antimony and arsenic contamination with lead contamination. The concentrations of antimony and arsenic in the surface soil samples had a linear relationship with the lead contamination.

Individuals can ingest surface soil in yards as an incidental consequence of general outdoor activities that include working in the yard, gardening and playing. People occupying homes with contaminated yards can also be exposed to lead, arsenic and antimony by ingesting household dust that originally came from outside soil. The soil exposure pathway is an especially important pathway for children, who often exhibit hand-to-mouth behavior and consequently have higher ingestion rates of soil than older children and adults.

In 2005, PADOH prepared a community fact sheet for the Hamburg area that offered guidance for preventing exposure to lead in soil (see Appendix B). Maintaining a healthy grass sod on play areas and covering bare soil areas with a sufficient amount of woodchips, mulch or clean sand was one general recommendation made in the fact sheet. PADOH also recommended testing the BLLs of young children as a preventive measure because individuals may exhibit no symptoms of lead poisoning.

Lead-Based Paint

Even though exposure to lead-based paint is not related to the Price Battery site, it is a very important source of exposure to lead for many children in the Hamburg area. Therefore, the lead-based paint exposure pathway is described so that the community will be familiar with this source of lead in and around the home.

Persons can be exposed to lead-based paint through ingestion of dust or soil contaminated with small particles of the paint or through direct ingestion of paint chips. Potential exposure to lead-based paint occurs in and around homes that had interiors and/or exteriors painted with lead-based paint that is not intact (paint that is peeling, chipping, chalking or otherwise deteriorating).
Homes constructed prior to 1950 are most likely to have surfaces painted with lead-based paint, but lead was still used in some homes built from 1950 to 1978. Use of lead-based paint in homes was banned in the United States in 1978. As indicated earlier, approximately 56.6 percent of the housing in Hamburg was built prior to 1950, with a median construction date of 1941. Therefore, a child living in the Price Battery Site investigation area could be exposed to lead from lead-based paint.

In 2005, PADOH developed a community fact sheet for the residents of Hamburg that contains guidelines for preventing exposures to lead-based paint and dust (see Appendix B). These guidelines recommended keeping the paint on surfaces intact, keeping the home clean by wet mopping and damp dusting, and always using lead-safe work practices prior to initiating and while performing any renovation or remodeling work.

**Price Battery Facility Emissions**

When the smelting operation at the Price Battery facility was functioning from the 1940s to approximately 1971, airborne emissions were likely a completed exposure pathway. The plant and facility are no longer in operation, so this past exposure pathway is no longer an issue. The boundaries of the soil contamination (displayed on Figures 1 and 2 in Appendix A) also likely represent the extent of exposure to airborne emissions from the facility. Individuals living or working in this area of Hamburg may have inhaled (breathed in) lead particulates, as well as antimony and arsenic contaminated particulates, from the time the smelter began operations until operations ceased.

**Ingestion of Homegrown Produce**

Some Hamburg residents grow edible fruits and vegetables in gardens located in yards with lead contaminated soil. Lead can be absorbed from the soil and taken up by plants [8]. In addition, lead-contaminated soil could adhere to plant surfaces, especially “root” vegetables, such as carrots, potatoes, turnips and similar vegetables. A recent study indicates that this exposure pathway is only of concern for children who consume large quantities (approximately one pound per day) of homegrown produce [26]. The study found that it does not appear to be a significant pathway for adults consuming vegetables grown in metal contaminated soils.

In 2005, PADOH produced a fact sheet for the community of Hamburg that provides guidelines on lead contaminated soil including gardening in and around soils contaminated with lead (see Appendix B). These guidelines recommend careful trimming and washing of plants grown in soil contaminated with lead. The guidelines also recommend that people grow produce in raised beds filled with uncontaminated soil to reduce contact with lead while gardening.
Eliminated Exposure Pathways

Eliminated exposure pathways are those where one or more of the five elements of an exposure pathway are missing. This means that exposure is not occurring. An exposure pathway for drinking water, surface water and fish uptake was not completed in the past or present and is not likely in the future. The eliminated pathways for the site are summarized in Table 2.

**Drinking Water**

A public water supplier provides drinking water for residents in Hamburg Borough [27]. The Hamburg Municipal Water and Sewer Authority, a local drinking water supplier, operates a surface water intake at an impounding dam and four groundwater wells for emergency purposes and back-up supply. The Hamburg Municipal Water Authority supplies drinking water to approximately 4,800 customers for the greater Hamburg Area at approximately 2,050 connections. However, some private wells do exist across from the Price Battery Site study area (i.e., west of and on the other side of the Schuylkill River). EPA sampled these wells and found they were not impacted by the smelter’s emissions. EPA has determined that the city of Hamburg is on public water and not using private wells [28].

**Table 2: Eliminated Exposure Pathways the Price Battery Site**

<table>
<thead>
<tr>
<th>Pathway</th>
<th>Media and Transport</th>
<th>Exposure Point</th>
<th>Exposure Route</th>
<th>Exposed Population</th>
<th>Period of Exposure</th>
<th>Exposure Status</th>
</tr>
</thead>
<tbody>
<tr>
<td>Drinking Water</td>
<td>Transfer of lead from soil to groundwater or surface water</td>
<td>Municipal drinking water</td>
<td>Ingestion (drinking)</td>
<td>Public water supply users</td>
<td>Past</td>
<td>Did not happen</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Present</td>
<td>Is not happening</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Future</td>
<td>Will not happen</td>
</tr>
<tr>
<td>Surface Water</td>
<td>Movement of lead from soil and groundwater to surface water</td>
<td>Schuylkill River</td>
<td>Incidental ingestion (accidently drinking)</td>
<td>Residents, visitors (recreational)</td>
<td>Past</td>
<td>Did not happen</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Present</td>
<td>Is Not happening</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Future</td>
<td>Will Not happen</td>
</tr>
<tr>
<td>Fish Uptake</td>
<td>Movement of lead from soil to surface water</td>
<td>Fish consumption</td>
<td>Ingestion (eating)</td>
<td>Residents, visitors (recreational)</td>
<td>Past</td>
<td>Did not happen</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Present</td>
<td>Is not happening</td>
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<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Future</td>
<td>Will not happen</td>
</tr>
</tbody>
</table>
Therefore, it is unlikely that any private well in the target area was contaminated with lead from the Price Battery Site.

The Hamburg Municipal Water and Sewer Authority routinely monitors drinking water as it leaves the treatment plant and at some taps to ensure contaminants, including lead, are below health-based levels established under the Safe Drinking Water Act [26]. Under certain conditions, the piping in older homes could contain lead solder that can introduce lead into the home’s water supply. However, the leaching of lead solder from pipes in this area does not appear to be occurring. The results of drinking water samples collected at the taps of approximately 20 residences in Hamburg indicated that lead was at concentrations of 1 ppb in six homes, 2 ppb in one home, and below the detection limit (less than 1 ppb) in the remaining homes [2]. In addition, the EPA START contractor sampled the taps of 23 residences in the Price Battery target area in December of 2003 and 2004. The results of these samples indicated that lead was present at two taps at concentrations of 7 ppb and 1.1 ppb, and the other 21 homes had sample results of less than 1 ppb for lead in drinking water. Prudent public health practice dictates achieving the lowest lead levels possible.

Surface Water

In general, surface water runoff eventually enters the Schuylkill River, which is just west of Hamburg. The Schuylkill River supports recreational fishing and boating. The limited duration and frequency of recreational activities that involve contact with surface water further reduces the potential for exposure.

Fish Ingestion

Although specific fish tissue data are not available, PADOH does not expect eating fish from the Schuylkill River to result in hazardous exposures to lead. However, persons should follow the fish advisories issued by the Commonwealth of Pennsylvania. Individuals should follow the general, statewide one-meal-per-week fish consumption advisory for all species recreationally caught in Pennsylvania. To determine if recommendations that are more protective apply to the fish that a person might consume, refer to the Pennsylvania Fish and Boat Commission’s website online at: http://www.fish.state.pa.us/fishpub/summary/sumconsumption.pdf.

Toxicological Evaluation of Contaminants

Lead

Table 3 (on the following page) shows the range of soil lead levels and average soil lead levels in the site release area and background area. These are the soil data collected at the site between November 2002 and March 2007.
In developing this PHA, PADOH considered evaluating the relationship between soil and BLLs at the same geographic locations to determine the significance, if any, of elevated environmental concentrations of lead in surface soil on childhood BLLs. However, PADOH decided not to conduct these spatial analyses because:

1. The connection between elevated lead concentrations in soil and BLLs is very difficult to establish through a direct comparison of soil and BLLs at the same locations. A number of factors affect BLL status. The evaluation of all the factors would require an exhaustive investigation of each property. Such an investigation is beyond the scope and purpose of this PHA. Some of the factors to consider include:
   - the ingestion rates of contaminated soil by a child varies throughout the year;
   - the degree of cleanliness and housekeeping in the home;
   - additional sources of lead exposure in the home; and
   - the diet and age of children in the home.

2. The soil lead data were collected in a systematic manner. However, the BLL data may be biased because of voluntary participation. This difference could introduce uncertainty into the analysis. Children whose parents chose to have them tested may have a significantly different chance of living at a location with elevated soil lead levels than children whose parents did not have them tested. Therefore, any evaluation might not reflect the actual relationship between BLLs and soil lead levels.

3. The results of such analyses would not alter or clarify the recommendations and public health actions proposed in this PHA.

Figure 1 and Figure 2 of Appendix A show the presumed boundaries of the lead contaminated surface soil (0 to 6 inches) at the Price Battery site when EPA proposed the site for placement on the NPL. As discussed earlier in this PHA, the lead contamination was not fully characterized at the time of proposal to the NPL. Since then, lead contamination has been determined to extend beyond the north site boundary of Pine Street.

<table>
<thead>
<tr>
<th>Location</th>
<th>Number of Samples Analyzed by XRF &amp; Lab Data</th>
<th>Range of Soil Lead Levels in Parts per Million</th>
<th>Mean Soil Lead Level in Parts per Million</th>
</tr>
</thead>
<tbody>
<tr>
<td>Site Release Area</td>
<td>3,844</td>
<td>46.9-173,978</td>
<td>1,656</td>
</tr>
<tr>
<td>Background Area</td>
<td>147</td>
<td>26.8-176</td>
<td>92</td>
</tr>
</tbody>
</table>

Soil samples (0-6 inch depth) data in this assessment were collected from November 2002 to May 2003 and December 2003 to January 2004, December 2004, November 2006, and February and March 2007 by EPA Region 3 Superfund and Technical Assistance and Response Team (EPA START).

Source of data: U.S. Environmental Protection Agency (EPA) Region III, EPA Remedial Project Manager. Philadelphia, PA.
Table 4 shows the percent of children in the **Price Battery Site Release Area** and all children tested in the **Hamburg ZIP Code 19526 area** with BLLs 10 µg/dL or greater (i.e., elevated BLL). From 1998 through 2000, 25 children were tested in the Hamburg ZIP code (19526). In 2001, according to the PADOH Bureau of Family Health tracking system, 38 children from the Hamburg ZIP code (19526) were screened for BLLs; two of these children had BLLs greater than or equal to 10 µg/dL. In 2002, 49 children were screened from the same Hamburg ZIP code area; eleven of these children had BLLs greater than or equal to 10 µg/dL. In 2003, 77 children were screened from this area; nine of these children had BLLs greater than or equal to 10 µg/dL. Data for 2004 indicate that a total of 60 children were screened from the Hamburg ZIP code area, with four of these children having BLLs greater than or equal to 10 µg/dL.

For the **Price Battery Site Release Area**, from May 2002 through April 2005, 11 percent (9/80) of children tested from the Price Battery Site study area had BLLs of 10 µg/dL or greater. In the **ZIP code of 19526 (Hamburg)**, from 1998 through September 2004, 17 percent (42/249) of the children tested had BLLs of 10 µg/dL or greater reported to PADOH (see Table 4 below).

**Table 4: Blood Lead Level Data for Price Battery Site (May 2002 through April 2005) and ZIP Code 19526 (1998 through September 2004)**

<table>
<thead>
<tr>
<th>Location</th>
<th>Testing Dates</th>
<th>Number of Blood Samples</th>
<th>Percent with Elevated BLLs*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Site Release Area+</td>
<td>May 2002 – April 2005</td>
<td>80</td>
<td>11% (n=9)</td>
</tr>
<tr>
<td>ZIP Code 19526† (Hamburg)</td>
<td>1998 – September 2004</td>
<td>249</td>
<td>17% (n=42)</td>
</tr>
</tbody>
</table>

*=Elevated BLLs mean Blood Lead Levels (BLLs) at or above 10 µg/dL.

+ Source: PADOH, Childhood Lead Poisoning Prevention Center (May 2002 – April 2005).

† Sources: PADOH, Bureau of Family Health Tracking System (1998 – September 2004); Pennsylvania’s National Electronic Disease Surveillance System (PA-NEDSS), as of 2004.

No comparisons or conclusions can be made regarding the BLL data shown in Table 4 from the Site Release Area data and the ZIP code data. This is in part because it is not clear whether the BLL data collected from the children in the site release area are replicated in the ZIP code cohort. This BLL data also occur over different testing date ranges, which make them impossible to compare. The data from this table must be used for qualitative purposes only to establish that blood lead poisoning is occurring and adverse health effects may result.

Data from Table 5 (on the following page) identify the number and percent of children with BLLs at or above 10 µg/dL for the Price Battery Site and the ZIP code 19526 (Hamburg) areas. These children were re-sampled for confirmation of the previous BLLs above 10 µg/dL via venous blood, which is considered the more accurate method. Most initial BLLs on children are taken using the capillary or finger stick method, which is quicker and easier to administer; however, this method is also more likely to produce biased high readings.
Table 5: Blood Lead Level Data for Price Battery Site Within the Hamburg ZIP Code 19526 (2004 through March 2008)

<table>
<thead>
<tr>
<th>Testing Dates</th>
<th>Number of Blood Samples</th>
<th>Percent With Elevated BLLs*</th>
</tr>
</thead>
<tbody>
<tr>
<td>2004</td>
<td>60</td>
<td>10% (n = 6)</td>
</tr>
<tr>
<td>2005</td>
<td>92</td>
<td>3.27% (n = 3)</td>
</tr>
<tr>
<td>2006</td>
<td>87</td>
<td>6.90% (n = 6)</td>
</tr>
<tr>
<td>2007</td>
<td>67</td>
<td>2.99% (n = 2)</td>
</tr>
<tr>
<td>2008 Qtr 1</td>
<td>12</td>
<td>8.33% (n = 1)</td>
</tr>
</tbody>
</table>

*= Elevated BLLs mean Blood Lead Levels (BLLs) at or above 10 µg/dL.
Source: Pennsylvania’s National Electronic Disease Surveillance System (PA-NEDSS).

A breakdown of the Table 5 BLLs by year, amount, and confirmation are shown in Table 6 and Table 7 (on this and the following pages).

Table 6: Child Blood Lead Levels (BLLs) 10 µg / dL and Above (2004 through March 2008)

<table>
<thead>
<tr>
<th>Reported to be residing in ZIP Code 19526 during</th>
<th>Percent of children tested by Blood Lead Level (BLL) category</th>
<th>Total Children Tested (see Table 5)</th>
</tr>
</thead>
<tbody>
<tr>
<td>0-9.9 µg/dL</td>
<td>10-14.9 µg/dL</td>
<td>15-19.9 µg/dL</td>
</tr>
<tr>
<td>2004</td>
<td>90.00%</td>
<td>1.67%</td>
</tr>
<tr>
<td>2005</td>
<td>96.74%</td>
<td>1.09%</td>
</tr>
<tr>
<td>2006</td>
<td>93.10%</td>
<td>3.45%</td>
</tr>
<tr>
<td>2007</td>
<td>97.01%</td>
<td>2.99%</td>
</tr>
<tr>
<td>2008 Qtr 1</td>
<td>91.67%</td>
<td>0%</td>
</tr>
</tbody>
</table>

µg/dL = micrograms lead per deciliter of blood tested.
*= Elevated BLLs mean Blood Lead Levels (BLLs) at or above 10 µg/dL.
Source: Pennsylvania’s National Electronic Disease Surveillance System (PA-NEDSS).
Table 7: Maximum Confirmed Child Blood Lead Levels (BLLs), 2004 through March 2008

<table>
<thead>
<tr>
<th>Measure</th>
<th>Children, &lt; 7 years of age (0 through 83 months of age), tested for lead</th>
</tr>
</thead>
</table>

<table>
<thead>
<tr>
<th>Reported to be residing in Zip Code 19526 during 2004</th>
<th>Category Basis: Child’s Single Highest (Maximum) Blood Lead Level</th>
<th>Total Children Tested</th>
<th>Confirmed Elevated (Confirmed Highs)</th>
<th>Percentage of tested Children with Confirmed Elevated Results</th>
</tr>
</thead>
<tbody>
<tr>
<td>2004</td>
<td>Low(^5)</td>
<td>0-9.9 μg/dL</td>
<td>10-14.9 μg/dL</td>
<td>15-19.9 μg/dL</td>
</tr>
<tr>
<td>2005(^10)</td>
<td>89</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>2006(^11)</td>
<td>81</td>
<td>3</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>2007(^12)</td>
<td>65</td>
<td>2</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>2008 Qtr 1(^13)</td>
<td>11</td>
<td>0</td>
<td>1</td>
<td>0</td>
</tr>
</tbody>
</table>

Key to Table 7:

1Single Highest (Maximum) blood lead level (BLL) basis - Children are often tested for lead more than one time, and this is especially true when the child’s initial test result is high (elevated). All measurements of lead BLLs in μg/dL reflect a single test result per child for the time period identified. That single result is the child’s highest (maximum) blood lead level (BLL).

2Total Children Tested = (Lows) + (Highs) + (Nulls) = (0-9.9) + (10-14.9) + (15-19.9) + (20-44.9) + (≥ 45) + (Nulls)

3Confirmed Elevated - As used within this report and defined by the Council for State and Territorial Epidemiologists (CSTE), Confirmed Elevated BLL refers to a child with one venous blood specimen ≥ 10 mg/dl, or any combination of two capillary and/or unknown blood specimens ≥ 10 mg/dl drawn within 12 weeks of each other. There is a difference between children whose highest blood lead level (max) was elevated and those children who meet the definition of Confirmed Elevated. Testing a child by venous method is more reliable (than by capillary method). Therefore, initial high capillary test results are often followed up by a venous test. That subsequent follow-up test does not always produce the same quantitative test result as the initial test did. However, if the first was a capillary and the second was a venous, the second is more reliable as a reflection of the child’s true BLL. For example, if a child’s initial test was done by capillary method and the BLL result is 12 micrograms per deciliter of blood (elevated), and the child’s second follow-up test is done soon thereafter by venous method and produced a quantitative test result value of 9 micrograms per deciliter of blood (low), then the child is not considered elevated.

4Percentage of Tested Children with Confirmed Elevated Results = (Confirmed Elevated) ÷ (Total Children Tested) X 100

5Lows - Those children whose highest (maximum) blood lead level for the time period indicated, was a quantitative test result of less than 10 micrograms per deciliter of blood.

6Highs - The Centers for Disease Control and Prevention (CDC) has established a threshold of concern of 10 micrograms per deciliter of blood, for children.

Key to Table 7 continues on the following page.
Potential Health Effects Associated with Elevated BLLs

All nine of the children in the Price Battery Site investigation area, who were identified beginning in May 2002 through April 2005 with elevated BLLs, had BLLs in the 10 to 14.9 µg/dL range. Children with an elevated BLL, based on the Pennsylvania National Electronic Disease Surveillance System (PA-NEDSS) reporting, had follow-up regarding their BLLs by the PADOH Children’s Blood Lead Program.

Sources of Lead Exposure for Children with Elevated BLLs

The PADOH review of available information indicates there are two major sources of lead for children living in the Price Battery site area: lead contaminated soil and lead-based paint. The findings indicate that most of these children with elevated BLLs live in areas where the mean soil concentration exceeds 400 ppm. As of July 2005, EPA’s soil sampling results indicated that approximately 73 percent (279 per 384) of the properties sampled in the Price Battery Site investigation area exceeded 400 ppm of lead in soil and approximately 69 percent (264 per 384) of these properties exceeded EPA’s preliminary remediation goal of 572 ppm [4]. Based on the number of properties with lead soil contamination, the scope of sampling was expanded by EPA over the years to find the contamination borders. Further, according to an EPA report completed in 2008, approximately 68 percent (579 per 856) of the properties sampled exceeded 400 ppm of lead in the soil and approximately 57 percent (490 per 856) of the properties exceeded 572 ppm of lead in the soil [28]. The EPA was denied access to 82 properties in the area for sampling, and it is not known if these 82 properties have soil lead above or below 400 ppm. The lead results from sampling indicate that soil is likely a significant source of exposure to lead in this site.

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Key to Table 7 continued:

7 Nulls - Those children tested for lead whose highest (maximum) BLL for the time period indicated was a blank (null) quantitative test result. Blank quantitative test results arrive in the Pennsylvania National Electronic Disease Surveillance System (PA-NEDSS) for various reasons. In some situations, a blank arrives because the child’s blood sample was analyzed at the laboratory and the blood lead content was extremely low -- too low for a number value. In these cases, the child’s result is considered a “low.” In other situations, a childhood lead test arrives in the PA-NEDSS with a blank (null) quantitative test result because the laboratory never actually analyzed the blood sample. That happens for various reasons. For example, the tube containing the blood sample may have broken, or the quantity of blood within the sample was too low (insufficient). In these cases, nothing is known about the child’s actual BLL. These children need to be re-tested for lead.

8 Total Highs = Those children whose highest (maximum) BLL for the time period indicated was a quantitative test result of 10 or more micrograms per deciliter of blood. Those children with Lows or Nulls (blanks) are not included as part of the Total Highs.

9 Specimen Collection Dates during Calendar Year = 01/01 through 12/31 of each respective year, inclusive.

10 Specimen Collection Dates during Calendar First Quarter in 2008 = 01/01/2008 through 03/31/2008.
As discussed earlier, a majority of the homes in Hamburg were constructed prior to 1950; many homes were constructed around 1941 [7]. Nearly all pre-1950 homes had exteriors and interiors painted with paint that could contain up to 50 percent lead [8]. Thus, children 6 years and younger living in homes constructed prior to 1950 likely are exposed to lead from paint if the lead-based paint has not been sealed or removed. It is also possible for individuals to be exposed to lead-based paint inside or outside of homes constructed prior to 1978. The Consumer Products and Safety Commission banned the sale and use of residential lead paint in September of 1977 [29].

Additionally, 98 percent (368 of 374) of the residential locations where the interior dust was sampled exceeded 40 µg/ft² lead in dust, which is EPA’s A. L. for lead in dust on floors [30]. The source of the leaded dust is more than likely a result of the two primary sources mentioned above (i.e. lead paint and lead-contaminated soil). The tracking of lead-contaminated soil via foot traffic from outside to inside the homes is a likely source of the elevated lead in dust concentrations inside the homes. Lead-based paint that is not intact also creates a leaded dust, which could also contribute to the elevated lead dust concentrations in the home.

Elimination or Reduction of Children’s Exposure to Lead

The elimination or reduction of children’s BLLs in or near the Price Battery Site involves identifying specific locations where exposure to lead-contaminated soil and lead-based paint is occurring. This was accomplished through the following activities:

- Primary prevention activities that identify, evaluate and promote control of lead hazards through ongoing temporary mitigation (i.e., sealing or repainting) or permanent elimination (i.e., removal); and
- Effective interventions for children with known lead exposure to reduce or prevent further exposure to mitigate adverse health effects.

Previously, CDC had requested that every state develop a plan to eliminate lead poisoning by 2010. As a follow-up, PADOH had developed a case management plan for every child reported to have a BLL of 10 µg/dL or higher, which includes [30]:

- Provide health education by contacting and mailing a fact sheet to each affected family;
- Coordinate care and follow-up testing following CDC guidelines between patient, physician or other primary medical provider, and PADOH;
- Discuss with each affected family the importance of testing the BLLs of all children and pregnant and lactating women living in the same household with a child with an elevated BLL;
- Conduct family education, including a home visit with assessment of possible sources of exposure and history of exposure for confirmed BLLs of 15 µg/dL or higher. Refer individuals and families as needed for follow-up care or intervention;
• Perform an environmental assessment for sources and pathways of lead exposure (e.g., paint, dust, soil, water) with lead hazard reduction follow-up and enforcement (confirmed BLLs of 15 µg/dL or greater);

• Coordinate free venous or capillary retesting; and

• Refer their address to EPA for soil testing, if not already conducted, and potential remediation if the child’s home is within the area that encompasses the Price Battery Site focus area (see Appendix A: Figures 1 and 2).

Public health actions that deal with lead exposure to children in the Price Battery site area have been focused on increasing the percentage of children, 6 years of age and younger, who are tested. This helps locate and then mitigate exposures due to lead-contaminated soil and/or lead-based paint. The various agencies involved with this site have been promoting awareness to the community of Hamburg regarding the known lead contamination and strong recommendations regarding BLL testing for children. Other public health actions have emphasized prevention messages to families and the medical community in this area.

**Arsenic**

A few residential soils sampled showed arsenic contamination but not lead. PADOH believes that these few exceptions are not site-related and that the arsenic was most likely from coal ash cinders from home heating stoves or from treated lumber placed in the yards. EPA detected arsenic in most of the surface soil samples in a linear relationship with the concentration of lead in the same sample (see Graph 1).

Graph 1 (on the following page) illustrates this overall linear relationship between the lead and arsenic concentration in the surface soil of residential yards at the site. This relationship is linear as the concentration of lead in surface soil approaches 5,000 ppm from 0 ppm. As the concentration of lead in the surface soil exceeds 5,000 ppm, the relationship between lead and arsenic concentrations is not as linearly correlated as at the lower concentrations of lead in the surface soil. Using the linear equation from the line on the graph, the average soil concentration of 5,166 ppm of lead equates to approximately 20 ppm arsenic. The expected average concentration of arsenic in the soil also is very close to the average concentration in the laboratory results, which was 18 ppm [2]. This estimate of 20 ppm is slightly more conservative and is based on an extrapolation from environmental samples, as opposed to the lower actual sampling results (18 ppm) from 40 environmental samples.

For the evaluation in this PHA, PADOH used the ATSDR CVs such as CREGs and Environmental Media Evaluation Guides (EMEGs) as screening tools to identify contaminants of concern. ATSDR’s EMEGs are estimated contaminant concentrations that are not expected to result in adverse noncarcinogenic health effects. EMEGs are based on ATSDR MRLs or EPA RfDs and conservative assumptions about exposure, such as intake rate, exposure frequency and duration, and body weight. Since average concentrations of expected arsenic concentrations of 20 ppm exceeded ATSDR CVs and this soil exposure pathway is completed, it necessitated further evaluation of exposure to arsenic. The ATSDR health-based CVs exceeded were the
arsenic EMEG for acute exposure for a child with **pica behavior** (a craving to eat nonfood items such as soil) and the arsenic CREG.

PADOH used ATSDR MRLs to further evaluate if noncarcinogenic health effects, associated with the exposures, are likely. For chemicals that are known, probable or possible human carcinogens, PADOH uses EPA’s chemical specific cancer slope factors (CSFs) to calculate theoretical excess cancer risks.

**Graph 1: Linear Relationship between Arsenic and Lead Concentration in Surface Soil* (0-6”) at Price Battery Site**

\[ y = 414.89x - 3145.5 \]

* ppm = parts per million.

* = Laboratory Results from Surface Soil (0-6 Inch Depth) Data Collected by EPA Region 3 in December 2003 and January 2004.

Source: U.S. Environmental Protection Agency (EPA) Region III. Information was from the EPA Region III Remedial Project Manager.

Average soil arsenic concentrations were used to determine potential exposures to contaminants in surface soil at this site. The exposure scenarios for children were based on a young child (1-6 years of age) and an older child (7 years of age or older) playing in private yards. For adults, PADOH assumed an exposure scenario that included daily residential exposure in private yards and, further, that persons are not exposed to surface soil three months per year because of snow cover and decreased outdoor activities during the cold winter months. An exposure dose, which is the amount of contaminant that might have entered a person’s body, is estimated using exposure duration, body weight and age. For example, young children between 1-6 years of age are known to exhibit hand to mouth behaviors and to ingest or put items in their mouths, including soil. Children who engage in this behavior to a greater degree exhibit pica behavior. These children have an increased likelihood of exposure (and increased exposure doses) to soil contaminants. The assumptions used to calculate exposure for a young child with soil pica behavior are a body weight of 16 kg (approximately 35 pounds) with an average ingestion rate of 5,000 milligrams (mg) of soil per day (compared to 200 mg of soil per day for a non-pica child). The assumptions of an older child (7-18 years of age) are a body weight of 45 kg (approximately
100 pounds) and a soil ingestion rate of 200 mg per day. The assumptions for an adult are a body weight of 70 kg (approximately 155 pounds) and a soil ingestion rate of 100 mg per day. These assumptions are summarized in Table 8.

**Table 8: Exposure Assumptions Summary**

<table>
<thead>
<tr>
<th>Exposure Assumptions</th>
<th>Population</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Children under 7 years of age</td>
</tr>
<tr>
<td>Body Weight (kg)</td>
<td>16</td>
</tr>
<tr>
<td>Surface Soil</td>
<td>Ingestion rate (mg/day)</td>
</tr>
<tr>
<td>Exposure Frequency</td>
<td></td>
</tr>
<tr>
<td>Exposure Duration (Years)†</td>
<td>1</td>
</tr>
</tbody>
</table>

*assuming the average lifetime is 70 years
†assuming people lived in the Hamburg area for about 30 years

**Evaluation of Arsenic Exposure**

The human body absorbs various forms of arsenic differently depending on the environmental media in which it is contained. Arsenic in soil is more difficult to absorb than the soluble arsenic forms found in groundwater on some sites. Health studies demonstrate that the bioavailability of arsenic in soil might be quite small in some soil types. These studies suggest that arsenic in soil may be imbedded in minerals or occur as insoluble compounds and, therefore, not be taken up by the body from the gastrointestinal tract [24]. This is important for estimating human doses. For the noncancerous chronic ATSDR EMEG (for a child) for soil, 20 ppm was used in this PHA. Some of the levels in the yards exceeded the EMEG, but based on 40 sample results, the actual average was 18 ppm. Bioavailability studies are based on various types of soils containing arsenic and determining the amount of soluble arsenic after exposure to gastrointestinal fluids [24]. Based on the studies, PADOH considered the bioavailability of arsenic from soil to be 50 percent of soluble arsenic in soils (range from 3 percent to 50 percent); however, PADOH assumed 100 percent, for a very conservative amount, in the dose calculations.

Age-adjusted soil ingestion rates were used to calculate arsenic exposure doses. Young children playing in the residential yards daily could have an estimated exposure dose as high as 0.0002
mg/kg/day over the course of a year (see Appendix C for PADOH calculations). The estimated average lifetime daily exposure to average levels of arsenic in soil for people living within the Price Battery Site study area for 30 years is 0.00002 mg/kg/day. These estimated doses are lower than the chronic MRL of 0.0003 mg/kg/day. ATSDR does not have an intermediate (15 to 364 days of exposure) MRL for arsenic. A young child, exhibiting soil pica behavior, would have an estimated exposure dose that is lower than the (provisional) acute (also for pica assessments) ATSDR arsenic MRL of 0.005 mg/kg/day.

The dose and end point used in the study to calculate the acute MRL was based on a lowest observable adverse effect level (LOAEL) of 0.05 mg/kg/day. The LOAEL is based on a 1956 study that summarized findings from 220 poisoning cases associated with an episode of arsenic contamination of soy sauce in Japan [24]. The clinical symptoms recorded were swelling of the face and gastrointestinal and upper respiratory symptoms, initially, followed in some patients by skin lesions and neuropathy. Other effects included mild anemia and leukopenia (an abnormally low number of white blood cells in the circulating blood), mild degenerative liver lesions and liver dysfunction, abnormal electrocardiogram and ocular lesions. For derivation of the MRL, facial swelling and gastrointestinal symptoms (nausea, vomiting, diarrhea) were considered to be the critical effects [24].

The acute MRL is supported by an incident in upstate New York. In this incident, people that were intermittently exposed to arsenic-contaminated drinking water at an estimated dose of 0.05 mg/kg/day, experienced gastrointestinal symptoms almost immediately (Franzblau and Lilis 1989) [24]. Gastrointestinal symptoms have been widely reported in other acute arsenic poisoning studies as well, although in some cases the doses were higher and effects were more severe, while in other cases the dose information was not available.

The ATSDR CV CREG used in this PHA to assess exposures to arsenic in the soil was 0.5 ppm. This CV is a theoretical calculation that assumes a consumption of 100 mg of soil per day by a 70 kg person (adult) over a lifetime without consideration of absorption rate or the bioavailability of arsenic from soil. The estimated cancer risk from the average lifetime daily exposure to the average soil arsenic concentration (20 ppm) is approximately one excess cancer per 100,000 people exposed or classified as no apparent theoretical increased risk (see Appendix C for PADOH calculations). After reviewing numerous human studies, as reported in ATSDR’s Toxicological Profile for arsenic, the lowest arsenic Cancer Effect Level (CEL) for lung cancer is 0.0011 mg/kg/day; for bladder cancer it is 0.0033 mg/kg/day; and for skin cancer it is 0.0075 mg/kg/day. The average lifetime exposure dose is far less than the lowest CELs for arsenic.

PADOH does not expect elevated cancer risk from exposure to the average levels of arsenic in the surface soils, depth 0-to-6 inches, at the site.

**Antimony**

Antimony was detected in surface soil samples in a linear relationship with the concentration of lead in the same sample. Graph 2 illustrates this linear relationship between the lead and antimony concentration in the surface soil of residential yards at the site. This relationship between antimony and lead concentration is linear as the concentration of lead in surface soil approaches 5,000 ppm from 0 ppm. As the concentration of lead exceeds 5,000 ppm, the
relationship is not as linearly correlated as at the lower concentrations. Using the linear equation from the line on the graph, the average soil concentration of 5,166 ppm of lead equates to approximately 58 ppm antimony. PADOH used the value of 58 ppm antimony. This estimate of 58 ppm is based on an extrapolation from 481 environmental samples, as opposed to the lower actual average (determined by EPA in 2005) sampling results (48 ppm) from 40 environmental samples.

In 2005, PADOH found that the estimated average (58 ppm) and the actual average (48 ppm) exceeded the CV, so further evaluation is warranted. Based on an exposure scenario of a 16 kg (approximately 35 pounds) child ingesting 200 mg daily of soil contaminated with 58 ppm antimony, the estimated exposure dose would be about 0.00072 mg/kg/day (see Appendix C for PADOH calculations). The actual average sampling results was 48 ppm antimony, so this evaluation would be similar though less conservative. The estimated dose exceeds the EPA RfD. Exposure doses that exceed EPA’s RfDs do not necessarily imply that health effects will occur. Antimony in soil is difficult to absorb, and only a small amount of the antimony that is ingested enters the blood a few hours after exposure. After the antimony enters the blood, it goes into the liver, lungs, intestines and spleen, where it is metabolized. Antimony leaves the body over the course of several weeks after exposure. EPA’s RfD for antimony is based on an uncertainty factor of 1,000 (i.e., the LOAEL was divided by: 10 for animal study, 10 to protect sensitive individuals, and 10 because the effect level was based on a LOAEL). A no observable adverse effect level (NOAEL) was not established for antimony [13]. Given that the estimated child dose is only slightly higher than the RfD, the conservative evaluation for this PHA, and the protective

ppm = parts per million

* Laboratory Results from Surface Soil (0-6 Inch Depth) Data Collected by EPA Region 3 in December 2003 and January 2004.

Source: U.S. Environmental Protection Agency (EPA) Region III. Information was from the EPA Region III Remedial Project Manager, August 2008.
uncertainly factor used in developing the RfD, PADOH would not expect adverse health effects to occur in young children from oral exposure to expected average concentrations of antimony in surface soil at the Price Battery Site.

Children’s Health Concerns

PADOH has established an ongoing initiative to protect children from exposure to hazardous substances. PADOH recognizes that the unique vulnerabilities of infants and children demand special emphasis in communities faced with contamination of their water, soil, air or food. Because of their immature and developing organs, infants and children are usually more susceptible to toxic substances than are adults. Children are also smaller, which results in higher exposure doses per body weight when compared with adults. Moreover, children depend entirely on adults for identification of hazards and management decisions, housing decisions, and access to medical care. PADOH’s evaluation contained within this PHA considered children as a susceptible subpopulation.

As indicated earlier, the occurrence of elevated lead contamination in soil and homes likely containing lead-based paint is prevalent in residential locations near the former Price Battery facility. In addition, there have been children 6 years of age and younger identified with elevated BLLs living near this site. These children are at risk of a variety of lead-related adverse health effects that include decreases in intelligence, impaired hearing and growth, behavioral changes, and other effects. The main sources of exposure to lead from the Price Battery Site appear to be soil contaminated with lead from the former Price Battery facility and possibly lead-based paint from homes constructed prior to 1978, especially those homes constructed prior to 1950. Ongoing efforts to reduce or eliminate exposures to both these sources should continue.

Health Outcome Data Evaluation

A health outcome data evaluation or review of health statistics is the analysis of existing health information (i.e., from cancer registries, death certificates, birth defect registries, blood lead screening databases, etc.) to determine if there is excess disease in a specific population, geographic area or time period. The evaluation of health outcome data may determine whether the occurrences of certain adverse health effects are higher than expected in the area potentially affected by the site contaminants. Health outcome data evaluation might also give a general picture of the health of a community. However, elevated rates of a particular disease may not necessarily be caused by exposure to hazardous substances. Other factors, such as personal habits, socioeconomic status, genetic pre-disposition and occupation, may also influence the onset of a particular disease. In contrast, even if elevated rates of disease are not found, a contaminant may still have caused illness or disease.

The Superfund law requires that evaluation of health outcome data be considered in a PHA [31]. ATSDR developed guidance that recommends an evaluation of health outcome data be done only if all the criteria listed below are met. The criteria and the determination of whether these were met at this site are as follows:
1. The presence of a completed or potential human exposure pathway is required. There are four completed or potential exposure pathways at the Price Battery Site (residential soil, lead-based paint, past facility air emissions and ingestion of homegrown produce).

2. Elevated contaminant levels must exist that result in measurable effects.
   - As described earlier, soil and BLLs are high enough to either result in adverse health effects or increase a child’s risk of having a BLLs above CDC’s level at which public health interventions are appropriate. The principle route of exposure is ingestion for the residential soil and lead-based paint exposure pathways.
   - Exposure to lead, arsenic and antimony from the Price Battery facility emissions pathway among residents of Price Battery site study area in Hamburg occurred from the 1940s to 1971. The Price Battery facility emissions pathway is the exposure route of concern for carcinogenic effects, because most human studies identify inhalation as the route of exposure for carcinogenic endpoints (although the levels of contaminants found in the soil at the site would not increase cancer risks) [14].

3. People must be in the completed or potential pathway for health effects to be measured.
   - PADOH estimates that 200 or more children 6 years of age and younger are at risk for exposure to lead in residential soil and lead-based paint exposure pathways in the Price Battery Site area.
   - PADOH estimates that more than 4,000 individuals were exposed to lead, arsenic and antimony in air emissions from the Price Battery facility until it closed in 1971. The Price Battery Site boundary best approximates the area where this past exposure occurred.

4. A health outcome database exists from which disease rates for population of concern can be identified.
   - PADOH’s Bureau of Family Health Tracking System includes BLL data from 1998–2001, and the PA-NEDSS database that contains BLL data in Pennsylvania since 2002. Prior to 1998, BLL data were not collected in a centralized database and were not available for review.
   - Since 2002, PADOH’s Childhood Blood Lead Poisoning Prevention Center has been collecting and maintaining ongoing blood lead data for the site.
   - Data on cancer rates in ZIP Code 19526 (Hamburg) are available from the Pennsylvania Cancer Registry. [Note: the levels of contaminants found in the soil at the site would not support any theoretical increase in cancer risks.]

PADOH determined that the Price Battery site met the criteria for conducting an evaluation of health outcome data. The PADOH Childhood Blood Lead Poisoning Center provided the BLL data that have been collected and maintained for the site since 2002. The evaluation of these data was an integral portion of this PHA.
Health Activities to Address the Community Concerns

PADOH has conducted a number of activities to communicate information about exposures related to the Price Battery site and to address the community’s concerns lead. These included:

1. Conducting BLL screening for children 6 years of age and younger at Hamburg Safety Fairs and individual residences to identify children who could be at a health risk from lead poisoning;

2. Meeting with community members, elected officials, educators, physicians and other health professionals to brief them on PADOH’s health education activities, identify their health-related concerns and questions, and determine the most effective means to deliver health education to the Hamburg community and surrounding areas;

3. Coordinating the development of the PADOH health education plan with ATSDR’s Division of Health Education and Promotion, ATSDR Region 3 and EPA Region 3 to better meet the needs of the Hamburg community and surrounding areas; and

4. Participating in site visits, physician meetings and other community meetings to gather information and provide technical assistance and health education.

Conclusions

PADOH reached two important conclusions in the October 2009 public comment PHA and in this final Price Battery Site PHA. PADOH concluded that:

1. Consistently ingesting (accidently eating) the contaminated soil containing lead at this site for a year or more could harm people’s health. The site is a public health hazard for those residential properties that have not yet been fully remediated for lead.

   PADOH bases this conclusion on the following: The ongoing exposure to lead of children 6 years of age and younger in the Price Battery site study area is a public health concern. Review of environmental sampling data indicates that soil at or near the surface is at levels that could cause adverse health effects. The elevated levels of lead deposited on the surface of the soil warrants remediation to eliminate the exposure pathway or reduce the levels of lead in the surface soil to below those associated with adverse health effects. Prior to 2010, there was a collaborative goal from the CDC to eliminate childhood lead poisoning by the year 2010. Progress has been made, and a new goal has been put in place by CDC to eliminate elevated BLLs in children and to reduce BLLs in children by 2020.

2. Ingesting (accidently eating) the contaminated soil containing antimony and arsenic at the average levels found in the surface soil at this site is not expected to harm people’s health.
Exposures to the average levels of antimony and arsenic in the surface soil at the site would not be expected to result in noncancerous (not cancer causing) adverse health effects in children and adults or to result in increased lifetime cancers. The arsenic levels in the soil are not high enough to cause an increased incidence of cancer. Antimony is not known to cause cancer.

Recommendations

PADOH recommended the following in this final Price Battery Site PHA and in the October 2009 public comment PHA:

1. Public health actions should continue to focus on the main sources of lead at the Price Battery Site: lead contaminated residential surface soil from former facility emissions and battery waste off-site.

2. Although not related to the former Price Battery facility, lead-based paint from pre-1978 constructed homes is a source of lead at the site. PADOH should continue to make information available about health effects from lead exposures available to the homeowners and residents in this area.

3. EPA should continue to sample and remediate the lead-contaminated soil from properties, particularly homes with children with elevated BLLs, residential yards with children, childcare facilities, schools and homes with pregnant women.

4. PADOH, ATSDR and EPA should continue to collaborate and focus on public health actions on testing the BLLs of as many children 6 years of age and younger as possible in and around the Price Battery Site and northern Berks County. This would help locate and then remediate lead-contaminated soil or mitigate exposures due to lead-based paint at residences where children with elevated BLLs live. These ongoing actions should continue to focus on primary prevention. This is making homes lead safe through removal of lead-contaminated soil and/or reduction of the exposure to lead-based paint.

5. PADOH, ATSDR and EPA should continue to work together on increasing the knowledge in the Hamburg community regarding the identified lead hazards, encouraging primary prevention activities, and promoting and facilitating ongoing BLL testing for all children 6 years of age and younger.

6. Health education activities should continue to focus on residents living in the higher risk areas who have both soil contamination and older housing. These activities should continue and center around reducing health risk in the long-term (by implementing primary prevention methods) as well as the short-term (by increasing knowledge of interim strategies to reduce exposure to lead).

7. EPA, PADOH and ATSDR should continue having discussions with Hamburg Borough officials regarding call before digging projects.
Public Health Actions

The public health action plan describes the activities designed to mitigate or prevent adverse human health effects that might result from exposure to hazardous substances associated with the site. PADOH is committed to the completed, on-going and planned public health actions at the Price Battery site that are listed below.

Completed Actions

Because of the widespread nature of the contamination in this region, PADOH has worked collaboratively to design and implement a comprehensive health education and outreach effort in this area. The purpose of this effort is to educate health care providers and community residents in the region about the local sources of lead contamination, the health effects from lead exposure and the resources available to prevent or reduce lead exposure. A special focus of these activities is on promoting childhood lead poisoning prevention and surveillance in this area.

The following public health actions have been completed at the Price Battery Site:

1. Activities in 2003 and 2004 included personal visits to local family practice, medical and pediatrician offices to increase physicians' awareness of the regional sources of lead contamination and to identify any local barriers to blood lead screening.


3. A community/health professional task force was developed to provide advice and support regarding sustaining the lead poisoning prevention efforts in the region. PADOH and ATSDR sponsored the first meeting of the Northern Berks Childhood Lead Poisoning Prevention Committee Meeting on Sept. 24, 2004. A total of 16 participants attended the meeting, including representatives from local pediatric and family practices, the local Women, Infant and Children program, Pennsylvania’s Early Childhood Screening Program, the Hamburg Elementary School Nurse, the Borough of Hamburg, EPA Region 3, ATSDR, and PADOH. One of the clinicians on the Task Force recommended that information be developed to assist residents in evaluating possible contamination in their homes on their own and to help them reduce possible exposures. As a result, PADOH developed three Fact Sheets entitled “Reducing Exposure to Lead in Drinking Water”, “Prevention Exposure to Lead in Soil”, and “Preventing Exposure to Lead in Dust and Paint “ (see Appendix B). These Fact Sheets have been distributed to members of the task force, EPA and at local community locations and events. In addition, PADOH updated health care professionals at a local family medicine practice in 2007.

4. PADOH and ATSDR presented an update on the local surveillance results at a May 2004 Reading Hospital Pediatric Department staff meeting.
5. As of Dec. 19, 2008, EPA’s ongoing investigation has identified 492 properties in the Price Battery Study area that have lead in surface soil exceeding the site remediation goal of 572 ppm from the sampling. In addition, 230 properties with lead paint above 40 µg/ft² were cleaned and interior lead dust removed from inside the homes. EPA addressed the residential properties with the highest lead contamination and/or with the most vulnerable populations (i.e., one child under 6 years of age and/or one pregnant woman) in the home.

6. As a result of these efforts, PADOH and ATSDR have been able to increase community awareness of lead poisoning sources in the region and lead poisoning prevention strategies, as well as to demonstrate a general increased level of blood lead surveillance in the area. For example, according to PADOH’s Bureau of Family Health tracking system, 38 children from the Hamburg ZIP code (19526) were screened for lead poisoning in 2001 (two of these children had BLLs greater than or equal to 10 µg/dL), 49 were screened from the same Hamburg ZIP code area in 2002 (eleven of these children had BLLs greater than or equal to 10 µg/dL), and 77 children were screened from this area in 2003 (nine of these children had BLLs greater than or equal to 10 µg/dL). The reporting for 2004 indicates that a total of 60 children were screened from the Hamburg ZIP code area, with four of these children with BLLs greater than or equal to 10 µg/dL (see Table 4 on page 22). In 2005, 92 children were screened with one confirmed elevated BLL greater than or equal to 10 µg/dL. In 2006, 87 children from this area were screened with three confirmed elevated BLLs. In 2007, 67 children were screened and one confirmed with an elevated BLL. PA-NEDSS allows more efficient and better data to prevent duplicate records and identify the correct number of children with BLLs above 10 µg/dL. As additional children were identified over the years with BLLs greater than 10 µg/dL, more parents and doctors apparently became aware of the site and of PADOH, ATSDR and EPA efforts, such as providing free BLL screenings (see Tables 5, 6 and 7 in this PHA).

7. A public meeting (public availability session) was held for the community on Oct. 14, 2009 in Hamburg, PA. The original 30-day public comment period for this PHA ended Sept. 30, 2009; however, the comment period was extended until after the public meeting was held in Oct. 2009. No public comments were received during this public comment period.

Ongoing and Planned Actions

The following public health actions are on-going or planned at the Price Battery site:

1. PADOH is preparing and plans to publish a Price Battery site HC, which will be published under cooperative agreement with ATSDR. The Price Battery site HC will contain the updated BLL screening data (through 2010). The HC will also contain updated EPA information and soil data.
2. PADOH will continue working with EPA on increasing the knowledge of the community on the lead hazards that have been identified in the Price Battery Site study area. PADOH and ATSDR will continue working to ensure that continuing education is directed to people in the area of concern about how to reduce their short and long-term risk by implementing primary prevention strategies that are outlined in community Fact Sheets (Appendix B). PADOH and ATSDR will continue to provide shorter-term, interim strategies for reducing risk through the distribution of the community Fact Sheets in physician offices, health fairs and site related events.

3. PADOH will continue BLL screening efforts as exposed populations, especially children 6 years of age and younger, are identified in the area of concern. This plan continues to encourage residents to have children 6 years of age and younger tested annually to detect possible exposure above the level of concern. In addition, as the site is characterized and new properties are identified with lead contamination, BLL screening will be strongly encouraged for residents 6 years of age and younger or who are pregnant. PADOH will continue the ongoing analysis of the site-related BLL data.

4. EPA will continue to investigate lead contamination in the surface soil of additional properties to the north of Pine Street in Hamburg. In addition, EPA will continue removal actions in the target area, concentrating on properties with children and pregnant women.

5. PADOH will update this public health action plan for the Price Battery Site as additional data or site conditions warrant.
References

1. United States Environmental Protection Agency. 2012. Price Battery Site. This document is online at: [http://www.epa.gov/reg3hscd/npl/PAN000305679.htm](http://www.epa.gov/reg3hscd/npl/PAN000305679.htm) [Reader’s note: please click on “EPA’s Administrative Record Database” from the webpage to view an online database of other documents and reports].


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Appendix A: Figures
Figure 1: (2009) Price Battery Site Location in Hamburg, Berks County

Map source: U.S. Environmental Protection Agency (EPA) Region III, EPA Remedial Project Manager. Philadelphia, PA.
Figure 2: Price Battery Site Layout in Hamburg, Berks County, Pennsylvania

Map source: U.S. Environmental Protection Agency (EPA) Region III, EPA Remedial Project Manager. Philadelphia, PA.
Map source: U.S. Environmental Protection Agency (EPA) Region III, EPA Remedial Project Manager. Philadelphia, PA.
Appendix B: Fact Sheets

Pennsylvania Department of Health

Hamburg Community Fact Sheets
Overview

This fact sheet will inform you about ways to minimize exposure to lead in paint and dust to protect your health and the health of your family.

Lead

Lead is a bluish-gray metal found in the earth’s crust. Lead has many uses and can be found in all parts of our environment, including in and around homes.

Lead in the Environment

Lead occurs naturally in the environment. It can be present in the ground, stuck to soil particles, in surface waters, and in underground or drinking water.

Lead in Paint and Dust

Deteriorated lead-based paint and lead contaminated dust (coming from windblown soil, dirt containing lead, and dry-scraped, dry sanded, or heated lead-based paint) are the most common sources of lead contamination in the home.

Exposure to Lead in Paint and Dust

Generally, a person can be exposed to lead in paint and lead dust by:
- Breathing or inhaling airborne dust containing lead
- Accidentally ingesting or eating paint chips or dust containing lead

However, not much lead can get into your body through the skin.
Other Ways a Person Can Be Exposed (in contact with) Lead

People can also be exposed to lead by:

- Eating food or drinking water containing lead.
- Spending time in areas where lead-based paints have been used and are deteriorating.
- Working in a job where lead is used.
- Using health care products or folk remedies that contain lead.
- Engaging in certain hobbies in which lead is used (for example, stained glass, refinishing furniture, pottery, fishing sinkers, and ammunition).
- Playing with some imported toy necklaces or near some imported plastic mini-blinds.
- Lead is also found in some food, beverages, and cigarette smoke.

Health Effects

General Population:
Lead can affect almost every organ and system in your body. The most sensitive is the central nervous system, especially in children.

At high levels, lead may:
- Decrease reaction time
- Cause weakness in fingers, wrists, or ankles
- Possibly affect memory
- Damage the kidneys
- Damage the reproductive system

Lead may also cause anemia, a disorder of the blood, behavior problems, and learning disorders.

Children:
Children are more vulnerable to lead poisoning than adults.
- As babies, they can be exposed in the womb if their mothers have lead in their bodies.
- Children are at a greater risk for ingesting dust by putting their hands in their mouths after touching dust, dirt, or sand contaminated with lead.
- Lead-based paint may also be a hazard when found on surfaces that children can chew or that get a lot of wear and tear, such as windows, doors, stairs, and porches.

Additionally, babies exposed to lead in the womb of their mothers may be born prematurely and have lower weights at birth.
Measures to Take if Your House Was Built Before 1978

If your house was built before 1978, it often contains lead-based paint and the following actions are recommended:

- Ask your doctor about a blood lead test for you and your family, especially for young children less than six years old.
- To determine whether your home contains lead, get your house tested for lead paint by a certified inspector. Call 1-800-424-LEAD or visit the web www.leadlisting.com to obtain a list of trained inspectors.
  - It is likely that samples collected by a certified inspector will be of better quality than those collected by a resident. Nevertheless, if the resident decides on collecting the paint or dust samples, we recommend sending them to a laboratory certified by EPA. A list of certified laboratories can be obtained by calling 1-800-424-LEAD.
- Always practice lead-safe work practices before beginning and while doing any renovation or remodeling work. Both children and adults can develop extremely high blood lead levels when renovation and remodeling activities take place.
  - Educate yourself on lead-safe practices by calling 1-800-424-LEAD and asking for the brochure: "Reducing Lead Hazards When Remodeling Your Home". This brochure will provide you with guidelines regarding appropriate equipment, protective clothing, and other measures to be followed while renovating or remodeling your home.
- Keep painted surfaces intact.

Interpreting Results

The standard for lead in dust in floor samples is 40 micrograms (mcg) of lead per square foot (ft²). The standard for lead in dust in window sills is 250 micrograms (mcg) per square foot (ft²).

The standard for lead in paint samples in weight of lead per unit area (mg/cm²) is 1.0 mg/cm² or above.

If the results show higher levels of lead than the standards, you might want a risk assessment of your home to be done. A risk assessor will tell you if there are any sources of serious lead exposure (such as peeling paint and lead dust). They will also tell you what actions to take to address any hazards. You can find a list of risk assessors by calling: 800-440-LEAD.
Cleaning of Areas Contaminated With Lead

If you have (or suspect) a lead dust problem, lead-contaminated dust will need to be cleaned up. Some recommendations for proper cleaning include:

- Hire a lead-based paint contractor to do the initial cleaning of surfaces and cracks where lead is settled.
  - Windows, worn floors, carpets, and upholstered furnishings seem to collect most of the lead dust. It is very hard to clean these surfaces thoroughly, and dust settles on them rapidly after they are cleaned.
  - Pregnant women and children should not be present while the initial cleaning is done.
  - If you do the cleaning yourself, call the 800-424-LEAD information line for more detailed information on how to clean.

- Retest following cleanup to ensure levels are safe.

- Vacuum with a HEPA filter-equipped vacuum cleaner. This special type of vacuum will trap lead particles and prevent them from being released back into the air. A household vacuum will not do this. When you finish vacuuming, carefully empty the dust collected in the vacuum cleaner, being sure to dampen it with water first to control the spread of collected dust.

- Wet clean exposed areas with a solution of water and an all-purpose cleaner or a cleaner made specifically for lead.
  - Use one bucket for the cleaning solution and one bucket for rinsing.
  - Change the rinse water frequently (at least once for each room being cleaned) and replace rags, sponges, and mops often.

- Clean the surface until no dust is visible. After cleaning, rinse the surface with clean water and a new sponge or cloth.

- At the same time that you undertake a cleaning project, have all the drapes, curtains, and carpets professionally cleaned.

- Replace the filters in heating and air-conditioning units.

Additional Measures to Reduce Exposure to Lead

- Keep your home clean by washing (wet mopping and damp dusting) floors, window frames, windowsills, and other surfaces at least once a week.

- Keep play areas clean: wash bottles, pacifiers, toys, and stuffed animals regularly.

- Make sure you and your family members eat a well-balanced diet that is low in fat and high in calcium and iron.
  - Include foods such as fish, green vegetables, milk, and cheese.
  - Children with good diets absorb less lead.

- And remember, if you think that your house might have high lead levels, get your children and your home tested.
Lead in the Environment

This fact sheet will explain actions you can take to minimize exposure to lead in soil and protect your health and the health of your family.

- Lead is a common metal, has many uses and can be found in all parts of our environment, including in and around homes.

- Lead occurs naturally in the environment. It can be present in the ground, stuck to soil particles, in surface waters, and in underground or drinking water.

Exposure (being in contact) to Lead in Dust and Soil

Outdoor activities such as gardening or playing where soils are contaminated may result in harmful exposures. Indoor activities can also expose people to lead in dust, especially children.

Generally, you can be exposed to lead in soil and dust by:
- Breathing or inhaling dust and soil contaminated with lead
- Incidentally ingesting or eating soil or dust contaminated with lead

Children are at a greater risk for ingesting soil and dust by putting their hands in their mouths after touching soil or dust contaminated with lead.
Other Ways a Person Can Be Exposed to Lead

People can also be exposed to lead by:
- Eating food or drinking water containing lead.
- Spending time in areas where lead-based paints have been used and are deteriorating.
- Working in a job where lead is used.
- Using health-care products or folk remedies that contain lead.
- Engaging in certain hobbies in which lead is used (for example, stained glass, refinishing furniture, pottery, fishing sinkers, and ammunition).
- Playing with some imported toy necklaces or near some imported plastic mini-blinds.
- Lead is also found in cigarettes.

Health Effects

General Population:
Lead can affect almost every organ and system in your body. The most sensitive is the central nervous system, especially in children.

At high levels, lead may:
- Decrease reaction time
- Cause weakness in fingers, wrists, or ankles
- Possibly affect memory
- Damage the kidneys
- Damage the reproductive system

Lead may also cause anemia, a disorder of the blood, behavior problems, and learning disorders.

Children:
Children are more vulnerable to lead poisoning than adults.
- As babies, they can be exposed in the womb if their mothers have lead in their bodies.
- Children are at a greater risk for ingesting dust by putting their hands in their mouths after touching dust, dirt, or sand contaminated with lead.
- Lead-based paint may also be a hazard when found on surfaces that children can chew or that get a lot of wear and tear, such as windows, doors, stairs, and porches.

Additionally, babies exposed to lead in the womb of their mothers may be born prematurely and have lower weights at birth.
What You Can Do

If you suspect high levels of lead in the soil around your home:

- Get yourself and your family members, especially young children, tested for lead in blood.
  - Contact your doctor or call Pennsylvania’s Lead Program at 1-800-440-LEAD
  - This test will not identify exposure or possible health effects but will help you and your doctor take actions to protect health.
- Contact Greg Ham at the Environmental Protection Agency at 215-814-3194 for a possible evaluation of lead levels in soil.

Soil testing can help identify lead as a problem and determine actions to minimize exposure.

Collecting Your Own Soil Samples

It is likely that the samples collected by a certified inspector will be of more quality than those collected by the resident. Nevertheless, if a resident decides to collect the soil samples, we recommend:

- To send the samples to a laboratory recognized by EPA
  - A list of certified laboratories can be obtained by calling 1-800-424-LEAD.
- To strictly follow the instructions provided by the laboratory

Interpreting Results

If the results of the soil tests are more than 100 (ppm, ug/g, or mg/kg), then follow these measures:

- Children under six years old who are exposed to the soil should be tested for lead and examined by a physician.
- Follow the general recommended actions listed in this fact sheet to limit exposure and protect health.

On the other hand, 400 (ppm, ug/g, or mg/kg) is the clean-up Pennsylvania State’s standard for residential soils. If the tests are more than 400 (ppm, ug/g, or mg/kg), then:

- Test children under six years old and get them examined by a physician.
- Restrict access of children and pets to this area.
- Do not grow vegetables for consumption in this soil.
- Follow the general recommended actions listed in the following section to limit exposure and protect health.
General Recommendations to Limit Exposure and Protect Health

It is always important to follow these preventive measures:

- Test Young Children for Blood Lead
  - Because many children with lead poisoning do not show any obvious symptoms, a blood lead test should be done on children under six years old. Additionally, children may be exposed to lead from other sources, especially inside homes built before 1978.

- Follow These Strategies for Outdoor Activities:
  - Establish a clean-hands policy
    - Children should wash their hands when coming in from playing outside and before eating
  - Provide children with a covered sandbox
  - Maintain a healthy grass sod on play areas. Bare play areas, such as those under a swing set, can also be covered with woodchips, mulch, or clean sand.
  - Do not eat or smoke in areas with contaminated soil.

- Reduce Lead Contact while Gardening
  - Use raised beds filled with uncontaminated soil.
  - Keep dust to a minimum by maintaining a moist soil and using mulch in the garden.
  - Keep the soil pH from being acidic to prevent uptake of lead into vegetables. In general plants themselves do not absorb or accumulate large amounts of lead, but when they do, the soils usually test acidic. Maintain pH of 6.5 to 7.0 to help minimize absorption of lead by plants.
  - Grow vegetables that pose low risk from lead contamination including fruiting crops such as tomato, sweet corn, squash, eggplant, or pepper. Fruit from orchard-type trees such as apples do not accumulate significant amounts of lead.
  - Wash vegetables and fruits carefully to remove soil and dust deposits and peel all root crops. Unpeeled root crops such as potatoes or carrots are likely to have soil attached to them and may lead to accidental ingestion of lead-contaminated soil. Discard the outer and older leaves of leafy vegetables.

- Prevent Tracking Lead Indoors:
  - Avoid tracking soil into the house on shoes and clothing.
  - Remove garden gloves outside and designate a safe storage place for them out of reach of children.
  - Ask family members to remove their shoes by the door.

- Minimize Levels of Soil Lead in the House:
  - Regularly damp mopping and damp dusting of surfaces
  - If you have carpets, use a vacuum with a HEPA (High Efficiency Particulate Air) filter. Vacuuming without this filter can increase the amount of lead-contaminated dust in the air.
Appendix C: Calculations

Dose and Increased Cancer Risk Calculations

Dose (mg/kg/day) = \( C \text{ (mg/kg)} \ast \text{ IR (mg/day)} \ast \text{ EF (units cancel)} \ast \text{ CF (kg/mg)} \ast \frac{\text{BW (kg)}}{\text{}} \)

<table>
<thead>
<tr>
<th>Variables; (units)</th>
<th>Ingestion rate (IR); milligrams per day (mg/day)</th>
<th>Exposure frequency (EF); (exposures in days per year/total possible days per year)</th>
<th>Conversion factor (CF)</th>
<th>Body weight (BW); kilograms (kg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Child (1–6)</td>
<td>200</td>
<td>Chronic exposures: EF = (350 days/year) / (365 days/year)</td>
<td>0.000001</td>
<td>16</td>
</tr>
<tr>
<td>Child (1–6) pica†</td>
<td>1,000</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Adult</td>
<td>100</td>
<td></td>
<td></td>
<td>70</td>
</tr>
</tbody>
</table>

Theoretical Increased Cancer Risk = Adj D (mg/kg/day) \times CSF (mg/kg/day)^{-1} \times \frac{ED (y)}{AT (y)}

<table>
<thead>
<tr>
<th>Variables; (units)</th>
<th>Adjusted dose (Adj D); (mg /kg)</th>
<th>Cancer Slope Factor (CSF)</th>
<th>Exposure duration (ED); years (y)</th>
<th>Averaging time (AT); year (y)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Adult</td>
<td>Adjusted concentration per EF above†</td>
<td>Chemical-specific</td>
<td>30</td>
<td>70</td>
</tr>
</tbody>
</table>

* = Concentrations in parts per million (ppm) may be expressed as milligrams per kilograms (mg/kg).
† = Exposure frequencies are based on assumed exposure time (in days) per year per total possible time (in days) per year.
Appendix D: Glossary

Absorption
The process of taking in. For a person or animal, absorption is the process of a substance getting into the body through the eyes, skin, stomach, intestines or lungs.

Acute
Occurring over a short time [compare with chronic].

Acute exposure
Contact with a substance that occurs once or for only a short time (up to 14 days) [compare with intermediate duration exposure and chronic exposure].

Adverse health effect
A change in body function or cell structure that might lead to disease or health problems.

Aerobic
Requiring oxygen [compare with anaerobic].

Ambient
Surrounding (for example, ambient air).

Analyte
A substance measured in the laboratory. A chemical for which a sample (such as water, air or blood) is tested in a laboratory. For example, if the analyte is mercury, the laboratory test will determine the amount of mercury in the sample.

Analytic epidemiologic study
A study that evaluates the association between exposure to hazardous substances and disease by testing scientific hypotheses.

Background level
An average or expected amount of a substance or radioactive material in a specific environment, or typical amounts of substances that occur naturally in an environment.

Biodegradation
Decomposition or breakdown of a substance through the action of microorganisms (such as bacteria or fungi) or other natural physical processes (such as sunlight).

Biologic indicators of exposure study
A study that uses (a) biomedical testing or (b) the measurement of a substance [an analyte], its metabolite, or another marker of exposure in human body fluids or tissues to confirm human exposure to a hazardous substance [also see exposure investigation].
**Biologic monitoring**
Measuring hazardous substances in biologic materials (such as blood, hair, urine or breath) to determine whether exposure has occurred. A blood test for lead is an example of biologic monitoring.

**Biologic uptake**
The transfer of substances from the environment to plants, animals and humans.

**Biomedical testing**
Testing of persons to find out whether a change in a body function might have occurred because of exposure to a hazardous substance.

**Body burden**
The total amount of a substance in the body. Some substances build up in the body because they are stored in fat or bone or because they leave the body very slowly.

**Cancer**
Any one of a group of diseases that occurs when cells in the body become abnormal and grow or multiply out of control.

**Cancer risk**
A theoretical risk of getting cancer if exposed to a substance every day for 70 years (a lifetime exposure). The true risk might be lower.

**Carcinogen**
A substance that causes cancer.

**Case study**
A medical or epidemiologic evaluation of one person or a small group of people to gather information about specific health conditions and past exposures.

**Case-control study**
A study that compares exposures of people who have a disease or condition (cases) with people who do not have the disease or condition (controls). Exposures that are more common among the cases may be considered as possible risk factors for the disease.

**Central nervous system**
The part of the nervous system that consists of the brain and the spinal cord.

**CERCLA** [see Comprehensive Environmental Response, Compensation, and Liability Act of 1980]

**Chronic**
Occurring over a long time (more than 1 year) [compare with acute].
**Chronic exposure**
Contact with a substance that occurs over a long time (more than 1 year) [compare with acute exposure and intermediate duration exposure].

**Comparison value (CV)**
Calculated concentration of a substance in air, water, food or soil that is unlikely to cause harmful (adverse) health effects in exposed people. The CV is used as a screening level during the public health assessment process. Substances found in amounts greater than their CVs might be selected for further evaluation in the public health assessment process.

**Completed exposure pathway** [see exposure pathway].

**Comprehensive Environmental Response, Compensation, and Liability Act of 1980 (CERCLA)**
CERCLA, also known as Superfund, is the federal law that concerns the removal or cleanup of hazardous substances in the environment and at hazardous waste sites. ATSDR, which was created by CERCLA, is responsible for assessing health issues and supporting public health activities related to hazardous waste sites or other environmental releases of hazardous substances.

**Concentration**
The amount of a substance present in a certain amount of soil, water, air, food, blood, hair, urine, breath or any other media.

**Contaminant**
A substance that is either present in an environment where it does not belong or is present at levels that might cause harmful (adverse) health effects.

**Delayed health effect**
A disease or injury that happens as a result of exposures that might have occurred in the past.

**Dermal contact**
Contact with (touching) the skin [see route of exposure].

**Detection limit**
The lowest concentration of a chemical that can reliably be distinguished from a zero concentration.

**Disease prevention**
Measures used to prevent a disease or reduce its severity.

**Disease registry**
A system of ongoing registration of all cases of a particular disease or health condition in a defined population.
**Dose** (for chemicals that are not radioactive)
The amount of a substance to which a person is exposed over some time period. Dose is a measurement of exposure. Dose is often expressed as milligram (amount) per kilogram (a measure of body weight) per day (a measure of time) when people eat or drink contaminated water, food or soil. In general, the greater the dose, the greater the likelihood of an effect. An “exposure dose” is how much of a substance is encountered in the environment. An “absorbed dose” is the amount of a substance that actually got into the body through the eyes, skin, stomach, intestines or lungs.

**Dose-response relationship**
The relationship between the amount of exposure [dose] to a substance and the resulting changes in body function or health (response).

**Environmental media**
Soil, water, air, biota (plants and animals), or any other parts of the environment that can contain contaminants.

**Environmental media and transport mechanism**
Environmental media include water, air, soil, and biota (plants and animals). Transport mechanisms move contaminants from the source to points where human exposure can occur. The environmental media and transport mechanism is the second part of an exposure pathway.

**EPA**
United States Environmental Protection Agency.

**Epidemiologic surveillance**
The ongoing, systematic collection, analysis and interpretation of health data. This activity also involves timely dissemination of the data and use for public health programs.

**Epidemiology**
The study of the distribution and determinants of disease or health status in a population; the study of the occurrence and causes of health effects in humans.

**Exposure**
Contact with a substance by swallowing, breathing or touching the skin or eyes. Exposure may be short-term [acute exposure], of intermediate duration or long-term [chronic exposure].

**Exposure assessment**
The process of finding out how people come into contact with a hazardous substance, how often and for how long they are in contact with the substance, and how much of the substance they are in contact with.

**Exposure-dose reconstruction**
A method of estimating the amount of people’s past exposure to hazardous substances. Computer and approximation methods are used when past information is limited, not available or missing.
**Exposure investigation**  
The collection and analysis of site-specific information and biologic tests (when appropriate) to determine whether people have been exposed to hazardous substances.

**Exposure pathway**  
The route a substance takes from its source (where it began) to its end point (where it ends), and how people can come into contact with (or get exposed to) it. An exposure pathway has five parts: a source of contamination (such as an abandoned business); an environmental media and transport mechanism (such as movement through groundwater); a point of exposure (such as a private well); a route of exposure (eating, drinking, breathing or touching), and a receptor population (people potentially or actually exposed). When all five parts are present, the exposure pathway is termed a completed exposure pathway.

**Feasibility study**  
A study by EPA to determine the best way to clean up environmental contamination. A number of factors are considered, including health risk, costs and what methods will work well.

**Geographic information system (GIS)**  
A mapping system that uses computers to collect, store, manipulate, analyze and display data. For example, GIS can show the concentration of a contaminant within a community in relation to points of reference such as streets and homes.

**Grand rounds**  
Training sessions for physicians and other health care providers about health topics.

**Groundwater**  
Water beneath the earth’s surface in the spaces between soil particles and between rock surfaces [compare with surface water].

**Hazard**  
A source of potential harm from past, current or future exposures.

**Hazardous waste**  
Potentially harmful substances that have been released or discarded into the environment.

**Health consultation**  
A review of available information or collection of new data to respond to a specific health question or request for information about a potential environmental hazard. Health consultations are focused on a specific exposure issue. Health consultations are therefore more limited than a public health assessment, which reviews the exposure potential of each pathway and chemical [compare with public health assessment].

**Health education**  
Programs designed with a community to help it know about health risks and how to reduce these risks.
Health investigation
The collection and evaluation of information about the health of community residents. This information is used to describe or count the occurrence of a disease, symptom or clinical measure and to estimate the possible association between the occurrence and exposure to hazardous substances.

Health promotion
The process of enabling people to increase control over and to improve their health.

Health statistics review
The analysis of existing health information (i.e., from death certificates, birth defects registries and cancer registries) to determine if there is excess disease in a specific population, geographic area and time period. A health statistics review is a descriptive epidemiologic study.

Indeterminate public health hazard
This category means a lack of data or information. The category used in ATSDR’s public health assessment documents when a professional judgment about the level of health hazard cannot be made because information critical to such a decision is lacking.

Incidence
The number of new cases of disease in a defined population over a specific time period [contrast with prevalence].

Ingestion
The act of swallowing something through eating, drinking or mouthing objects. A hazardous substance can enter the body this way [see route of exposure].

Inhalation
The act of breathing. A hazardous substance can enter the body this way [see route of exposure].

Intermediate duration exposure
Contact with a substance that occurs for more than 14 days and less than a year [compare with acute exposure and chronic exposure].

In vitro
In an artificial environment outside a living organism or body. For example, some toxicity testing is done on cell cultures or slices of tissue grown in the laboratory, rather than on a living animal [compare with in vivo].

In vivo
Within a living organism or body. For example, some toxicity testing is done on whole animals, such as rats or mice [compare with in vitro].
Lowest-observed-adverse-effect level (LOAEL)
The lowest tested dose of a substance that has been reported to cause harmful (adverse) health effects in people or animals.

Medical monitoring
A set of medical tests and physical exams specifically designed to evaluate whether an individual’s exposure could negatively affect that person’s health.

Metabolized
The conversion or breakdown of a substance from one form to another by a living organism.

Metabolite
Any product of metabolism.

mg/kg
Milligram per kilogram.

µg/m³
Microgram per cubic meter; a measure of the concentration of a chemical in a known volume (a cubic meter) of air, soil or water.

Migration
Moving from one location to another.

Minimal risk level (MRL)
An ATSDR estimate of daily human exposure to a hazardous substance at or below which that substance is unlikely to pose a measurable risk of harmful (adverse), noncancerous effects. MRLs are calculated for a route of exposure (inhalation or oral) over a specified time period (acute, intermediate or chronic). MRLs should not be used as predictors of harmful (adverse) health effects [see reference dose].

Morbidity
State of being ill or diseased. Morbidity is the occurrence of a disease or condition that alters health and quality of life.

Mortality
Death. Usually the cause (a specific disease, condition or injury) is stated.

National Priorities List for Uncontrolled Hazardous Waste Sites (National Priorities List or NPL)
EPA’s list of the most serious uncontrolled or abandoned hazardous waste sites in the United States. The NPL is updated on a regular basis.
No apparent public health hazard
A category used in ATSDR’s public health assessments for sites where human exposure to contaminated media might be occurring, might have occurred in the past, or might occur in the future, but where the exposure is not expected to cause any harmful health effects.

No-observed-adverse-effect level (NOAEL)
The highest tested dose of a substance that has been reported to have no harmful (adverse) health effects on people or animals.

No public health hazard
A category used in ATSDR’s public health assessment documents for sites where people have never and will never come into contact with harmful amounts of site-related substances.

Pica
A craving to eat nonfood items, such as dirt, paint chips and clay. Some children exhibit pica-related behavior.

Plume
A volume of a substance that moves from its source to places farther away from the source. Plumes can be described by the volume of air or water they occupy and the direction they move. For example, a plume can be a column of smoke from a chimney or a substance moving with groundwater.

Point of exposure
The place where someone can come into contact with a substance present in the environment [see exposure pathway].

Population
A group or number of people living within a specified area or sharing similar characteristics (such as occupation or age).

Potential exposure pathway [see exposure pathway].

ppb
Parts per billion.

ppm
Parts per million.

Prevalence
The number of existing disease cases in a defined population during a specific time period [contrast with incidence].

Prevention
Actions that reduce exposure or other risks, keep people from getting sick, or keep disease from getting worse.
**Public comment period**
An opportunity for the public to comment on agency findings or proposed activities contained in draft reports or documents. The public comment period is a limited time period during which comments will be accepted.

**Public availability session**
An informal, drop-by meeting at which community members can meet one-on-one with PADOH and ATSDR staff members to discuss health and site-related concerns.

**Public health action**
A list of steps to protect public health.

**Public health advisory**
A statement made by PADOH or ATSDR to EPA or a state regulatory agency that a release of hazardous substances poses an immediate threat to human health. The advisory includes recommended measures to reduce exposure and reduce the threat to human health.

**Public health assessment (PHA)**
A PADOH or an ATSDR document that examines hazardous substances, health outcomes and community concerns at a hazardous waste site to determine whether people could be harmed from coming into contact with those substances. The PHA also lists actions that need to be taken to protect public health [compare with health consultation].

**Public health hazard**
A category used in ATSDR’s public health assessments for sites that pose a public health hazard because of long-term exposures (greater than 1 year) to sufficiently high levels of hazardous substances or radionuclides that could result in harmful health effects.

**Public health hazard categories**
Public health hazard categories are statements about whether people could be harmed by conditions present at the site in the past, present or future. One or more hazard categories might be appropriate for each site.

**Public meeting**
A public forum with community members for communication about a site.

**Receptor population**
People who could come into contact with hazardous substances [see exposure pathway].

**Reference dose (RfD)**
An EPA estimate, with uncertainty or safety factors built in, of the daily lifetime dose of a substance that is unlikely to cause harm in humans.

**Registry**
A systematic collection of information on persons exposed to a specific substance or having specific diseases [see exposure registry and disease registry].
Remedial Investigation
The CERCLA process of determining the type and extent of hazardous material contamination at a site.

This Act regulates management and disposal of hazardous wastes currently generated, treated, stored, disposed of or distributed.

Risk
The probability that something will cause injury or harm.

Route of exposure
The way people come into contact with a hazardous substance. Three routes of exposure are breathing [inhaling], eating or drinking [ingesting] or contact with the skin [dermal contact].

Sample
A portion or piece of a whole. A selected subset of a population or subset of whatever is being studied. For example, in a study of people the sample is a number of people chosen from a larger population [see population]. An environmental sample (for example, a small amount of soil or water) might be collected to measure contamination in the environment at a specific location.

Sample size
The number of units chosen from a population or environment.

Source of contamination
The place where a hazardous substance comes from, such as a landfill, waste pond, incinerator, storage tank or drum. A source of contamination is the first part of an exposure pathway.

Special populations
People who might be more sensitive or susceptible to exposure to hazardous substances because of factors such as age, occupation, gender or behaviors (for example, cigarette smoking). Children, pregnant women and older people are often considered special populations.

Statistics
A branch of mathematics that deals with collecting, reviewing, summarizing and interpreting data or information. Statistics are used to determine whether differences between study groups are meaningful.

Substance
A chemical.

Surface water
Water on the surface of the earth, such as in lakes, rivers, streams, ponds and springs [compare with groundwater].
Surveillance [see epidemiologic surveillance]

Toxic agent
Chemical or physical (for example, radiation, heat, cold, microwaves) agents which, under certain circumstances of exposure, can cause harmful effects to living organisms.

Toxicological profile
An ATSDR document that examines, summarizes, and interprets information about a hazardous substance to determine harmful levels of exposure and associated health effects. A toxicological profile also identifies significant gaps in knowledge on the substance and describes areas where further research is needed.

Toxicology
The study of the harmful effects of substances on humans or animals.

Uncertainty factor
Mathematical adjustments for reasons of safety when knowledge is incomplete. For example, factors used in the calculation of doses that are not harmful (adverse) to people. These factors are applied to the lowest-observed-adverse-effect-level (LOAEL) or the no-observed-adverse-effect-level (NOAEL) to derive a minimal risk level (MRL). Uncertainty factors are used to account for variations in people’s sensitivity, for differences between animals and humans, and for differences between a LOAEL and a NOAEL. Scientists use uncertainty factors when they have some, but not all, of the information from animal or human studies to decide whether an exposure will cause harm to people [also sometimes called a safety factor].

Urgent public health hazard
A category used by ATSDR for sites where short-term acute exposures to hazardous substances or conditions could result in harmful health effects that require rapid intervention.

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United States Environmental Protection Agency http://www.epa.gov/glossary/.