

# Letter Health Consultation

## Pollock Park Adjacent Residential Soil Evaluation

POLLOCK PARK ADJACENT RESIDENTIAL  
PROPERTIES  
POTTSTOWN, MONTGOMERY COUNTY,  
PENNSYLVANIA

September 2020

Prepared by:



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Room 933 | Health and Welfare Building 625 Forster Street | Harrisburg, PA  
17120-0701

## **Health Consultation: A Note of Explanation**

The Pennsylvania Department of Health (PADOH) prepared this Letter Health Consultation for residences adjacent to the Pollock Park in Pottstown, Montgomery County, Pennsylvania. This publication was made possible by grant number CDC-RFA-TS17-170103CONT19 from the Agency for Toxic Substances Disease Registry (ATSDR). The PADOH evaluated data of known quality using approved methods, policies, and ATSDR procedures existing at the date of publication. Its contents are solely the responsibility of the authors and do not necessarily represent the official views of ATSDR or the U.S. Department of Health and Human Services.

### **Contact Information**

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To: Michael A. Lenhart  
Parks and Recreation Director  
Borough of Pottstown

C. David Brown P.G. | Professional Geologist Manager  
Environmental Cleanup & Brownfields Program  
Department of Environmental Protection | Southeast Regional Office

Dear Mr. Lenhart,

This Letter Health Consultation (LHC) is written in response to your request to the Agency for Toxic Substances and Disease Registry (ATSDR) and the Pennsylvania Department of Health (PADOH) to evaluate residential surface soil data to determine whether exposures may result in adverse health effects. The Environmental Protection Agency (EPA) provided the soil sample data collected from four residential yards adjacent to Pollock Park in Pottstown, Montgomery County, Pennsylvania. Pollock Park (the park) is a recreational park constructed on a former iron scrap yard. In October 2017, Environmental Standards conducted soil quality investigation at the park and detected polycyclic aromatic hydrocarbons (PAHs), polychlorinated biphenyls (PCBs), volatile organic compounds (VOCs), metals, and other chemicals. In May 2018, EPA conducted soil sampling in adjacent residential locations to determine if these contaminants were also present off the park property. This letter provides our public health evaluation of only residential surface soil sampling results and includes recommendations for stakeholders and property owners where contaminants were detected.

Based on PADOH's data evaluation, chronic exposure to PAHs and arsenic detected in residential surface soil is not expected to cause harmful noncancer health effects. However, chronic exposure to PAHs and arsenic may cause low increased cancer risk. Exposure to the detected levels of lead may cause adverse noncancer health effects, particularly in children. The PADOH recommends residents to perform proper personal hygiene (frequent washing of hands, arms, and face with soap and water) habits after activities in their yard area. The PADOH also recommends residents, particularly pregnant women and parents of children under six years of age, take steps to reduce their lead exposures from other sources such as lead-based paint (living in homes built before 1978), brass and lead-containing toys, ceramics, plastic products, jewelry, decorative ornaments, candies imported from other countries or traditional home remedies, hobby and occupationally related exposures. Residents who are pregnant and children should obtain blood lead testing and talk to their health professional or call PADOH's Lead Information Line at 1-800-440-LEAD (5323) if they have concerns.

The remainder of this LHC presents detailed information in support of the PADOH's analysis, conclusions, and recommendations.

## **Site Background and Description**

The park is approximately two acres in size and is located in a mixed-use, mainly industrial and residential area, between Cross and South Streets of Pottstown in Montgomery County, Pennsylvania. The park includes asphalt paved basketball courts, a grassed playing field, a paved trail, and two paved parking lots/playing areas. Most of the park is covered with grass. Trees and shrubs are present along the perimeter and in the center of the park. The known historical use of the park dated to at least 1909 when the park appeared to have been used as an iron scrap yard by Mayer Pollock Steel Company. The Borough of Pottstown acquired the property in 1979 and subsequently converted the parcels into the park. In October 2017, an environmental site assessment (ESA) identified multiple contaminants in the park's surface and subsurface soils, requiring soil remedial action [EPA 2018]. At present, the park perimeter includes high visibility fencing, and signs are present warning the public from access to the park.

On May 15, 2018, the EPA further delineated the results of the ESA to the adjacent residential areas, warranting an EPA removal action. Based on soil sampling results and EPA's cleanup standards evaluation, EPA concluded that the detected contaminant levels at the adjacent residential areas do not warrant a removal action. The PADOH used the same sampling data for public health perspective analysis by considering site-specific and age-specific exposure conditions to determine whether harmful health effects are expected from contaminants in the environment and make recommendations to protect public health (ATSDR 2005).

## **Soil Data**

The EPA collected discrete surface soil samples (depth of less than three inches) from the following residential yards: SB18, SB19, SB20, and SB21. During the same time EPA also collected surface and subsurface soil samplings from the park. The EPA sampling identified site-related contaminants such as PAHs, PCBs, VOCs, and metals detected both in park soils (surface and subsurface) and in the surface soils of four residential yards [EPA 2018]. In August 2018, the EPA analyzed the soil sample data and sent letters to all four homeowners. This letter health consultation focuses only on the analysis of four residential yard surface soil sampling results (SB18, SB19, SB20, SB21).

## **Exposure Pathway**

An exposure pathway is a description of the way that an environmental release moves from its source (where it began), to where, and how people can come into contact with (or get exposed to) the environmental contaminant. To determine whether residents are likely to be exposed to contaminants on their property, the PADOH evaluated the environmental and human components that could lead to human exposure. An exposure pathway includes the following five elements [ATSDR, 2005]:

1. A contaminant source (e.g., industrial facilities utilizing hazardous materials, landfills, waste sites);

2. An environmental medium (or media) and transport mechanisms (e.g., water, soil, or air);
3. A point of exposure (e.g., ambient air, private residential well water or a building into which vapors enter, indoor air);
4. A route of exposure (e.g., ingestion or inhalation or dermal); and
5. A receptor population (e.g., residents, children, workers).

Exposure pathways are categorized as completed, potential, or eliminated. A completed exposure pathway is one in which all five elements are present. In a potential exposure pathway, at least one of the pathways elements is uncertain, indicating that exposure to a contaminant could have occurred in the past, is occurring, or could occur in the future. A pathway is eliminated when one or more elements are missing or prevented and are unlikely to be present [ATSDR, 2005]. The PADOH identified a completed exposure pathway via ingestion and dermal contact of contaminants present in soil based on past and present exposure condition, since all five parts of an exposure pathway are present, as seen in Table 1 below.

**Table 1: Exposure Pathway Analysis**

Source	Medium	Point of Exposure	Route of Exposure	Receptor Population	Exposure Pathway Status		
					Past	Present	Future
Pollock Park-historic contamination	Soil	Residential yards	Ingestion, Dermal contact	Residents and visitors	Completed	Completed	Potential
	Water	Drinking and showering	None (Ingestion and dermal)	None	Eliminated	Eliminated	Eliminated
	Air	Air emissions	Inhalation	Residents and visitors	Potential	Potential	Potential

From our exposure analysis, exposure to soil contaminants is expected to occur by ingestion and dermal contact from the contaminated residential soil near the park. The PADOH further evaluated the completed ingestion and dermal exposure pathway to determine whether site-specific exposures were at high concentrations, frequent enough, and for a long period of time, to result in adverse health effects. Ingestion of contaminants through surface and groundwater has been eliminated since all residents near the park are on the public water system and there are no private wells identified near these residential locations [Lenhart AM, email communication, July 21<sup>st</sup>, 2020]. Residents could inhale/breathe-in contaminated dust from their residential soil. However, the PADOH is not able to evaluate the potential inhalation exposure pathway of analysis at this time, since air quality data were not available. Children are generally more susceptible to harmful environmental exposures owing to physiological and behavioral reasons [ATSDR 2005]. However, the EPA was informed by the Borough of Pottstown that no children live in these four single-family homes tested. Although no children live at any of these four residential locations, the PADOH evaluated the potential adverse health effects for children to be protective of any children that might visit or move into the neighborhood.

## Data Screening and Evaluation

Following the identification of a completed exposure pathway, the PADOH screened the soil sample results against ATSDR's health-based comparison values (CVs), such as environmental media evaluation guides (EMEGs), reference dose media evaluation guides (RMEGs), and cancer risk evaluation guides (CREGs) to identify the contaminants that require further analysis as part of this assessment. The EMEGs are estimated contaminant concentrations in specific media (i.e., air, water, soil) that are not expected to result in adverse noncarcinogenic health effects after exposure for a given period of time, i.e., acute (14 days or less), intermediate (15 to 365 days), and chronic (365 days or more) exposure durations [ATSDR 2005]. The EMEGs are based on ATSDR minimal risk levels (MRLs) for a given period of time (acute, intermediate and chronic). An MRL is 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 noncancerous health effects. The RMEGs are estimated contaminant concentrations at which a chronic human exposure is not likely to result in adverse noncarcinogenic effects based on EPA's reference dose (RfD). A RfD is an EPA estimate of the daily dose of a substance over a lifetime of exposure that is unlikely to cause harm in humans. The CREGs are estimated contaminant concentrations that would be expected to cause no more than one excess cancer in a million persons exposed during their lifetime (78 years). These health guidelines are derived based on data drawn from the epidemiologic and toxicologic literature with many uncertainty or safety factors applied to ensure that they are amply protective of human health. When an ATSDR CV is not available, screening values are acquired from the environmental agency's medium-specific concentration or cleanup standards (EPA or PADEP). However, the basis for values obtained from the environmental agencies were not reviewed/approved by ATSDR. If contaminant levels are found above health-based CVs, it does not mean adverse health effects are likely, but further evaluation is required. Contaminants that exceeded health-based screening values were selected as contaminants of concern (COC) for further evaluation of site-specific exposures to derive the daily exposure dose. Exposure doses were calculated in accordance with ATSDR exposure dose guidance and standard default exposure assumptions for soil ingestion and dermal contact [ATSDR 2005]. Equations and exposure parameters utilized in this assessment are presented in Appendix A.

The PADOH also used ATSDR's Public Health Assessment Site Tool to calculate exposure dose concentrations, the noncancer hazard quotients, and the cancer risk estimates. Calculations were based on reasonable maximum exposure concentrations. Please refer to Appendix B, Tables B1, and B2 for additional information.

## Public Health Implications

The PADOH identified **arsenic, lead, and PAHs** as COCs (contaminants that exceeded health-based screening values). These soil contaminants are further evaluated in this section. VOCs and manmade chemicals PCBs were detected in residential soil, but at an estimated value (J value), which means that the analyte was present in the field sample, but the concentration reported is an estimate. As a conservative approach, the PADOH evaluated these estimated values. The detected

J values for all the VOCs and PCBs were below the health-based screening values. Metals such as aluminum, antimony, calcium, chromium, copper, iron, magnesium, manganese, sodium, and vanadium were also detected at an estimated value (J value), and the levels were below the screening values. Metals such as beryllium, cadmium, cobalt, mercury, nickel, and zinc were detected below health CVs or cleanup standards. Potassium was detected at a maximum concentration of 811 milligrams per kilogram (mg/kg); however, a health CV or cleanup standard is not available for potassium. Potassium is an essential micronutrient for both plants and humans. Chronic ingestion of potassium supplements (e.g., up to 15,600 mg for 5 days) in healthy individuals could lead to increase in potassium levels in plasma [NIH 2020]. Short-term potassium supplementation of approximately 2,500 mg/day (31.2 mg/kg/day) along with the recommended daily intake of 2,600 mg/day (32.5 mg/kg/day) appears to be safe for generally healthy individuals [NAP 2019]. The calculated exposure dose from the maximum detected level of potassium (811 mg/kg) in contaminated soil is 0.001 mg/kg/day, which is a trivial amount when compared to the short-term potassium supplementation of 31.2 mg/kg/day, so it is not considered as a COC. A summary of contaminants detected, and their respective screening values are given in Appendix A Table A1.

### ***Arsenic***

Arsenic is a naturally occurring element that is widely distributed in the earth's crust. Organic forms of arsenic are less harmful than the inorganic forms [ATSDR 2007]. Some forms of arsenic may be tightly attached to particles or embedded in minerals that they are not taken up by plants and animals. In general, arsenic concentrations in soil range from 1–40 mg/kg with a mean of 5 mg/kg, although much higher levels may occur in mining areas, at waste sites, near high geological deposits of arsenic-rich minerals, or from pesticide applications. The detected levels arsenic at these four residential yards were within the background range. The United States Department of Health and Human Services, the International Agency for Research on Cancer, and the EPA have determined that inorganic arsenic is carcinogenic to humans [ATSDR 2007].

Ingestion of arsenic contaminated soil or consumption of foods grown in arsenic contaminated soil is one way that arsenic can enter the body. Chronic arsenic exposures in people have been linked to lung cancer, basal and squamous cell skin cancers, liver cancer, urinary tract cancers (bladder, kidney, ureter, and all urethral cancers), and intra-epidermal cancers [ATSDR 2007].

Dermal exposure to metals is typically not considered a major exposure pathway due to the limited ability of metals to penetrate the skin barrier. However, as a conservative approach, the PADOH calculated both ingestion and dermal exposures by using the default bioavailability factor of 0.6 and dermal absorption factor of 0.03 for arsenic contaminated soil exposure evaluation [PHAST 2020a]. Ingestion and dermal dose calculations, equations, and exposure parameters utilized in this assessment are presented in Appendix A.

As seen in Table 2, the calculated exposure doses for arsenic (mg/kg/day) in adults are as follows:  $1.7 \times 10^{-5}$  (SB18),  $6.0 \times 10^{-6}$  (SB19),  $4.1 \times 10^{-6}$  (SB20), and  $4.3 \times 10^{-6}$  (SB21). The calculated exposure doses at all four locations were below ATSDR's MRL ( $3.0 \times 10^{-4}$  mg/kg/day). The calculated

exposure doses in children were also below ATSDR’s MRL at all locations (Appendix B, Table B1).

Noncancerous health effects are evaluated based on hazard quotient (HQ). The HQ for a chemical is the ratio of the estimated exposure dose to the MRL or the RfD. The higher the concentration (above MRL or RfD), the greater the chance for noncancerous health effects. If the HQ is less than 1.0, noncancerous harmful effects are unlikely to occur. If a HQ exceeds 1.0 the exposure dose concentrations are then compared to levels in the scientific literature that cause health effects in laboratory animals and human epidemiological studies.

**Table 2. Calculated Exposure Dose, Hazard Quotient and Cancer Risk Estimates**

Residence	Contaminant of Concern	Concentration mg/kg	Exposure Dose (ED) mg/kg/day	Hazard Quotient (ED/cMRL)	Excess Cancer Risk <sup>a</sup>
SB18	Arsenic	19.2	$1.7 \times 10^{-5}$	0.06	$1.1 \times 10^{-5}$
SB19	Arsenic	6.6	$6.0 \times 10^{-6}$	0.02	$3.8 \times 10^{-6}$
SB20	Arsenic	4.5	$4.1 \times 10^{-6}$	0.01	$2.6 \times 10^{-6}$
SB21	Arsenic	4.7	$4.3 \times 10^{-6}$	0.01	$2.7 \times 10^{-6}$

cMRL= ATSDR chronic MRL of 0.0003 mg/kg/day for arsenic; a=cancer risk estimates were calculated using EPA’s cancer risk slope factors of  $1.5 \text{ (mg/kg/day)}^{-1}$  for arsenic.

As shown in Table 2, the HQ for adults and children at all locations was below 1.0. (Appendix B, Table B1). Therefore, noncancer health effects are not likely to occur for both children and adults.

The PADOH calculated the cancer risk estimates using the EPA’s arsenic oral cancer slope factor (CSF) of  $1.5 \text{ (mg/kg/day)}^{-1}$ . Based on the arsenic levels detected, adult cancer risks at the following locations were as follows: SB18 ( $1.1 \times 10^{-5}$ ), SB19 ( $3.8 \times 10^{-6}$ ), SB20 ( $2.6 \times 10^{-6}$ ), and SB21 ( $2.7 \times 10^{-6}$ ). The cancer risk estimates for children below 21 years (Appendix B, Table B1) were as follows: SB18 ( $3.4 \times 10^{-5}$ ), SB19 ( $1.2 \times 10^{-5}$ ), SB20 ( $8.1 \times 10^{-5}$ ), and SB21 ( $8.4 \times 10^{-5}$ ). The cancer risk estimates indicate that exposure to arsenic contaminated soil at these residential yards may cause low increased cancer risk. However, the cancer risk estimates for both adults and children were low and fall below or within the EPA’s acceptable cancer risk range of  $1 \times 10^{-4}$  and  $1 \times 10^{-6}$ .

The exposure dose equations and assumptions used for cancer risk estimations are given in Appendix A.

**Lead**

Lead is a naturally occurring element in the earth’s crust and can be found throughout our environment in the air, water, and soil. This element can also be present in the environment due to pollution, particularly in urban areas, from sources related to human activity such as traffic emissions, industrial emissions, weathering of buildings, and pavement surfaces. Background levels of lead can vary greatly depending on the surrounding landscape. Soil in developed areas such as urban soil often has lead concentrations much greater than naturally occurring background levels. These concentrations frequently range from 150 mg/kg to as high as 10,000 mg/kg at the base of structures painted with lead-based paint (<https://extension.psu.edu/lead-in-residential->



[soils-sources-testing-and-reducing-exposure](#)). The detected levels of lead at these four residential yards were within the urban background range.

Lead has no nutritional benefits for humans and has its greatest adverse effect on the nervous system, especially in children. An unborn child can also be exposed to lead if the mother has lead levels in her body. This exposure can cause problems such as premature births, low birth weight, decreased mental ability, learning difficulties, and reduced growth as young children. Young children can also be exposed to lead through their mother's breast milk if the mother has an elevated blood-lead level in her system [ATSDR 2007a, ATSDR 2009]. Additionally, exposure to lead from residences built before 1978 that contain lead-based paint that may lead to additional lead exposure at levels of a health concern. Even extremely low levels of lead exposure have been shown to affect intelligence quotient (IQ), ability to pay attention, and academic achievement as the nervous system is the main target for lead toxicity in adults and children [Lanphear et al. 2005].

Since no ATSDR health-based CV exists for screening lead, PADOH evaluated lead using EPA's Integrated Exposure Uptake Biokinetic (IEUBK) model. The IEUBK model calculates exposure from lead in air, water, soil, dust, diet, paint, and other sources using site-specific data and default values and predicts the risk of elevated blood lead levels in children six months to seven years of age. As it is true for all models, the accuracy of the results obtained using the IEUBK model is highly dependent on the selection of the various coefficients and default values that are used [EPA 1994].

Based on the IEUBK model, exposure to lead in soil at 330 mg/kg could result in a blood lead level (BLL) of 5.0 µg/dL in children aged one to two years (Appendix B, Table B3). The screening value of 330 mg/kg was identified for this evaluation by running the model with incrementally increasing concentrations of soil lead along with the model's default variables for exposure to lead in air and drinking water until the resulting BLL reached a minimum of 5.0 µg/dL. While no safe BLL in children has been identified, a BLL of 5.0 µg/dL is considered "elevated" [CDC 2012]. The detected lead levels at location SB18 (395 mg/kg) and SB19 (356 mg/kg) exceeded 330 mg/kg.

### ***Polycyclic Aromatic Hydrocarbons (PAHs)***

Polycyclic Aromatic Hydrocarbons (PAHs) are produced by the incomplete combustion of organic materials such as coal and wood [ATSDR 1995]. They are also found in petrochemical products such as asphalt, coal tar, creosote, and roofing tar. PAHs are released into the environment from processes such as volcanic eruptions, forest fires, home wood burning, and vehicle exhaust. As a result, they are commonly found in the environment as mixtures. Background soil concentrations of some of the PAHs in urban soil in the United States are given below in Table 3 [ATSDR 1995]. The detected PAHs levels at these four residential yards were well below or within the background concentration range for most of the PAHs except benzo(a)pyrene.

**Table 3: Background Soil Concentration of Polycyclic Hydrocarbons in Urban Soil**

Polycyclic Aromatic Hydrocarbons	Concentrations at each Residential Locations (mg/kg)				Background Concentration (mg/kg) *
	SB18	SB19	SB20	SB21	
Benzo(a)anthracene	0.400	0.450	0.470	0.120	0.169–59
Benzo(a)pyrene	0.400	0.430	0.420	0.130	0.165–0.220
Benzo(b)fluoranthene	0.550	0.600	0.610	0.190	15–62
Indeno(1,2,3-cd) Pyrene	0.240	0.290	0.280	0.008	8–61
Chrysene	0.460	0.520	0.500	0.130	0.251–0.640
Fluoranthene	0.760	0.930	0.900	0.220	0.200–166
Pyrene	0.810	1.0	0.950	0.220	0.145–147

\*Source [ATSDR 1995]

The most studied PAH is benzo(a)pyrene. Benzo(a)pyrene has been classified as carcinogenic to humans by EPA [EPA 2017]. The noncarcinogenic effects of PAHs involve primarily the pulmonary, gastrointestinal, renal, and dermatologic systems [ATSDR 1995]. The PAHs detected in soil were evaluated as a mixture using a calculated benzo(a)pyrene toxic equivalent/potency factor. Each PAH concentration is multiplied by its toxic/potency equivalent factor to obtain calculated toxic equivalents. These calculated toxic equivalents are then summed as benzo(a)pyrene equivalents for each location (see Appendix A, Table A2). The PADOH used the default dermal exposure factor of 0.13 and bioavailability factor of 1.0 for benzo(a)pyrene equivalents contaminated soil evaluation [EPA 2004, PHAST 2020a]. Ingestion and dermal dose calculations, equations, and exposure parameters utilized in this assessment are presented in Appendix A.

As seen in Table 4 below, the calculated exposure doses for benzo(a)pyrene equivalents (mg/kg/day) in adults are as follows: SB18 (1.17), SB19 (1.12), SB20 (1.36), and SB21 (0.43). The calculated exposure doses for children in each age group are given in Appendix B, Table B2. The site-specific exposure dose at all four locations for both adults and children were lower than the EPA's RfD of  $3 \times 10^{-4}$  mg/kg/day. As shown in Table 4, and Table B2 the HQ's were below 1.0, indicating noncancerous health effects are unlikely to occur based on the exposure dose concentrations.

The PADOH calculated the cancer risk estimates using the EPA's benzo(a)pyrene oral CSF of  $1.0 \text{ (mg/kg/day)}^{-1}$ . Based on the benzo(a)pyrene equivalent levels, adult cancer risks at the following locations were as follows: SB18 ( $9.6 \times 10^{-7}$ ), SB19 ( $9.2 \times 10^{-7}$ ), SB20 ( $1.1 \times 10^{-6}$ ), and SB21 ( $3.5 \times 10^{-7}$ ). The cancer risk estimates for children below 21 years (Appendix B, Table B2) were as follows: SB18 ( $1.3 \times 10^{-5}$ ), SB19 ( $1.3 \times 10^{-5}$ ), SB20 ( $1.5 \times 10^{-5}$ ), and SB21 ( $4.0 \times 10^{-6}$ ). The cancer risk estimates indicate that exposure to PAH contaminated soil at these residential yards may cause low increased cancer risk. However, the cancer risk estimates for both adults and children were low and below the EPA's acceptable cancer risk range of  $1 \times 10^{-4}$  and  $1 \times 10^{-6}$ .

The exposure dose equations and assumptions used for cancer risk estimations are given in Appendix A.

**Table 4. Calculated Exposure Dose, Hazard Quotient and Cancer Risk Estimates**

Residence	Contaminants of Concern	Concentration mg/kg	Ingestion and Dermal Exposure Dose (ED) mg/kg/day	Hazard Quotient (ED/RfD)	Excess Cancer Risk <sup>a</sup>
SB18	B(a)P equivalent	1.17	2.3x10 <sup>-6</sup>	0.0076	9.6x10 <sup>-7</sup>
SB19	B(a)P equivalent	1.12	2.2x10 <sup>-6</sup>	0.0072	9.2x10 <sup>-7</sup>
SB20	B(a)P equivalent	1.36	2.6x10 <sup>-6</sup>	0.0088	1.1x10 <sup>-6</sup>
SB21	B(a)P equivalent	0.43	8.3x10 <sup>-7</sup>	0.0028	3.5x10 <sup>-7</sup>

EPA's RfD of 0.0003 mg/kg/day for benzo(a)pyrene; a=cancer risk estimates were calculated using EPA'S cancer risk slope factors of 1.0 (mg/kg/day)<sup>-1</sup> for benzo(a)pyrene.

## Child Health Consideration

The PADOH recognizes that infants and children have unique vulnerabilities compared to adults. Children are small and likely to play outdoors more and receive higher doses of potential contaminants than adults. Children are more sensitive and susceptible than adults to the effects of some of the toxic contaminants such as lead and mercury. These contaminants can be passed from a pregnant mother to the developing child. These contaminants can also pass into breast milk. Although currently no children reside at these four residences, if the demographic of these residential area changes or the recreational visitors with children visit these residences, precautions should be taken to limit the soil exposure by children and adults as much as possible as a protective measure.

## Limitations

The conclusion is based on a single surface soil sample from each residential property. The full nature and extent of soil contamination at these 4 residences are not known. The samples from other portions of the property/residential yards might have different results. Data gaps are present in terms of missing air data, subsurface soil data, and groundwater data beneath the soil.

## Conclusions

Based on PADOH's data evaluation, chronic exposure to PAHs and arsenic detected in residential surface soil is not expected to cause harmful noncancer health effects. However, chronic exposure to PAHs and arsenic may cause low increased cancer risk. Exposure to lead at the detected levels may cause adverse noncancer health effects, particularly in children.

## Recommendations

- The PADOH recommends residents to follow proper personal hygiene (frequent washing of hands, arms and face with soap and water) after activities in their yard area.

- The PADOH also recommends residents, particularly pregnant women and parents of children under six years of age, take steps to reduce lead exposures from other sources such as lead-based paint (especially in homes built before 1978), brass and lead-containing toys, plastic products, ceramics, jewelry, decorative ornaments, candies imported from other countries or traditional home remedies, hobby and occupationally related exposures. Close supervision of children visiting these residences along with the proper practice of personal hygiene must be followed to avoid exposure to contaminants.
- Residents who are pregnant and children should consider blood lead testing and talk to their health professional or call PADOH's Lead Information Line at 1-800-440-LEAD (5323) if they have concerns.
- The PADOH also recommends residents using clean soil and compost for gardening, covering (or mulching) bare soil, and putting a barrier under play areas.
- The PADOH will continue to work with EPA on public health evaluation of any environmental samples as necessary.
- The PADOH will continue to work with community needs and provide health education materials within the community surrounding the Pollock Park site, as needed and requested.

Sincerely,

Sasidevi Arunachalam, MS PHS | Health Assessor, Pennsylvania Department of Health  
Bureau of Epidemiology | Division of Environmental Health Epidemiology

cc: Anil Nair, Ph.D., MPH, Director, Division of Environmental Health Epidemiology

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Lenhart AM. Email communication from Michael A Lenhart, Borough of Pottstown, Parks and Recreation Director to Sasidevi Arunachalam/PADOH, on July 21, 2020.

## Appendix A

### Exposure Dose Equation for incidental ingestion and dermal contact

#### *Exposure Dose Equation for Ingestion*

$$ED = (C \times IR \times EF \times CF \times AF)/BW$$

ED = Exposure dose (mg/kg/day)

C = Contaminant Concentration (mg/kg)

IR = Ingestion Rate of contaminated soil (100 mg/day was used)

EF = Exposure Factor (1)

CF = Conversion Factor ( $10^{-6}$  kg/mg)

AF = Bioavailability Factor 0.6 for arsenic and 1.0 for Benzo(a)pyrene equivalent

BW = Body Weight (80 kg)

#### *Exposure Dose Equation for Dermal Absorption*

$$ED = (C \times A \times AF \times EF \times CF)/BW$$

ED = Exposure Dose (mg/kg/day)

C = Contaminant Concentration (mg/kg)

A = Total soil adhered (mg) = 326 mg

AF = Absorption Factor of 0.03 for arsenic and 0.13 for Benzo(a)pyrene equivalent

EF = Exposure Factor (1)

CF = Conversion Factor ( $10^{-6}$  kg/mg)

BW = Body Weight (80 kg)

#### *Calculation for the evaluation of excess cancer risk*

$$CR = ED \times CSF \times \text{Estimated exposure years}/78 \text{ years}$$

CR = Cancer Risk; ED = Exposure Dose; CSF = Cancer Slope Factor:  $1.5 \text{ (mg/kg/day)}^{-1}$ ;  $1.0 \text{ (mg/kg/day)}^{-1}$  for arsenic and benzo(a)pyrene equivalent respectively. Estimated exposure years for adults 33 years and for children 21 years.

## Appendix A

**Table A1: Summary of the residential surface soil contaminants and health comparison values (CVs)**

Contaminants Detected	Concentrations at each Residential Locations (mg/kg)				CV (mg/kg)	Source
	SB18	SB19	SB20	SB21		
<b>Polycyclic AromaticHydrocarbons (PAHs)</b>						
Acenaphthene	0.012	0.014	0.013	0.003J	3,100	ATSDR Child RMEG
Acenaphthylene	0.021	0.015	0.019	0.005	NA	NA
Anthracene	0.046	0.059	0.061	0.012	16,000	ATSDR Child RMEG
<i>Benzo(a)anthracene</i>	0.400	0.450	0.470	0.120	NA	NA
<i>Benzo(b)fluoranthene</i>	0.550	0.600	0.610	0.190	NA	NA
<i>Benzo(g,h,i)perylene</i>	0.240	0.270	0.270	0.076	NA	NA
<i>Benzo(k)fluoranthene</i>	0.160	0.140	0.190	0.058	NA	NA
<i>Benzo(o)pyrene</i>	0.400	0.430	0.420	0.130	16 240 0.11	ATSDR Child RMEG ATSDR Adult RMEG ATSDR CREG
<i>Chrysene</i>	0.460	0.520	0.500	0.130	NA	NA
<i>Dibenzo(a,h)anthracene</i>	0.063	0.053	0.078	0.025	NA	NA
<i>Fluoranthene</i>	0.760	0.930	0.900	0.220	2,100 32,000	ATSDR Child RMEG ATSDR Adult RMEG
Fluorene	0.002	0.002	0.002	0.004J	2,100	ATSDR Child RMEG
<i>Indeno(1,2,3-cd)pyrene</i>	0.240	0.290	0.280	0.008	NA	NA
<i>2-Methylnaphthalene</i>	0.008	0.006	0.005	ND	2,100	ATSDR Child c.EMEG
Naphthalene	0.009	0.008	0.007	ND	1,000	ATSDR Child RMEG
Phenanthrene	0.370	0.500	0.440	0.009	NA	NA
<i>Pyrene</i>	0.810	1.0	0.950	0.220	1,600 24,000	ATSDR Child RMEG ATSDR Adult RMEG
<b>Polychlorinated Biphenyls (PCBs)</b>						
Aroclar-1254	ND	ND	ND	0.061J	1.0	ATSDR Child c.EMEG
Aroclar-1260	ND	0.015J	ND	ND	NA	NA
<b>Volatile Organic Compounds (VOCs)</b>						
unknown-01	0.940J	1.110J	0.800J	1.00J	NA	NA
unknown-02	0.840J	1.30J	0.750J	0.550J	NA	NA
unknown-03	0.430J	1.60J	0.45J	0.72J	NA	NA
unknown-04	0.24J	0.64J	0.50J	0.25J	NA	NA
unknown-05	0.22J	0.90J	0.21J	0.20J	NA	NA
unknown-06	0.24J	0.23J	0.16J	NA	NA	NA
unknown-07	NA	0.16J	0.13J	NA	NA	NA
unknown-08	NA	0.39J	NA	NA	NA	NA
9,10-Anthracenedione	NA	0.11J	NA	NA	NA	NA
7H-Benzo[c]fluorene	NA	0.12J	NA	NA	NA	NA
.beta.-Myrcene	NA	0.37J	NA	NA	NA	NA
Bicyclo[4.2.0]octa-1,3,5-triene	NA	0.85J	NA	NA	NA	NA
Bicyclo[3.1.1]heptane, 6,6-Dimethyl	NA	0.36J	NA	NA	NA	NA
Cyclohexane, 1,5-diethenyl-3-methy	NA	0.15J	NA	NA	NA	NA
Cyclohexene, 4-methylene-1-(1-Meth	NA	0.19J	0.25J	NA	NA	NA
3,5-Diamino-2-methylbenzoic Acid	NA	0.95J	NA	NA	NA	NA



Diisooctyl phthalate	0.10J	0.12J	NA	NA	NA	NA
(2,3-Diphenylcyclopropyl) methyl Ph	NA	0.34J	NA	NA	NA	NA
Eucalyptol	NA	0.51J	NA	NA	NA	NA
Fluoranthene, 2-methyl-	0.13J	NA	NA	NA	NA	NA
n-Hexadecanoic acid	0.18J	0.19J	NA	0.21J	NA	NA
D-Limonene	NA	0.13J	NA	NA	NA	NA
2-Naphthalenemethanol, decahydro-	NA	0.20J	NA	NA	NA	NA
Phenanthrene, 1-methyl-	NA	NA	89J	NA	NA	NA
Phenanthrene, 2-methyl-	NA	0.10J	NA	NA	NA	NA
Silane, dichloromethyl-	NA	NA	NA	1.10J	NA	NA
Supraene	0.93J	NA	NA	NA	NA	NA
2-Thioxo-dihydropyrimidine-4,6-dio	0.97J	NA	NA	NA	NA	NA
.beta.-Pinene	NA	0.012J	0.030J	NA	NA	NA
Bicyclo[3.1.1]hept-2-ene, 3,6,6-tr	NA	NA	0.004J	NA	NA	NA
Dimethylsulfide	NA	NA	NA	0.009J	NA	NA
(1R)-2,6,6-Trimethylbicyclo[3.1.1]	NA	0.006J	0.02J	NA	NA	NA
<b>Metals</b>						
Aluminum	8,690J	7,970J	7,700	6,760J	52,000	ATSDR Child c.EMEG
Antimony	0.92J	0.71J	0.58	0.5J	21	ATSDR Child RMEG
<b>Arsenic</b>	19.2	6.6	4.5	4.7	16 240 0.26	ATSDR Child c.EMEG ATSDR Adult c.EMEG ATSDR CREG
Barium	0.92J	181	111J	139J	10,000 160,000	ATSDR Child c.EMEG ATSDR Adult c.EMEG
Beryllium	1.0	1.1	1.1	0.68	100 1,600	ATSDR Child c.EMEG ATSDR Adult c.EMEG
Cadmium	1.1	1.5	0.75	0.84	5.26 80	ATSDR Child c.EMEG ATSDR Adult c.EMEG
Calcium	3,530J	2,710J	4,640J	6,870J	NA	NA
Chromium	21.9J	21.6J	16J	25.2J	78,000	ATSDR Child RMEG
Cobalt	11.8	13.3	12.3	9.2	520	ATSDR Child i.EMEG
Copper	56.6J	44.1	37J	34.6J	520	ATSDR Child i.EMEG
Iron	20,400J	20,500J	21,900J	14,800J	NA	NA
<b>Lead</b>	395	356	160	205	400 330	EPA's screening guidance level in bare soil play areas IEUBK Screen
Magnesium	1,850J	1,560J	2,500J	1,800J	NA	NA
Manganese	628J	731J	826J	391J	NA	NA
Mercury	0.55	0.91	0.28	0.18	35	PADEP MSC
Nickel	19.8	19.8	19.7	15.7	1,000 16,000	ATSDR Child c.RMEG ATSDR Adult c.RMEG
Potassium	653	811	300J	791	NA	NA
Sodium	164J	144J	153J	126J	NA	NA
Vanadium	33.2J	29.1J	40.7J	22.9J	520	ATSDR Child i.EMEG
Zinc	373	333J	195	157	16,000 240,000	ATSDR Child c.EMEG ATSDR Adult c.EMEG

mg/kg=milligram per kilogram; c.EMEG=chronic Environmental Media Evaluation Guide; i.EMEG=intermediate Environmental Media Evaluation Guide; RMEG=chronic Reference Dose Media Evaluation Guide; CREG=Cancer Risk Evaluation Guide; PADEP=Pennsylvania Department of Environmental Protection; MSC=Medium Specific Concentration; NA=not available; J=estimated value; Potential contaminants of concern are in **bold Italics**.

**Table A2: Benzo(a)pyrene Toxicity/Potency Equivalency Factors**

Polycyclic Aromatic Hydrocarbons (PAHs)	Toxicity Equivalency Factor (TEF)*	Locations and Calculated Toxicity Equivalents			
		SB18	SB19	SB20	SB21
Benzo(a)pyrene	1.0	0.40	0.43	0.42	0.13
Benzo(a)anthracene	0.1	0.04	0.045	0.047	0.012
Benzo(b)fluoranthene	0.1	0.055	0.060	0.061	0.019
Benzo(k)fluoranthene	0.1	0.016	0.014	0.019	0.0058
Indeno(1,2,3-cd)pyrene	0.1	0.023	0.029	0.028	0.008
Benzo(g,h,i)perylene	0.01	0.0024	0.0027	0.0027	0.001
Chrysene	0.01	0.0046	0.0052	0.0050	0.0013
Dibenzo(a,h)anthracene	10#	0.630	0.530	0.780	0.250
Phenanthrene	0.001	0.00037	0.0005	0.00044	0.00009
Fluoranthene	0.001	0.00076	0.00093	0.00090	0.00022
Pyrene	0.001	0.000810	0.001	0.00095	0.00028
Benzo(a)pyrene equivalents [B(a)P]		1.17294	1.11833	1.36499	0.42769


\*Source: TEF from ATSDR 1995; # This value is potency equivalent factor based on the ratio of Office of Environmental Health Hazard Assessment's (OEHHA's) oral Cancer Slope Factors for dibenzo(a,h)anthracene (i.e., 4.1 [mg/kg/day]) and Benzo(a)Pyrene (i.e., 1.77 [mg/kg/day]). Each PAH concentration is multiplied by its TEF to get the calculated toxic equivalents. These toxic/potency equivalent factors are then summed to get benzo(a)pyrene equivalent at each location.

## Appendix B


### Site-specific Soil Residential Results for Chronic Exposure

**Table B1: Arsenic Combined (ingestion and dermal) Soil Exposure**

**Location SB18: Site-specific combined ingestion and dermal exposure doses for chronic exposure to arsenic in soil at 19.2 mg/kg along with noncancer hazard quotients and cancer risk estimates\***


 Exposure Group	RME Dose (mg/kg/day)	RME Non-cancer Hazard Quotient	RME Cancer Risk	Exposure Duration for Cancer (yrs)
Birth to < 1 year	0.00025	0.83	-	1
1 to < 2 years	0.00023	0.75	-	1
2 to < 6 years	0.00015	0.50	-	4
6 to < 11 years	8.6E-05	0.29	-	5
11 to < 16 years	3.1E-05	0.10	-	5
16 to < 21 years	2.6E-05	0.086	-	5
Total Child	-	-	3.4E-5 †	21
Adult	1.7E-05	0.058	1.1E-5 †	33

**Location SB19: Site-specific combined ingestion and dermal exposure doses for chronic exposure to arsenic in soil at 6.6 mg/kg along with noncancer hazard quotients and cancer risk estimates\***


 Exposure Group	RME Dose (mg/kg/day)	RME Non-cancer Hazard Quotient	RME Cancer Risk	Exposure Duration for Cancer (yrs)
Birth to < 1 year	8.5E-05	0.28	-	1
1 to < 2 years	7.7E-05	0.26	-	1
2 to < 6 years	5.1E-05	0.17	-	4
6 to < 11 years	3.0E-05	0.099	-	5
11 to < 16 years	1.1E-05	0.036	-	5
16 to < 21 years	8.9E-06	0.030	-	5
Total Child	-	-	1.2E-5 †	21
Adult	6.0E-06	0.020	3.8E-6 †	33

## Appendix B

**Location SB20: Site-specific combined ingestion and dermal exposure doses for chronic exposure to arsenic in soil at 4.5 mg/kg along with noncancer hazard quotients and cancer risk estimates\***

 Exposure Group	RME Dose (mg/kg/day)	RME Non-cancer Hazard Quotient	RME Cancer Risk	Exposure Duration for Cancer (yrs)
Birth to < 1 year	5.8E-05	0.19	-	1
1 to < 2 years	5.3E-05	0.18	-	1
2 to < 6 years	3.5E-05	0.12	-	4
6 to < 11 years	2.0E-05	0.067	-	5
11 to < 16 years	7.3E-06	0.024	-	5
16 to < 21 years	6.1E-06	0.020	-	5
Total Child	-	-	8.1E-6 †	21
Adult	4.1E-06	0.014	2.6E-6 †	33

**Location SB21: Site-specific combined ingestion and dermal exposure doses for chronic exposure to arsenic in soil at 4.7 mg/kg along with noncancer hazard quotients and cancer risk estimates\***


 Exposure Group	RME Dose (mg/kg/day)	RME Non-cancer Hazard Quotient	RME Cancer Risk	Exposure Duration for Cancer (yrs)
Birth to < 1 year	6.1E-05	0.20	-	1
1 to < 2 years	5.5E-05	0.18	-	1
2 to < 6 years	3.7E-05	0.12	-	4
6 to < 11 years	2.1E-05	0.070	-	5
11 to < 16 years	7.7E-06	0.026	-	5
16 to < 21 years	6.3E-06	0.021	-	5
Total Child	-	-	8.4E-6 †	21
Adult	4.3E-06	0.014	2.7E-6 †	33

RME = reasonable maximum exposure (higher); mg/kg/day = milligram chemical per kilogram body weight per day; mg/kg = milligram chemical per kilogram soil.\* The calculations in this table were generated using ATSDR's PHAST v1.5.1.0. The noncancer hazard quotients were calculated using the chronic (greater than 1 year) minimal risk level of 0.0003 mg/kg/day and the cancer risks were calculated using the cancer slope factor of 1.5 (mg/kg/day)<sup>-1</sup>. The cancer risk for child is derived by summing all the cancer risks for each age group from birth to < 21 years. A shaded cell indicates that the cancer risk exceeds one extra case in a million people similarly exposed, which ATSDR evaluated further.


## Appendix B

**Table B2: Benzo(a)p Combined (ingestion and dermal) Soil Exposure**

**Location SB18: Site-specific combined ingestion and dermal exposure doses for chronic exposure to benzo(a)pyrene in soil at 1.17 mg/kg along with noncancer hazard quotients and cancer risk estimates\***


 Exposure Group	RME Dose (mg/kg/day)	RME Noncancer Hazard Quotient	RME Cancer Risk	Exposure Duration for Cancer (yrs)
Birth to < 1 year	2.9E-05	0.098	-	1
1 to < 2 years	2.7E-05	0.089	-	1
2 to < 6 years	1.8E-05	0.060	-	4
6 to < 11 years	1.1E-05	0.037	-	5
11 to < 16 years	5.0E-06	0.017	-	5
16 to < 21 years	4.2E-06	0.014	-	5
Total Child	-	-	1.3E-5 ‡	21
Adult	2.3E-06	0.0076	9.6E-7	33

**Location SB19: Site-specific combined ingestion and dermal exposure doses for chronic exposure to benzo(a)pyrene in soil at 1.12 mg/kg along with noncancer hazard quotients and cancer risk estimates\***


 Exposure Group	RME Dose (mg/kg/day)	RME Noncancer Hazard Quotient	RME Cancer Risk	Exposure Duration for Cancer (yrs)
Birth to < 1 year	2.8E-05	0.094	-	1
1 to < 2 years	2.6E-05	0.085	-	1
2 to < 6 years	1.7E-05	0.057	-	4
6 to < 11 years	1.1E-05	0.035	-	5
11 to < 16 years	4.8E-06	0.016	-	5
16 to < 21 years	4.0E-06	0.013	-	5
Total Child	-	-	1.3E-5 ‡	21
Adult	2.2E-06	0.0072	9.2E-7	33

## Appendix B

**Location SB20: Site-specific combined ingestion and dermal exposure doses for chronic exposure to benzo(a)pyrene in soil at 1.36 mg/kg along with noncancer hazard quotients and cancer risk estimates\***

 PHAST PUBLIC HEALTH ASSESSMENT SITE TOOL	RME Dose (mg/kg/day)	RME Noncancer Hazard Quotient	RME Cancer Risk	Exposure Duration for Cancer (yrs)
<b>Exposure Group</b>				
Birth to < 1 year	3.4E-05	0.11	-	1
1 to < 2 years	3.1E-05	0.10	-	1
2 to < 6 years	2.1E-05	0.069	-	4
6 to < 11 years	1.3E-05	0.042	-	5
11 to < 16 years	5.7E-06	0.019	-	5
16 to < 21 years	4.9E-06	0.016	-	5
Total Child	-	-	1.5E-5 ‡	21
Adult	2.6E-06	0.0088	1.1E-6 ‡	33

**Location SB21: Site-specific combined ingestion and dermal exposure doses for chronic exposure to benzo(a)pyrene in soil at 0.43 mg/kg along with noncancer hazard quotients and cancer risk estimates\***

 PHAST PUBLIC HEALTH ASSESSMENT SITE TOOL	RME Dose (mg/kg/day)	RME Noncancer Hazard Quotient	RME Cancer Risk	Exposure Duration for Cancer (yrs)
<b>Exposure Group</b>				
Birth to < 1 year	1.1E-05	0.035	-	1
1 to < 2 years	9.6E-06	0.032	-	1
2 to < 6 years	6.5E-06	0.022	-	4
6 to < 11 years	4.0E-06	0.013	-	5
11 to < 16 years	1.8E-06	0.0060	-	5
16 to < 21 years	1.5E-06	0.0050	-	5
Total Child	-	-	4.E-6 ‡	21
Adult	8.3E-07	0.0028	3.5E-7	33

RME = reasonable maximum exposure (higher); mg/kg/day = milligram chemical per kilogram body weight per day; mg/kg = milligram chemical per kilogram soil; \*The calculations in this table were generated using ATSDR's PHAST v1.5.1.0. The noncancer hazard quotients were calculated using the chronic (lifetime) reference dose of 0.0003 mg/kg/day and the cancer risks were calculated using the cancer slope factor of 1.0 (mg/kg/day)<sup>-1</sup> and age-dependent adjustment factors. A shaded cell indicates that the cancer risk exceeds one extra case in a million people similarly exposed, which ATSDR evaluate d further.

## Appendix B

**Table B3: Calculated Blood Lead and Lead Uptakes Using the IEUBK Model,  
Soil Lead Concentration: 330 mg/kg**

Year	Air (µg/day)	Diet (µg/day)	Alternate (µg/day)	Water (µg/day)	Soil+Dust (µg/day)	Total (µg/day)	Blood (µg/dL)
.5-1	0.021	1.032	0.000	0.365	6.544	7.962	4.3
1-2	0.034	0.881	0.000	0.899	10.235	12.050	5.0
2-3	0.062	0.971	0.000	0.948	10.380	12.361	4.6
3-4	0.067	0.942	0.000	0.979	10.513	12.501	4.4
4-5	0.067	0.923	0.000	1.042	7.983	10.015	3.6
5-6	0.093	0.980	0.000	1.109	7.252	9.433	3.0
6-7	0.093	1.066	0.000	1.133	6.883	9.175	2.7

330 mg/kg resulted in BLL of 5.0 µg/dL in children aged one to two years highlighted in red