# Health Consultation

NORWOOD SITE
NORWOOD BOROUGH, DELAWARE COUNTY, PENNSYLVANIA

Public Health Evaluation of EPA 2017-2018 Environmental Sampling Data at the Norwood Landfill Site and 1985-2019 Cancer Incidence Data Review

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# Health Consultation: A Note of Explanation

A health consultation is a verbal or written response from the Agency for Toxic Substances Disease (ATSDR) or ATSDR's Cooperative Agreement Partners to a specific request for information about health risks related to a specific site, a chemical release, or the presence of hazardous material. In order to prevent or mitigate exposures, a consultation may lead to specific actions, such as restricting the use of or replacing water supplies, intensifying environmental sampling, restricting site access, or removing the contaminated material.

In addition, consultations may recommend additional public health actions, such as conducting health surveillance activities to evaluate exposure or trends in adverse health outcomes, conducting biological indicators of exposure studies to assess exposure, and providing health education for health care providers and community members.

The Pennsylvania Department of Health (PADOH) prepared this health consultation for Norwood residents. This publication was made possible by grant number CDC-RFA-TS17-170103CONT19 from ATSDR. Its contents are solely the responsibility of the authors and do not necessarily represent the official views of the ATSDR, or the U.S. Department of Health and Human Services. The PADOH evaluated data sampled/monitored/estimated using approved methods, policies, and procedures existing at the date of publication.

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## Summary

The Norwood site ("the site") is located in a suburban residential neighborhood in lower Norwood, Delaware County, Pennsylvania. Beginning in 2014 community residents expressed concerns to the US Environmental Protection Agency (EPA), the Agency for Toxic Substances and Disease Registry (ATSDR), and the Pennsylvania Department of Health (PADOH) that historic landfill contamination was the source of cancer and multiple sclerosis cases in their community. Historical imagery and records indicated that what are now wooded areas adjacent to the community were formerly used as a 15-acre landfill ("Norwood landfill"), and 10-acre town dump between 1950 and 1963. In 2017-2018 EPA began a Site Assessment to determine whether the site qualified for the National Priorities List (NPL). In the NPL process, Site Assessments do not typically fully characterize a site area but are designed to determine whether there has been a release of hazardous substances that is threatening human health and the environment. As part of its Site Assessment, EPA sampled residential soil, non-residential soil, and Darby and Muckinipattis Creek sediment and surface water for more than 80 chemicals commonly found at landfills.

PADOH evaluated EPA's 2017-2018 sampling results of residential surface soil (21 samples), non-residential surface soil (17 samples), and creek sediment (8 samples) and surface water (8 samples) to determine whether the chemical concentrations at these locations posed a threat to human health. (Note: of the 8 collated creek and surface water samples, 7 were taken in Darby Creek.)

As a health protective approach and in accordance with ATSDR guidance, we (PADOH) considered each residential soil sample as a separate unit and independent of one another to estimate the potential for health effects from residential-based exposures. Conversely, we designated each non-residential location (non-residential soil; creek sediment and surface water) as a distinct exposure area to estimate health risks from non-residential areas.

For chemicals that exceeded screening levels, we designated them as chemicals of concern (CoCs) warranting further evaluation. For each CoC we calculated combined incidental ingestion and dermal exposure estimates (also known as "exposure doses") to assess the potential for cancer and non-cancer health effects. Using PADOH's cancer registry, we also analyzed age-adjusted standardized incidence ratios (SIRs) by sex for 22 cancer types for four time periods (1985-1994, 1995-2004, 2005-2014, 2015-2019) from 1985-2019. We compared cancer incidence rates at Norwood as well as surrounding boroughs (Folcroft, Prospect Park) to Delaware County and state rates. Based on our analysis of EPA's 2017-2018 environmental sampling data for the site and review of the PADOH cancer registry, we reached the following conclusions:

**Conclusion 1**

Except for lead for which there is no presumed safe level of exposure, adverse non-cancer health effects are unlikely to occur from dermal and incidental ingestion exposures to detected chemical concentrations at sampled site locations.

*Basis for conclusion*
Lead is a naturally occurring element in the earth's crust and can be found throughout our environment in the air, water, and soil from anthropogenic sources such as fossil fuels, including past use of leaded gasoline, some types of industrial facilities and past use of lead-based paint in homes. Lead at Norwood was lower than that typically found in more urban Pennsylvania areas, such as Philadelphia. At Norwood, 3 of 21 residential and 5 of 17 non-residential samples exceeded a soil lead model threshold level of 245 milligram/kilogram (mg/kg) that could result in a child blood lead level (BLL) of 3.5 microgram/deciliter (µg/dL). While there is no safe blood lead level for children, a BLL of 3.5 µg/dL and above is considered "elevated" according to the PADOH, and children could experience adverse health effects, including nervous system effects, from potential lead exposure.

Regarding other CoCs, the highest exposure doses for 3 chemicals – 1) benzo[a]pyrene-equivalent polycyclic aromatic hydrocarbons (PAHs), 2) di (2-ethylhexyl) phthalate (DEHP), and 3) chromium(VI) – exceeded chronic and/or intermediate health guidelines for young children, but not adults:

- The highest exposure doses for children age 1 year and younger exceeded chronic and intermediate-duration health guidelines for PAHs and DEHP, respectively.
- The highest exposure doses for children ages 0-12 months (to residential soil) and 6-10 years (to creek sediment) exceeded chronic health guidelines for chromium(VI).

However, the exposure doses for these chemicals were several orders of magnitude (70-270 times) below reported effect levels from which their chronic and intermediate health guidelines are derived. In addition, our chromium(VI) estimate assumed that detected total chromium at Norwood was 100% chromium(VI), a "worst case" and unlikely scenario. Therefore, it is unlikely that children or adults would experience adverse non-cancer health effects from these chemicals.

**Conclusion 2**
A young-child engaging in soil-pica behavior (which is uncommon and involves eating large amounts of soil) may experience adverse non-cancer health effects such as gastrointestinal or nervous system effects if consuming the highest detected concentrations of copper, iron, or lead at site sampling locations.

*Basis for conclusion*

In toxicological evaluations for the chemicals listed above, soil-pica exposure scenarios approached or exceeded health effect thresholds. For copper, this conclusion is based on intermediate-duration and single occasion pica behavior involving the highest copper residential soil sample; for iron, the conclusion is based on intermediate-duration pica behavior involving the highest iron residential soil sample. As there is no presumed safe exposure to lead, pica behavior could result in adverse health effects from lead exposure regardless of site location.

Soil-pica behavior involves eating soil and can be found in some children 1-5 years old, though this behavior is uncommon. We assessed an intermediate and single (1 time) soil-pica scenario. Our intermediate-duration pica estimates assume a child consuming 5,000 mg soil (equivalent to 5 packets of artificial sweetener used in coffee or tea) for 3 days per week for up to one year. Our single pica estimate assumes consumption of 5,000 mg in soil once. These are health protective assumptions. Please see Appendix E for a more detailed discussion.

**Conclusion 3**

Community members' long-term exposure to several chemicals of concern (CoCs) that are known or probable human carcinogens poses an increased cancer risk in children and adults.

*Basis for conclusion*

We estimated lifetime excess cancer risk for CoCs that are known or probable human carcinogens and have an EPA or ATSDR-recommended cancer slope factor (CSF). Those CoCs were: benzo[a]pyrene-equivalent PAHs, DEHP, polychlorinated biphenyls (PCBs), aldrin and dieldrin, arsenic, and chromium(VI).

PADOH's current approach for assessing lifetime cancer risk assumes that there's some level of risk associated with exposure to each molecule and that a threshold for cancer effects does not exist. While there will always be some risk, risk increases with the amount of exposure, frequency of exposures, and how many years a person is exposed. Cancer risk estimates are expressed as the proportion of a population that ***may*** be affected by a carcinogen during a lifetime of exposure. For example, an estimated cancer risk of 2 in 100,000 represents ***potentially*** 2 additional cancer cases above expected cases in a population of 100,000 over a lifetime of continuous exposure. Lifetime cancer risk is a theoretical estimate and not a prediction of the number of cancers in a community.

As a health protective approach, we assessed residential cancer risk based on each residential unit with the assumption that residential soil samples were independent from one another. Accordingly, for residential soil exposure, the highest excess cancer risk estimates were 3 in 10,000 for children and 2 in 100,000 for adults, based on the maximum benzo[a]pyrene-equivalent PAHs detected. These estimates are based on benzo[a]pyrene-equivalent PAHs at a single (maximum) household that had concentrations 3.5 times above the next highest

household. Lifetime excess cancer risk based on the next highest benzo[a]pyrene-equivalent PAH sample was 7 in 100,000 for children and 5 in a million for adults.

For the remaining CoCs, the highest residential excess cancer risk estimates were 2 in 10,000 for children (and 2 in 100,000 for adults) based on the highest chromium sample, and 5 in 100,000 for children (and 2 in 100,000 for adults) based on the highest dieldrin sample. Our chromium estimates assumed that all detected total chromium concentrations were chromium(VI). In most soils, total chromium is in its much less toxic, chromium(III) form.

For non-residential exposures, the highest excess cancer risk was 2 in 10,000 for children and 6 in 100,000 for adults based on daily, year-round exposures to creek sediment at sampled locations and a 100% chromium(VI) assumption.

If we assume that maximum detected chromium concentrations are 75% chromium(III) and 25% chromium(VI), a more likely chromium speciation scenario, excess cancer risk from chromium exposure is lower for children and adults for residential (6 in 100,000 for children, 6 in a million for adults) and sediment-based exposures (6 in 100,000 for children, 1 in 100,000 for adults).

For adults, all Norwood cancer risk estimates (whether from residential soil, non-residential soil or creek sediment) assume daily, year-round exposures to a CoC at that location and concentration for 33 consecutive years. For children, *residential* soil estimates assume daily exposure from birth through age 20 years, while *non-residential* soil and sediment estimates assume exposures from ages 6 through 20 years. Estimates would be lower for exposures of shorter duration.

**Conclusion 4**

Age-adjusted cancer data analysis for 1985-2019 did not show consistent patterns for the 22 cancer types analyzed, except for lung cancer. Lung cancer incidence rates were mostly higher and statistically significant for all the four time-periods (1985-1994; 1995-2004; 2005-2014; 2015-2019) for both men and women.

*Basis for Conclusion*

Lung cancer incidence for men at Norwood, Folcroft and Prospect Park Boroughs combined for all four time periods (1985-1994; 1995-2004; 2005-2014; 2015-2019) was statistically significantly higher at 30%, 24%, 59%, and 83%, respectively, than expected compared to Delaware County. For women, except for one time period (1985-1994), lung cancer incidence at these 3 boroughs combined was statistically significantly higher (24%, 44%, 39% and 59%, respectively) than expected when compared to Delaware County.

The cancer registry does not account for smoking, the most common risk factor for lung cancer. Environmental risk factors for lung cancer have typically involved inhalation exposures to radon or certain workplace-based chemicals, or ingestion of contaminated drinking water. Exposure to contaminated drinking water is highly unlikely because community residents are and have historically been served by a public water system.

**Conclusion 5**
There is no registry similar to a cancer registry to evaluate Multiple Sclerosis (MS). For identified CoCs, exposure doses were below thresholds that induced neurological or immune system effects in human or laboratory animal studies.

*Basis for conclusion*
Due to community concerns regarding MS, we compared CoC daily exposure estimates to levels in human and or laboratory animal studies that have found immune system and neurological effects. There is no registry to evaluate MS. In addition, while researchers have identified several risk factors for MS, its exact cause remains unknown and there are currently no definitive data showing that MS is caused by environmental contamination. **Our evaluation was <u>not</u> meant to prove or disprove a CoC's association with MS. Rather, it was to provide information on the <u>types and thresholds</u> of immune and neurological health effects that have been found for Norwood CoCs from human and/or laboratory animal studies.** For CoCs for which these data were available, exposure doses were below health effect levels identified in scientific studies.

## Limitations

Our analysis has several assumptions/limitations:

- Our conclusions are based on single soil, sediment and surface water samples taken between 2017-2018. They cannot be extrapolated to past concentrations or account for possible fluctuation or variance in concentrations. Sampling from different parts of a residential yard could have produced a different result. In 2018, EPA sampled residential soil from 0-12.'' ATSDR notes that ideally, surface soil should be sampled at depths of 0-3'' (ATSDR 2005). For surface water, our conclusions are based on the amount of inorganic metals detected, as organic compounds were not sampled.
- Our conclusions for chromium assume that total chromium concentrations at Norwood were 100% chromium(VI), the more toxic form of chromium. Without chromium speciation, the proportion of chromium(VI) or chromium(III) concentrations at Norwood is uncertain, and by extension, refined estimates of cancer and non-cancer risk. In most soils, chromium is in its less toxic, chromium(III) form.
- Although we estimated exposures to residential soil, non-residential soil, and Darby and Muckinipattis Creek sediment and surface water, our estimates don't account for

movement *between* these locations. We assumed maximum exposure at one of these four locations. If someone frequently traversed to and from one location to another (e.g., residential soil to creek sediment), daily exposures for each distinct location would presumably be lower. As noted, a majority (7) of the 8 creek samples were taken in Darby Creek.

- We evaluated a daily, 12 week creek swimming scenario and year-round, 10-year wading scenario. Exposures would be higher for someone engaging in these activities for a longer duration.
- We included "J" data values in our screening and analysis. "J" values were considered detected (as opposed to non-detected), and these values were considered part of exposure estimates and concentration ranges (e.g. minimums and maximums). J values indicate that the chemical was present in the field sample but its concentration is an estimate; the true concentration may be higher or lower. Only two *maximum* CoC J values – one for DEHP in residential soil and one for manganese in Darby Creek sediment – were featured in our health effects evaluations.
- We were not able to estimate quantitative cancer risk from mercury or lead exposures, which are considered possible and probable human carcinogens but lack a cancer slope factor.
- Our cancer incidence analysis does not account for other contributors to cancer such as genetic pre-disposition, occupational exposures or other environmental exposures such as to radon, residential history, behaviors, and diet; and whether incidence rates are related to the former Norwood landfill.
- Currently, there is no registry available to assess Norwood MS rates, and the causes and risk factors for MS are not well understood.
- Our exposure estimates assume combined incidental ingestion and dermal exposures only (e.g., not exposures by inhalation, ingestion from fish or crops, or soil vapor intrusion).
- This analysis is based on 2017-2018 results, which assessed soil at 21 homes and did not assess soil or sediment at the former Old Norwood Dump, along Muckinipattis Creek nearer to the former Muckinipattis Wastewater Treatment Plant, or at Norwood Borough Park. We agree with EPA's decision to expand sampling to these and other site locations, and to sample more media (e.g., groundwater, deep soil) and residential locations (70 total), which they completed in 2020. We will assess EPA's 2020 expanded sampling results, released in December 2021, as an addendum to this report.

## Recommendations

Based on this Health Consultation, we recommend that:

1. Parents monitor the outdoor behavior of their children (ages 1 to 5 years old) if the child is suspected of engaging in soil-pica activity.

2. Crop uptake from chemicals in soil is likely to be minimal; however, to reduce potential exposure to soil chemicals when gardening, PADOH suggests adhering to EPA's suggested best practices such as using raised garden beds and pots filled with clean soil, mixing additional compost into in-ground gardens, and washing produce, peeling root crops and removing outer leaves of leafy vegetables before eating.
3. To reduce or eliminate potential exposures to lead from soil, residents should:
    a. Remove shoes before entering the house to prevent bringing lead-contaminated soil from outside.
    b. Avoid allowing their children to play in bare soil (e.g., if possible, use sandboxes).
    c. Plant grass (if possible) on any bare soil, or cover the soil with seed, mulch, or wood (CDC 2022a).
4. Parents have children under 6 tested for lead poisoning via a simple blood test. PADOH has a lead information line (1-800-440-LEAD) to respond to questions about lead poisoning and other environmental hazards.
5. Whenever possible, residents avoid or limit other potential lead exposure sources, such as old or imported toys that may still contain lead-based paint, certain imported consumer products (e.g., some jewelry, cosmetics, candies, or spices), or certain hobbies in which lead exposure can occur. If engaging in hobbies or certain occupations in which lead exposure is common, efforts should be made to avoid tracking it into the home from clothing or equipment. Any renovation of homes containing lead-based paint should be done by a qualified lead abatement professional. Additional information on lead exposure sources can be found in this report.
6. EPA consider additional sampling of the site that speciates chromium valence form, and sample in residential areas closer to the surface (e.g., 0-3'') than it did in its 2017-2018 sampling (0-12'').

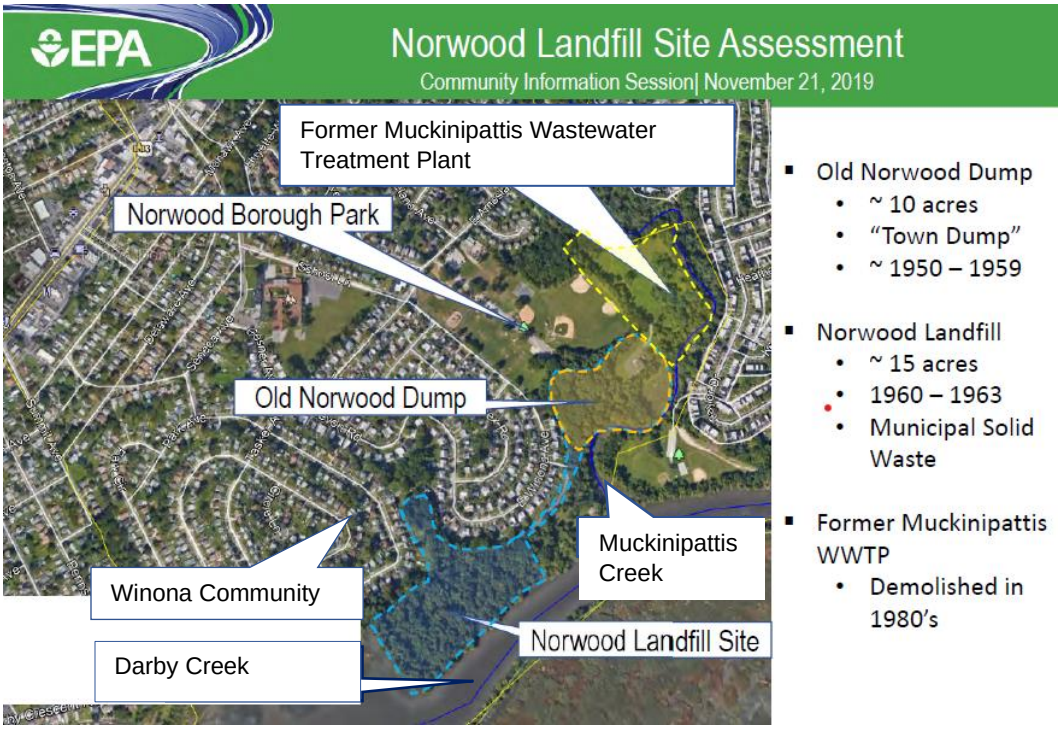
## Next Steps

- PADOH will present the findings of this HC and provide health education outreach to the lower Norwood community.
- PADOH will assess EPA's expanded 2020 Norwood sampling results as an addendum.
- PADOH will continue to assist site stakeholders when requested to evaluate additional environmental or health data from the site.

## 1. Background and Site Overview

The Norwood site ("the site") encompasses the Winona residential community and surrounding areas in lower Norwood, Delaware County, Pennsylvania. The community was constructed in the 1950s and is surrounded by undeveloped wooded areas to the south and east. Bordering these wooded areas are Muckinipattis Creek to the east and Darby Creek to the south (EPA 2019, Figure 1). Muckinipattis Creek flows into Darby Creek.

**Figure 1. EPA map of the site, including the former Norwood landfill area and Darby and Muckinipattis Creeks to the South and East (Source: EPA, Community Information Session, 2019).**




The site is primarily residential, though visitor gatherings and recreation activity occur at several adjacent areas including Norwood Borough Park to the northeast, a public fishing dock along Muckinipattis Creek, and the Historic Morton House to the southeast (Figure 2).

**Figure 2. EPA 2017-2018 Sampling Locations for the Site (Residential locations redacted; Source: EPA, 2018)**


From approximately 1950-1963 the wooded areas surrounding Winona were used as a 15-acre landfill ("Norwood landfill") to the south (from est. 1960-1963) and 10-acre town dump to the east (from est. 1950-1959; Figure 1).

Beginning in 2014, several Norwood citizens contacted the US Environmental Protection Agency (EPA), the Agency for Toxic Substances and Disease Registry (ATSDR), and the Pennsylvania Department of Health (PADOH) to express concerns that historic landfill contamination was the source of reported cancer and multiple sclerosis (MS) cases in their community. Residents were concerned that the landfill was contaminated from historical unregulated sources, including from nearby Glenolden Laboratories (and subsequently Merck, Sharp, and Dohme Pharmaceutical Laboratories) and the former Muckinipattis Wastewater Treatment Plant (WWTP). Residents also alleged that their homes were built on soil material used for the construction of Walt Whitman Bridge in the 1950s and potentially contained harmful substances such as polychlorinated biphenyls (PCBs) and heavy metals (Weston 2018).

### 1.1. Previous investigations

In 1993 and 1999 the US Fish and Wildlife Service conducted a Level I and Level II contamination survey, respectively, of two tracts of land (tract 24 and 35) located between the residential neighborhoods of lower Norwood and Darby Creek (Figure 2; Tetra Tech 2020). The 1993 Level I survey noted debris including glass jars and bottles, automobile frames and parts, aluminum siding, asphalt, concrete, and tires (Tetra Tech 2020). The 1999 Level II contamination survey sampled tracts 24 and 35 soil, sediment, groundwater, and surface water for chemicals such as semi volatile organic compounds (SVOCs), pesticides, PCBs and metals. Although several of these chemicals exceeded EPA Region III Risk-Based Concentration (RBC)

screening levels, their concentrations were attributed to natural conditions of the area, surface water runoff from adjacent properties and streets, and non-hazardous materials previously disposed on the property (Tetra Tech 2020).

### 1.2. Norwood Borough Demographics

According to American Community Survey estimates, Norwood borough, which encompasses lower Norwood, had a population of approximately 6,000 people as of July 2019. The population changed little (0.1% increase) from 2010-2019. Residents are by majority Non-Hispanic White (89.6%), followed by Asian (5.2%), two or more races (3.9%), Hispanic or Latino (3.0%) and Black or African American (1.2%). An estimated 5% of the population are children 5 years old and younger, while 19.8% are under 18 and 12.5% are over 65 years of age (American Community Survey 2019).

### 1.3 Environment and Environmental Justice Indicators

We assessed environmental justice (EJ) indicators within a mile radius of the site. Certain at-risk communities are more vulnerable to environmental pollution due to their proximity to environmental hazards as well as other factors (e.g., restricted access to health care and healthy foods, etc.) that impact community health outcomes. EPA's EJ screen tool (EPA 2020) combines publicly available environmental and demographic indicators to identify communities that might be disproportionately burdened by environmental hazards. Eleven EJ indices allow for percentile-based comparisons of a community to the rest of Pennsylvania, the EPA's region 3 (Pennsylvania, Virginia, West Virginia, District of Columbia, Maryland and Delaware), and the USA. The environmental factors included in the EJ indices are: particulate matter 2.5 (PM2.5), ozone, National Air Toxic Assessment (NATA) diesel PM, NATA air toxics cancer risk, NATA respiratory Hazard Index (HI), traffic proximity, lead paint indicator (percentage of pre-1960s housing), Superfund proximity, risk management plan (RMP) proximity, hazardous waste proximity, and wastewater discharge proximity.

Based on EPA's EJ screen report, within a one-mile radius of the approximate center of the site, EJ indices for a) particulate matter (PM) 2.5, b) ozone air pollution, and c) NATA Air Toxics Cancer Risk and d) NATA Respiratory Hazard Index exceeded the 50$^{th}$ percentile for Pennsylvania (Appendix Table A1). The remaining 7 EJ indices did not exceed the 50$^{th}$ percentile for Pennsylvania, EPA region 3 or the USA. By contrast, when considering the 11 environmental indicators solely (e.g., without the demographic indicators), all indicators except for particulate matter pollution exceeded the 50$^{th}$ percentile for Pennsylvania, EPA Region 3, and the rest of the USA (Appendix Table A2). Several indicators exceeded the 70$^{th}$ percentile of all comparison groups, including NATA diesel pollution, traffic proximity and volume, lead paint indicator, RMP proximity, Superfund site proximity, hazardous waste proximity, and wastewater discharge indicators. Appendix A, Tables A1-A2 display the results of EPA's EJ screen report.

## 2. EPA's 2017-2018 Norwood Sampling and Comparison of EPA Risk Assessments to PADOH Health Assessments

In February 2017 EPA began screening the site as part of its Site Assessment process to determine whether it qualified for the National Priorities List (NPL). On September 26-27, 2017, and May 23-24, 2018, EPA collected the following samples:

- 17 non-residential surface (0-6'' below ground) soil samples, in addition to 2 background and 1 duplicate sample
- 8 co-located subsurface, non-residential soil samples (24-48''), in addition to 1 duplicate sample
- 21 residential surface soil samples (0-12'')
- 8 co-located sediment and surface water samples along Darby and Muckinipattis Creeks, in addition to 3 background samples and 1 duplicate sample. Note: of the 8 co-located samples, 7 were taken in Darby Creek and 1 was taken in Muckinipattis Creek, near the convergence of both creeks (Figure 2).

The sampling locations encompassed 21 Winona residences, mostly along E. Winona Ave and W. Martin lanes adjacent to the former Norwood landfill, as well as locations at the landfill area and Darby Creek to its immediate south (Figure 2). For its 2018 residential sampling, EPA contacted 37 residences and 21 residences gave permission to access their properties. A few residential samples were taken at Love Lane, Essex Road, and Mohawk Avenue, which were further away from the former landfill but consistent with homeowners interested in having their property sampled (Weston 2018).

EPA sampled for chemicals (including isomer variations) commonly found at landfills, which included 13 Volatile Organic Compounds (VOCs), 29 Semi Volatile Organic Compounds (SVOCs, including 17 PAHs), 2 PCBs (Aroclor 1254 and Aroclor 1260), 20 pesticides and 23 inorganic metals, for a total of more than 80 chemicals. Although more than 80 chemicals were sampled overall, volatile organic compounds (VOCs) and PCBs were not sampled in sediment, and only inorganic metals were sampled in surface water. EPA released its sampling results in September 2018 (Weston 2018).

EPA sampled non-residential surface soil at 0-6'' below ground surface (bgs) and residential surface soil at 0-12'' bgs. ATSDR recommends that surface soil depths be sampled at 0-3'' for human exposure health effects evaluation (ATSDR 2005).

The PADOH evaluated EPA's 2017-2018 sampling results and considered site- and age-specific exposure conditions to determine whether chemical levels at these locations could pose a threat to public health (ATSDR 2005). **We considered results from EPA's surface soil (residential and non-residential), sediment, and surface water assessments for our public health evaluation**. Although EPA also took 8 subsurface soil samples, we did not consider them

in our evaluation due to the low likelihood that the public would be exposed to soil at the sub-surface sample depths (24-48'').

*Evaluation of Cancer Incidence*

Using Pennsylvania's cancer registry, we also evaluated 1985-2019 cancer incidence rates for Norwood, Prospect Park and Folcroft Boroughs combined compared to Delaware County and Pennsylvania, and for Norwood compared to Pennsylvania. A similar surveillance system does not exist for Multiple Sclerosis (MS); however, in 2019 the CDC received Congressionally-appropriated funds to initiate development of a National Neurological Conditions Surveillance System (NNCSS). Pending continued funding, the full NNCSS would be implemented and include MS and other neurological conditions in fiscal year 2022 and beyond (CDC 2020a).

In November 2020 EPA completed additional sampling that included additional site locations, including the Old Norwood Dump (to the east of the Winona community, Figure 1), Norwood Borough Park, additional Muckinipattis Creek (and former WWTP area) locations, and a larger residential area of the community. This HC encompasses EPA's 2017 and 2018 results. We will assess EPA's 2020 results, released in December 2021, as an addendum to this report.

*Comparison of EPA Risk Assessment and PADOH Health Assessments*

EPA collected screening data at the Norwood site to determine whether it qualified for the National Priorities List (NPL). As part of this process EPA conducts a Risk Assessment, which provides a numerical (quantitative) estimate of theoretical risk or hazard for a community.

PADOH's Public Health Assessments, while using the same environmental data as EPA, are more focused on a community's specific health concerns. For example, for this Health Consultation, PADOH assessed the community concern of MS and cancer; for cancer, PADOH assessed lifetime cancer risk and data from the cancer registry. PADOH assessments evaluate these concerns both quantitatively and qualitatively, and determine whether there is an exposure of concern and health impact from that exposure. For this Health Consultation, PADOH considered exposures to soil, sediment and surface water. If an exposure of concern is identified, PADOH's assessments provide recommendations on methods to reduce that exposure. Unlike EPA, PADOH does not have regulatory authority.

Both EPA Risk Assessments and PADOH Public Health Assessments evaluate exposure scenarios that are meant to be public health protective, though PADOH assessments can at times include scenarios that are more health protective and/or represent "worst-case scenario" exposure conditions (including those that could affect sensitive subpopulations) to determine if such conditions could harm human health. For example, for this Heath Consultation PADOH considered exposures from both pica and non-pica behavior in young children, and scenarios that children as young as 6 years could regularly (e.g., daily) interact with soil and sediment at non-residential locations. In addition, for certain chemicals, PADOH utilizes ATSDR guidance in which stricter (lower) and more health protective values than EPA's are used to screen

chemicals and/or estimate their lifetime cancer risk; an example is benzo[a]pyrene, for which a stricter value is used than EPA's to estimate lifetime cancer risk. For all exposure scenarios, PADOH assessments perform in-depth analyses to determine whether harmful health effects are possible.

## 3. Methods

### 3.1 Exposure Pathway Analysis

An exposure pathway describes how people are or may be exposed to a hazardous substance from an environmental release. We conducted an exposure pathway analysis to evaluate conditions in which Winona residents and visitors could have been exposed to chemicals from the former Norwood landfill. An exposure pathway analysis contains the following components (ATSDR 2005):

1. A chemical source (e.g., industrial waste sites emitting hazardous materials, landfills)
2. An environmental medium (e.g., air, water, soil)
3. An exposure point (e.g., use of a water supply source, outdoor or indoor air)
4. An exposure route (e.g., dermal, ingestion, inhalation)
5. A receptor population (e.g., residents, children, visitors)

Exposure pathways are categorized as completed, potential, or eliminated. A completed exposure pathway has all five of the above components. In a potential exposure pathway, one or more of the pathway components are uncertain but could have occurred in the past, at present, or may occur in the future. In an eliminated exposure pathway, one or more of the five components are missing or absent. A "completed" or "potential" exposure pathway does not indicate that the exposure will negatively affect health; rather, chemicals identified in these pathways are further evaluated. For the Norwood landfill area, we identified soil, sediment and surface water exposure as potential exposure pathways for the Winona community (Table 1). We deemed the above exposure pathways as "potential" instead of "completed" due to limited historical information on the specific chemicals present in the Norwood landfill during its operation.

**Table 1. Exposure Pathway Analysis for Norwood Landfill Site**

| **Exposure Pathway Components** | | | | | **Exposure Pathway Status** | | |
|---|---|---|---|---|---|---|---|
| **Source** | **Medium** | **Point of Exposure** | **Route of Exposure** | **Receptor Population** | **Past** | **Present** | **Future** |
| Norwood Landfill (1960-1963) historic contamination | Soil and Sediment | Residential Yards, Non-Residential Wooded Areas, Sediment along Darby or | Dermal contact, Ingestion | Residents and visitors | Potential | Potential | Potential |

| | | | | | | | |
|---|---|---|---|---|---|---|---|
| | | Muckinipattis Creek | | | | | |
| | Drinking Water | Drinking and showering | None (ingestion, dermal) | None | Eliminated | Eliminated | Eliminated |
| | Surface Water and sediment | Swimming or Wading in Darby or Muckinipattis Creeks | Ingestion, dermal | Residents and visitors | Potential | Potential | Potential |
| | Air | Air emissions | Inhalation | Residents and visitors | Potential | Potential | Potential |
| | Food | Fruits or vegetables grown in contaminated soil | Ingestion | Residents | Potential | Potential | Potential |

Our exposure pathway analysis concluded that **dermal or incidental ingestion** to **residential soil, non-residential soil, creek surface water or creek sediment** could have occurred, may be occurring presently, or could occur in the future from:

- Community member exposures to chemicals in the **residential soil of their yards**, or nearby **non-residential soil** if venturing into the wooded brush areas or nearby land surrounding their homes (Figure 2).
- Resident and visitor exposures to chemicals from wading or swimming **in creek surface water** or interacting with **creek sediment.**

We assessed combined **dermal and incidental ingestion** exposures to Norwood chemicals from the activities listed above. Dermal exposure occurs by making skin contact a chemical. Incidental ingestion occurs by inadvertently eating, drinking and/or swallowing small amounts of a chemical. Dermal and ingestion exposures can occur from contact with soil, sediment, or surface water. Children ingest small amounts of soil and indoor dust daily as they interact with their environment, usually by hand-to-mouth activity, mouthing toys, eating dropped food, or similar activities. Our dermal and incidental ingestion assessments for adults assumed general interactions with Norwood soil and sediment or wading or swimming exposures to creek surface water.

We also evaluated a **soil-pica** scenario for children. Pica is a craving to eat nonfood items, such as dirt and paint chips, that some young children exhibit (ATSDR 2005). Children who engage in

soil-pica behavior ingest soils at unusually high rates compared to the rest of the population (1,000-5,000 mg/kg day; ATSDR 2001). Soil-pica exposure assessment is further discussed in Appendix B.

We evaluated the potential exposure pathways discussed in this section (3.1) to determine whether 1) exposure occurred long enough, 2) exposure was frequent enough, or 3) chemical levels were high enough among Winona adult and child residents to result in adverse health effects from the site.

Winona residents or visitors could also potentially inhale certain chemicals from dust suspended from surface soil during recreation or other activities; however, because no air emissions data were collected, we did not evaluate this pathway. Most residents within a 4-mile radius of the site received their drinking water from Aqua Pennsylvania, whose source water is drawn from groundwater and surface water intakes outside of the 4-mile radius of the former landfill (Weston 2018). Therefore, we deemed the drinking water pathway as eliminated. Although some residents expressed concerns with gardening-based exposures, we did not have data on homegrown food (garden) contamination to fully evaluate this ingestion pathway. Appendix H of this HC briefly discusses the potential for garden crop contamination based on chemicals detected in residential surface soil.

### 3.2. Data Screening and Evaluation of Chemicals of Concern (CoC)

Following our identification of exposure pathways for the site, we screened EPA's maximum chemical concentrations for surface soil, sediment and surface water against ATSDR's health-based comparison values (CVs). ATSDR CVs are health protective estimates of a chemical level below which no adverse health effects are expected to occur. If a chemical exceeds a CV, it doesn't necessarily indicate that the exposure will be harmful, but that further evaluation is needed. ATSDR establishes CVs for chemicals based on the environmental media in which they occur (soil, water, air). For non-cancer health effects, ATSDR CVs consider acute (1-14 days), intermediate (15-364 days) and chronic (≥365 days) exposure scenarios. CVs for cancer effects consider lifetime exposure to a substance. CVs are based on epidemiological and toxicological studies and account for sensitive populations (e.g., pregnant women, children) and other uncertainty factors. They are health protective estimates.

There are several ATSDR CVs, listed below.

- **Environmental Media Evaluation Guides (EMEGs)** are estimated concentrations of a substance in a particular media (water, soil or air) to which humans may be exposed during a specified time period (acute, intermediate, chronic) without experiencing adverse non-cancer health effects (ATSDR 2005). EMEGs are derived from ATSDR minimal risk levels (MRLs) which are daily exposures to a substance that are unlikely to cause non-cancer health effects. In tables of this report, acute, intermediate and chronic EMEGs are denoted as aEMEG, iEMEG, and cEMEG, where appropriate.

- **Reference Dose Media Evaluation Guides (RMEGs)** are concentrations of a substance at which daily exposure over a chronic time period is unlikely to result in adverse, non-cancer health effects. RMEGs are calculated from EPA's Oral Reference doses (RfDs) and are used when there is no MRL to derive an EMEG.
- **Cancer Risk Evaluation Guides (CREGs)** are estimated concentrations of a substance that would be expected to cause no more than 1 excess cancer risk in a million persons exposed during a lifetime of 78 years (ATSDR 2005). ATSDR derives CREGs from EPA Cancer Slope Factors (CSFs) for oral exposures.

For chemicals in which ATSDR CVs were not available, we referred to other sources of health-based screening levels. These included EPA's Regional Screening Levels (RSLs) for residential soil, California's Department of Toxic Substances Control (DTSC) screening levels, and Pennsylvania's Department of Environmental Protection (PADEP) Medium Specific Concentrations (MSCs). RSL, DTSC and MSC screening levels are not verified by ATSDR.

PADOH screened EPA's 2017-2018 sampling results using ATSDR's Public Health Site Assessment Tool (PHAST). Chemicals that exceeded a CV were identified as Chemicals of Concern (CoC) and were further evaluated. Lead was also retained for further evaluation as a CoC, as there is no ATSDR-based screening value.

CoCs also include chemicals for which there is widespread community concern or for which there are no CVs. We included the two sampled PCBs (Aroclor 1254 and Aroclor 1260) and multiple heavy metals as CoCs due to community concerns that their homes may have been built on soil containing these chemicals in the 1950s. Although these chemicals can remain in the environment for long periods, EPA's 2017-2018 sampling may not represent prior concentrations.

Of detected Norwood chemicals, there are currently no ATSDR, MSC, RSL or DTSC CVs for sodium, magnesium, potassium, calcium, ethanol and dimethyl phthalate (DMP). Ethanol was detected in a single non-residential soil sample (SS-14) but not its duplicate (SS-20). The single detection was at a low concentration (0.0066 ppm); however we briefly evaluated it as a CoC (Section 5.12 of this HC). Because no CV was identified for DMP, we also evaluated it as a CoC. Essential nutrients sodium, magnesium, potassium and calcium are typically not harmful under most environmental exposure scenarios (ATSDR 2005).

### 3.3. Defining Exposure Areas / Units

***3.3.1 Exposure areas.*** In our CoC evaluation we deemed each of the sampled 21 residences as an exposure area, or its own "exposure unit." Soil-based exposures to residents at one home may not reflect exposures at a second home, and each residential soil concentration was considered independent of other residential concentrations. This was also done as a health protective approach to gauge the potential for health effects based on the highest residential sample.

By contrast, we considered the 17 non-residential soil samples as one exposure area and derived CoC exposure estimates from the 95% Upper Confidence Limit (95UCL) of the arithmetic mean concentration. If a non-residential CoC was detected in less than 20% and fewer than four samples, we evaluated the maximum concentration for that CoC. We similarly used this approach to derive exposure estimates for the 8 collated creek sediment and surface water samples. We note one exception: we estimated PAH exposure from the highest non-residential soil sample (SS6) as it was the sole sample not to use selective ion monitoring (SIM), resulting in higher soil concentrations than the remaining non-residential soil samples (explained further in Appendix D1).

For each CoC we calculated daily exposure doses, which represent the estimated amount of a chemical found in the medium (soil, sediment or water) that enters a person's body based on the levels found at Norwood. All exposure dose estimates assumed a Reasonable Maximum Exposure (RME) scenario. RME refers to people at the high end of the exposure distribution (approximately the 95$^{th}$ percentile). The RME dose is intended to assess exposures that are higher than average but still within a realistic range (ATSDR 2005). Further information on exposure assumptions, including adult, child, and soil-pica assumptions, as well as sample calculations, are provided in Appendix sections B1, B2 and B5.

***3.3.2. Process for Health Effects Evaluation.*** For each CoC we assessed the potential for non-cancer and cancer health effects. For non-cancer effects we calculated a Hazard Quotient (HQ). An HQ consists of a CoC exposure dose divided by an ATSDR Minimal Risk Level (MRL) or EPA oral reference dose (RfD). HQs below 1 indicate that non-cancer health effects are unlikely to occur from the estimated level of exposure to a specific CoC. If a HQ exceeded 1 or if there was no MRL or RfD, we conducted a health effects (also known as a toxicological) evaluation to determine whether adverse health effects could occur. For further details on non-cancer hazard calculations and health effect evaluations, please see Appendix B3. Because of the community concern of MS, we also compared daily CoC exposure dose estimates to exposures known to induce adverse neurological or immunological effects based on the available toxicological literature.

For CoCs that are known or probable human carcinogens with an EPA cancer slope factor (CSF), we calculated lifetime excess cancer risk estimates. Lifetime cancer risk estimates are theoretical proportions of the population that *may* be affected from lifetime exposure to a chemical based on its detected concentration. They are health protective estimates; actual (true) risk is unknown, but may be substantially lower, perhaps by several orders of magnitude (ATSDR 2017). Further details on lifetime cancer risk estimates are discussed at the beginning of section 5 as well as in Appendix section B4.

## 4. Norwood Screening Results and Identified Chemicals of Concern (CoCs)

***4.1 Screening Results Overview.*** We screened chemicals in all media evaluated (residential soil, non-residential soil, sediment, and surface water). Table 2 displays the Norwood screening

results for **residential soil** that exceeded a CV. The table displays the detection percentages, ranges (in parts per million, or mg/kg) and number of samples exceeding a CV. Full screening results for residential soil, as well as results for non-residential soil, creek sediment, and creek surface water can be found in Appendix C. For residential soil, the sample numbers are not shown, as we omitted residential sampling numbers from this report.

**Table 2. Norwood Residential Surface Soil Concentrations that exceeded Comparison Values (CVs)**

| Chemical Sampled in Residential Soil | Number of homes detected (% of total homes assessed) | Range (ppm) | CV (ppm) | CV Type (non ATSDR CV listed if no CV available) | Number Samples Above a CV out of 21 samples (CV type exceeded) |
|---|---|---|---|---|---|
| **Semi-Volatile Organic Compounds and Polycyclic Aromatic Hydrocarbons (PAHs)** | | | | | |
| Benzo(a)anthracene | 21 (100%) | 0.018-9.1 | 1.1 | RSL | 3 |
| Benzo(b)fluoranthene | 21 (100%) | 0.03-15.0 | 1.1 | RSL | 3 |
| Benzo(a)pyrene | 21 (100%) | 0.024-9.7 | 0.065<br>16 | CREG<br>RMEGc | 9 (CREG) |
| Indeno(1,2,3-cd)pyrene | 21 (100%) | 0.0095-6.9 | 1.1 | RSL | 3 |
| bis(2-Ethylhexyl)phthalate (DEHP) | 21 (100%) | 0.029-6.8 J | 5.2<br>28<br>0.53 | iEMEGc<br>CREG<br>iPica | 1 (iEMEGc)<br>0 (CREG)<br>1 (iPica) |
| **Polychlorinated Biphenyls (PCBs)** | | | | | |
| Aroclor-1260 | 16 (76.2%) | ND-0.31 | 0.24<br>9.0 | RSL<br>PADEP MSC | 1 (RSL) |
| **Pesticides** | | | | | |
| Gamma-BHC (Lindane) | 6 (28.6%) | ND-0.065 | 0.52<br>0.053 | iEMEGc<br>iPica | 1 (iPica) |
| Aldrin | 6 (28.6%) | ND-0.053 J | 0.023 | CREG | 1 |
| Dieldrin | 19 (90.5%) | ND-1.3 | 0.024<br>0.53 | CREG<br>iPica | 2 (CREG: 2; iPica: 1) |
| **Metals** | | | | | |
| Aluminum | 21 (100%) | 8990-13600 | 5,300<br>52,000 | iPica<br>cEMEGc | All (iPica) |
| Antimony | 1 (4.8%) | 4.2-4.2 J | 3.2<br>21 | iPica<br>RMEGc | 1 (iPica) |
| Arsenic | 21 (100%) | 4.2-9.7 | 0.26<br>16 | CREG<br>cEMEGc | 21 (CREG)<br>0 (cEMEGc) |
| Cadmium | 20 (95.2%) | ND-3.4 | 5.2<br>2.7 | cEMEGc<br>iPica | 0 (cEMEGc)<br>1 (iPica) |
| Copper | 21 (100%) | 10.2-264 | 53<br>1,000 | aPica, iPica<br>iEMEGc | 2 (aPica, iPica)<br>0 (iEMEGc) |
| Iron | 21 (100%) | 12,300-25300 | 5,500 | RSL | 21 |

| | | | | | |
|---|---|---|---|---|---|
| Lead | 21 (100%) | 30-1800 | 400 | RSL | 1 |
| Manganese | 21 (100%) | 105-553 | 180<br>10,000 | RSL<br>PADEP MSC | 18 (RSL)<br>0 (PADEP MSC) |
| Thallium | 0 (0.0%) | ND | 0.078 | RSL | Non-detect quantitation limit exceeds RSL |
| RMEGc=Reference Dose Media Evaluation Guide (Child); cEMEGc = Chronic Evaluation Media Guide (Child); iEMEGc = Intermediate Evaluation Media Guide (Child); CREG = Cancer Risk Evaluation Guide; aPica = Acute Pica; iPica = Intermediate Pica; RSL = EPA Regional Screening Value; CV = Comparison Value; ND = Not Detected; U = Not Detected Quantitation Limit; J = Reported value is estimated and actual value may be higher or lower, PADEP MSC = PADEP Medium Specific Concentration | | | | | |

Generally, across all sampled media, few of the sampled chemicals exceeded a CV (Appendix Tables C1-C4). Among residential soil samples, 3 samples most commonly exceeded a CV, most often for one or more PAHs. Among non-residential soil samples, SS6 most often exceeded a CV (Appendix Table C2). Creek sediment had lower PAHs but often higher heavy metal concentrations than did residential or non-residential soil (Appendix Table C3). Very few surface water samples exceeded a CV; as noted, only inorganic metals were sampled in surface water (Appendix Table C4).

***4.2 Chemicals of concern (CoCs)***. Table 3 below displays the chemicals of concern (CoCs) selected for further evaluation. As discussed in section 3.2, we retained CoCs that exceeded a non-ATSDR CV if an ATSDR CV was unavailable.

**Table 3. Chemicals of concern (CoCs) selected for further evaluation**

| No. | Chemical of Concern (CoC) | Why Evaluated |
|---|---|---|
| 1 | Benzo[a]pyrene / PAHs | Multiple residential and non-residential samples exceeded soil CVs (CREGs or EPA RSLs). |
| 2 | di(2-ethylhexyl) phthalate (DEHP) | Two residential samples exceeded child intermediate soil CVs (EMEGs), one for pica and one for non-pica exposures. These samples were estimated (J) values. |
| 3 | PCBs: Aroclor 1254 and Aroclor 1260 | A single residential sample exceeded a soil CV (EPA RSL), as did 2 non-residential samples; PCBs were identified as a community concern. |
| 4 | Aldrin and Dieldrin | Two residential and one non-residential sample exceeded a CV (CREG) for Dieldrin. One residential sample exceeded a CREG for Aldrin. |
| 5 | Copper | Several residential, non-residential, and sediment samples exceeded an acute and intermediate soil-pica CV for copper. Non-pica CVs were not exceeded. |
| 6 | Iron | All samples exceeded a soil CV (an EPA RSL) for iron. |
| 7 | Manganese | Most samples exceeded a CV (an EPA RSL) for manganese. |
| 8 | Arsenic | All soil/sediment samples exceeded a soil CV (CREG), which is set below background levels. Two sediment samples and one surface water sample exceeded a chronic EMEG CV. Heavy metals were identified as a community concern. |
| 9 | Chromium | Although CVs were not exceeded for chromium(III), the valence state (chromium VI versus chromium III) was not specified; heavy metals were identified as a community concern. |

| | | |
|---|---|---|
| 10 | Mercury | A single sediment sample (a duplicate sample) exceeded a CA Toxic Substances Control CV; heavy metals were identified as a community concern. |
| 11 | Dimethyl Phthalate | A CV could not be identified. |
| 12 | Ethanol | Ethanol was detected in a single non-residential sample, but not its duplicate. A CV could not be identified. |
| 13 | Lead | There is no ATSDR screening value for lead, since no blood lead level is presumed safe; it is always retained for further evaluation. Heavy metals were identified as a community concern. |
| CV=comparison value; EMEG = Environmental Media Evaluation Guide; CREG = Cancer Risk Evaluation; EPA RSL = Regional Screening Level for Residential Soil | | |

***4.3. Additional chemicals evaluated.*** A few chemicals (aluminum, antimony, lindane, and cadmium) only exceeded an ATSDR CV for intermediate-duration soil-pica behavior; in these instances, ATSDR CVs for non-pica behavior were not exceeded. For these chemicals we evaluated the potential health effects from an intermediate-duration and single soil-pica scenario. An intermediate-duration soil-pica scenario assumes consumption of 5,000 mg soil for 3 days per week for up to a year and is an uncommon scenario.

We also evaluated thallium, which was not detected at Norwood but whose maximum non-detect threshold exceeded EPA's RSL CV comparison value of 0.078 mg/kg.

***4.4. Summary of CV Exceedances.*** Table 4 displays the number of samples that exceeded CVs based on media evaluated (residential soil, non-residential soil, creek sediment, and creek surface water). Many more samples were not exceeded, which is shown fully in Appendix Tables C1-C4.

**Table 4. Number of samples that exceeded a comparison value based on location**

| Chemical | Number of Samples | | | |
|---|---|---|---|---|
| | Location | | | |
| | RS (n=21) | NRS (n=17) | SED (n=8) | SW (n=8) |
| Benzo(a)anthracene | 3 | 2 | 0 | NS |
| Benzo(b)fluoranthene | 3 | 2 | 0 | NS |
| Benzo(a)pyrene | 9 | 12 | 0 | NS |
| Indeno(1,2,3-cd)pyrene | 3 | 1 | 0 | NS |
| Dibenzo(ah)anthracene | 0 | 3 | 0 | NS |
| di(2-ethylhexyl) phthalate (DEHP) | 2* | 0 | NS | NS |
| Aroclor-1254 | 0 | 2(i)pi | NS | NS |
| Aroclor-1260 | 1 | 2 | NS | NS |

| | | | | |
|---|---|---|---|---|
| Gamma-BHC (Lindane) | 1 (i)pi | NS | NS | NS |
| Aldrin | 1 | 0 | NS | NS |
| Dieldrin | 2 | 1 | NS | NS |
| Aluminum | 21 (i)pi | 17 (i)pi | 8 (i)pi | 0 |
| Antimony | 1 (i)pi | 1 (i)pi | 0 | ND |
| Arsenic | 21 | 17 | 8 | 8 |
| Cadmium | 1 (i)pi | 0 | 6 (i)pi | 0 |
| Chromium (Total Chromium)** | NA | NA | NA | NA |
| Copper | 2 (pi) | 2 (pi) | 8 (pi) | 0 |
| Iron | 21 | 17 | 8 | NA |
| Lead | 1*** | 0 | 0 | NA |
| Manganese | 18 | 16 | 8 | NA |
| Mercury | 0 | 0 | 1 | ND |
| Thallium | 21(U) | 17(U) | 8(U) | ND |

RS = Residential Soil; NRS = Non-residential Soil; SED = Creek Sediment; SW = Creek Surface Water; U = Not Detected, but the quantitation limit exceeded a CV, NA = not applicable (no CV); ND = Not Detected; NS = Not Sampled, (pi) = acute and intermediate pica (i)pi = intermediate pica
*1 of the 2 exceedances was for intermediate pica only
**There is no total chromium CV, but no samples exceeded a chromium(III) CV.
***Based on an EPA Residential Screening Level of 400 mg/kg; however, PADOH always retains lead for further evaluation.

As shown in Table 4, benzo[a]pyrene was the most common PAH that exceeded CVs in residential (9 exceedances) and non-residential soil (12 exceedances). We further examined the lowest, highest, and second highest benzo[a]pyrene samples that exceeded screening levels (Appendix D1). We report on the potential for health effects from benzo[a]pyrene and remaining PAHs based on the highest sample (Appendix D1 and Section 5 of this HC). For most other chemicals (e.g., non-benzo[a]pyrene PAHs, Aroclor 1254 and 1260, aldrin, dieldrin, copper, mercury, lindane, antimony, aluminum, cadmium, thallium), CV exceedances are limited to 1-3 samples, pertain only to soil pica behavior (e.g., aluminum), or were not detected (thallium) but had a quantitation limit that exceeded a CV. Arsenic, iron and manganese were the most common metals that exceeded CVs. In the case of arsenic, all Norwood samples exceeded an ATSDR CREG CV; however, these CVs are set below background levels. There are no ATSDR CVs for iron and manganese; CV exceedances for these chemicals are based on EPA residential RSL CVs listed in its 2017-2018 Norwood report (Weston 2018). Total chromium was

not speciated between its less toxic chromium(III) and more toxic chromium(VI) forms, but no Norwood samples exceeded a chromium(III) CV.

## 5. Public Health Evaluation - Summary

We conducted a health effects evaluation for each chemical of concern (CoC) identified in section 4.2 (Table 3). Our results are summarized below. Our full health effect evaluations can be found in Appendix D (for non-pica exposures) and Appendix E (for pica exposures). Where appropriate, estimated exposure doses are presented as microgram per kilogram per day (µg/kg/day) to aid interpretation of low exposures.

Table 5 below displays a summary of the calculated exposure doses, chronic hazard quotients, and excess cancer risk estimates for CoCs based on the highest detected concentrations from EPA's 2017-2018 sampling. The maximum chemical concentrations for residential soil are displayed. If *non-residential* concentrations (e.g., non-residential soil, creek sediment) exceeded *residential* maximums, the *non-residential* result is displayed instead, as either a maximum value (if the sample was detected in fewer than 4 or 20% of samples) or the 95UCL if the chemical was detected with greater frequency. At non-residential locations, the highest concentrations were most often found in creek sediment.

For residential soil, two CoCs exceeded a chronic health guideline for young children up to 1 year of age: 1) benzo[a]pyrene-equivalent PAHs, and 2) total chromium, under a chromium(VI) assumption. This means chronic hazard quotients (HQs) exceeded 1. The remaining CoCs with MRLs or RfDs did not exceed chronic health guidelines (HQs<1) indicating that chronic exposure is unlikely to result in adverse non-cancer health effects. The maximum benzo[a]pyrene-equivalent sample produced the highest residential excess cancer risk estimate, of 3 in 10,000, for children less than 21 years of age. An explanation of cancer and non-cancer health effect estimates, including Hazard Quotients and Lifetime Excess Cancer Risk, is provided in Appendix B.

**Table 5**: **Calculated Exposure Doses, Chronic Hazard Quotients and Excess Cancer Risk Estimates for Norwood Chemicals of Concern (CoCs) based on the highest single sample and 95 UCL concentrations detected during 2017-2018 sampling**

| Chemical of Concern (CoC) | Sampling location with highest concentration | Concentration (µg/kg) | Exposed Population[1] | Ingestion and Dermal Exposure Dose (ED) (µg/kg/day) | CoC Chronic RfD or MRL[2](µg/kg/day) | Chronic Hazard Quotient (ED/RfD or MRL) | Excess Cancer Risk[3,4] |
|---|---|---|---|---|---|---|---|
| Benzo[a]pyrene Equivalent-PAHs | RS | 13,490 (max) | Adult | 0.026 | 0.30 (RfD) | <1 | 2 in 100,000 |
| | | | Child | 0.34 | | **1.1** | 3 in 10,000 |
| DEHP | RS | 6,800 (J) (max) | Adult | 0.012 | 20 (RfD) | <1 | < 1 in 1 million |
| | | | Child | 0.16 | | <1 | < 1 in 1 million |
| Aroclor 1254 | NRS | 450 | Adult | 0.00089 | 0.02 | <1 | < 1 in 1 million |

| | | | | | | | |
|---|---|---|---|---|---|---|---|
| (PCB Mixture) | | (max) | Child | 0.0043 | (MRL) | <1 | 1 in 1 million |
| Aroclor 1260 (PCB Mixture) | RS | 310 (max) | Adult | 0.00062 | NA | NA | < 1 in 1 million |
| | | | Child | 0.0079 | | | 2 in 1 million |
| Aldrin | RS | 53 (max) | Adult | 0.000094 | 0.04 (MRL) | <1 | < 1 in 1 million |
| | | | Child | 0.0013 | | <1 | 2 in 1 million |
| Dieldrin | RS | 1,300 (max) | Adult | 0.0023 | 0.05 (MRL) | <1 | 2 in 100,000 |
| | | | Child | 0.031 | | <1 | 5 in 100,000 |
| Copper | RS | 264,000 (max) | Adult | 0.35 | NA | NA | NA |
| | | | Child | 5.3 | | | |
| Iron | SED | 34,255,000 (95 UCL) | Adult | 43 | 700 (RfD) | <1 | NA |
| | | | Child | 220 | | <1 | |
| Manganese | SED | 813,200 (J) (95 UCL) | Adult | 2.1 | 160 (Int Value) | <1 | NA |
| | | | Child | 10 | | <1 | NA |
| Arsenic | SED | 18,000 (95 UCL) | Adult | 0.016 | 0.3 | <1 | 1 in 100,000 |
| | | | Child | 0.081 | | <1 | 1 in 100,000 |
| Chromium(VI)[5] | SED | 80,900 (95 UCL) | Adult | 0.27 | 0.9 | <1 | 6 in 100,000 |
| | | | Child | 1.3 | | **1.4** | 2 in 10,000 |
| Mercury | SED | 958 (95 UCL) | Adult | 0.0019 | 0.3 | <1 | NA |
| | | | Child | 0.0093 | | <1 | NA |
| Dimethyl Phthalate (DMP) | SED | 862 (95 UCL) | Adult | 0.0015 | NA | NA | NA |
| | | | Child | 0.0075 | | | |
| Ethanol | NRS | 6.6 (max) | Adult | 0.0000083 | 62,000[6] | <1 | NA |
| | | | Child | 0.000042 | | <1 | NA |
| Lead | RS | 1,800,000 (max) | Adult and child | NA | NA | NA | NA |

**Bold** = exceedance of a chronic hazard quotient (please see Appendix B3). 95 UCL = the 95$^{th}$ percent upper confidence limit of the mean; ED = Exposure Dose; MRL = ATSDR's Minimal Risk Level; Int Value = ATSDR Interim Guidance Value; PAHs = Polycyclic Aromatic Hydrocarbons; RfD = EPA's Chronic Reference Dose; RS = Residential Sample; NRS = Non-residential Sample; SED = Sediment; SW = Surface Water; NA= Not Applicable.

[1]All child daily exposure dose estimates (in µg/kg/day) represent ages up to 12 months age for residential soil, and ages 6-10 years for sediment.

[2]Only chronic health guidelines are shown in this table. Intermediate and Acute health guidelines for each CoC are discussed in Appendix D.

[3]Excess cancer risk was calculated by multiplying each exposure dose estimate by the U.S. Environmental Protection Agency's (EPA's) cancer slope factor (CSF) in (mg/kg/day)$^{-1}$. For benzo[a]pyrene-equivalent PAHs and chromium(VI), we multiplied the exposure dose estimate by CSFs derived from the California EPA, which are ATSDR-recommended and more health protective. Excess cancer risk was not calculated for chemicals that lack a CSF. For Aroclor 1254 and Aroclor 1260, excess cancer risk was calculated using EPA's CSF for PCBs.

[4]The childhood cancer risk estimate applies to children of all ages (<21 years of age) for residential soil, and children ages 6 to <21 years for creek sediment. Adult cancer risk estimates pertain to persons ages 21 years and older.

[5]The values for chromium assume Norwood total chromium concentrations are 100% chromium(VI) as opposed to the less toxic chromium(III).

[6]RfD Source: Michigan Department of Environment, Great Lakes, and Energy (2015)

For CoCs that are known or probable human carcinogens with an EPA or ATSDR-recommended cancer slope factor (CSF), we calculated excess cancer risk estimates. Under quantitative cancer

risk assessment methodology, cancer risk estimates are expressed as a probability. They are expressed as the proportion of a population that ***may*** be affected by a carcinogen during a lifetime of exposure (24 hours/day, 365 days/year). An estimated cancer risk of 2 in 1 million represents potentially 2 additional cancer cases above expected cases in a population of 1 million over a lifetime of continuous exposure. Lifetime cancer risk is a theoretical estimate and not a prediction of the number of cancers in a community. For adults, Norwood cancer risk estimates assume 33 consecutive years of exposure. For children, cancer risk estimates from *residential soil* assume daily exposure from birth through age 20 years, while estimates from *non-residential soil and sediment* assume exposures from ages 6 through age 20 years.

A summary of our health effects evaluation for each CoC is below. Our full CoC descriptions are provided in Appendix D.

### 5.1 Benzo[a]pyrene and Polycyclic Aromatic Hydrocarbons (PAHs)

Polycyclic aromatic hydrocarbons (PAHs) are a group of semi-volatile organic compounds (SVOCs), found throughout the environment. The burning of wood in homes and vehicle exhaust are common sources of PAHs; meat cooked under high temperature can also release PAHs. Most members of the U.S. are exposed from food or inhalation from tobacco smoke, wood smoke, or contaminated air (ATSDR 1995).

The highest Norwood PAH concentrations were found in residential soil. We evaluated PAHs as a benzo[a]pyrene-equivalent mixture (explained in Appendix D1). Based on estimated exposure doses to the highest benzo[a]pyrene-equivalent mixture (13,490 µg/kg; Table 5), adverse non-cancer effects are unlikely to occur among children or adults. Exposure doses for children 1 year old or younger exceeded EPA's reference dose (RfD) for benzo[a]pyrene. Our subsequent toxicological evaluation revealed that adverse non-cancer health effects for this age group are unlikely.

Benzo[a]pyrene is among the most widely studied PAHs, and the EPA has deemed it a human carcinogen based on strong animal and human evidence (EPA 2017). Lung and skin cancer are common cancer risk sites in humans, mostly from certain occupational-based exposures to higher benzo[a]pyrene-based PAH mixtures. At Norwood, the highest lifetime excess cancer risk from benzo[a]pyrene-equivalent PAH exposures was 3 in 10,000 for children and 2 in 100,000 for adults based on concentrations from a single (maximum) residential soil sample. This maximum sample was 3.5 times higher than the next highest residential sample; thus it is an overestimate of PAH risk for most Norwood residential soil exposures. The next highest sample produced an excess cancer risk estimate of 7 in 100,000 for children and 5 in a million for adults.

### 5.2 Di(2-ethylhexyl)phthalate (DEHP)

DEHP is a man-made chemical used to make plastics flexible. People are primarily exposed through food. DEHP has mostly been phased out of U.S. commercial production, but it can enter the environment from disposal into landfills.

The highest Norwood DEHP concentrations were found in residential soil, and the maximum concentration was an estimated ("J") value of 6,800 µg/kg (Table 5). Based on estimated exposure doses to this maximum concentration, adverse non-cancer effects are unlikely to occur among children or adults. ATSDR does not have a recommended chronic health guideline for DEHP, but Norwood exposure doses fell well below thresholds known to cause health effects from chronic (a year or longer) exposures. Exposure doses were also below ATSDR's acute health guideline, pertaining to exposures of 14 days or fewer. Conversely, both pica and non-pica exposure dose estimates for intermediate exposures (15 days to a year) exceeded ATSDR's intermediate minimal risk level (MRL). These intermediate MRL exceedances pertained to children 1-5 years old (pica) and children 1 year and younger (non-pica) and prompted us to conduct a toxicological evaluation. The evaluation revealed that adverse non-cancer health effects are unlikely. This is discussed further in Appendix D for non-pica and E for pica exposures.

The EPA and Department of Health and Human Services (DHHS) have classified DEHP as a probable/reasonably anticipated human carcinogen based on sufficient evidence in animals, but few human studies have evaluated cancer endpoints (ATSDR 2022). At Norwood, the highest excess cancer risk from exposures to the maximum DEHP concentration was 2 in 10 million.

**5.3 Polychlorinated Biphenyls (PCBs)**

PCBs are a group of man-made chemicals that have not been produced in the U.S. since the 1970s. Because they can remain in the environment for long periods, PCBs can be found in small amounts in air, water, and soils. People are primarily exposed from old transformers or related equipment, breathing contaminated air, or by eating contaminated fish, meat or poultry.

EPA sampled two chemically-similar PCBs in Norwood residential and non-residential soil: Aroclor 1254 and Aroclor 1260. These PCBs were detected at similar concentrations. Based on estimated exposure doses to the highest detected concentrations, adverse non-cancer health effects are unlikely to occur among children or adults. Exposure doses for Aroclor 1254 did not exceed chronic or intermediate health guidelines. Aroclor 1260 lacks health guidelines, so we conducted a toxicological evaluation. Our evaluation for both Aroclors indicated that neither non-pica (Appendix D) nor pica exposures (Appendix E) were of concern for adverse non-cancer health effects.

In 2013 the International Agency for Research on Cancer (IARC) deemed PCBs as carcinogenic to humans due to strong evidence for malignant melanoma risk and sufficient evidence in animals. DHHS classifies PCBs as "reasonably anticipated to be human carcinogens" based on evidence

in laboratory animals, and the EPA classifies them as probably carcinogenic (ATSDR 2000). At Norwood, use of EPA's cancer slope factor for PCBs resulted in a maximum lifetime excess cancer risk estimate of 2 in a million (Table 5).

**5.4 Aldrin and Dieldrin**

Aldrin and dieldrin are insecticides that were primarily used in the 1950s and 60s. Though these chemicals have since been phased out, they can remain in the environment for long periods. The most common human exposures are from food or drinking water containing these compounds, but current U.S. exposures are generally low to undetectable.

Aldrin and dieldrin were sampled in Norwood residential and non-residential soil, with residential soil having slightly higher concentrations. Based on estimated exposure doses to the highest aldrin (53 µg/kg) and dieldrin (1,300 µg/kg) concentrations, adverse non-cancer health effects are unlikely to occur among children or adults. Aldrin and dieldrin doses were below chronic minimal risk levels (MRLs) (Table 5). An intermediate soil-pica scenario for dieldrin exceeded ATSDR's intermediate MRL, prompting us to conduct a toxicological evaluation for pica behavior (Appendix E); this evaluation revealed that adverse health effects from pica behavior are unlikely.

Based on laboratory animal studies, EPA and IARC have deemed aldrin and dieldrin as probable human carcinogens (ATSDR 2021). At Norwood, the highest lifetime excess cancer risk from the compounds was 5 in 100,000 for children and 2 in 100,000 for adults (for dieldrin).

**5.5 Copper**

Copper occurs naturally in the environment and is an essential nutrient, although excess copper can cause gastrointestinal and other ailments.

A residential soil sample had the highest copper concentration. Based on estimated exposure doses to this concentration, adverse health effects from *non-soil-pica behavior* are unlikely to occur among children or adults (Table 5). Conversely, intermediate or acute consumption of this soil concentration from *pica behavior could* result in gastrointestinal effects in children 1 to 5 years old. Further details are provided in Appendices D for non-pica and E for pica exposures.

Copper has not been classified as a carcinogen due to lack of adequate human or animal cancer studies.

**5.6 Iron**

Iron is an essential nutrient naturally present in many foods. Either insufficient or excess iron levels in the body can be harmful to health.

Creek sediment had the highest iron concentrations. Based on estimated exposure doses to these concentrations, adverse health effects from *non-pica* behavior are unlikely among children or adults. Conversely, *intermediate-soil-pica behavior* resulting in consumption of the

highest amounts of iron at Norwood *could* result in gastrointestinal effects in children 1 to 5 years old, and could also be a general health concern for adults who have a condition called hemochromatosis. (Note: we considered an intermediate pica-scenario for residential soil exposures.) Further details are discussed in Appendix D for non-pica, and E for pica-based exposures.

Iron is not considered a carcinogen.

**5.7 Manganese**

Manganese occurs naturally in many rocks and soil and is a normal constituent of air, water, and food. It is also used in steel production and can be found in consumer products. Humans are primarily exposed through diet, and a certain amount of manganese is needed for good health. At high exposures, manganese can be harmful to health.

Creek sediment had the highest manganese concentrations, which were all estimated ("J") values. Based on estimated exposure doses to these concentrations, adverse health effects are unlikely to occur among children or adults. There is no chronic MRL for manganese, but the highest exposure doses from sediment (10 µg/kg/day among children 6-10 years old) and residential soil (17 µg/kg/day among children up to 1 year old) fell below ATSDR's interim guidance value of 160 µg/kg/day (Table 5), which is recommended for use in health assessments (ATSDR 2012a). They also fell below a California Office of Environmental Health and Hazard Assessment RfD of 30 µg/kg/day which is protective for childhood manganese exposure (OEHHA 2006). These comparisons are discussed further in Appendix D.

Manganese is not considered a carcinogen.

**5.8 Arsenic**

Arsenic is a naturally occurring element widely distributed in the Earth's crust. It can also be released from coal-fired power plants or at hazardous waste sites if not properly disposed. People are exposed to small amounts through food, drinking water, or inhalation. Inorganic arsenic is harmful to health and has been widely studied.

Of all environmental media, creek sediment had the highest arsenic concentrations. Based on estimated exposure doses to these concentrations, adverse non-cancer health effects are unlikely to occur among children or adults. Doses did not exceed chronic or acute health guidelines. There is no intermediate health guideline for arsenic, but Norwood exposure doses were orders of magnitude below reported thresholds that induced adverse health effects in intermediate-duration studies.

Arsenic is a well-known human carcinogen and is classified as such by the IARC, DHHS, EPA, and other agencies. Chronic exposures in people have most commonly been linked to lung, skin and bladder cancers, though increased risk of bladder or respiratory tumors has not been found in the U.S. following oral exposure (ATSDR 2007). At Norwood, the highest excess cancer risk for

arsenic in residential soil was 2 in 100,000 for children and 6 in a million for adults (Appendix Table D.8.1). Further details on our arsenic health effects evaluation are available in Appendix D for non-pica exposures and E for pica exposures.

**5.9. Chromium**

Chromium is a naturally-occurring element of rocks, animals, plants and soil, and it can also be found in air and drinking water. It is widely used in manufacturing and to make products such as stainless steel. Food is the most common source of chromium for the public. A certain amount of chromium(III) is needed for good health, but chromium(VI) is more harmful to health and widely studied.

Creek sediment had the highest total chromium concentrations. Since chromium's valence state (chromium(III) or chromium(VI)) was not specified, we assumed all detected total chromium was chromium(VI), its more toxic form. This assumption was made as a health protective and "worst-case scenario" approach, however, in most soils, chromium is in its less toxic chromium(III) form (ATSDR 2012b).

Based on our calculations using assumptions mentioned above, we would not expect adults or children to experience adverse non-cancer health effects from exposures to the highest estimated chromium(VI) concentrations. Exposure doses for children 0-12 months based on the highest residential soil sample exceeded ATSDR's chronic MRL for chromium(VI). An intermediate soil-pica scenario for a 1 year old child exceeded ATSDR's intermediate MRL for chromium(VI). Our subsequent toxicological evaluations revealed that adverse non-cancer health effects for these age groups are unlikely.

The IARC and DHHS have classified chromium(VI) as carcinogenic to humans, and EPA has classified it as a carcinogen by inhalation. Inhalation exposure to chromium(VI) has been shown to cause lung cancer in occupationally-exposed workers, who can be exposed at two orders of magnitude higher than the general population. At Norwood, the highest excess residential cancer risk estimate based on our chromium(VI) assumption was 2 in 10,000 for children and 2 in 100,000 for adults. These estimates assume daily, year-round exposures to the highest detected residential chromium as 100% chromium(VI). In creek sediment (which had the highest total chromium concentrations), the highest excess cancer risk was 2 in 10,000 for children and 6 in 100,000 for adults. For children, cancer risk estimates based on sediment exposures assume daily exposures at EPA sampling locations from ages 6 through 20 years. Our toxicological evaluation for chromium is provided in Appendices D (non-pica) and E (pica).

We also assessed an exposure scenario under an assumption that total chromium was primarily in its less toxic, chromium(III) form. This is a more likely scenario chromium speciation scenario. Under an assumption that detected Norwood total chromium was 75% chromium(III) and 25% chromium(VI), the highest excess cancer risk from residential soil exposures was 6 in 100,000 for children and 6 in a million for adults.

Because total chromium was not speciated, we could not calculate more refined estimates of health risk.

**5.10. Mercury**

Mercury is found throughout the environment from natural and man-made sources. Humans are exposed to low levels from air, water, and food. The form of mercury most harmful to health is methylmercury, which can build up in contaminated fish. Commercial fish in the U.S. cannot be sold unless mercury levels are below 1 part per million.

Creek sediment had the highest mercury concentration. Based on estimated exposure doses to this concentration, it is unlikely that adverse non-cancer health effects would occur among children or adults. Doses did not exceed chronic, intermediate and acute health guidelines for inorganic mercury, the presumed form of mercury sampled. Residential mercury concentrations did not exceed a CV. Further discussion on our mercury health effects evaluation is available in Appendices D (non-pica) and E (pica).

EPA considers some forms of mercury (mercuric chloride and methylmercury) as possible human carcinogens but has not derived a mercury cancer slope factor.

**5.11 Dimethyl phthalate (DMP)**

Dimethyl phthalate (DMP) has many uses, including in the manufacture of products such as plastics, safety glasses, and insect repellants (EPA 2007, EPA 2000). There are very limited data on the oral or dermal toxicity of DMP and it has not been classified as a carcinogen.

Creek sediment had the highest DMP concentrations. Although DMP lacks health guidelines, the highest estimated Norwood exposure doses were well below an EPA oral subchronic reference screening value for DMP (EPA 2007). Therefore, it is unlikely that adverse health effects would occur from the highest Norwood DMP exposures.

**5.12 Ethanol**

Ethanol has many uses, including as an additive to gasoline and component of alcoholic beverages. Ethanol was detected in a single non-residential soil sample (SS-14) at a low concentration (6.6 µg/kg), and was not detected in the duplicate sample (SS-20). There are no oral CVs or chronic RfDs/MRLs for ethanol, but the highest Norwood exposure estimates for children (0.000042 µg /kg/day) and adults (0.0000083 µg/kg/day) were over a million times below a state reference dose (RfD) of 62 mg/kg/day (Michigan Department of Environment, Great Lakes, and Energy, 2015). Therefore, adverse health effects are unlikely to occur from this single Norwood ethanol sample. Due to the low exposure doses for ethanol and the lack of its detection in the duplicate sample, we did not consider it further for our toxicological evaluations.

**5.13 Lead**

Lead is found in ore deposits and distributed widely throughout the world. It is released from man-made sources such as coal and oil combustion, mining and smelting of ore, and waste incineration. Human lead exposures include outdoor air, food, drinking water, soil and dust. Lead found in paint in older homes in densely populated areas creates a greater lead exposure risk in cities than in suburban or rural areas. Further, lead's use in gasoline prior to its ban in 1995, which deposited lead in soil where homes are closer to the road, creates an additional increased risk for exposure in more urban areas. Urban lead exposures are typically higher than rural due to housing characteristics and proximity to roads.

No safe blood lead level has been identified, and children, whose bodies are still developing, are particularly susceptible from lead exposures. Children also exhibit behaviors (e.g., hand mouth activity) that increase ingestion of lead surface dusts, and have a closer breathing zone to soil and surface dust, increasing their risk. Lead can adversely affect the neurological, immunological, and other organ systems in children and adults.

Additional risk factors that contribute to lead body burden, in children and adults, include living in older buildings with deteriorating lead paint, certain occupational exposures (which can then track lead into a home from clothing or tools), socioeconomic status, lead in water service lines, living in areas where lead was produced or disposed, or second-hand smoke exposures. Lead is also sometimes found in certain foods, cosmetics, traditional/alternative medicines, toys or jewelry imported into the U.S. from other countries. Certain hobbies can also increase exposure to lead hazards, including casting or soldering (e.g., bullets, fishing weights, stained glass), mixing or applying glaze or pigments containing lead, shooting firearms during target practice, drinking home distilled liquids (e.g., moonshine), and consuming certain traditional medicines (CDC 2022b). Conducting home renovations, remodeling or painting on structures built prior to 1978 can lead to lead exposure and should only be done by a qualified lead abatement professional. Further details on these and other sources of lead exposure can be found from the U.S. Centers for Disease Control and Prevention website: https://www.cdc.gov/nceh/lead/prevention/sources.htm. **PADOH recommends reducing the possibility for lead exposure whenever possible.**

The concentration of lead in the top layers of soil varies widely due to deposition and accumulation of atmospheric particulates from anthropogenic sources (ATSDR 2020). 2017-2018 lead concentrations at Norwood sampling locations were within ranges for U.S. soil, though mean and median lead levels were higher than national background averages (please see section Appendix Tables D.12.1 or H.3.1 for further details). Median lead for Norwood residential soil (54.7 mg/kg) slightly exceeded Pennsylvania's median of 46.4 mg/kg. Median lead in non-residential soil was higher (145 mg/kg). Pennsylvania's median lead value is based on 2007-2010 data from the U.S. Geological Survey (EPA n.d.). Lead at Norwood was lower than that found in more urban Pennsylvania areas, such as Philadelphia (O'Shea et al. 2021). It is possible that older housing stock and proximity to nearby roadways, including from I-95 across from Darby Creek, are contributing to lead levels in this general region.

We used EPA's Integrated Exposure Uptake of Biokinetic Model (IEUBK version 2.0) to estimate a lead threshold in soil that would result in an at or greater than 3.5 µg/dL childhood blood lead level. A 3.5 µg/dL threshold is based on the U.S. 97.5$^{th}$ percentile blood lead distribution for children 1-5 years old (Ruckart et al. 2021), and PADOH considers this threshold as "elevated." Use of the IEUBK model revealed that exposure to 245 mg/kg lead in soil, when combined with other default childhood assumptions for dietary intake, outdoor air, water consumption and lead absorption, could result in a 3.5 µg/dL blood lead level in children 6-12 months old. At Norwood, 3 of 21 residential samples (of 248 mg/kg, 283 mg/kg, and 1,800 mg/kg) and 5 of 17 non-residential samples exceeded this 245 mg/kg threshold (Table 6). In combination with other potential lead exposures (e.g., outdoor air, indoor dust, dietary sources) this exposure to lead in soil may contribute to a child's overall blood lead level.

**Table 6. Lead concentrations (mg/kg) in Norwood residential soil, non-residential soil, and creek sediment**

| | Residential Soil | Non-Residential Soil | Creek Sediment |
|---|---|---|---|
| Samples (% Detected) | 21 (100%) | 17 (100%) | 8 (100%) |
| Range | 30-1800 | 20.7-358 | 74.5-214 |
| Median | 54.7 | 145.0 | 89.1 |
| Mean | 162.5 | 147.3 | 111.2 |
| Geometric Mean | 78.9 | 102.6 | 101.9 |
| Samples exceeding 245 mg/kg IEUBK threshold | 3 | 5 | 0 |
| IEUBK = EPA's Integrated Exposure Uptake of Biokinetic Model, version 2.0. | | | |

Median creek surface water lead (3.9 µg/L) was similar to mean lead found in U.S. surface waters (3.9-4.0 µg/L; ATSDR 2020).

Lead is a probable human carcinogen but lacks a cancer slope factor to assess excess cancer risk. Human studies provide some evidence that lead is a carcinogen, but cancer results are inconsistent and often confounded by other factors (e.g., smoking status, family history of cancer, co-exposure to other carcinogens; ATSDR 2020). Further discussion of our lead health effects evaluation is available in Appendix D12.

### 5.14 Consideration of additional chemicals

As mentioned in section 4.3, we also assessed **aluminum, antimony, lindane, and cadmium,** because they exceeded an intermediate soil-pica comparison value only. **Based on our review, none of these chemicals are likely to be of concern for soil-pica scenarios.**

We also assessed thallium. Thallium was not detected, but its quantitation limits for residential soil, non-residential soil and creek sediment exceeded an EPA RSL comparison value. Our assessment of the above compounds is provided in Appendix E2.

### 5.15 Summary

We assessed the potential for cancer and non-cancer health effects based on EPA's 2017-2018 sampling data of Norwood soil and Darby and Muckinipattis Creek sediment and surface water. (Note: sediment and surface water samples were primarily taken along Darby Creek, with a single Muckinipattis Creek sample taken near the convergence of both creeks.) As a health protective approach, we assumed high exposure scenarios. This included an unlikely, "worst case" scenario that all detected total chromium was in its more toxic chromium(VI) form. Based on EPA's 2017-2018 sampling data, dermal and incidental-ingestion exposures to detected Norwood chemicals of concern (CoCs) are not expected to result in adverse non-cancer health effects. The one exception is lead, for which there is no safe level of exposure, particularly in children. Soil pica exposures among young children may result in gastrointestinal effects from the highest copper or iron exposures, or neurological or other effects from lead exposures.

The highest lifetime excess cancer risk was 3 in 10,000 children and 2 in 100,000 adults, based on the maximum benzo[a]pyrene-equivalent PAH residential soil sample. This risk is based on a sample 3.5 times above the next highest sample; we considered residential soil samples as independent from one another. Excess cancer risk is separate from U.S. background cancer risk; on average, approximately 1 in 2 men and 1 in 3 women are at risk of developing cancer during their lifetime (American Cancer Society 2020).

The cancer risk estimates described in this section are based on the maximum detected soil concentrations in residential locations and the 95th Upper Confidence Limit of the mean (95 UCL) concentrations in non-residential locations (or the maximum in non-residential locations if a CoC was detected in fewer than 4 or 20% of samples). They are also based on the smaller percentage of samples overall that exceeded comparison values (CVs). They also assume daily exposures to the highest concentrations at sampled site locations for 20 consecutive years among children (to residential soil; 15 consecutive years at non-residential locations), and 33 consecutive years among adults. Estimated risks would be lower for exposures of shorter duration. Cancer risk estimates are theoretical estimates and not predictions of actual cancer cases. PADOH's current approach for assessing lifetime cancer risk assumes that there is some level of risk associated with exposure to each molecule. While there will always be some risk, the risk increases with the amount of exposure, frequency of exposures, and how many years a person is exposed.

# 6. Chemical Mixtures / Interactions

A discussion on the potential health effects from exposures to multiple Norwood CoCs is presented in Appendix F.

# 7. Health Outcome Data Evaluation

## 7.1 Cancer Registry Review

Cancer was a major concern of the Winona homes community. We reviewed PADOH's Cancer Registry ("registry") incidence data from 1985 – 2019 across 4 time periods (1985-1994; 1995-2004; 2005-2014; 2015-2019) (Note: 2019 is the most recent year of Pennsylvania registry data at the time of this report.) We evaluated which cancers were higher or lower than expected at 1) Norwood, Folcroft and Prospect Park boroughs compared to Delaware County and Pennsylvania and 2) Norwood compared to Pennsylvania. To do so, we assessed age-adjusted Standardized Incidence Ratios (SIRs), comparing rates between men and women. SIRs are used to determine whether observed cancers are higher or lower than expected for a specific community compared to the larger reference community, but they cannot determine the cause for a cancer. SIRs greater than 1.0 indicate that more cancer cases were observed than expected over a defined time period; SIRs less than 1.0 indicate fewer cases were observed than expected. For example, an SIR of 1.20 indicates that observed cancer cases were 20% greater than expected. An SIR of 0.70 indicates that observed cancer cases were 30% less than expected. The 95% confidence interval (CI) surrounding an SIR determines the precision of the SIR estimate. The narrower the CI the more precise the cancer estimate. A CI that does not include 1.0 is considered statistically significant. Whether higher or lower than expected, differences in cancer rates can be due to chance, known risk factors, unknown factors, or other reasons not captured in the data analysis (ATSDR 2017). Statistically significant SIRs are less likely to have occurred by chance, though chance or other risk factors cannot be ruled out.

Multiple risk factors can influence someone's risk of developing cancer, including genetic (e.g., family history), lifestyle (e.g., smoking, diet, physical activity), and environmental factors (e.g., biological, physical, or chemical agents). On average in the U.S., approximately 1 in 2 men and 1 in 3 women are at risk for developing cancer at some point during their lifetime (American Cancer Society 2020). Cancer takes many years to develop before it's diagnosed. Some people with known risk factors may not develop cancer; others may develop cancer even without a known risk factor (ATSDR 2015). Due to these factors and many limitations it is often difficult to understand exactly why one person develops cancer and another person does not (National Cancer Institute 2015). The registry (and SIRs) account for age and sex but no other risk factors.

Table 7 lists the registry incidence results for the 4 assessed time periods between 1985-2019 for the 3 boroughs combined compared to Delaware County. This was done to obtain a sense of the lower Norwood area, specifically. Combining the 3 boroughs as part of the analysis allowed us to examine a greater number of cancer cases and better assess potential trends. SIRs are highlighted in **bold** for cancer types that were higher than expected and statistically significant. In *italics and underlined* are SIRs that were lower than expected and statistically significant. We also examined registry results for the 3 boroughs combined compared to Pennsylvania (Appendix Table G1) and Norwood Borough compared to Pennsylvania (Appendix Table G2).

**Table 7. Age-adjusted standardized incidence rates (SIRs) and 95% confidence intervals (CI) for cancers by sex at Norwood, Prospect Park and Folcroft Boroughs combined compared to Delaware County – 1985-2019**

| Time Period | | 1985 – 1994 | | 1995-2004 | | 2005-2014 | | 2015-2019 | |
|---|---|---|---|---|---|---|---|---|---|
| **Gender** | | **Male** | **Female** | **Male** | **Female** | **Male** | **Female** | **Male** | **Female** |
| **Bladder** | **Exp.** | 32.4 | 13.2 | 39.4 | 15.4 | 36.3 | 13.0 | 16.8 | 6.2 |
| | **Obs.** | 39 | 17 | 46 | 15 | 43 | 17 | 20 | 5 |
| | **SIR** | 1.20 | 1.28 | 1.17 | 0.98 | 1.18 | 1.31 | 1.19 | 0.80 |
| | **95% CI** | 0.86 - 1.65 | 0.75 - 2.06 | 0.86 - 1.56 | 0.55 - 1.61 | 0.86 - 1.59 | 0.76 - 2.10 | 0.73 - 1.84 | 0.26 - 1.87 |
| | | | | | | | | | |
| **Brain** | **Exp.** | 8.5 | 6.6 | 8.8 | 8.5 | 18.3 | 24.1 | 8.9 | 12.5 |
| | **Obs.** | 4 | 5 | 7 | 10 | 21 | 26 | 14 | 8 |
| | **SIR** | 0.47 | 0.76 | 0.79 | 1.18 | 1.15 | 1.08 | 1.57 | 0.64 |
| | **95% CI** | 0.13 - 1.21 | 0.25 - 1.77 | 0.32 - 1.63 | 0.57 - 2.17 | 0.71 - 1.75 | 0.71 - 1.58 | 0.86 - 2.63 | 0.28 - 1.26 |
| | | | | | | | | | |
| **Breast (Female Pop. Only)** | **Exp.** | N/A | 164.7 | N/A | 194.0 | N/A | 199.0 | N/A | 95.3 |
| | **Obs.** | N/A | 168 | N/A | 185 | N/A | 198 | N/A | 82 |
| | **SIR** | N/A | 1.02 | N/A | 0.95 | N/A | 1.00 | N/A | 0.86 |
| | **95% CI** | N/A | 0.87 - 1.19 | N/A | 0.82 - 1.1 | N/A | 0.86 - 1.14 | N/A | 0.68 - 1.07 |
| | | | | | | | | | |
| **Cervix (Female Population Only)** | **Exp.** | N/A | 33.7 | N/A | 11.9 | N/A | 8.7 | N/A | 3.5 |
| | **Obs.** | N/A | 42 | N/A | 16 | N/A | 8 | N/A | 4 |
| | **SIR** | N/A | 1.25 | N/A | 1.35 | N/A | 0.92 | N/A | 1.16 |
| | **95% CI** | N/A | 0.9 - 1.68 | N/A | 0.77 - 2.19 | N/A | 0.4 - 1.81 | N/A | 0.32 - 2.96 |
| | | | | | | | | | |
| **Colon** | **Exp.** | 70.5 | 67.0 | 73.2 | 68.3 | 54.9 | 53.2 | 25.5 | 23.0 |
| | **Obs.** | 75 | 69 | 82 | 77 | 73 | 51 | 37 | 23 |
| | **SIR** | 1.06 | 1.03 | 1.12 | 1.13 | **1.33** | 0.96 | **1.45** | 1.00 |
| | **95% CI** | 0.84 - 1.33 | 0.8 - 1.3 | 0.89 - 1.39 | 0.89 - 1.41 | **1.04 - 1.67** | 0.71 - 1.26 | **1.02 – 2.0** | 0.63 - 1.5 |
| | | | | | | | | | |
| **Esophagus** | **Exp.** | 6.9 | 2.8 | 8.6 | 3.4 | 8.4 | 2.8 | 3.7 | 1.3 |
| | **Obs.** | 7 | 3 | 5 | 2 | 7 | 4 | 2 | 2 |
| | **SIR** | 1.02 | 1.08 | 0.58 | 0.59 | 0.83 | 1.45 | 0.53 | 1.52 |
| | **95% CI** | 0.41 - 2.11 | 0.22 - 3.17 | 0.19 - 1.35 | 0.07 - 2.13 | 0.33 - 1.71 | 0.4 - 3.72 | 0.06 - 1.93 | 0.18 - 5.5 |
| | | | | | | | | | |
| **Hodgkin's Lymphoma** | **Exp.** | 4.4 | 3.6 | 3.9 | 2.9 | 3.8 | 3.2 | 1.8 | 1.4 |
| | **Obs.** | 1 | 2 | 2 | 1 | 3 | 3 | 0 | 5 |
| | **SIR** | 0.23 | 0.56 | 0.51 | 0.35 | 0.79 | 0.94 | - | **3.46** |
| | **95% CI** | 0.01 - 1.27 | 0.07 - 2.02 | 0.06 - 1.85 | 0.01 - 1.92 | 0.16 - 2.32 | 0.19 - 2.74 | - | **1.13 - 8.09** |
| | | | | | | | | | |
| **Kidney** | **Exp.** | 13.6 | 8.1 | 19.1 | 11.4 | 25.6 | 14.8 | 11.6 | 6.4 |
| | **Obs.** | 6 | 5 | 18 | 16 | 22 | 14 | 11 | 1 |

| | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| | **SIR** | *0.44* | 0.61 | 0.94 | 1.40 | 0.86 | 0.95 | 0.95 | 0.16 |
| | **95% CI** | *0.16 - 0.96* | 0.20 - 1.43 | 0.56 - 1.49 | 0.80 - 2.28 | 0.54 - 1.30 | 0.52 - 1.59 | 0.47 - 1.70 | 0 - 0.87 |
| | | | | | | | | | |
| **Laryngeal** | **Exp.** | 10.6 | 3.2 | 10.2 | 2.5 | 7.1 | 1.9 | 3.4 | 0.8 |
| | **Obs.** | 15 | 3 | 13 | 6 | 9 | 3 | 3 | 1 |
| | **SIR** | 1.41 | 0.93 | 1.28 | 2.40 | 1.26 | 1.55 | 0.89 | 1.24 |
| | **95% CI** | 0.79 - 2.32 | 0.19 - 2.72 | 0.68 - 2.19 | 0.88 - 5.22 | 0.58 - 2.4 | 0.32 - 4.52 | 0.18 - 2.60 | 0.03 - 6.90 |
| | | | | | | | | | |
| **Leukemia** | **Exp.** | 11.4 | 9.6 | 14.1 | 11.5 | 15.7 | 11.2 | 7.4 | 5.9 |
| | **Obs.** | 11 | 6 | 10 | 13 | 15 | 13 | 6 | 8 |
| | **SIR** | 0.97 | 0.62 | 0.71 | 1.13 | 0.95 | 1.16 | 0.81 | 1.37 |
| | **95% CI** | 0.48 - 1.73 | 0.23 - 1.35 | 0.34 - 1.30 | 0.60 - 1.94 | 0.53 - 1.57 | 0.62 - 1.98 | 0.30 - 1.76 | 0.59 - 2.69 |
| | | | | | | | | | |
| **Liver** | **Exp.** | 3.6 | 2.2 | 7.8 | 3.0 | 13.6 | 4.9 | 8.1 | 2.5 |
| | **Obs.** | 5 | 1 | 10 | 4 | 15 | 9 | 8 | 2 |
| | **SIR** | 1.41 | 0.45 | 1.28 | 1.32 | 1.10 | 1.83 | 0.99 | 0.80 |
| | **95% CI** | 0.46 - 3.28 | 0.01 - 2.49 | 0.61 - 2.35 | 0.36 - 3.39 | 0.62 - 1.82 | 0.84 - 3.48 | 0.43 - 1.96 | 0.10 - 2.88 |
| | | | | | | | | | |
| **Lung** | **Exp.** | 84.5 | 58.0 | 87.8 | 70.9 | 74.6 | 75.0 | 30.7 | 35.3 |
| | **Obs.** | 110 | 72 | 109 | 102 | 119 | 104 | 56 | 56 |
| | **SIR** | **1.30** | 1.24 | **1.24** | **1.44** | **1.59** | **1.39** | **1.83** | **1.59** |
| | **95% CI** | **1.07 - 1.57** | 0.97 - 1.56 | **1.02 - 1.50** | **1.17 - 1.75** | **1.32 - 1.91** | **1.13 - 1.68** | **1.38 - 2.37** | **1.20 - 2.06** |
| | | | | | | | | | |
| **Melanoma** | **Exp.** | 13.7 | 9.2 | 31.2 | 26.3 | 59.2 | 50.0 | 27.8 | 21.0 |
| | **Obs.** | 17 | 5 | 27 | 22 | 44 | 36 | 27 | 12 |
| | **SIR** | 1.25 | 0.54 | 0.86 | 0.84 | 0.74 | 0.72 | 0.97 | 0.57 |
| | **95% CI** | 0.73 - 1.99 | 0.18 - 1.27 | 0.57 - 1.26 | 0.52 - 1.26 | 0.54- 1.0 | 0.50 -1.0 | 0.64 - 1.41 | 0.29- 1.0 |
| | | | | | | | | | |
| **Myeloma** | **Exp.** | 4.0 | 4.3 | 6.1 | 6.0 | 6.7 | 6.0 | 3.5 | 3.2 |
| | **Obs.** | 3 | 5 | 3 | 5 | 10 | 6 | 1 | 2 |
| | **SIR** | 0.76 | 1.18 | 0.49 | 0.83 | 1.49 | 1.00 | 0.29 | 0.63 |
| | **95% CI** | 0.16 - 2.22 | 0.38 - 2.74 | 0.10 - 1.44 | 0.27 - 1.94 | 0.71 - 2.74 | 0.37 - 2.18 | 0.01 - 1.61 | 0.08 - 2.26 |
| | | | | | | | | | |
| **Non-Hodgkin's Lymphoma** | **Exp.** | 16.5 | 15.2 | 22.4 | 19.1 | 24.1 | 19.7 | 11.3 | 10.2 |
| | **Obs.** | 16 | 18 | 19 | 26 | 24 | 13 | 19 | 10 |
| | **SIR** | 0.97 | 1.19 | 0.85 | 1.36 | 0.99 | 0.66 | **1.68** | 0.98 |
| | **95% CI** | 0.55 - 1.58 | 0.70 - 1.87 | 0.51 - 1.32 | 0.89 - 1.99 | 0.64 - 1.48 | 0.35 - 1.13 | **1.01 - 2.62** | 0.47 - 1.81 |
| | | | | | | | | | |

| | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| **Oral** | **Exp.** | 15.0 | 7.9 | 15.9 | 7.1 | 18.3 | 7.6 | 8.5 | 3.3 |
| | **Obs.** | 23 | 6 | 15 | 8 | 17 | 4 | 14 | 5 |
| | **SIR** | 1.53 | 0.76 | 0.94 | 1.12 | 0.93 | 0.53 | 1.65 | 1.50 |
| | **95% CI** | 0.97 - 2.30 | 0.28 - 1.64 | 0.53 - 1.55 | 0.48 - 2.21 | 0.54 - 1.49 | 0.14 - 1.35 | 0.90 - 2.76 | 0.49 - 3.49 |
| | | | | | | | | | |
| **Ovary (Female Population Only)** | **Exp.** | N/A | 19.3 | N/A | 17.7 | N/A | 16.2 | N/A | 6.8 |
| | **Obs.** | N/A | 14 | N/A | 18 | N/A | 14 | N/A | 11 |
| | **SIR** | N/A | 0.72 | N/A | 1.02 | N/A | 0.87 | N/A | 1.62 |
| | **95% CI** | N/A | 0.40 - 1.21 | N/A | 0.60 - 1.61 | N/A | 0.47 - 1.45 | N/A | 0.81 - 2.90 |
| | | | | | | | | | |
| **Pancreas** | **Exp.** | 8.1 | 9.8 | 11.5 | 11.4 | 13.9 | 13.6 | 7.7 | 7.3 |
| | **Obs.** | 12 | 10 | 8 | 7 | 12 | 15 | 9 | 9 |
| | **SIR** | 1.48 | 1.03 | 0.70 | 0.61 | 0.86 | 1.10 | 1.17 | 1.23 |
| | **95% CI** | 0.77 – 2.59 | 0.49 - 1.89 | 0.30 - 1.37 | 0.25 - 1.26 | 0.45 - 1.51 | 0.62 - 1.82 | 0.54 - 2.23 | 0.56 - 2.33 |
| | | | | | | | | | |
| **Prostate (Male Population Only)** | **Exp.** | 120.2 | N/A | 167.4 | N/A | 157.6 | N/A | 61.3 | N/A |
| | **Obs.** | 115 | N/A | 129 | N/A | 130 | N/A | 49 | N/A |
| | **SIR** | 0.96 | N/A | *0.77* | N/A | *0.82* | N/A | 0.80 | N/A |
| | **95% CI** | 0.79 - 1.15 | N/A | *0.64 - 0.92* | N/A | *0.69 - 0.98* | N/A | 0.59 - 1.06 | N/A |
| | | | | | | | | | |
| **Stomach** | **Exp.** | 11.4 | 6.5 | 10.1 | 6.2 | 8.9 | 5.0 | 4.1 | 2.7 |
| | **Obs.** | 9 | 4 | 13 | 4 | 18 | 3 | 3 | 4 |
| | **SIR** | 0.79 | 0.62 | 1.28 | 0.64 | **2.01** | 0.60 | 0.73 | 1.48 |
| | **95% CI** | 0.36 - 1.49 | 0.17 - 1.58 | 0.68 - 2.19 | 0.17 - 1.64 | **1.19 - 3.18** | 0.12 - 1.75 | 0.15 - 2.13 | 0.4 - 3.8 |
| | | | | | | | | | |
| **Testis (Male Pop. Only)** | **Exp.** | 6.3 | N/A | 6.0 | N/A | 6.4 | N/A | 3.0 | N/A |
| | **Obs.** | 4 | N/A | 8 | N/A | 2 | N/A | 3 | N/A |
| | **SIR** | 0.63 | N/A | 1.33 | N/A | 0.31 | N/A | 1.01 | N/A |
| | **95% CI** | 0.17 - 1.61 | N/A | 0.57 - 2.62 | N/A | 0.04 - 1.14 | N/A | 0.21 - 2.96 | N/A |
| | | | | | | | | | |
| **Thyroid** | **Exp.** | 2.9 | 8.1 | 5.4 | 14.6 | 9.4 | 27.4 | 4.6 | 14.7 |
| | **Obs.** | 2 | 6 | 0 | 10 | 7 | 20 | 6 | 16 |
| | **SIR** | 0.69 | 0.74 | - | 0.69 | 0.74 | 0.73 | 1.31 | 1.09 |
| | **95% CI** | 0.08 - 2.49 | 0.27 - 1.60 | - | 0.33 - 1.26 | 0.30 - 1.53 | 0.45 - 1.13 | 0.48 - 2.84 | 0.62 - 1.77 |
| | | | | | | | | | |
| **Uterus (Female** | **Exp.** | N/A | 25.0 | N/A | 31.7 | N/A | 35.5 | N/A | 18.8 |
| | **Obs.** | N/A | 28 | N/A | 30 | N/A | 36 | N/A | 24 |
| | **SIR** | N/A | 1.12 | N/A | 0.95 | N/A | 1.01 | N/A | 1.27 |

| | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| **Population Only)** | **95% CI** | N/A | 0.75 - 1.62 | N/A | 0.64 - 1.35 | N/A | 0.71 - 1.4 | N/A | 0.82 - 1.9 |
| Exp = Expected, Obs = Observed, SIR = Standardized Incidence Ratio, CI = Confidence Interval, N/A = Not Applicable | | | | | | | | | |

As shown in Table 7, lung cancer was higher than expected and statistically significant for most of the assessed periods, and affected men and women similarly. For the four time periods (1985-1994; 1995-2004; 2005-2014; 2015-2019) assessed between 1985-2019, it was 30%, 24%, 59% and 83% higher than expected in men, and 24%, 44%, 39% and 59% higher than expected in women. Similar statistically significant lung cancer patterns were seen at the 3 boroughs combined compared to Pennsylvania (Appendix G1) as well as at Norwood compared to Pennsylvania (Appendix G2). The remaining cancer types that were higher and statistically significant included colon cancer in men from 2005-2014 (SIR 1.33, 95% CI 1.04-1.67) and 2015-2019 (SIR 1.45, 95% CI 1.02-2.0), stomach cancer in men from 2005-2014 (SIR 2.01, 95% CI 1.19-3.18), Non-Hodgkin's Lymphoma in men from 2015-2019 (SIR 1.68, 95% CI 1.01-2.62), and Hodgkin's Lymphoma in women from 2015-2019 (SIR 3.46, 95% CI 1.13-8.09; Table 7); Hodgkin's Lymphoma was based on a small number of total cases (n=5). Prostate cancer was lower in men and statistically significant from 1995-2004 (SIR 0.77, 95% CI 0.64-0.92) and from 2015-2019 (SIR 0.82, 95% CI 0.69-0.98), as was kidney cancer from 1985-1994 (SIR 0.44, 95% CI 0.16-0.96); kidney cancer was based on a small number of total cases (n=6). Appendix Tables G1 and G2 show additional cancer types that were higher and statistically significant at Norwood or the 3 boroughs compared to Pennsylvania, which were also isolated to one time period or sex and included brain, laryngeal, liver, oral cancer, and melanoma.

Though multiple cancer types were higher and statistically significant, only lung cancer showed a consistent pattern of significance for the time periods assessed. It also affected men and women similarly, lending greater credence to the possibility for an environmental risk factor. As is the case in the U.S., lung cancer is the 3rd most common cancer in the Pennsylvania and leading cause of cancer death (National Cancer Institute n.d.; American Cancer Society 2021). Smoking is by far the biggest risk factor for lung cancer, which is not accounted for in the registry. Various environmental chemicals, mostly in certain occupational settings, have been known to influence lung cancer risk in humans. Typically these risks have been found from regular and long-term inhalation of certain workplace chemicals (e.g., asbestos, silica, arsenic, chromium(VI), or coal products), or ingestion of contaminated drinking water (arsenic). Such workplace exposures represent much higher and/or more frequent than those likely for most members of the general population and Norwood residents. Contaminated drinking water exposures are also unlikely for Norwood residents, who are and have historically been served by a public water system. Other well-known environmental risk factors for lung cancer include exposures to high radon levels and outdoor air pollution.

Several other cancer types such as melanoma, liver, Non-Hodgkin's lymphoma and stomach cancers, were higher and statistically significant during a time period and have environmental in addition to family history, behavioral or other risk factors. For these cancer types, most chemical-based risk has also occurred from certain (higher and more frequent) workplace-based exposures to compounds such as benzene, PCBs, vinyl chloride, trichloroethylene, or lead, or long-term consumption of contaminated drinking water (e.g., arsenic).

For the cancer types that were higher (or lower) and statistically significant, their true exposure or risk factor sources are unknown. PADOH cannot attribute their cause to Norwood CoCs, or attribute these cancers to other risk factors associated with the cancer type, since these risk factors (other than age) are not collected or evaluated in the registry. Demographic, family history, or other differences between the areas of study (Norwood and surrounding boroughs) and the reference areas (Pennsylvania and/or Delaware County) may be contributing to statistical differences for some cancer types.

## 7.2. Multiple Sclerosis

Winona community members identified Multiple Sclerosis (MS), attributed to the former Norwood landfill, as a concern. MS is the most common inflammatory disease of the Central Nervous System (CNS), which consists of the brain, optic nerves, and spinal cord. In MS the immune system mistakenly attacks the myelin sheath that protects critical CNS nerve fibers (NIH 2021). "Multiple Sclerosis" refers to the areas of plaque or scar tissue that remain from the attacks.

MS is most common in young adults (e.g. ages 20 – 40) and more common in women than men (NIH 2021). Although we know some of the risk factors for MS, its true cause is unknown (CDC 2011; National MS Society, n.d.). Research shows that genetic vulnerabilities in combination with environmental factors may cause MS (NIH 2021).

The three main environmental risk factors that have been identified are:

- **Sunlight/vitamin D exposure.** Studies show that people exposed to more sunlight and/or who live closer to the equator are less likely to develop MS. Having higher levels of vitamin D (which can come from sunlight) is also thought to lower risk for MS. Vitamin D may help regulate the immune system (NIH 2021).

- **Prior Epstein-Barr Virus infection (particularly in adolescents)**. MS risk is elevated in people who develop Epstein Barr-Virus (EBV), particularly during adolescence or adulthood. EBV is the virus that causes infectious mononucleosis, and the *type* of exaggerated immune response to EBV may lead to the MS, rather than the virus itself. However, these exact mechanisms are still poorly understood, and there is still no proof that EBV causes MS (NIH 2021).

- **Smoking.** People who smoke are more likely to develop MS and have a more aggressive form of MS, although the exact reasons are unclear (NIH 2021).

Despite continued research on MS and knowledge of these risk factors, its exact cause remains unknown. There are no definitive studies at this time showing that MS is caused by exposure to environmental contamination (EPA 2021) including heavy metals such as lead, mercury and manganese (National MS Society n.d.). This hinders the ability to make any conclusions about the Norwood Chemicals of concern (CoC) in relation to MS, specifically.

Although we cannot make definitive conclusions about MS, we compared CoCs identified from EPA's 2017-2018 sampling data to the scientific literature to see whether ingestion or dermal exposure to these CoCs have induced immune system or neurological effects in toxicological studies. These results are discussed further in Appendix D. **This evaluation was not meant to prove or disprove a CoC's association with MS. Rather, it was to provide information on the types and thresholds of immune and neurological health effects that have been found in human and/or laboratory animal studies.** For instance, some chemicals are known to suppress the immune system, while others can stimulate it. Our evaluation revealed that multiple CoCs have been shown to affect the immune and/or neurological systems but that these effects often occurred at much higher exposures (oral and dermal) than estimated exposure doses at Norwood. **Because of the lack of definitive data on the chemical contributions to MS, we cannot say whether a Norwood CoC caused, or even contributed to, MS.**

The U.S. Centers for Disease Control and Prevention is seeking to better understand the characteristics and causes of MS. As part of this effort and with Congressional funding it has begun developing a National Neurological Conditions Surveillance System (NNCSS) for both Multiple Sclerosis and Parkinson's Disease (CDC 2020a). This initiative is part of an effort to improve MS surveillance, research into its causes, and diagnosis and treatment.

Further details on the CDC NNCSS initiative can be found here:
https://www.cdc.gov/surveillance/neurology/index.html

Additional details on MS from the National Institutes of Health and U.S. Centers for Disease Control (discussed above) can be found here:

- U.S. National Institutes of Health (2022). Multiple Sclerosis: Hope Through Research. Available from: https://www.ninds.nih.gov/Disorders/Patient-Caregiver-Education/Hope-Through-Research/Multiple-Sclerosis-Hope-Through-Research#whatisMS
- U.S. Centers for Disease Control (2011; Appendix C). Multiple Sclerosis Cluster Evaluation in an Inpatient Oncology Ward – Wisconsin. Available from: https://www.cdc.gov/niosh/hhe/reports/pdfs/2011-0047-3143.pdf

## 8. Community Concerns

A discussion of Winona community concerns is presented in Appendix H.

## 9. Child Health Considerations

Children are not small adults, and developing fetuses, infants and children have unique vulnerabilities to environmental contamination. Children breathe more air, drink more water, and eat more food per their body weight than adults. They also crawl and play closer to the ground and regularly engage in behaviors such as mouthing of hands and objects, putting them at risk for exposures of concern. Because children's bodies are rapidly developing, exposure to high levels of certain compounds during critical exposure periods can lead to lasting effects.

Several Norwood chemicals of concern (CoCs) such as DEHP, lead, mercury, and manganese are known to have unique effects on certain childhood health outcomes. As part of our health effects evaluation we considered a few of these critical outcomes (e.g., neurological outcomes) relative to estimated exposures from Norwood soil or sediment. For residential soil we assessed childhood exposure doses starting from birth to 12 months, the age range with the highest estimated exposures. We also considered soil-pica and non-pica scenarios and assumed pica behavior was possible. Overall, based on chemical levels from EPA's 2017-2018 site sampling, it is unlikely that adverse non-cancer effects would occur among children who do not engage in pica-behavior. The only exception to this is for lead, for which no safe screening level exists. Iron, copper and lead were of potential health concern for soil-pica behavior.

Two CoC carcinogens – benzo[a]pyrene and chromium(VI) – are carcinogenic by a mutagenic mode of action, meaning that they can change DNA in a cell. Children are more susceptible to these types of chemicals. ATSDR's Public Health Site Assessment Tool (PHAST) incorporated age-dependent adjustment factors (ADAFs) as part of the excess child lifetime cancer risk estimates for these chemicals. The highest excess cancer risk estimates were 3 in 10,000 for children, based on the maximum residential sample of benzo[a]pyrene-equivalent PAHs at Norwood. This maximum sample was approximately 3.5 times above the next highest benzo[a]pyrene-equivalent sample. The next highest excess cancer risk estimate was 2 in for 10,000 children based on the highest chromium sample. Our chromium assumption assumed that all detected chromium at Norwood was in its more toxic, chromium(VI) form; this is an unlikely scenario, as chromium is more commonly in its less toxic, chromium(III) form in soil.

## 10. Conclusions

After evaluating 2017-2018 chemical concentrations detected in Norwood residential soil, non-residential soil, and Darby and Muckinipattis Creek sediment and surface water, we (PADOH) reached the following conclusions:

**Conclusion 1**
Except for lead for which there is no presumed safe level of exposure, adverse non-cancer health effects are unlikely to occur from dermal and incidental ingestion exposures to detected chemical concentrations at sampled site locations.

*Basis for conclusion*
Lead is a naturally occurring element in the earth's crust and can be found throughout our environment in the air, water, and soil from anthropogenic sources such as fossil fuels, including past use of leaded gasoline, some types of industrial facilities and past use of lead-based paint in homes. Lead at Norwood was lower than that typically found in more urban Pennsylvania areas, such as Philadelphia. At Norwood, 3 of 21 residential and 5 of 17 non-residential samples exceeded a soil lead model threshold level of 245 milligram/kilogram (mg/kg) that could result in a child blood lead level (BLL) of 3.5 microgram/deciliter (µg/dL). While there is no safe blood lead level for children, a BLL of 3.5 µg/dL and above is considered "elevated" according to the PADOH, and children could experience adverse health effects, including nervous system effects, from potential lead exposure.

Regarding other CoCs, the highest exposure doses for 3 chemicals – 1) benzo[a]pyrene-equivalent polycyclic aromatic hydrocarbons (PAHs), 2) di (2-ethylhexyl) phthalate (DEHP), and 3) chromium(VI) – exceeded chronic and/or intermediate health guidelines for young children, but not adults:

- The highest exposure doses for children age 1 year and younger exceeded chronic and intermediate-duration health guidelines for PAHs and DEHP, respectively.
- The highest exposure doses for children ages 0-12 months (to residential soil) and 6-10 years (to Darby Creek sediment) exceeded chronic health guidelines for chromium(VI).

However, the exposure doses for these chemicals were several orders of magnitude (70-270 times) below reported effect levels from which their chronic and intermediate health guidelines are derived. In addition, our chromium(VI) estimate assumed that detected total chromium at Norwood was 100% chromium(VI), a "worst case" and unlikely scenario. Therefore, it is unlikely that children or adults would experience adverse non-cancer health effects from these chemicals.

**Conclusion 2**
A young-child engaging in soil-pica behavior (which is uncommon and involves eating large amount of soil) may experience adverse non-cancer health effects such as gastrointestinal or nervous system effects if consuming the highest detected concentrations of copper, iron, or lead at site sampling locations.

*Basis for conclusion*

In toxicological evaluations for the chemicals listed above, soil-pica exposure scenarios approached or exceeded health effect thresholds. For copper, this conclusion is based on intermediate-duration and single occasion pica behavior involving the highest copper residential soil sample; for iron, the conclusion is based on intermediate-duration pica behavior involving the highest iron residential soil sample. As there is no presumed safe exposure to lead, pica behavior could result in adverse health effects from lead exposure regardless of site location.

Soil-pica behavior involves eating soil and can be found in some children 1-5 years old, though this behavior is uncommon. We assessed an intermediate and single (1 time) soil-pica scenario. Our intermediate-duration pica estimates assume a child consuming 5,000 mg soil (equivalent to 5 packets of artificial sweetener used in coffee or tea) for 3 days per week for up to one year. Our single pica estimate assumes consumption of 5,000 mg in soil once. These are health protective assumptions. Please see Appendix E for a more detailed discussion.

**Conclusion 3**

Community members' long-term exposure to several chemicals of concern (CoCs) that are known or probable human carcinogens poses an increased cancer risk in children and adults.

*Basis for conclusion*

We estimated lifetime excess cancer risk for CoCs that are known or probable human carcinogens and have an EPA or ATSDR-recommended cancer slope factor (CSF). Those CoCs were: benzo[a]pyrene-equivalent PAHs, DEHP, polychlorinated biphenyls (PCBs), aldrin and dieldrin, arsenic, and chromium(VI).

PADOH's current approach for assessing lifetime cancer risk assumes that there's some level of risk associated with exposure to each molecule and that a threshold for cancer effects does not exist. While there will always be some risk, risk increases with the amount of exposure, frequency of exposures, and how many years a person is exposed. Cancer risk estimates are expressed as the proportion of a population that ***may*** be affected by a carcinogen during a lifetime of exposure. For example, an estimated cancer risk of 2 in 100,000 represents ***potentially*** 2 additional cancer cases above expected cases in a population of 100,000 over a lifetime of continuous exposure. Lifetime cancer risk is a theoretical estimate and not a prediction of the number of cancers in a community.

As a health protective approach, we assessed residential cancer risk based on each residential unit with the assumption that residential soil samples were independent from one another. Accordingly, for residential soil exposure, the highest excess cancer risk estimates were 3 in 10,000 for children and 2 in 100,000 for adults, based on the maximum benzo[a]pyrene-

equivalent PAHs detected. These estimates are based on benzo[a]pyrene-equivalent PAHs at a single (maximum) household that had concentrations 3.5 times above the next highest household. Lifetime excess cancer risk based on the next highest benzo[a]pyrene-equivalent PAH sample was 7 in 100,000 for children and 5 in a million for adults.

For the remaining CoCs, the highest residential excess cancer risk estimates were 2 in 10,000 for children (and 2 in 100,000 for adults) based on the highest chromium sample, and 5 in 100,000 for children (and 2 in 100,000 for adults) based on the highest dieldrin sample. Our chromium estimates assumed that all detected total chromium concentrations were chromium(VI). In most soils, total chromium is in its much less toxic, chromium(III) form.

For non-residential exposures, the highest excess cancer risk was 2 in 10,000 for children and 6 in 100,000 for adults based on daily, year-round exposures to creek sediment and a 100% chromium(VI) assumption.

If we assume that maximum detected chromium concentrations are 75% chromium(III) and 25% chromium(VI), a more likely chromium speciation scenario, excess cancer risk from chromium exposure is lower for children and adults for residential (6 in 100,000 for children, 6 in a million for adults) and sediment-based exposures (6 in 100,000 for children, 1 in 100,000 for adults).

For adults, all Norwood cancer risk estimates (whether from residential soil, non-residential soil or creek sediment) assume daily, year-round exposures to a CoC at that location and concentration for 33 consecutive years. For children, *residential* soil estimates assume daily exposure from birth through age 20 years, while *non-residential* soil and sediment estimates assume exposures from ages 6 through 20 years. Estimates would be lower for exposures of shorter duration.

**Conclusion 4**

Age-adjusted cancer data analysis for 1985-2019 did not show consistent patterns for the 22 cancer types analyzed, except for lung cancer. Lung cancer incidence rates were mostly higher and statistically significant for all the four time-periods (1985-1994; 1995-2004; 2005-2014; 2015-2019) for both men and women.

*Basis for Conclusion*

Lung cancer incidence for men at Norwood, Folcroft and Prospect Park Boroughs combined for all four time periods (1985-1994; 1995-2004; 2005-2014; 2015-2019) was statistically significantly higher at 30%, 24%, 59%, and 83%, respectively, than expected compared to Delaware County. For women, except for one time period (1985-1994), lung cancer incidence at these 3 boroughs combined was statistically significantly higher (24%, 44%, 39% and 59%, respectively) than expected when compared to Delaware County.

The cancer registry does not account for smoking, the most common risk factor for lung cancer. Environmental risk factors for lung cancer have typically involved inhalation exposures to radon or certain workplace-based chemicals, or ingestion of contaminated drinking water. Exposure to contaminated drinking water is highly unlikely because community residents are and have historically been served by a public water system.

**Conclusion 5**

There is no registry similar to a cancer registry to evaluate Multiple Sclerosis (MS). For identified CoCs, exposure doses were below thresholds that induced neurological or immune system effects in human or laboratory animal studies.

*Basis for conclusion*

Due to community concerns regarding MS, we compared CoC daily exposure estimates to levels in human and or laboratory animal studies that have found immune system and neurological effects. There is no registry to evaluate MS. In addition, while researchers have identified several risk factors for MS, its exact cause remains unknown and there are currently no definitive data showing that MS is caused by environmental contamination. **Our evaluation was not meant to prove or disprove a CoC's association with MS. Rather, it was to provide information on the types and thresholds of immune and neurological health effects that have been found for Norwood CoCs from human and/or laboratory animal studies.** For CoCs for which these data were available, exposure doses were below health effect levels identified in scientific studies.

## 11. Limitations

Our analysis has several assumptions/limitations:

- Our conclusions are based on single soil, sediment and surface water samples taken between 2017-2018. They cannot be extrapolated to past concentrations or account for possible fluctuation or variance in concentrations. Sampling from different parts of a residential yard could have produced a different result. In 2018, EPA sampled residential soil from 0-12.'' ATSDR notes that ideally, surface soil should be sampled at depths of 0-3'' (ATSDR 2005). For surface water, our conclusions are based on the amount of inorganic metals detected, as organic compounds were not sampled.
- Our conclusions for chromium assume that total chromium concentrations at Norwood were 100% chromium(VI), the more toxic form of chromium. Without chromium speciation, the proportion of chromium(VI) or chromium(III) concentrations at Norwood is uncertain, and by extension, refined estimates of cancer and non-cancer risk. In most soils, chromium is in its much less toxic, chromium(III) form.

- Although we estimated exposures to residential soil, non-residential soil, and Darby and Muckinipattis Creek sediment and surface water, our estimates don't account for movement *between* these locations. We assumed maximum exposure at one of these four locations. If someone frequently traversed to and from one location to another (e.g., residential soil to creek sediment), daily exposures for each distinct location would presumably be lower. As noted, a majority (7) of the 8 creek samples were taken in Darby Creek.
- We evaluated a daily, 12 week creek swimming scenario and year-round, 10-year wading scenario. Exposures would be higher for someone engaging in these activities for a longer duration.
- We included "J" data values in our screening and analysis. "J" values were considered detected (as opposed to non-detected), and these values were considered part of exposure estimates and concentration ranges (e.g. minimums and maximums). J values indicate that the chemical was present in the field sample but its concentration is an estimate; the true concentration may be higher or lower. Only two *maximum* CoC J values – one for DEHP in residential soil and one for manganese in Darby Creek sediment – were featured in our health effects evaluations.
- We were not able to estimate quantitative cancer risk from mercury or lead exposures, which are considered possible and probable human carcinogens but lack a cancer slope factor.
- Our cancer incidence analysis does not account for other contributors to cancer such as genetic pre-disposition, occupational exposures or other environmental exposures such as to radon, residential history, behaviors, and diet; and whether incidence rates are related to the former Norwood landfill.
- Currently, there is no registry available to assess Norwood MS rates, and the causes and risk factors for MS are not well understood.
- Our exposure estimates assume combined incidental ingestion and dermal exposures only (e.g., not exposures by inhalation, ingestion from fish or crops, or soil vapor intrusion).
- This analysis is based on 2017-2018 results, which assessed soil at 21 homes and did not assess soil or sediment at the former Old Norwood Dump, along Muckinipattis Creek nearer to the former Muckinipattis Wastewater Treatment Plant, or at Norwood Borough Park. We agree with EPA's decision to expand sampling to these and other site locations, and to sample more media (e.g., groundwater, deep soil) and residential locations (70 total), which they completed in 2020. We will assess EPA's 2020 expanded sampling results, released in December 2021, as an addendum to this report.

## 12. Recommendations

Based on the findings of this Health Consultation, we recommend that:

1. Parents monitor the outdoor behavior of their children (ages 1 to 5 years old) if the child is suspected of engaging in soil-pica activity.
2. Crop uptake from chemicals found in soil is likely to be minimal; however, to reduce potential exposure to soil chemicals when gardening, PADOH suggests adhering to EPA's suggested best practices such as using raised garden beds and pots filled with clean soil, mixing additional compost into in-ground gardens, and washing produce, peeling root crops, and removing outer leaves of leafy vegetables before eating.
3. To reduce or eliminate exposure to lead from soil, residents should:
    a. Remove shoes before entering the house to prevent bringing lead-contaminated soil from outside.
    b. Avoid allowing their children to play in bare soil (e.g., if possible, use sandboxes).
    c. Plant grass (if possible) on any bare soil, or cover the soil with seed, mulch, or wood (CDC, 2022a).
    d. Have children under 6 tested for lead poisoning via a simple blood test. PADOH has a lead information line (1-800-440-LEAD) to respond to questions about lead poisoning and other environmental hazards.
4. Whenever possible, residents avoid or limit additional potential lead exposure sources, such as old or imported toys that may still contain lead-based paint, certain imported consumer products (e.g., some jewelry, cosmetics, candies, or spices), or certain hobbies in which lead exposure can occur. If engaging in hobbies or certain occupations in which lead exposure is common, efforts should be made to avoid tracking it into the home from clothing or equipment. Any renovation of homes containing lead-based paint should be done by a qualified lead abatement professional. Additional information on lead exposure sources can be found in this report.
5. EPA consider additional sampling of the site that speciates chromium valence form, and sample in residential areas closer to the surface (e.g., 0-3'') than it did in its 2017-2018 sampling (0-12'').

## Next Steps

- PADOH will present the findings of this HC and provide health education outreach to the lower Norwood community.
- PADOH will assess EPA's expanded 2020 Norwood sampling results as an addendum.
- PADOH will continue to assist site stakeholders when requested to evaluate additional environmental or health data from the site or surrounding communities.

## Report Preparation

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# Appendix A. EPA's EJ Screen Report (Version 2020) for mile radius surrounding former Norwood Landfill

**Figure A1. EPA's EJScreen Report for 1 mile radius surrounding approximate Norwood Landfill area**

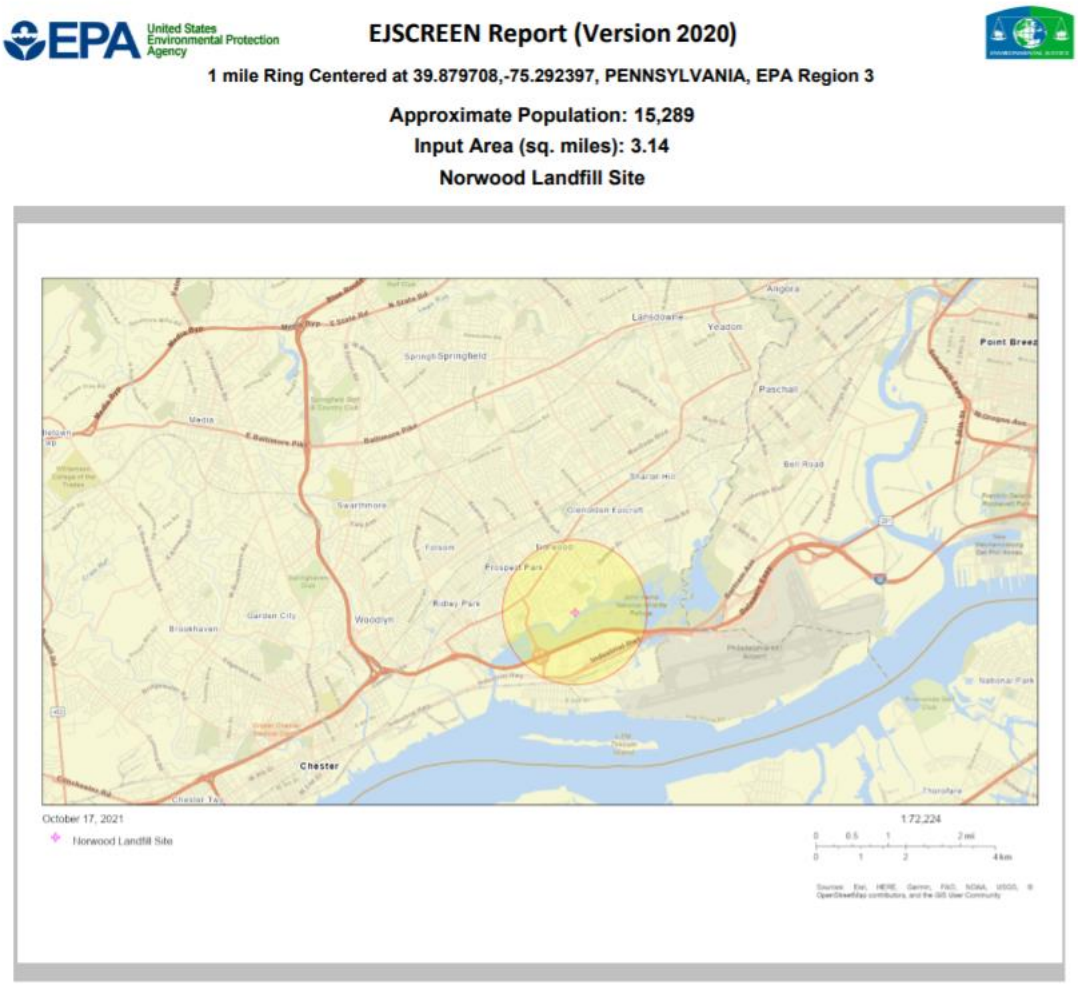


**Table A1. Environmental Justice (EJ) percentiles for the 1 mile radius surrounding the former Norwood Landfill**

| Selected Variables | State Percentile | EPA Region 3 Percentile | USA Percentile |
|---|---|---|---|
| Norwood Landfill (1 mile radius) EJ Percentiles for: | | | |
| Particulate Matter (PM 2.5) | 65 | 54 | 43 |
| Ozone | 65 | 55 | 44 |
| NATA Diesel PM | 47 | 40 | 30 |
| NATA Air Toxics Cancer Risk | 60 | 51 | 41 |
| NATA Respiratory Hazard Index | 58 | 50 | 41 |
| Traffic Proximity and Volume | 11 | 10 | 7 |

| | | | |
|---|---|---|---|
| Lead Paint Indicator | 41 | 24 | 14 |
| Superfund Proximity | 27 | 18 | 12 |
| RMP Proximity | 41 | 27 | 23 |
| Hazardous Waste Proximity | 20 | 18 | 17 |
| Wastewater Discharge Indicator | 9 | 6 | 5 |
| NATA = National Air Toxics Assessment; RMP = Proximity to Risk Management Plan Sites; EJ = Environmental Justice indexes. EJ indexes are calculated by combining environmental and demographic information for a place. | | | |

**Table A2. Environmental and Demographic Percentiles for the 1 mile surrounding the former Norwood landfill**

| **Selected Variables** | **State Percentile** | **EPA Region 3 Percentile** | **USA Percentile** |
|---|---|---|---|
| Norwood Landfill (1 mile radius) Environmental Indicators | | | |
| Particulate Matter (PM 2.5 in $\mu gm^3$) | 40 | 75 | 74 |
| Ozone (ppb) | 54 | 59 | 61 |
| NATA Diesel PM ($\mu gm^{3)}$ | 76 | 70-80th | 70-80th |
| NATA Air Toxics Cancer Risk (lifetime risk per million) | 72 | 70-80th | 60-70th |
| NATA Respiratory Hazard Index | 81 | 70-80th | 50-60th |
| Traffic Proximity and Volume (daily traffic count/distance to road) | 85 | 82 | 82 |
| Lead Paint Indicator (% Pre-1960 Housing) | 71 | 82 | 87 |
| Superfund Proximity (site count/site distance) | 92 | 94 | 95 |
| RMP Proximity (facility count/km distance) | 71 | 78 | 74 |
| Hazardous Waste (facility count/km distance) | 83 | 79 | 73 |
| Wastewater Discharge Indicator (toxicity-weighted concentration/m distance) | 86 | 90 | 90 |
| Norwood Landfill (1 mile radius) Demographic Indicators | | | |
| Demographic Index | 61 | 48 | 37 |
| People of Color Population | 65 | 47 | 38 |
| Low Income Population | 51 | 54 | 44 |
| Linguistically Isolated Population | 72 | 69 | 57 |
| Population with Less than High School Education | 48 | 47 | 42 |
| Population Under 5 Years of Age | 72 | 68 | 64 |
| Population Over 64 Years of Age | 27 | 36 | 42 |
| NATA = National Air Toxics Assessment; RMP = Risk Management Plan<br>#Based on EPA 2019 National Air Toxics Assessments (NATA); EJ = Environmental Justice indexes are calculated by combining environmental and demographic information for a place. | | | |

## Appendix A Reference:

EPA (2020). Environmental Justice Screening and Mapping Tool (Version 2020). https://ejscreen.epa.gov/mapper/. Accessed October 17, 2021.

# Appendix B. Process for Evaluating Exposures and Health Effects from Norwood Chemicals of Concern (CoCs), based on EPA's 2017-2018 Environmental Sampling Data

## B1. Exposure Assumptions

We assessed EPA's data using the Agency for Toxic Substances and Disease Registry's (ATSDR)'s Public Health Site Assessment Tool (PHAST). Using PHAST, we calculated combined dermal and incidental ingestion exposure estimates (also known as "exposure doses") for each Chemical of Concern (CoC). An exposure dose is an estimate of the amount of a chemical that enters a person's body over a specific period of time (ATSDR 2017). Exposure doses encompass multiple factors, including incidental intake rates, chemical absorption into the body, chemical and soil adherence to the skin, skin surface areas, and differences in body weight and age. We used PHAST's default assumptions for these variables (ATSDR 2018a, 2018b, 2016). Our exposure dose estimates for adults also applied to adult gardeners, who are assumed a default soil incidental intake rate of 100 mg/day. The adult gardening scenario does not account for consumed crops grown in Norwood soil, as crops were not evaluated.

All estimated exposure doses assumed a Reasonable Maximum Exposure (RME) scenario. RME refers to people at the high end of the exposure distribution (approximately the 95$^{th}$ percentile). RME doses represent exposures that are higher than average but still within a realistic range (ATSDR 2005). Our chronic estimated exposure doses assume year-round exposure to the detected chemical in soil, sediment, or surface water, though for Norwood residents, chronic exposures to residential soil (as opposed to the other media) are most applicable.

Equations for combined soil ingestion and dermal exposures to Norwood soil and Darby and Muckinipattis Creek sediment and surface water are shown in this Appendix section B2, with examples provided in Appendix section B5.

The exposure doses account for children (<21 years) and adults (21 years and older). For non-pica behavior, children up to 12 months had the highest combined dermal-ingestion exposure doses of all childhood ages. Although we assessed a 0-12 month child exposure scenario to residential soil, we assumed that children as young as 0-12 months were **not** regularly exposed to non-residential soil or to Darby or Muckinipattis Creek sediment or surface water; for these locations, the youngest childhood ages we considered were 6-10 years.

***Soil-pica exposure assumptions.*** Soil-pica involves eating soil. Although uncommon, it is most likely to occur in preschool children, with estimates between 4 and 20% of preschool children exhibiting soil-pica behavior (ATSDR 2018a). Children ages 1-2 years have the greatest tendency to exhibit this behavior, which diminishes as they become older. As a health protective approach, we assumed that pica-behavior could occur at the site. We calculated soil-pica estimates for children 1 to 5 years of age. We considered an intermediate-duration and single

occasion soil-pica scenario. An intermediate (15-364 day) soil-pica scenario assumes a child intake of 5,000 mg soil for 3 of 7 days per week for up to a year. This is likely a rare scenario because recurrent soil-pica behavior is not well characterized (ATSDR 2001). A 5,000 mg soil intake per pica event is an estimate of average ingested soil based on very few soil ingestion studies (ATSDR 2001), and this uncommon scenario may not represent true exposure. 5,000 mg of soil is equivalent to 5 packets of artificial sweeteners used for coffee or tea.

For residential soil, we evaluated an intermediate and single soil-pica scenario, with the assumption that although rare, an intermediate soil-pica scenario of 3 days per week could still occur. For non-residential soil and creek sediment, we evaluated a single occasion soil-pica scenario.

## B2. Equations for estimating incidental ingestion and dermal exposures at Norwood

Soil ingestion can occur by the inadvertent consumption of soil on hands or food items, mouthing of objects, or the ingestion of unusually high amounts of soil (e.g., soil-pica; ATSDR 2005). All children mouth or ingest non-food items to some extent (ATSDR 2005). Residential and recreational areas provide access for soil incidental ingestion exposure. Soil tracked into homes can also become indoor dust particles, leading to exposure. Our soil and sediment estimates assumed that soil/sediment could be tracked into the home.

Incidental ingestion and dermal exposures were calculated using equations below and summed in PHAST to generate daily exposure estimates.

### B.2.1 Estimated ingestion exposures from soil and sediment were calculated as follows (ATSDR 2018a):

**D = (C x IR x EF x AF x CF) / BW**

where,

- **D =** estimated exposure, or exposure dose, in milligrams per kilogram body weight per day (mg/kg/day)
- **C =** chemical concentration (mg/kg)
- **IR =** intake rate of contaminated soil (mg/day). Default soil intake rates under a Reasonable Maximum Exposure Scenario are:
    i. 100 mg/day (adult soil intake rate)
    ii. 150 mg/day (child ages birth to <1 year intake rate)
    iii. 200 mg/day (children ages 1-10 years' soil intake rate)
    iv. 100 mg/day (child 11-20 years' soil intake rate)
    v. 5,000 mg/day (child pica soil intake rate estimate)
- **EF =** exposure factor (unitless); the EF accounts for frequency of exposure (e.g., days per year). In many instances, the EF will equal 1, representing a daily exposure to the chemical (ATSDR 2005).
- **CF =** Conversion factor ($10^{-6}$ kg/mg)

- **AF** = Bioavailability factor, which represents, as a percent, the total amount of an ingested substance that actually enters the bloodstream and is available to possibly harm a person
- **BW =** Body weight (kg)
    i. For a child: birth to <1 year: 7.8 kg
    ii. For an adult: 80 kg

**B.2.2. Estimated dermal exposures from soil and sediment contact were calculated as follows:**

**$D = (C \times EF \times CF \times AF \times ABS_d \times SA) / BW * ABSGI$**

**where,**

- **C =** chemical concentration (mg/kg)
- **D =** estimated absorbed dermal dose, in milligrams per kilogram body weight per day (mg/kg/day)
- **EF =** exposure factor (unitless); the EF accounts for frequency of exposure (e.g., days per year). In many instances, the EF will equal 1, representing a daily exposure to the chemical (ATSDR 2005).
- **CF =** Conversion factor for mg to kg ($1x10^{-6}$ kg/mg)
- **AF =** Adherence factor of soil/sediment to skin ($mg/cm^2$ per event)
- **$ABS_d$** = Dermal absorption fraction for soil and sediment
- **SA** = Surface Area available for contact ($cm^2$)
- **BW =** Body weight (kg)
- **ABSGI =** the fraction of chemical absorbed by the gastrointestinal tract, based on dermal exposure

**B.2.3. Equations for estimating exposures from Creek Surface Water**

For CoCs detected in creek surface water, we evaluated a seasonal swimming and year-round wading scenario. A swimming scenario assumes that incidental ingestion and dermal exposures occur; a wading scenario assumes that only dermal exposure occurs. A swimming scenario assumes a single one hour per day swim, 7 days per week, for 12 weeks of the year. A wading scenario assumes a 1 hour per day wading session, 7 days per week, year-round for 10 years. Both assumptions are health protective estimates.

Per ATSDR methodology, chronic-based exposures for swimming scenarios are only assessed for year-round swimming in warm climates (e.g., California, Florida, Puerto Rico). For other climates, only acute or intermediate exposure scenarios are evaluated because swimming exposures are more intermittent (ATSDR 2018b). As a result, we considered intermediate and acute health effects from a 12-week swimming scenario. Because we did not consider year-round exposures, cancer risks were not calculated for the swimming scenario.

Our surface water exposure estimates pertain only to inorganic metals, as organic compounds were not sampled.

**Estimated exposures for a creek <u>swimming</u> scenario (accounting for dermal and ingestion exposures), were calculated as follows (ATSDR 2018b):**

**$D = (C \times IR \times t_{event} \times EV \times EF) / BW$**

Where,

- D = Exposure Dose (mg/kg/day)
- C = Chemical Concentration (mg/L)
- IR = Intake Rate (L/hr),
- $t_{event}$ = Event Duration (hr/event)
- EV = Event Frequency (events/day)
- EF = Exposure Factor (unitless)
- BW = Body Weight (kg)

**Estimated exposures for a creek <u>wading</u> scenario (dermal exposures only), were calculated as follows:**

$ADD = (DA_{event} \times SA \times EV \times EF) / (BW \times ABS_{GI})$, where,

- ADD = Administered Dermal Dose (mg/kg/day)
- $DA_{event}$ = Absorbed Dose per Event ($mg/cm^2$/event)
- SA = Surface Area Available for Contact ($cm^2$)
- EV = Event Frequency (events/day)
- EF = Exposure Factor (unitless)
- BW = Body Weight (kg)
- $ABS_{GI}$ = Gastrointestinal Absorption Factor (unitless)

## B3. Evaluation of Potential Health Effects - Non-cancer

Estimated exposure doses were calculated using ATSDR's Public Health Site Assessment Tool (PHAST) from equations detailed in Appendix Section B2. Estimates were then compared to ATSDR Minimal Risk Levels (MRLs) or EPA's oral Reference Doses (RfDs).

MRLs and RfDs are estimates of the amount of a chemical a person can eat, drink, or breathe each day without experiencing an appreciable adverse risk to health (ATSDR 2018c). These estimates are usually expressed in milligram per kilogram of body weight per day. ATSDR sets MRLs based on exposures of acute (1-14 day), intermediate (15-364 days) or chronic (1 year or longer) duration. EPA RfDs are daily exposure estimates over the course of a lifetime. MRLs and RfDs refer to non-cancer effects and are health protective estimates. Often, but not always, laboratory animal studies are used to derive MRLs and RfDs because relevant human studies are lacking (ATSDR 2018c); additionally, daily exposure to a chemical can be better assessed in a laboratory setting involving experimental animals, such as rats or mice. MRLs and RfDs are

often set hundredfold below levels shown to be non-toxic in animals (ATSDR 2018c). In deriving MRLs or RfDs, ATSDR and EPA apply uncertainty factors (UFs) meant to be protective for several factors, such as use of an animal study to derive the MRL, differences in human variability (e.g., children versus adults), and other factors. MRLs and RfDs undergo extensive scientific peer review.

For each CoC, we compared the highest incidental ingestion and dermal combined exposure doses to the CoC's MRL or RfD to produce a Hazard Quotient (HQ). A HQ less than 1 indicates that the exposure estimate is below the MRL of RfD, and non-cancer health effects are unlikely to occur. If an HQ exceeded 1, we conducted a toxicological evaluation of the "principal" study or studies used to derive the MRL or RfD to determine the possibility for adverse non-cancer health effects. In these circumstances we also compared Norwood estimates (in µg/kg/day) to levels of significant exposure (LSE), which are compiled in ATSDR's toxicological or EPA's Integrated Risk Information System (IRIS) profiles. If no MRL or RfD was available, we also compared Norwood exposure doses to reported LSEs. **Comparing site-specific exposures (or "exposure doses") to MRLs and LSEs is the primary basis for determining whether estimated exposures are likely to harm human health (ATSDR 2017).** For each CoC we considered acute (1-14 day), intermediate (15-364 day) and chronic (1 year or longer) duration exposures, where data was available.

### B4. Process for Evaluating Excess Cancer Risk

To evaluate excess cancer risk for each known or probable CoC carcinogen, we multiplied combined dermal and ingestion exposure estimates by EPA's oral Cancer Slope Factor (CSF) and divided it by age-specific exposure duration and lifetime years (78 years). This equation is displayed below. (Note: for two CoCs, benzo[a]pyrene and chromium(VI), we used ATSDR recommended CSFs, which are more health protective than EPA's CSF.)

*Excess Cancer Risk calculation based on Norwood exposure doses:*

$CR = (D \times CSF) \times (ED / LY)$

Where,

- CR = Cancer Risk
- D = Age-Specific Exposure Dose (mg/kg/day)
- CSF = Cancer Slope Factor $(mg/kg/day)^{-1}$
- ED = Age-Specific Exposure Duration (years),
- LY = Lifetime in Years (78 years)

Under quantitative cancer risk assessment methodology, cancer risk estimates are expressed as a probability. They are expressed as the proportion of a population that may be affected by a carcinogen during a lifetime of exposure (24 hours/day, 365 days/year, for life) to the amount of a carcinogen specified. They represent "excess" or "increased" population cancer risk in

respect to background cancer risk. U.S. background cancer risk is relatively high; on average, approximately 1 in 2 men and 1 in 3 women are at risk for developing cancer over the course of their lifetimes (American Cancer Society, 2020). Using quantitative risk assessment methodology, an estimated cancer risk of 2 in one million represents potentially two additional cancer cases above expected cases in a population of one million over a lifetime of continuous exposure.

Lifetime excess cancer risk is a theoretical value of the proportion of the population that may be affected during a lifetime of exposure to a specific chemical (ATSDR 2005). It is not a prediction of the number of cancer cases in a community. Estimates greater than one in a million are reviewed as part of the toxicological evaluation (ATSDR 2005). Excess cancer risk is a health protective estimate; the actual (true) risk is unknown, but may be substantially lower, perhaps by several orders of magnitude (ATSDR 2017).

Our calculated risk estimates for soil or sediment-based exposures are health protective estimates. For children, they assume up to 20 consecutive years (from birth up through age 20) at a given residence. For adults, they assume living at a residence for 33 consecutive years, which is the U.S. 95$^{th}$ percentile for residential occupancy. As a health protective approach, we applied these scenarios to non-residential locations too, although such daily, regular exposures are unlikely. For childhood cancer risk estimates at these non-residential locations, we assessed age 6 up through age 20 years, as we presumed children younger than 6 years would not be regularly exposed to these media. However, Appendix D of this report also provides excess cancer risk estimates for children from *birth* up through age 20 years where applicable, despite this unlikely daily exposure scenario at non-residential locations for children younger than 6.

## B5. Sample Calculations

### B.5.1 Example 1 – non-cancer and cancer health effect estimates based on the highest residential arsenic soil concentration (9.7 mg/kg).

*Example:* Assessing chronic (year-round) dermal and incidental ingestion exposures based on the maximum residential soil sample for arsenic, of 9.7 mg/kg. Note: for arsenic in soil, a relative bioavailability (RBA) of 0.6 is used as part of the ingestion equation (ATSDR 2018a). Input parameters are shown in Table B.5.1.1 below and derived from ATSDR (2016, 2018a), using exposure parameter guidance from EPA (2004, 2011).

**Table B.5.1.1 Input parameters for children and adults - Soil and Sediment Exposures**

| Parameter | Abbreviation | Assessed for | Children (birth to 1 year) | Adults (21 or older) |
|---|---|---|---|---|
| Soil intake rate | IR | Ingestion | 150 mg/day | 100 mg/day |
| Body Weight | BW | Ingestion/Dermal | 7.8 kg | 80 kg |
| Relative Bioavailability factor for arsenic | RBA | Ingestion/Dermal | 0.6 | 0.6 |

| | | | | |
|---|---|---|---|---|
| Exposure Factor (1 = daily exposures) | EF | Ingestion/Dermal | 1 | 1 |
| Conversion Factor (CF) (mg to kg) | CF | Ingestion/Dermal | $1x10^{-6}$ | $1x10^{-6}$ |
| Gastrointestinal Absorption Factor based on dermal arsenic exposure | $ABS_{GI}$ | Dermal | 1 | 1 |
| Adherence Factor of a chemical to skin | AF | Dermal | 0.2 mg/$cm^2$ | 0.07 mg/$cm^2$ |
| Skin surface area | SA | Dermal | 1,772 $cm^2$ | 6,030 $cm^2$ |
| Dermal Absorption Factor for soil and sediment | $ABS_d$ | Dermal | 0.03 | 0.03 |
| Cancer slope factor for arsenic | CSF | Cancer Risk | $(1.5 mg/kg)^{-1}$ | $(1.5 mg/kg)^{-1}$ |
| Exposure duration | ED | Cancer Risk | 1 | 33* |
| Lifetime Years | LY | Cancer Risk | 78 | 78 |
| *95th percentile for occupancy period (EPA 2011)<br>Sources: ATSDR 2018a; ATSDR 2016; EPA 2011; EPA 2004. | | | | |

### *B.5.1.2 Child (birth to <1 year) estimated exposure dose and cancer risk estimate based on highest residential arsenic soil concentration (9.7 mg/kg)*

***Ingestion exposure to highest residential arsenic concentration at Norwood (9.7 mg/kg)***

$$\text{Dose} = \frac{\text{(Concentration x Intake Rate x Exposure Factor x Relative Bioavailability Factor x Conversion Factor)}}{\text{Body Weight}}$$

$$D = \frac{(C \times IR \times EF \times RBA \times CF)}{BW}$$

$$\text{Child, birth to 1 year} = \frac{(9.7\ mg/kg \times 150\ mg/day \times 1 \times 0.6 \times 1x10^{-6})}{7.8\ kg}$$

= 0.00011 mg/kg/day x (1000 ug/kg) **= 0.11 µg/kg/day**

***Dermal exposure to highest arsenic concentration at Norwood (9.7 mg/kg)***

$$\text{Dose} = \frac{\text{(Concentration x Exposure Factor x Conversion Factor x Adherence Factor x Dermal Absorption Factor x Skin Surface Area)}}{\text{Body Weight * Gastrointestinal Absorption Factor from dermal arsenic}}$$

$$D = \frac{(C \times EF \times CF \times AF \times ABS_d \times SA)}{BW \times ABSGI}$$

$$\text{Child, birth to <1 year} = \frac{(9.7mg/kg * 1 * 1x10^{-6} \times 0.2\ mg/cm^2 \times 0.03 \times 1{,}772\ cm^2)}{7.8kg \times 1}$$

= 0.000013 mg/kg/day x (1000 ug/mg) = **0.013 µg/kg/day**

***Child (birth to <1 year) combined ingestion and dermal exposures***

= exposure (ingestion) + exposure (dermal)
= 0.11 µg/kg/day + 0.013 µg/kg/day = **0.13 µg/kg/day**

***Child Cancer Risk Estimate***

Cancer Risk = (Dose x Arsenic Cancer Slope Factor) x (Exposure Duration / Lifetime Years)

Cancer Risk = (D x CSF) x (ED / LY)

Ingestion Cancer risk = $((0.00011 \text{ mg/kg/day} \times 1.5 \text{ mg/kg/day})^{-1}) \times (1/78) = 2.2 \times 10^{-6}$ , or 2 excess cases per 1 million people

Dermal Cancer risk = $((0.000013 \text{ mg/kg/day} \times 1.5 \text{mg/kg/day})^{-1}) \times (1/78) = 2.5 \times 10^{-7}$, or 3 excess cases per 10 million people

Combined Cancer Risk (CR) = CR (ingestion) + CR (dermal)

= $2.2 \times 10^{-6} + 2.5 \times 10^{-7} = 2.4 \times 10^{-6}$ or **2 excess cases per 1 million people***

***Note:** CDC's Public Health Assessment Site Tool (PHAST), estimates child excess cancer risk for all children under the age of 21, based on combined cancer risk each separate age group (birth to <1 year, 1 to <2 years, 2 to <6 years, 6 to <11 years, 11 to <16, and 16 to <21) to encompass all ages up to age 21**.** When combining childhood excess cancer risk for each age group in addition to children up to age 1, using the formulas of this section, the total combined excess cancer risk for children is **$1.7 \times 10^{-5}$, or 2 excess cases per 100,000 people.**

### *B.5.1.3 Adult (21 and up) estimated exposure dose and cancer risk estimate based on highest residential arsenic soil concentration (9.7 mg/kg)*

***Adult ingestion exposure to highest residential arsenic concentration at Norwood (9.7 mg/kg)***

$$\text{Dose} = \frac{\text{(Concentration x Intake Rate x Exposure Factor x Relative Bioavailability Factor x Conversion Factor)}}{\text{Body Weight}}$$

$$D = \frac{\text{(C x IR x EF x RBA x CF)}}{\text{BW}}$$

$$\text{Adult (21 and up)} = \frac{\text{(9.7 mg/kg x 100 mg/day x 1 x 0.60 x } 1 \times 10^{-6})}{\text{80kg}}$$

= 0.000073 mg/kg/day x (1000 µg/kg) **= 0.0073 µg/kg/day**

***Adult dermal exposure to highest residential arsenic concentration at Norwood (9.7 mg/kg)***

$$\text{Dose} = \frac{\text{(Concentration x Exposure Factor x Conversion Factor x Adherence Factor x Dermal Absorption Factor x Skin Surface Area)}}{\text{Body Weight * Gastrointestinal Absorption Factor from dermal arsenic}}$$

$$D = \frac{\text{(C x EF x CF x AF x ABSd x SA)}}{\text{BW x ABSGI}}$$

$$\text{Adult (21 and up)} = \frac{\text{(9.7 mg/kg x 1 x } 1 \times 10^{-6} \text{ x 0.07mg/cm}^2 \text{ x 0.03 x 6,030cm}^2)}{\text{80kg x 1}}$$

= 0.000015 mg/kg/day x (1000 µg/kg) **= 0.0015 µg/kg/day**

***Adult combined ingestion and dermal exposures***

= exposure (ingestion) + exposure (dermal)

= 0.0073 µg/kg/day + 0.0015 µg/kg/day = **0.0088 µg/kg/day**

***Adult cancer Risk Estimate***

Cancer Risk = (Dose x Arsenic Cancer Slope Factor) x (Exposure Duration / Lifetime Years)

Cancer Risk = (D x CSF) x (ED / LY)

Ingestion Cancer risk = $((0.000073 \text{ mg/kg/day} \times 1.5 \text{ mg/kg/day})^{-1}) \times (33/78) = 4.6 \times 10^{-6}$ , or 5 excess cases per 1 million people

Dermal Cancer risk = $((0.000015 \text{ mg/kg/day} \times 1.\ 5\text{mg/kg/day})^{-1}) \times (33/78) = 9.7\text{x}10^{-7}$, or 10 excess cases per 10 million people

Combined Cancer Risk (CR) = CR (ingestion) + CR (dermal)

= $4.6\text{x}10^{-6} + 9.7\text{x}10^{-7}$ = **$5.6\text{x}10^{-6}$ or 6 excess cases per 1 million people**

**Table B.5.1.4 Summary of Cancer and Non-cancer health effects for children and adults based on the highest residential soil sample (9.7 mg/kg)**

| Sample type, and Conc | Exposed Population and Time Period | Estimated Ingestion and Dermal Exposure Dose (ED) µg/kg/day | Chronic Hazard Quotient<br><br>MRL: 0.3 | Acute Hazard Quotient<br><br>MRL: 5 | Excess Cancer Risk Estimate |
|---|---|---|---|---|---|
| Residential 9.7 mg/kg, or 9,700 µg/kg (highest) | Adult | 0.0088 | <1 | <1 | 6 in 1,000,000 |
| | Child (birth to <1y) | 0.13 | <1 | <1 | 2 in 100,000[1] |

Conc = Concentration in surface soil; MRL = Minimal Risk Level; y = year of age. MRLs are expressed in µg/kg/day. There is no intermediate MRL for arsenic.

[1] Combined cancer risk for children up to 21 years old

## B.5.2 Example 2 – non-cancer and cancer health effect estimates based on the highest creek chromium concentration (found in Darby Creek), under a swimming and wading scenario. Assumes that the maximum creek sample of 24.3 µg/L is 100% chromium(VI), an unlikely scenario.

**Table B.5.2. Input parameters for children and adults – Surface Water Exposures based on highest chromium concentration, assuming total chromium detected is 100% chromium(VI)***

| Parameter | Abbreviation | Assessed for | Children (6 to 10 years) | Adults (21 or older) |
|---|---|---|---|---|
| Concentration in Darby Creek (mg/L) | C | Swimming/Wading | 0.0243 | 0.0243 |
| Intake Rate (L/hr) | IR | Swimming | 0.12 | 0.071 |
| Body Weight (kg) | BW | Swimming/Wading | 31.8 | 80 |

| | | | | |
|---|---|---|---|---|
| Event Duration (hr/event) | $t_{event}$ | Swimming/Wading | 1 hour | 1 hour |
| Event frequency (events/day) | EV | Swimming/Wading | 1 event | 1 event |
| Exposure Factor (EF) (1 = daily exposures) | EF | Swimming/Wading | 1 | 1 |
| Absorbed Dose Per Event for chromium(VI) | $DA_{event}$ | Swimming/Wading | $4.86x10^{-8}$ | $4.86x10^{-8}$ |
| Surface Area Available for Contact, swimming scenario ($cm^2$) | SA | Swimming | 10,800$cm^2$ | 19,811$cm^2$ |
| Surface Area Available for Contact, wading scenario ($cm^2$) | SA | Wading | 3,824$cm^2$ | 7,325$cm^2$ |
| Gastrointestinal Absorption Factor (unitless) for chromium(VI) | $ABS_{GI}$ | Swimming/Wading | 0.025 | 0.025 |
| Cancer slope factor for chromium(VI) | CSF | Cancer Risk | $(0.5\ mg/kg)^{-1}$ | $(0.5\ mg/kg)^{-1}$ |
| Age-Dependent Adjustment Factor for chromium(VI) | ADAF** | Cancer Risk | 3 | 1 |
| Exposure duration | ED | Cancer Risk | 5 | 10 |
| Lifetime Years | LY | Cancer Risk | 78 | 78 |

Sources: ATSDR 2018b; ATSDR 2016; EPA 2011; EPA 2004
*Estimates assume a Reasonable Maximum Exposure Scenario
**Age-dependent adjustment factors (ADAFs) account for chemicals that act with a mutagenic mode of action (MOA) for carcinogenesis. Younger children are more susceptible to these chemicals. Chromium(VI) is a chemical identified as having an MOA. As shown, the ADAF for children ages 6-10 years is 3 (ATSDR 2018b).

### B.5.2.1. Exposure Dose Calculation for a Swimming Scenario, for children and adults

The calculation assumes a daily, 1 hour per day, 12 week swimming scenario in Darby Creek.

### *B.5.2.2 Child (ages 6-10) exposure dose and cancer risk estimates based on a 12 week swimming scenario in Darby Creek, under a 100% chromium(VI) assumption*

***Dermal exposure for a child 6-10 based on a swimming scenario to the highest total chromium concentration (24.3 ug/L), under a chromium(VI) assumption***

Administered Dermal Dose = (Absorbed Dose per Event x Surface Area Available for Contact x Event Frequency x Exposure Factor) / (Body Weight x Gastrointestinal Absorption Factor)

$$ADD = \frac{DA_{event} \times SA \times EV \times EF}{BW \times ABS_{GI}}$$

$$\text{Child, ages 6-10} = \frac{4.86x10^{-8}\ mg/cm^2/event \times 10{,}800cm^2 \times 1\ event/day \times 1}{31.8\ kg \times 0.025}$$

= 0.00066 mg/kg/day x (1000 ug/kg) **= 0.66 µg/kg/day**

***Ingestion exposure for a child 6-10 based on a swimming scenario to the highest total chromium concentration (24.3 ug/L), under a chromium(VI) assumption***

$$\text{Dose} = \frac{\text{(Concentration (mg/L) x Intake Rate x Event Duration x Event Frequency x Exposure Factor)}}{\text{Body Weight}}$$

$$D = \frac{(C \times IR \times ED \times EV \times EF)}{BW}$$

$$\text{Child, ages 6-10} = \frac{\text{(0.0243 mg/L x 0.12 L/hr x 1 hr/event x 1 event/day x1)}}{\text{31.8 kg}}$$

$= 9x10^{-5}$ x (1000 µg/kg) **= 0.091** µg/kg/day

***Combined child (6-10 years of age) ingestion and dermal exposures (swimming scenario)***

= exposure (ingestion) + exposure (dermal)

= 0.66 µg/kg/day + 0.091 µg/kg/day **= 0.75 µg/kg/day , or 0.00075 mg/kg/day**

## *B.5.2.3. Adult (ages 21 and over) exposure dose and cancer risk estimates based on a 12 week swimming scenario in Darby Creek, under a 100% chromium(VI) assumption*

***Dermal exposure for an adult based on a swimming scenario to the highest total chromium concentration (24.3 ug/L), under a chromium(VI) assumption***

$$\text{Administered Dermal Dose} = \frac{\text{(Absorbed Dose per Event x Surface Area Available for Contact x Event Frequency x Exposure Factor)}}{\text{(Body Weight x Gastrointestinal Absorption Factor)}}$$

$$ADD = \frac{(DA_{event} \times SA \times EV \times EF)}{BW \times ABS_{GI}}$$

$$\text{Adult} = \frac{\text{(4.86x10}^{-8}\text{ mg/cm}^2\text{/event x 19,811cm}^2\text{ x 1 event/day x 1)}}{\text{80 kg x 0.025}}$$

= 0.00048mg/kg/day x (1000 ug/kg) **= 0.48 µg/kg/day**

***Ingestion exposure for an adult based on a swimming scenario to the highest total chromium concentration (24.3 ug/L), , under a chromium(VI) assumption***

$$\text{Dose} = \frac{\text{(Concentration (mg/L) x Intake Rate x Event Duration x Event Frequency x Exposure Factor)}}{\text{Body Weight}}$$

$$D = \frac{(C \times IR \times ED \times EV \times EF)}{BW}$$

$$\text{Adult} = \frac{\text{(0.0243 mg/L x 0.07 L/hr x 1 hr/event x 1 event/day)}}{\text{80 kg}}$$

= 0.000022 mg/kg/day x (1000 ug/kg) **= 0.02 µg/kg/day**

***Combined adult (21 years of age and older) ingestion and dermal exposures (swimming scenario)***
= exposure (dermal) + exposure (ingestion)
= 0.48 μg/kg/day + 0.02 μg/kg/day = **0.50 μg/kg/day** , or **0.0005 mg/kg/day**

### *B.5.2.4 Child (ages 6-10) exposure dose and cancer risk estimates based on a year-round wading scenario and exposed to the highest total chromium concentration (24.3 μg/L), under the assumption it is 100% chromium(VI)*

***Dermal exposure for a child 6-10 based on a swimming scenario to the highest total chromium concentration, under a chromium(VI) assumption (24.3 ug/L)***

Administered Dermal Dose = (Absorbed Dose per Event x Surface Area Available for Contact x Event Frequency x Exposure Factor)

(Body Weight x Gastrointestinal Absorption Factor)

$$ADD = \frac{(DA_{event} \times SA \times EV \times EF)}{BW \times ABS_{GI}}$$

$$\text{Child, ages 6-10} = \frac{(4.86\times10^{-8}\ mg/cm^2/event \times 3{,}824 cm^2 \times 1\ event/day \times 1)}{31.8\ kg \times 0.025}$$

= 0.00023 mg/kg/day x (1000 ug/kg) **= 0.23 μg/kg/day**

***Child Cancer Risk Estimate based on Wading Scenario, under a chromium(VI) assumption***

Cancer Risk = [(Dose x Chromium(VI) Cancer Slope Factor / (Exposure Duration x Lifetime Years)] x Age-Dependent Adjustment Factor for Chromium(VI)

= CR = (D x CSF) x (ED / LY) x ADAF

- Wading/Dermal cancer risk = (0.00023 mg/kg/day) x $(0.5\ mg/kg/day)^{-1}$) x (5/78) x 3 = **$2.2\times10^{-5}$, or 2 excess cases per in 100,000 people**

**Note:** CDC's Public Health Assessment Site Tool (PHAST), estimates child excess cancer risk for all children under the age of 21, based on combined cancer risk each separate age group. When combining childhood excess cancer risk for each age group based on creek exposures (ages 6-10, ages 11-16, ages 16-21) in addition using the same formulas of this section, the total combined excess cancer risk for children over a period of 10 years is **$4.0\times10^{-5}$, or 4 excess cases per 100,000 people.**

### *B.5.2.5 Adult (ages 21 and over) exposure dose and cancer risk estimates based on a year-round wading scenario and exposed to the highest total chromium concentration (24.3 μg/L), under the assumption it is 100% chromium(VI)*

***Dermal exposure for a child 6-10 based on a swimming scenario to the highest total chromium concentration, under a chromium(VI) assumption (24.3 ug/L)***

Administered Dermal Dose = (Absorbed Dose per Event x Surface Area Available for Contact x Event Frequency x Exposure Factor)

(Body Weight x Gastrointestinal Absorption Factor)

$$ADD = \frac{(DA_{event} \times SA \times EV \times EF)}{BW \times ABS_{GI}}$$

$$Adult = \frac{(4.86 \times 10^{-8}\ mg/cm^2/event \times 7{,}325 cm^2 \times 1\ event/day \times 1)}{80\ kg \times 0.025}$$

= 0.00018 mg/kg/day x (1000 ug/kg) **= 0.18 µg/kg/day**

***Adult Cancer Risk Estimate based on Wading Scenario, under a chromium(VI) assumption***

Cancer Risk = [(Dose x Chromium(VI) Cancer Slope Factor / (Exposure Duration x Lifetime Years)] x Age-Dependent Adjustment Factor for Chromium(VI)

= CR = (D x CSF) x (ED / LY) x ADAF

- Wading/Dermal cancer risk = (0.00018 mg/kg/day) x $(0.5\ mg/kg/day)^{-1}$) x (10/78) x 1 = **$1.1 \times 10^{-5}$, or 1 excess case per 100,000 people**

**Table B.5.2.6. Summary of Cancer and Non-cancer health effects for children and adults based a swimming and wading scenario, and 100% chromium(VI) assumption**

| Sample type, and Conc (µg/kg for soil and sediment, µg/L for surface water) | Exposed Population and Time Period | Estimated Ingestion and Dermal Exposure Dose (ED), µg/kg/day | Chronic Hazard Quotient<br><br>MRL: 0.9 | Int Hazard Quotient<br><br>MRL: 5 | Excess Cancer Risk[5] |
|---|---|---|---|---|---|
| Surface Water 24.3 µg/L (max) | Adult, swimming | 0.5 | NA | <1 | NA |
| | Adult, wading | 0.18 | <1 | <1 | 1 in 100,000 |
| | Child (6-10y), swimming | 0.75 | NA | <1 | NA |
| | Child (6-10y), wading | 0.23 | <1 | <1 | 4 in 100,000 |

Conc = Concentration; 95UCL = 95th Upper Confidence Limit of the Mean; Int=Intermediate; NA, Not Applicable; y = year of age. MRLs expressed in µg/kg/day. Cancer risk estimates encompass children (6-21 years) and adults (21 years and older).

**Appendix B References:**

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# Appendix C. Norwood Screening Results (Based on 2017-2018 EPA Sampling)

**Note:** On multiple occasions in EPA's 2017-2018 dataset, chemical concentrations were estimated (e.g., assigned a "J" value; Weston 2018, Tables 1-9). This qualification means that the analyte was present in the field sample, but the concentration reported is an estimate and the true concentration may be higher or lower. As a health protective approach for all media sampled, we considered these J values during screening and as part of our health effects evaluation if a J value exceeded a CV.

A few samples were assigned "R" qualifiers in EPA's dataset, indicating that the data were rejected due to a sample jar being broken in transit, and thus the remaining soil in a baggie was analyzed (Weston 2018). We considered these values "detected" (as opposed to non-detected), but did not include their concentrations in our analysis (whether for screening or the health effects evaluation).

Below are the screening results for residential soil (Table C1), non-residential soil (Table C2), Darby/Muckinipattis Creek sediment (Table C3), and Darby/Muckinipattis surface water (Table C4). Sample numbers that exceed CVs are listed for non-residential locations.

**Appendix Table C1. Norwood Residential Surface Soil Concentrations and Comparison Values (CVs)**

| Chemical Sampled in Residential Soil | N and % Homes Detected out of 21 samples | Range (ppm) | CV (ppm) | CV Source (non ATSDR CV listed if no CV available) | Number of Samples above a CV out of 21 samples (CV Type) |
|---|---|---|---|---|---|
| *Volatile Organic Compounds** | | | | | |
| Acetone | 5 (24.0%) | ND-0.068 | 31,000 | iEMEGc | 0 |
| 2-Hexanone | 3 (14.3%) | ND-0.016 | 260 | RMEGc | 0 |
| 2-Butanone | 2 (9.5%) | ND-0.008 | 31,000 | RMEGc | 0 |
| Chloroform | 2 (9.5%) | ND-0.004 | 520 | RMEGc | 0 |
| 1,1,1 Trichloroethane | 9 (42.9%) | ND-0.004 J | 100,000 | RMEGc | 0 |
| Styrene | 4 (19.0%) | ND-0.046 J | 10,000 | RMEGc | 0 |
| *Semi-Volatile Organic Compounds and Polycyclic Aromatic Hydrocarbons (PAHs)* | | | | | |
| Naphthalene | 21 (100%) | 0.001-0.24 J | 1,000 | RMEGc | 0 |
| 2-Methylnaphthalene | 21 (100%) | 0.001-0.15 J | 210 | RMEGc | 0 |
| Acenaphthylene | 21 (100%) | 0.0011-0.04 | 13,000 | PADEP MSC | 0 |
| Acenaphthene | 21 (100%) | 0.0007-0.24 J | 3,100 | RMEGc | 0 |
| Fluorene | 10 (47.6%) | 0.004-0.27 J | 2,100 | RMEGc | 0 |
| Pentachlorophenol | 1 (5.6%) | ND-0.0017 J | 0.97 | CREG | 0 |
| Phenanthrene | 21 (100%) | 0.012-6.5 | 66,000 | PADEP MSC | 0 |
| Anthracene | 21 (100%) | 0.0021-1.3 | 16,000 | RMEGc | 0 |
| Fluoranthene | 21 (100%) | 0.038-18.0 | 2,100 | RMEGc | 0 |
| Pyrene | 21 (100%) | 0.031-15.0 | 1,600 | RMEGc | 0 |
| **Benzo(a)anthracene** | 21 (100%) | 0.018-9.1 | 1.1 | RSL | 3 |
| Chrysene | 21 (100%) | 0.02-9.9 | 110 | RSL | 0 |
| **Benzo(b)fluoranthene** | 21 (100%) | 0.03-15.0 | 1.1 | RSL | 3 |
| Benzo(k)fluoranthene | 21 (100%) | 0.01-4.6 | 11 | RSL | 0 |
| **Benzo(a)pyrene** | 21 (100%) | 0.024-9.7 | 0.065 | CREG | 9 (CREG) |

| | | | | | |
|---|---|---|---|---|---|
| | | | 16 | RMEGc | |
| **Indeno(1,2,3-cd)pyrene** | 21 (100%) | 0.0095-6.9 | 1.1 | RSL | 3 |
| Dibenzo(ah)anthracene | 0 (0.0%) | ND-0.0052 U | 0.11 | RSL | 0 |
| Benzo(ghi) perylene | 18 (85.7%) | ND-7.6 | 13,000 | PADEP MSC | 0 |
| Dibenzofuran | 1 (4.8%) | ND-0.25 J | 220 | PADEP MSC | 0 |
| Carbazole | 3 (14.3%) | ND-0.55 J | 930 | PADEP MSC | 0 |
| **bis(2-Ethylhexyl)phthalate (DEHP)** | 21 (100%) | 0.03-6.8 J | 5.2<br>28<br>0.53 | iEMEGc<br>CREG<br>iEMEG, iPica | 2 (1 for iEMEGc:1 for iPica) |
| *Polychlorinated Biphenyls (PCBs)* | | | | | |
| Aroclor-1254 | 1 (4.8%) | ND-0.016 J | 1.0 | RMEGc | 0 |
| **Aroclor-1260** | 16 (76.2%) | ND-0.31 | 0.24<br>9.0 | RSL<br>PADEP MSC | 1<br>0 (PADEP MSC) |
| **Pesticides** | | | | | |
| Alpha-BHC | 1 (4.8%) | ND-0.00048 J | 0.062 | CREG | 0 |
| Beta-BHC | 1 (4.8%) | ND-0.0028 J | 0.22 | CREG | 0 |
| Delta-BHC | 0 (0.0%) | ND-0.011U | No CV | No CV | NA |
| **Gamma-BHC (Lindane)** | 6 (28.6%) | ND-0.065 | 0.52<br>0.053 | iEMEGc<br>iPica | 1 (iPica)<br>0 (iEMEGc) |
| Heptachlor | 7 (33.3%) | ND-0.0063 J | 0.086 | CREG | 0 |
| **Aldrin** | 6 (28.6%) | ND-0.053 J | 0.023 | CREG | 1 |
| Heptachlor Epoxide | 15 (71.4%) | ND-0.029 J | 0.043 | CREG | 0 |
| Endosulfan I | 15 (71.5%) | ND-0.0024 J | 260 | cEMEGc | 0 |
| **Dieldrin** | 19 (90.5%) | ND-1.3 | 0.024<br>0.53 | CREG<br>iPica | 2 (CREG: 2; iPica: 1) |
| 4,4'DDE | 21 (100%) | 0.00044-0.29 | 1.1 | CREG | 0 |
| Endrin | 20 (95.2%) | ND-0.023 J | 16 | cEMEGc | 0 |
| Endosulfan II | 3 (14.3%) | ND-0.008 | 47 | RSL | 0 |
| 4',4'-DDD | 13 (61.9%) | ND-0.0025 J | 1.6 | CREG | 0 |
| Endosulfan Sulfate | 13 (61.9%) | ND-0.2 J | 47 | RSL | 0 |
| 4,4-DDT | 15 (71.4%) | ND-0.13 | 1.1 | CREG | 0 |
| Methoxychlor | 20 (95.2%) | ND-0.65 J | 260 | iEMEGc | 0 |
| Endrin ketone | 11 (52.4%) | ND-0.13 J | 1.9 | RSL | 0 |
| Endrin aldehyde | 18 (85.7%) | ND-0.0033 J | 1.9 | RSL | 0 |
| Cis-Chlordane | 17 (81.0%) | ND-0.12 | 1.1 | CREG | 0 |
| Trans-Chlordane | 20 (95.2%) | ND-0.066 | 1.1 | CREG | 0 |
| *Metals* | | | | | |
| **Aluminum** | 21 (100%) | 8990-13600 | 5,300<br>52,000 | iPica<br>cEMEGc | All (iPica)<br>0 (cEMEGc) |
| **Antimony** | 1 (4.8%) | 4.2-4.2 J | 3.2<br>21 | iPica<br>RMEGc | 1 (iPica)<br>0 (RMEGc) |
| **Arsenic** | 21 (100%) | 4.2-9.7 | 0.26<br>16 | CREG<br>cEMEGc | 21 (CREG)<br>0 (cEMEGc) |
| Barium | 21 (100%) | 26.8-181.0 | 10,000 | cEMEGc | 0 |
| Beryllium | 21 (100%) | 0.24-0.61 | 100 | cEMEGc | 0 |

| | | | | | |
|---|---|---|---|---|---|
| Cadmium | 20 (95.2%) | ND-3.4 | 5.2<br>2.7 (iPica) | cEMEGc<br>2.7 (iPica) | 1 (iPica) |
| Calcium | 21 (100%) | 209-8960 | No CV | No CV | NA |
| **Chromium** | 21 (100%) | 14.1-26.1 | 12,000 | RSL** | 0 |
| Cobalt | 21 (100%) | 3.6-9.4 | 520 | iEMEGc | 0 |
| **Copper** | 21 (100%) | 10.2-264 | 53<br>1,000 | aPica, iPica<br>iEMEGc | 2 (aPica, iPica)<br>0 (iEMEGc) |
| **Iron** | 21 (100%) | 7100-25300 | 5,500 | RSL | 21 (All) |
| **Lead** | 21 (100%) | 30.6-1800 | 400 | RSL | 1 |
| Magnesium | 21 (100%) | 1560-4250 | No CV | No CV | NA |
| **Manganese** | 21 (100%) | 105-553 | 180<br>10,000 | RSL<br>PADEP MSC | 18<br>0 (PADEP MSC) |
| Mercury | 21 (100%) | 0.032-0.88 | 1.0<br>1.1 | DTSC<br>RSL | 0 |
| Nickel | 21 (100%) | 9.2-29.3 | 1,000 | RMEGc | 0 |
| Potassium | 21 (100%) | ND-2500 | No CV | No CV | NA |
| Selenium | 15 (71.4%) | ND-1.0 J | 260 | cEMEGc | 0 |
| Silver | 12 (57.1%) | ND-3.7 | 260 | RMEGc | 0 |
| Sodium | 21 (100%) | ND-87.2 J | No CV | No CV | NA |
| **Thallium** | 0 (0.0%) | ND-0.76 U | 0.078 | RSL | Non-detect quantitation limit exceeds RSL |
| Vanadium | 21 (100%) | 16.2-37.9 | 520 | iEMEGc | 0 |
| Zinc | 21 (100%) | 49.0-914.0 | 16,000 | cEMEGc | 0 |

**Bold** = chemical exceeded a comparison value (CV) for one or more samples. RMEGc=Reference Dose Media Evaluation Guide (Child); cEMEGc = Chronic Evaluation Media Guide (Child); iEMEGc = Intermediate Evaluation Media Guide (Child); CREG = Cancer Risk Evaluation Guide; aPica = Acute Pica; iPica = Intermediate Pica; RSL = EPA Regional Screening Value; CV = Comparison Value; ND = Not Detected; U = Not Detected Quantitation Limit; J = Reported value is estimated and actual value may be higher or lower, PADEP MSC = PADEP Medium Specific Concentration, DTSC = California Department of Toxic Substances Control Screening value

*Percentages of detected Volatile Organic Compounds (VOCs) include a single sample (RS17) of "rejected data" due to a sample jar being broken in transit. We considered this sample "detected" as opposed to non-detected but did not consider its concentration. The "rejected data" concentrations for RS17, for all VOC samples, ranged from 0.0054-0.011 mg/kg, which is below applicable CVs.

**RSL pertains to chromium(III), as there is no RSL for total chromium.

**Appendix Table C2. Norwood Non-Residential Surface Soil Concentrations and Comparison Values (CVs)**

| Chemical Sampled in Non-Residential Soil | N and % Samples Detected out of 17 samples | Range (ppm) | CV (ppm) | CV Source (non ATSDR CV listed if no CV available) | Number of Samples at or above a CV out of 17 samples (Sample ID/CV Type Exceeded) |
|---|---|---|---|---|---|
| *Volatile Organic Compounds** | | | | | |
| Acetone | 2 (11.8%) | ND-0.032 | 31,000 | iEMEGc | 0 |
| Methylene chloride | 6 (35.3%) | ND-0.041 | 55 | CREG | 0 |
| Ethylbenzene | 1 (5.9%) | ND-0.0014 U | 5,200 | RMEGc | 0 |
| Toluene | 2 (11.8%) | ND-0.0062 U | 4,200 | RMEGc | 0 |
| Ethanol | 2 (5.9%) | ND-0.0066 J | No CV | No CV | NA |
| 2-Butanone | 1 (5.9%) | ND-0.0066 J | 31,000 | RMEGc | 0 |
| Chlorobenzene | 1 (5.9%) | ND-0.0013 U | 1,000 | RMEGc | 0 |
| 1,2-Dichlorobenzene | 1 (5.9%) | ND-0.0078 U | 4,700 | RMEGc | 0 |
| 1,4-Dichlorobenzene | 1 (5.9%) | ND-0.0078 U | 3,600 | iEMEGc | 0 |
| Isopropyl benzene | 1 (5.9%) | ND-0.0078 U | 5,200 | RMEGc | 0 |
| Styrene | 1 (5.9%) | ND-0.0078 U | 10,000 | RMEGc | 0 |
| *Semi-Volatile Organic Compounds and Polycyclic Aromatic Hydrocarbons (PAHs)* | | | | | |
| Naphthalene | 15 (88.2%) | ND-0.17 J | 1,000 | RMEGc | 0 |
| 2-Methylnaphthalene | 17 (100%) | ND-0.12 J | 210 | RMEGc | 0 |
| Acenaphthylene | 16 (94.1%) | 0.0014-0.065 J | 13,000 | PADEP MSC | 0 |
| Acenaphthene | 16 (94.1%) | ND-0.76 | 3,100 | RMEGc | 0 |
| Fluorene | 16 (94.1%) | ND-0.89 | 2,100 | RMEGc | 0 |
| Phenanthrene | 17 (100%) | 0.011-11 | 66,000 | PADEP MSC | 0 |
| Anthracene | 17 (100%) | 0.0018-1.4 | 16,000 | RMEGc | 0 |
| Fluoranthene | 17 (100%) | 0.026-5.0 | 2,100 | RMEGc | 0 |
| Pyrene | 17 (100%) | 0.031-8.6 | 1,600 | RMEGc | 0 |
| **Benzo(a)anthracene** | 17 (100%) | 0.017-2.9 | 1.1 | RSL | 2 (SS6, SS14) |
| Chrysene | 17 (100%) | 0.019-4.3 | 110 | RSL | 0 |
| **Benzo(b)fluoranthene** | 17 (100%) | 0.028-4.7 | 1.1 | RSL | 2 (SS6, SS14) |
| Benzo(k)fluoranthene | 17 (100%) | 0.0094-1.2 | 11 | RSL | 0 |
| **Benzo(a)pyrene** | 17 (100%) | 0.017-2.3 | 0.065<br>16 | CREG<br>RMEGc | 12 (CREG: SS1, SS3, SS5, SS6, SS8, SS9, SS10, SS11, SS12, SS14, SS16, SS19) |
| **Indeno(1,2,3-cd)pyrene** | 17 (100%) | 0.012-1.4 | 1.1 | RSL | 1 (SS6) |
| **Dibenzo(ah)anthracene** | 17 (100%) | 0.0042-0.41 | 0.11 | RSL | 3 (SS5, SS6, SS14) |
| Benzo(ghi) perylene | 17 (100%) | 0.012-1.3 | 13,000 | PADEP MSC | 0 |
| 4-Chloroaniline | 0 (0.0%) | ND-0.56 U | 93 | PADEP MSC | 0 |
| 1,1-Biphenyl | 1 (5.9%) | ND-0.051 | 48 | CREG | 0 |
| Carbazole | 6 (35.3%) | ND-0.91 | 930 | PADEP MSC | 0 |

| | | | | | |
|---|---|---|---|---|---|
| Dibenzofuran | 3 (17.6%) | ND-0.58 | 220 | PADEP MSC | 0 |
| bis(2-Ethylhexyl)phthalate | 11 (64.7%) | ND-0.24 | 5.2<br>28 | iEMEGc<br>CREG | 0 |
| Dimethyl phthalate | 16 (94.1%) | ND-0.54 | No CV | No CV | NA |
| Di-n-butylphthalate | 3 (17.6%) | ND-0.20 J | 5,200 | RMEGc | 0 |
| Butylbenzylphthalate | 1 (5.9%) | ND-0.17 J | 10,000 | RMEGc | 0 |
| Benzaldehyde | 1(5.9%) | ND-0.16 J | 5,200 | RMEGc | 0 |
| Acetophenone | 0 (0.0%) | ND-0.56 U | 5,200 | RMEGc | 0 |
| Phenol | 10 (58.8%) | ND-0.11 J | 16,000 | RMEGc | 0 |
| *Polychlorinated Biphenyls (PCBs)* | | | | | |
| **Aroclor-1254** | 3 (17.6%) | ND-0.45 | 1.0<br>0.16 | RMEGc<br>iPica | 2 (iPica: SS5, SS9) |
| **Aroclor-1260** | 15 (88.2%) | ND-0.28 | 0.24 | RSL | 2 (SS5; SS9) |
| **Pesticides** | | | | | |
| 4,4'DDE | 12 (70.6%) | ND-0.023 J | 1.1 | CREG | 0 |
| 4',4'-DDD | 4 (23.5%) | ND-0.13 | 1.6 | CREG | 0 |
| 4,4-DDT | 10 (58.8%) | ND-0.066 | 1.1 | CREG | 0 |
| Cis-Chlordane | 6 (35.3%) | ND-0.024 | 1.1 | CREG | 0 |
| Trans-Chlordane | 6 (35.3%) | ND-0.025 | 1.1 | CREG | 0 |
| Heptachlor | 2 (11.8%) | ND-0.0024 | 0.086 | CREG | 0 |
| Heptachlor Epoxide | 1 (5.9%) | ND-0.0035 | 0.043 | CREG | 0 |
| **Dieldrin** | 4 (23.5%) | ND-0.085 | 0.024 | CREG | 1 (SS5) |
| Aldrin | 3 (17.6%) | ND-0.018 | 0.023 | CREG | 0 |
| Endrin ketone | 1 (5.9%) | ND-0.0039 | 16 | cEMEGc | 0 |
| *Metals* | | | | | |
| **Aluminum** | 17 (100%) | 5700-16800 | 5,300<br>52,000 | iPica<br>cEMEGc | All (iPica)<br>0 (cEMEGc) |
| **Antimony** | 17 (100%) | 0.52-1.8 J | 3.2<br>21 | iPica<br>RMEGc | 1 (iPica: SS6) |
| **Arsenic** | 17 (100%) | **2.6-7.8** | **0.26**<br>16 | **CREG**<br>cEMEGc | All (CREG)<br>0 (cEMEGc) |
| Barium | 17 (100%) | 35.2-220 | 10,000 | cEMEGc | 0 |
| Beryllium | 17 (100%) | 0.42-1.0 | 100 | cEMEGc | 0 |
| Cadmium | 17 (100%) | 0.59-2.3 | 5.2<br>2.7 | cEMEGc<br>iPica | 0 |
| Calcium | 17 (100%) | 511-8450 | No CV | No CV | 0 |
| **Chromium** | 17 (100%) | 12.4-31 | 12,000 | RSL** | 0 |
| Cobalt | 17 (100%) | 4.3-16.3 | 520 | iEMEGc | 0 |
| **Copper** | 17 (100%) | 1.9-64.2 | 53<br>1,000 | aPica, iPica<br>iEMEGc | 2 (SS6, SS19) |
| **Iron** | 17 (100%) | 12700-35100 | 5,500 | RSL | All |
| **Lead** | 17 (100%) | 20.7-358 | 400 | RSL | 0 |
| Magnesium | 17 (100%) | 1,390-4760 | No CV | No CV | NA |
| **Manganese** | 17 (100%) | 165-710 | 180<br>10,000 | RSL<br>PADEP MSC | All except SS1 (RSL only) |
| Mercury | 17 (100%) | 0.042-0.64 | 1.0 | DTSC | 0 |

| | | | | | |
|---|---|---|---|---|---|
| | | | 1.1 | RSL | |
| Nickel | 17 (100%) | 8.7-29.3 | 1,000 | RMEGc | 0 |
| Potassium | 17 (100%) | 330-3180 | No CV | No CV | NA |
| Selenium | 0 (0%) | ND-3.9 U | 260 | cEMEGc | 0 |
| Silver | 17 (100%) | 0.39-1.3 | 260 | RMEGc | 0 |
| Sodium | 17 (100%) | 64-168 | No CV | No CV | NA |
| **Thallium** | 0 (0.0%) | ND-**2.9 U** | **0.078** | RSL | Non-detect quantitation limit exceeds RSL |
| Vanadium | 17 (100%) | 22.7-78.1 | 520 | iEMEGc | 0 |
| Zinc | 17 (100%) | 42.6-269 | 16,000 | cEMEGc | 0 |

**Bold** = chemical exceeded a comparison value (CV) for one or more samples. RMEGc=Reference Dose Media Evaluation Guide (Child); cEMEGc = Chronic Evaluation Media Guide (Child); iEMEGc = Intermediate Evaluation Media Guide (Child); CREG = Cancer Risk Evaluation Guide; aPica = Acute Pica; iPica = Intermediate Pica; RSL = EPA Regional Screening Value; CV = Comparison Value; ND = Not Detected; U = Not Detected Quantitation Limit; J = Reported value is estimated and actual value may be higher or lower, PADEP MSC = PADEP Medium Specific Concentration, DTSC = California Department of Toxic Substances Control Screening value

*Volatile Organic Compounds (VOCs) were largely undetected except for a single sample (SS19) that was listed as “rejected data” due to a sample jar being broken in transit. We considered this sample “detected” as opposed to non-detected, but did not consider its concentration. The “rejected data” concentration for SS19, for all VOC samples, was 0.0071ppm, which is below applicable CVs.

**RSL pertains to chromium(III), as there is no RSL for total chromium.

**Appendix Table C3. Creek Sediment Concentrations and Comparison Values (CVs)**

**Note:** Our evaluation of Darby/Muckinipattis Creek sediment samples, which consisted of 8 co-located sediment and surface water samples, incorporated a duplicate (SD12) as opposed to the non-duplicate sample (SD4). SD12 had slightly but consistently higher concentrations than SD4 and we incorporated it instead of SD4 as a health protective approach. SD4 (and the SD12 duplicate) represents the sole sample taken along Muckinipattis Creek, and this sample was taken next to a public fishing dock near the convergence of Darby and Muckinipattis Creeks (Figure 2 of the main report).

| Chemical Sampled in Sediment | N and % Samples Detected out of 8 samples | Range (ppm) | CV (ppm) | CV Source (non ATSDR CV listed if no CV available) | Number of Samples at or above a CV out of 8 samples (Sample ID/CV Type Exceeded) |
|---|---|---|---|---|---|
| *Semi-Volatile Organic Compounds and Polycyclic Aromatic Hydrocarbons (PAHs)* | | | | | |
| Dimethyl phthalate | 8 (100.0%) | 0.19-1.2 | NA | NA | NA |
| Naphthalene | 2 (25.0%) | ND-0.015 | 1,000 | RMEGc | 0 |
| 2-Methylnaphthalene | 2 (25.0%) | ND-0.0049 | 210 | RMEGc | 0 |
| Acenaphthylene | 1 (12.5%) | ND-0.0037 J | 13,000 | PADEP MSC | 0 |
| Acenaphthene | 0 (0.0%) | ND-0.0011 U | 3,100 | RMEGc | 0 |
| Fluorene | 1 (12.5%) | ND-0.0025 J | 2,100 | RMEGc | 0 |
| Phenanthrene | 7 (87.5%) | ND-0.02 | 66,000 | PADEP MSC | 0 |
| Anthracene | 3 (37.5%) | ND-0.0049 | 16,000 | RMEGc | 0 |
| Fluoranthene | 8 (100.0%) | 0.0059-0.043 | 2,100 | RMEGc | 0 |
| Pyrene | 8 (100.0%) | 0.008-0.055 | 1,600 | RMEGc | 0 |
| Benzo(a)anthracene | 7 (87.5%) | ND-0.029 | 1.1 | RSL | 0 |
| Chrysene | 7 (87.5%) | ND-0.029 | 110 | RSL | 0 |
| Benzo(b)fluoranthene | 7 (87.5%) | ND-0.043 | 1.1 | RSL | 0 |
| Benzo(k)fluoranthene | 6 (75.0%) | ND-0.013 | 11 | RSL | 0 |
| Benzo(a)pyrene | 7 (87.5%) | ND-0.029 | 0.065<br>16 | CREG<br>RMEGc | 0 |
| Indeno(1,2,3-cd)pyrene | 7 (87.5%) | ND-0.019 | 1.1 | RSL | 0 |
| Dibenzo(ah)anthracene | 4 (50.0%) | ND-0.064 | 0.11 | RSL | 0 |
| Benzo(ghi) perylene | 7 (87.5%) | ND-0.022 | 13,000 | PADEP MSC | 0 |
| Bis(2-ethylhexyl)phthalate | 0 (0.0%) | ND-0.59 U | 5.2<br>28 | iEMEGc<br>CREG | 0 |
| Phenol | 3 (37.5%) | ND-0.19J | 16,000 | RMEGc | 0 |
| *Pesticides* | | | | | |
| 4,4-DDE | 2 (25.0%) | ND-0.00053 J | 1.1 | CREG | 0 |
| 4,4-DDD | 0 (0.0%) | ND-0.011 U | 1.6 | CREG | 0 |
| 4,4-DDT | 0 (0.0%) | ND-0.011 U | 1.1 | CREG | 0 |
| Methoxychlor | 2 (25.0%) | ND-0.057 | 260 | iEMEGc | 0 |
| cis-Chlordane | 0 (0.0%) | ND-0.0059 U | 1.1<br>1.7 | CREG<br>RSL | 0 |
| trans-Chlordane | 0 (0.0%) | ND-0.0059 U | 1.1<br>1.7 | CREG<br>RSL | 0 |
| *Metals* | | | | | |

| | | | | | |
|---|---|---|---|---|---|
| **Aluminum** | 8 (100%) | 11800.0-25200 | 5,300<br>52,000 | iPica<br>cEMEGc | All (iPica)<br>0 (cEMEGc) |
| Antimony | 5 (62.5%) | ND-1.3 J | 3.2<br>21 | iPica<br>RMEGc | 0 |
| **Arsenic** | 8 (100%) | 9.4-26.6 | 0.26<br>16 | CREG<br>cEMEGc | All (CREG); 2 (cEMEGc: SD6, SD12) |
| Barium | 8 (100%) | 140.0-223 | 10,000 | cEMEGc | 0 |
| Beryllium | 8 (100%) | 1.1-2.2 | 100 | cEMEGc | 0 |
| Cadmium | 8 (100%) | 1.9-4.1 | 5.2<br>2.7 | cEMEGc<br>iPica | 6 (iPica)<br>0 (cEMEGc) |
| Calcium | 8 (100%) | 2580.0-6980 J | No CV | No CV | NA |
| Chromium* | 8 (100%) | 43.1-115 | 12,000 | RSL | 0 |
| Cobalt | 8 (100%) | 12.3-22.9 J | 520 | iEMEGc | 0 |
| **Copper** | 8 (100%) | 61.6-111 J | 53<br>1,000 | aPica, iPica<br>iEMEGc | All (aPica, iPica)<br>0 (iEMEGc) |
| **Iron** | 8 (100%) | 30000-37700 | 5,500 | RSL | All |
| **Lead** | 8 (100%) | 74.5-214.0 | 400 | RSL | 0 |
| Magnesium | 8 (100%) | 5720-6670 J | No CV | No CV | NA |
| **Manganese** | **8 (100%)** | 420-848 J | 180<br>10,000 | RSL<br>PADEP MSC | All (RSL) |
| **Mercury** | **8 (100%)** | 0.3-1.1 | 1.0<br>1.1 | DTSC<br>RSL | 1 (DTSC: SD12) |
| Nickel | 8 (100%) | 25.0-43.1 | 1,000 | RMEGc | 0 |
| Potassium | 8 (100%) | 1790-2150 J | No CV | No CV | NA |
| Selenium | 0 (0%) | ND-8.3 U | 260 | cEMEGc | 0 |
| Silver | 8 (100%) | 1.2-4.6 J- | 260 | RMEGc | 0 |
| Sodium | 8 (100%) | 318-394 J | No CV | No CV | 0 |
| **Thallium** | 0 (0.0%) | ND-5.9 U | **0.078** | RSL | Non-detect quantitation limit exceeds RSL |
| Vanadium | 8 (100%) | 37.5-71.7 | 520 | iEMEGc | 0 |
| Zinc | 8 (100%) | 239-418 J | 16,000 | cEMEGc | 0 |

**Bold =** chemical exceeded a comparison value (CV) for one or more samples. RMEGc=Reference Dose Media Evaluation Guide (Child); cEMEGc = Chronic Evaluation Media Guide (Child); iEMEGc = Intermediate Evaluation Media Guide (Child); CREG = Cancer Risk Evaluation Guide; aPica = Acute Pica; iPica = Intermediate Pica; RSL = EPA Regional Screening Value; CV = Comparison Value; ND = Not Detected; U = Not Detected Quantitation Limit; J = Reported value is estimated and actual value may be higher or lower, J- = Reported value is estimated, actual value is expected to be lower; PADEP MSC = PADEP Medium Specific Concentration; DTSC = California Department of Toxic Substances Control Screening value.

*RSL pertains to chromium(III), as there is no RSL for total chromium.

**Appendix Table C4. Creek Surface Water Concentrations and Comparison Values (CVs)**

**Note:** Our evaluation of Darby/Muckinipattis Creek surface water samples, which consisted of 8 co-located surface water and sediment samples, incorporated a duplicate (SD12) as opposed to the non-duplicate sample (SD4). SD12 had slightly but consistently higher concentrations than SD4 and we incorporated it instead of SD4 as a health protective approach. SD4 (and the SD12 duplicate) represent the sole sample taken along Muckinipattis Creek, and this sample was taken next to a public fishing dock near the convergence of Darby and Muckinipattis Creeks (Figure 2 of the main report).

| Chemical Sampled in Surface Water | N and % detected out of 8 samples | Range (ppb) | CV (ppb) | CV Source (non ATSDR CV listed if no CV available) | Number of Samples at or above a CV out of 8 samples (Sample ID/CV Type Exceeded) |
|---|---|---|---|---|---|
| *Metals* | | | | | |
| Aluminum | 8 (100.0%) | 202-674 | 7,000 | cEMEGc | 0 |
| Antimony | 0 (0.0%) | ND-2 U | 2.8 | RMEGc | 0 |
| **Arsenic** | 8 (100.0%) | 1.2-2.2 | 0.016<br>2.1 | CREG<br>cEMEGc | All (CREG)<br>1 (cEMEGc: SW11) |
| Barium | 8 (100.0%) | 54.8-75.3 | 1,400 | cEMEGc | 0 |
| Beryllium | 0 (0.0%) | ND-1 U | 14 | cEMEGc | 0 |
| Cadmium | 1 (12.5%) | ND-0.22 J | 0.70 | cEMEGc | 0 |
| Calcium | 8 (100.0%) | 28200-32700 | No CV | No CV | NA |
| Chromium | 3 (37.5%) | 2.5-24.3 | 100 | MCL | 0 |
| Cobalt | 8 (100.0%) | 0.4-6 J | 70 | iEMEGc | 0 |
| Copper | 8 (100.0%) | 2.9-15.7 | 70 | iEMEGc | 0 |
| Iron | 8 (100.0%) | 812-2130 | No CV | No CV | NA |
| Lead | 8 (100.0%) | 2.2-24.8 | No CV | No CV | NA |
| Magnesium | 8 (100.0%) | 10900-13300 | No CV | No CV | NA |
| Manganese | 8 (100.0%) | 141-297 | No CV | No CV | NA |
| Mercury | 0 (0.0%) | ND-0.2U | No CV | No CV | NA |
| Nickel | 8 (100.0%) | 1.8-18.1 | 140 | RMEGc | 0 |
| Potassium | 8 (100.0%) | 3830-4480 | 53,000 | No CV | 0 |
| Selenium | 0 (0.0%) | ND-5.0 U | 35 | RMEGc | 0 |
| Silver | 0 (0.0%) | ND-1.0 U | 35 | RMEGc | 0 |
| Sodium | 8 (100.0%) | 35400-40200 | No CV | No CV | 0 |
| Thallium | 0 (0/0%) | ND-1.0 U | 2 | MCL | 0 |
| Vanadium | 8 (100.0%) | 1.8-3.1 J | 70 | iEMEGc | 0 |
| Zinc | 8 (100.0%) | 9.6-104 | 2,100 | cEMEGc | 0 |
| **Bold =** chemical exceeded comparison value (CV) for one or more samples. RMEGc, Reference Dose Media Evaluation Guide (Child); cEMEGc, Chronic Evaluation Media Guide (Child); iEMEGc; Intermediate | | | | | |

Evaluation Media Guide (Child); CREG, Cancer Risk Evaluation Guide; CV, Comparison Value; ND, Not Detected; U, Not Detected Quantitation Limit; J, Estimated Value; MCL, EPA's Maximum Contaminant Limit

**Appendix C References**

Weston (2018). Norwood Landfill. Final Site Inspection Report, Revision 1. Norwood, Delaware County, Pennsylvania. Available From: https://www.epa.gov/sites/production/files/2020-02/documents/final_norwood_landfill_esi_report_redacted_part1.pdf

# Appendix D. Non-Pica Health Effects Evaluation for Norwood Chemicals of Concern, based on EPA's 2017-2018 Environmental Sampling Data

The following sections are in-depth toxicological evaluations based on EPA's 2017-2018 environmental sampling data of the site.

## D.1. Benzo[a]pyrene and PAHs

*Overview*

Polycyclic aromatic hydrocarbons (PAHs) are a group of semi-volatile organic compounds (SVOCs), found throughout the environment in air, water, and soil. They are formed by the incomplete burning of substances such as coal, oil, wood, garbage, tobacco, and charbroiled meat. There are over 100 types of PAHs, which commonly exist as mixtures. The burning of wood in homes and vehicle exhaust are common and large sources of PAHs. For most members of the U.S. population, the greatest sources of PAHs are ingestion from foodstuffs or inhalation from tobacco smoke, wood smoke, or contaminated air (ATSDR 1995). Absorption of benzo[a]pyrene through ingestion is low in humans.

Benzo[a]pyrene is among the most widely studied PAHs. The EPA has classified benzo[a]pyrene as "carcinogenic to humans" based on strong and consistent animal and human evidence (EPA 2017a). Cancer studies on humans have mostly involved workers exposed to benzo[a]pyrene and other PAHs from occupations such as chimney sweeping, coal tar distillation, or coal gasification. Often, but not always, the site of tumor induction for carcinogenic PAHs is influenced by the route of administration (e.g., stomach tumors following ingestion, lung tumors following inhalation, skin tumors following dermal exposure; ATSDR 1995).

*Health effects evaluation*

Several PAHs exceeded CVs in residential and non-residential soil (Appendix C1, C2). Sediment PAHs did not exceed CVs.

We evaluated PAH soil concentrations as a mixture using California's Office of Environmental Health Hazard Assessment's (OEHHA) Potency Equivalent Factors (PEFs) relative to benzo(a)pyrene (OEHHA 2015). For PAHs lacking OEHHA PEFs, we used Toxic Equivalency Factor (TEF) values from ATSDR (ATSDR 1995). Table D.1.1. shows the highest, second highest, and lowest benzo[a]pyrene-equivalent concentrations above screening for residential samples, and the highest non-residential sample (SS6). The residence with the highest benzo[a]pyrene-equivalent PAHs (13.49 mg/kg) had concentrations 3.5 times higher than that of the next highest residential concentrations (3.8 mg/kg, Table D.1.1).

**Table D.1.1. Benzo(a)pyrene Equivalent calculations for the residential and non-residential soil samples with detected soil benzo[a]pyrene concentrations above screening**

| **Polycyclic aromatic Hydrocarbon (PAH)** | **PEF/TEF[1]** | **PAH Concentrations in soil (mg/kg) multiplied by the PEF/TEF** | | | |
|---|---|---|---|---|---|
| | | Lowest Residential Sample Above Screening | **Highest Residential Sample** | Second Highest Residential Sample | Highest Non-residential Sample (SS-6) |
| Benzo(a)pyrene | 1 | 0.12 | 9.7 | 2.7 | 2.3 |
| Benzo(a)anthracene | 0.1 | 0.013 | 0.91 | 0.25 | 0.29 |
| Benzo(b)-fluoranthene | 0.1 | 0.017 | 1.5 | 0.44 | 0.47 |
| Benzo(k)-fluoranthene | 0.1 | 0.0046 | 0.46 | 0.14 | 0.12 |
| Indeno(1,2,3-cd)- pyrene | 0.1 | 0.0053 | 0.69 | 0.2 | 0.14 |
| Benzo(g,h,i)perylene | 0.01 | ND | 0.076 | 0.022 | 0.013 |
| Chrysene | 0.01 | 0.0012 | 0.099 | 0.032 | 0.043 |
| Anthracene | 0.01 | 0.00036 | 0.013 | 0.0046 | 0.14 |
| Dibenzo(a,h)anthracene | 2.44[2] | ND | NS | NS | 1.0004 |
| Phenanthrene | 0.001 | 0.00011 | 0.0065 | 0.0025 | 0.011 |
| Fluoranthene | 0.001 | 0.00025 | 0.018 | 0.0063 | 0.005 |
| Pyrene | 0.001 | 0.00019 | 0.015 | 0.0047 | 0.0086 |
| **Benzo(a)pyrene equivalent** | | **0.16** | **13.49** | **3.80** | **4.42** |

ND = Not Detected; NS = Not Sampled; PEF = Potency Equivalency Factor relative to Benzo(a)pyrene; TEF = Toxic Equivalency Factor relative to Benzo(a)pyrene; SS = the non-residential sample number.

[1]PEFs for benzo(a)pyrene, benzo(a)anthracene, benzo(b)fluoranthene, benzo(k)fluoranthene, indeno(1,2,3-cd)pyrene, dibenzo(a,h)anthracene and chrysene are derived from California's Office of Environmental Health and Hazard Assessment values (OEHHA, 2015 p. G-3). OEHHA PEFs do not exist for the remaining PAHs and therefore we used ATSDR Toxic Equivalency Factors for Benzo(g,h,i)perylene, phenanthrene, fluoranthene, and pyrene (ATSDR 1995).

**[2]** The TEF for dibenzo[a,h]anthracene is based on the ratio of OEHHA's oral CSFs for dibenzo(a,h)anthracene (i.e., over the age sensitivity factor of 1.7 $(mg/kg/day)^{-1}$ for benzo[a]pyrene.

We then used the benzo[a]pyrene equivalent values calculated in Table D.1.1 to estimate combined ingestion and dermal exposures. We used a default benzo[a]pyrene bioavailability of 1.0 and dermal absorption fraction of 0.13 as part of the calculation. To evaluate excess cancer risk, we used ATSDR's recommended cancer slope factor (CSF) of 1.7 $(mg/kg/day)^{-1}$, which is derived from an OEHHA factor that accounts for age sensitivity to benzo[a]pyrene (OEHHA 2010). Estimated exposure doses for the highest benzo[a]pyrene equivalent concentrations were calculated in PHAST and shown in Table D.1.2 below.

**Table D.1.2. Calculated Exposure Doses, Hazard Quotients and Cancer Risk Estimate for <u>highest</u> benzo[a]pyrene-equivalent PAH soil sample**

| Sample type | Conc (µg/kg) | Exposure Group | Ingestion and Dermal Exposure Dose (ED) µg/kg/day | Chronic Hazard Quotient (ED/RfD) RfD: 0.3 µg/kg/day | Excess cancer risk estimate[1] |
|---|---|---|---|---|---|

| | | | | | |
|---|---|---|---|---|---|
| Residential | 13,490 (highest) | Adult | 0.026 | <1 | 2 in 100,000 |
| | | Child (birth to <1 y) | **0.34** | **1.1** | 3 in 10,000 |
| | | Child (1 to <2 y) | **0.31** | **1.0** | |
| | | Child (2 to <21 y) | <0.3 | <1 | |

**Bold values =** Equivalent to or exceedance of an EPA's Reference Dose. Conc = Concentration in surface soil; RfD = EPA's reference dose; y = year of age. There are no intermediate or acute health guidelines for benzo[a]pyrene. [1]cancer risk estimates were calculated using ATSDR's recommended cancer risk slope factor of 1.7 $(mg/kg/day)^{-1}$ for benzo[a]pyrene. Estimates account for children (<21 years) and adults (21 years or older).

*Non-cancer health effects*

As shown in Table D.1.2, exposure doses for adults based on the highest benzo[a]pyrene equivalent concentrations were well below EPA's chronic RfD of 0.30 µg/kg/day. Conversely, childhood doses for birth to <1 year, of 0.34 µg/kg/day, and 1 to <2 years, of 0.31 µg/kg/day, exceeded EPA's benzo[a]pyrene oral RfD of 0.30 µg/kg/day.

EPA's RfD for benzo[a]pyrene is derived from a study that found altered neurodevelopment in Sprague Dawley rats (Chen et al. 2012; EPA 2017a). The rats were exposed to benzo[a]pyrene on postnatal days 5-11 days and those with higher exposures performed worse on neurobehavioral tests as juveniles and adults. From these studies EPA modeled a lower bound benchmark dose (BMDL) estimate of 92 µg/kg/day and then applied uncertainty factors to derive an RfD of 0.30 µg/kg/day. The highest Norwood exposure doses, for children up to age 1 year of 0.34 µg/kg/day, are at least 270 times lower than EPA's BMDL of the principal study (Chen et al. 2012). **Therefore, it is unlikely that adults and children chronically exposed to the highest benzo[a]pyrene-equivalent concentrations at Norwood would experience adverse non-cancer health effects.**

ATSDR has not derived intermediate or acute oral MRLs for benzo[a]pyrene. The highest Norwood exposure doses for adults, of 0.026 µg/kg/day, and children, of 0.34 µg/kg/day, are well below levels of significant exposure (LSE) of most intermediate animal studies, which have often found evidence for adverse health effects at 3,000 µg/kg/day or higher (EPA 2017b). One exception is a 90-day study by Chung et al. 2011 that observed histological changes in seminiferous tubules in male Sprague-Dawley rats. The rats were exposed orally to benzo[a]pyrene between 1-100 µg/kg/day. In its Integrated Risk Information System (IRIS) assessment for benzo[a]pyrene, EPA noted limited confidence in this study due to its reporting methods, and it did not include the study in its dose-response analysis for its assessment (EPA 2017b). We therefore do not consider this 1-100 µg/kg/day level relevant for our evaluation.

As for acute-duration studies, estimated Norwood exposure doses are at least 29,000 times below the lowest effect levels identified in a 10-day mouse study by Mackenzie and Angevine 1981 (ATSDR 1995). Acute studies emerging more recently have evaluated developmental endpoints in animal offspring, such as increases in blood pressure or cognitive effects from short-term gestational or postnatal exposures to benzo[a]pyrene (EPA 2017b). Other than the

Chen et al. 2012 postnatal study described in this section, the lowest thresholds for these effects often occurred at 300-2,000 µg/kg/day (EPA 2017b), still orders of magnitude above the highest Norwood estimates. **Based on these available toxicological studies, we would not expect adults or children exposed to the highest benzo[a]pyrene-equivalent exposures for acute or intermediate duration to experience adverse non-cancer health effects.**

*Neurological and Immunological considerations*

Benzo[a]pyrene is a developmental neurotoxicant (EPA 2017b), with the most sensitive thresholds derived from the Chen et al. 2012 study discussed in this section. As mentioned, EPA's lower bound estimate from this study of 92 µg/kg/day is more than 270 times' the highest exposure doses at Norwood, of 0.34 µg/kg/day.

Evidence suggests that benzo[a]pyrene immunotoxicity is a possible human health hazard (EPA 2017b), and ATSDR notes that it is prudent to consider that PAHs may pose an immunotoxic risk to humans in areas surrounding hazardous waste sites (ATSDR 1995). A few studies on coke oven workers found that PAH inhalation reduced certain antibody levels (EPA 2017b); however, these exposures are not directly comparable to Norwood. In studies in which rats were exposed orally to benzo[a]pyrene, researchers found significantly reduced antibody levels, changes in thymus weight, and other immune effects, often at doses of 10,000-90,000 µg/kg/day (EPA 2017b). These doses are a least 29,000 times' the highest Norwood exposure dose of 0.34 µg/kg/day.

Benzo[a]pyrene may also induce autoimmune responses (ATSDR 1995). A study on laboratory rats by Faiderbe et al. 1992 found evidence for increased autoimmune antibodies occurring in rats injected with 11,100 µg/kg benzo[a]pyrene and observed up to 50 days. This suggests that benzo[a]pyrene-induced neoplasia may alter the metabolism of endogenous substances, resulting in the production of autoimmune antibodies to those substances (ATSDR 1995). These effects from this direct injection study occurred at a far higher threshold (11,100 µg/kg, or >32,000 times above) the highest Norwood exposure dose of 0.34 µg/kg/day.

*Cancer health effects*

Lifetime excess cancer risk was calculated using ATSDR's-recommended cancer slope factor of 1.7 $(mg/kg/day)^{-1}$ for benzo[a]pyrene. Estimated excess cancer risks based on the highest beno[a]pyrene-equivalent PAH levels at Norwood were **3 in 10,000 for children and 2 in 100,000 for adults**. For the next highest residential benzo[a]pyrene-equivalent PAH sample, lifetime excess cancer was **7 in 100,000 for children and 5 in a million** for adults (data not shown).

Our cancer risk estimates assume daily, year-round exposure to the highest benzo[a]pyrene equivalent PAH concentrations detected in Norwood soil for 33 consecutive years of residential occupancy for adults, and 20 consecutive years for children. Thirty-three years is the 95th percentile of U.S. residential occupancy (EPA 2011); thus the estimate would be lower for an

adult who did not live in the same home for 33 years consecutively, or for a child who did not live at the same household up to 21 years of age.

On average in the U.S., approximately 1 in 2 men and 1 in 3 women are at risk for developing cancer at some point during their lifetime (American Cancer Society 2020).

## D.2. Di(2-Ethylhexyl)Phthalate (DEHP)

*Overview*

Di(2-Ethylhexyl)Phthalate (DEHP) is not found naturally in the environment but produced commercially as a plasticizer to make polyvinyl chloride (PVC) products soft and flexible (ATSDR 2022). It is found in many common items such as furniture upholstery, shower curtains, garden hoses, toys, shoes, automobile upholstery, medical tubing, and blood storage bags. Many manufacturers have phased out DEHP. Prior to its phase out, approximately 95% of the U.S. population was exposed to DEHP in foods, packaging and personal care products, though usually at very low levels.

DEHP predominately enters the environment by being disposed into industrial and municipal waste landfills, where it tends to stick to soil. Humans are primarily exposed through food; as with many phthalates, DEHP tends to leach from plasticizer-based containers or wraps and onto food. Once entering the body by ingestion, DEHP is rapidly broken down and usually excreted within 24 hours. Most information on DEHP toxicity comes from animal studies (ATSDR 2022).

Agencies have classified DEHP as a "probable human carcinogen" (EPA), reasonably anticipated to be a human carcinogen (U.S. Department of Health and Human Services) and "possibly carcinogenic to humans" (IARC) based on sufficient evidence in animals, but few human studies have evaluated cancer endpoints. The most consistent animal tumor site following high DEHP exposures (>350,000 µg/kg/day) is the liver (ATSDR 2022).

*Health effects evaluation*

DEHP was detected in all residential samples but only two exceeded a CV (Appendix C1). Both exceedances were estimated (J) values, indicating that actual concentrations may be higher or lower than the listed value. Non-residential samples did not exceed a CV. Estimated exposure doses from the highest residential DEHP concentration are shown in Table D.2.1 below.

**Table D.2.1. Calculated Exposure Doses, Hazard Quotients and Excess Cancer Risk Estimates based on the highest DEHP soil sample**

| Sample type and Conc (µg/kg) | Exposed Population | Estimated Ingestion and Dermal Exposure Dose (ED) µg/kg/day | Chronic Hazard Quotient<br><br>MRL: NA | Int Hazard Quotient (ED/MRL)<br><br>MRL: 0.1 | Acute Hazard Quotient<br><br>MRL: 3 | Excess Cancer Risk Estimate[1] |
|---|---|---|---|---|---|---|
| Residential, | Adult | 0.012 | NA | <1 | <1 | 7 in 100,000,000 |

| | | | | | | |
|---|---|---|---|---|---|---|
| 6,800 J (highest) | Child, birth to <1y | 0.16 | <1 | **1.6** | <1 | 2 in 10,000,000 |
| | Child, 1 to <2y | 0.15 | <1 | **1.5** | <1 | |
| | Child (2 to <21y, highest estimate) | 0.098 | <1 | <1 | <1 | |
| **Bold values =** Exceedance of a Minimal Risk Level (MRL; Please see Appendix B3 for more information). Conc = Concentration (µg/kg) in surface soil; Int = Intermediate-duration exposure; J = value is an estimate; NA = Not Applicable; y = year of age. MRLs are expressed in µg/kg/day.<br>[1]cancer risk estimates were calculated using EPA's cancer risk slope factors of 0.14 $(mg/kg/day)^{-1}$ for DEHP. Estimates account for children (<21 years) and adults (21 years or older). | | | | | | |

*Non-cancer health evaluation*

As shown in Table D.2.1, estimated Norwood exposure doses for adults and children were 0.012 µg/kg/day and 0.16 µg/kg/day, respectively. These doses are well below EPA's chronic oral reference dose of 20 µg/kg/day. As of January 2022, ATSDR does not recommend use of EPA's chronic oral RfD of 20 µg/kg/day for health assessments, and ATSDR has not derived its own chronic health guideline for DEHP due to insufficient data. We therefore compared the highest DEHP Norwood exposure doses to reported levels of significant exposure (LSEs) that induced health effects in chronic-duration, scientific studies.

The lowest chronic LSE identified from ATSDR's toxicological profile was from a study by Kamijo et al. 2007 that found elevated blood pressure and adverse kidney effects in mice exposed to 9,500 µg/kg/day DEHP for 22 months (ATSDR 2022). These reported effect levels are far higher (60,000-790,000 times) than the highest Norwood exposure doses for children (0.16 µg/kg/day) and adults (0.012 µg/kg/day). **Based on these reported effect levels, children and adults' exposure to the maximum DEHP soil concentrations for chronic duration (a year or more), are unlikely to experience adverse non-cancer health effects.**

In addition, Norwood exposure doses for children (0.16 µg/kg/day) and adults (0.012 µg/kg/day) were below ATDR's acute MRL of 3 µg/kg/day. **Therefore, children and adults exposed to the maximum DEHP soil concentrations for a short duration (1-14 days) are unlikely to experience adverse non-cancer health effects.**

By contrast, Norwood exposure doses for children up to age 2 years (highest: 0.16 µg/kg/day) exceeded ATSDR's intermediate MRL of 0.1 µg/kg/day. These doses produced hazard quotients (HQs) > 1.

ATSDR's intermediate MRL of 0.1 µg/kg/day is derived from a 2015 study by Zhang et al. finding altered reproductive system development among first and second generation female mice. Female mice offspring of mothers exposed to 40 µg/kg/day DEHP during gestation experienced altered folliculogenesis and other effects. These generational effects in mice are difficult to conceptualize or extrapolate for human populations.

Separate studies found that mice that were sensitized to ovalbumin, or egg allergy, and exposed to 30 µg/kg/day DEHP experienced increases in certain antibody and T-cell activity

(Han et al. 2014; Guo et al. 2012; ATSDR 2022). These effects weren't found in mice not sensitized to ovalbumin (OVA). This 30 µg/kg/day effect level is the lowest threshold for which adverse health effects were observed in intermediate-duration DEHP studies (ATSDR 2022).

The enhanced immune responses found in OVA-sensitized mice at 30 µg/kg/day DEHP are 188 times' the highest Norwood estimated exposure dose, of 0.16 µg/kg/day in children up to 1 year of age (Table D.2.1). Further, the human health relevance of findings from sensitized animals is uncertain in the absence of clear evidence that the immune system is a target of DEHP toxicity in humans or unsensitized animals (ATSDR 2022). **Therefore, for adults and children, adverse non-cancer health effects are unlikely to occur from intermediate-duration exposures to DEHP at Norwood.**

*Neurological and Immunological Considerations*

In human adults, epidemiological data are extremely limited regarding the neurological effects from DEHP exposure. In infants, the potential for DEHP to alter neurodevelopment has been studied through prenatal exposures, as measured by maternal urinary levels during pregnancy. These studies have contrasted in findings, limiting ability to draw firm conclusions. Studies on mice and rats have evaluated the effect of gestational or early postnatal exposures on effects such as impaired reflexes and altered neurobehavior. A study on mice by Barakat et al. 2018 found that a DEHP maternal dose of 200 µg/kg/day led to increased anxiety in offspring, though another measure of anxiety in the same study did not find this evidence until maternal exposures reached 750,000 µg/kg/day (ATSDR 2022). In other available studies, the lowest maternal doses associated with neurodevelopmental effects were 1,000 µg/kg/day in mice and 30,000 µg/kg/day in rats (ATSDR 2022). These above doses are more than a thousand to a million times higher than the highest Norwood exposure doses.

Studies on DEHP and human immune system effects are limited and have produced inconsistent results (ATSDR 2022). A few prenatal studies found associations between maternal DEHP and increased risk of wheezing, asthma, and higher serum immunoglobulin E (IgE) levels in their children. However, other studies have not found these associations. The animal immune system is a sensitive target for oral DEHP exposure, particularly for allergen-sensitive animals. As discussed, the relevance of these findings are still uncertain in humans, and the lowest effect levels in laboratory animal studies, of 30 µg/kg/day in OVA-sensitized mice, were 188 times above the highest Norwood exposure doses.

*Cancer health effects*

Excess cancer risk was calculated using EPA's Cancer Slope Factor of 0.14 $(mg/kg/day)^{-1}$. The highest excess cancer risk was **2 in 10 million for children and 7 in 100 million** for adults (Table D.2.1). Our excess cancer estimates assume daily, year-round exposure to Norwood DEHP concentrations for 33 consecutive years of adult and 20 years of child residency. The estimate

would be lower for an adult who did not live in the same home for 33 years consecutively, or for a child who did not live at the same household up to 21 years of age.

## D.3. Polychlorinated Biphenyls (PCBs)

*Overview*

PCBs are a group of man-made organic chemicals that were widely used as coolants and lubricants in transformers, capacitators, and other electrical equipment. The U.S. stopped their production in 1977 due to evidence that they persisted in the environment for a long period and caused human and environmental harm. Though no longer produced in the U.S., PCBs can be found in small amounts in air, water and soils (ATSDR 2000). People can also be exposed from old transformers, capacitators or related equipment, or by eating contaminated fish, meat and poultry. Once absorbed in the body, PCBs adhere to fat and some types may remain for multiple years. PCBs accumulate in fish and mammals at higher levels than initially found in the environment.

In 2013 the International Agency for Research on Cancer (IARC) deemed PCBs as carcinogenic to humans due to strong evidence for malignant melanoma risk and sufficient evidence in laboratory animals (IARC 2015). EPA lists PCBs as a probable human carcinogen (ATSDR 2000). The U.S. Department of Health and Human Services (DHHS) classifies PCBs as "reasonably anticipated to be human carcinogens" based on evidence in laboratory animals (NTP 2018).

*Health effects evaluation*

EPA sampled two chemically-similar PCB mixtures, Aroclor 1254 and Aroclor 1260, which contain 54% and 60% chlorine by weight, respectively. We evaluated them as CoCs due to community concerns about historic PCB contamination. Aroclor 1260 was detected with greater frequency in both residential (76%) and nonresidential soil (88%) than Aroclor 1254 was in residential (5%) and non-residential samples (18%) (Appendix Tables C1, C2); however, their concentrations were similar. Three Aroclor 1260 samples and one Aroclor 1254 sample exceeded a CV. We considered each Aroclor separately as opposed to the sum of their concentrations because Aroclor 1254 was detected much less frequently than Aroclor 1260.

Non-residential soil had the highest detected Aroclor 1254 concentration, of 450 µg/kg. Residential soil had the highest detected Aroclor 1260 concentration, of 310 µg/kg. Estimated exposure doses for these concentrations are shown in Table D.3.1.

**Table D.3.1. Calculated Exposure Doses, Hazard Quotients and Excess Cancer Risk Estimates based on the highest PCB soil samples**

| Chemical | Sample type, and conc (µg/kg). | Exposed Population | Estimated Ingestion and Dermal Exposure Dose (ED) µg/kg/day | Chronic Hazard Quotient<br>MRL[2]: 0.02 | Int Hazard Quotient<br>MRL: 0.03 | Excess Cancer Risk Estimate[3] |
|---|---|---|---|---|---|---|
| Aroclor 1254 | Non-residential, | Adult | 0.00089 | <1 | <1 | 8 in 10,000,000 |
| | | Child (6-10y)[1] | 0.0043 | <1 | <1 | 1 in 1,000,000 |

| | | | | | | |
|---|---|---|---|---|---|---|
| | 450 (highest) | | | | | |
| Aroclor 1260 | Residential, 310 (highest) | Adult | 0.00062 | NA | NA | 5 in 10,000,000 |
| | | Child (birth to <1y) | 0.0079 | | | 2 in 1,000,000 |

Conc = Concentration (µg/kg) in surface soil; Int = Intermediate; MRL = Minimal Risk Level; NA, Not Applicable; y = year of age

[1] The youngest ages we considered for childhood exposures to non-residential soil were ages 6-10. We assumed that children younger than this age were not regularly exposed to non-residential soil.

[2] MRLs are expressed in µg/kg/day and are shown for Aroclor 1254. There are no MRLs for Aroclor 1260, and no acute MRL for Aroclor 1254.

[3] There is no cancer slope factor specific to Aroclor 1254 or Aroclor 1260. As a result we calculated cancer risk using EPA's CSF for PCBs (nonspecified) of 2 $(mg/kg/day)^{-1}$. For residential soil, estimates account for children (birth to <21 years) and adults (21 years or older). For non-residential soil, estimates account for children (6 to <21 years) and adults (21 years or older). If assuming children from *birth* up to age 21 are exposed to the highest Aroclor 1254 concentrations in non-residential soil, this excess risk estimate increases from 1 in 1,000,000 to 2 in 1,000,000.

*Non-cancer health evaluation*

***Aroclor 1254.*** As shown in Table D.3.1, Aroclor 1254 exposure doses for adults, of 0.00089 µg/kg/day, and children, of 0.0043 µg/kg/day, did not exceed chronic or intermediate MRLs of 0.02 and 0.03 µg/kg/day, respectively. **Therefore, exposures to the maximum soil Aroclor 1254 concentrations for intermediate or chronic duration (15 days or more) are unlikely to result in adverse non-cancer health effects.**

ATSDR has not derived an acute MRL for Aroclor 1254, so we compared the highest Norwood doses to the lowest acute levels of significant exposure (LSE; ATSDR 2000). Norwood's highest exposure dose was 0.0043 µg/kg/day for children. This dose is more than 230,000 times lower than an LSE of 1,000 µg/kg/day reported in studies by Carter et al., which found increased relative liver weight and serum cholesterol in rats (ATSDR 2000). **Based on these reported effect levels, we would not expect these adverse health effects to occur from acute exposures (1-14 days) to the highest detected Aroclor 1254 concentrations in Norwood soil.**

***Aroclor 1260*.** There are no chronic, intermediate, or acute health guidelines for Aroclor 1260, so we compared Norwood exposure doses to the lowest levels of significant exposure (LSE) that induced toxicity (ATSDR 2000).

As shown in Table D.3.1, the highest Norwood exposure dose was 0.0079 µg/kg/day for infants up to 12 months. This dose is 126,000 times lower than a chronic 1,000 µg/kg/day LSE for hepatic and endocrinal effects found in rats exposed to Aroclor 1260 for 2 years in a study by Mayes et al. (ATSDR 2000). The Norwood exposure dose estimate is also below the lowest thresholds found in intermediate-duration Aroclor 1260 studies: in an 8 week study by Vos and de Roji (1972), guinea pigs exposed to 800 µg/kg/day Aroclor 1260 experienced decreases in gamma globulin-containing cells, indicative of some immunosuppression. Lower doses were

not tested but this 800 µg/kg/day LSE is more than 100,000 times' the highest Norwood exposure dose.

No toxicological acute-duration studies were identified to compare Norwood exposure doses against LSEs (ATSDR 2000). **Based on the lowest toxicity thresholds discussed in the above studies, we would not expect Aroclor 1260 exposures of intermediate or chronic duration (15 days or more) at Norwood sampling locations to induce these adverse health effects.**

*Neurological and Immunological Considerations*

Mounting evidence suggests that PCBs may contribute to neurodevelopmental alterations in neonates and infants of the highest exposed mothers. Animal studies have also found neurodevelopmental impacts. ATSDR's intermediate MRL for Aroclor 1254 is 0.03 µg/kg/day (ASTDR 2000). It is derived from a study on monkeys and protective against neurobehavioral effects. The highest Norwood exposure doses to Aroclor 1254 (0.0043 µg/kg/day), and Aroclor 1260 (0.0079 µg/kg/day), fell well below this intermediate MRL of 0.03 µg/kg/day (Table D.3.1).

Studies suggest that the human immune system is sensitive to PCBs, particularly in infants exposed *in utero* and/or breast feeding to mothers with high PCB levels. Animal studies also indicate that PCBs may suppress the immune system. ATSDR's chronic MRL for Aroclor 1254 is 0.02 µg/kg/day and derived from a two year study on Rhesus monkeys that reported decreased antibody response from Aroclor 1254 exposures (ATSDR 2000). The highest PCB exposure doses at Norwood were well below this 0.02 µg/kg/day chronic MRL, which is protective against these markers of immunosuppression.

*Cancer Health Effects*

There is no Cancer Slope Factor (CSF) for Aroclor 1254 or Aroclor 1260. We thus used EPA's CSF of 2 $(mg/kg/day)^{-1}$ for PCBs (nonspecified). Based on this CSF, the highest excess cancer risks for children and adults **were 2 in a million for children and 8 in 10 million for adults** (Table D.3.1.). Our residential calculated excess cancer risk estimates assume daily, year-round exposure to the highest Aroclor concentrations detected in soil for 20 consecutive years (child) or 33 consecutive years (adult). The estimate would be lower for an adult who did not live in the same home for 33 years consecutively, or for a child who did not live at the same household up to 21 years of age.

## D.4 Aldrin and Dieldrin

Aldrin and dieldrin are insecticides of the 1950s and 60s that are no longer produced or used. Though discontinued for several decades, these compounds remain in the environment for long periods. Aldrin readily changes to dieldrin when it enters the environment or absorbed in the

body. Once absorbed, dieldrin has a lengthy half-life of an estimated 369 days (ATSDR 2021), meaning it takes roughly a year for the body to eliminate half absorbed dieldrin.

Most people are exposed to aldrin or dieldrin by eating food or drinking water with either compound. Dieldrin has been detected in food such as root crops, dairy products and meat. Since these compounds are no longer produced or used, U.S. exposures are low to undetectable.

Based on animal studies, EPA and IARC have deemed aldrin and dieldrin as probable human carcinogens. Animal studies largely find tumors affecting the mouse liver, though evidence suggests that the liver of mice is uniquely susceptible to aldrin and dieldrin carcinogenicity. Human studies on cancer risk from these compounds, such as breast cancer risk, have been inconclusive.

*Health effects evaluation*

Aldrin was detected in 6 of 21 residential samples (29%) ranging from 0.28-53 µg/kg and 3 of 17 non-residential samples (18%) ranging from: 0.69-18 µg/kg. A single aldrin sample exceeded a CREG CV.

Dieldrin was detected in 19 of 21 residential samples (91%) ranging from 0.08-1,300 µg/kg and 4 of 17 non-residential samples (24%) ranging from 2.6-85 µg/kg. Two dieldrin samples exceeded a CREG (Appendix Tables C1, C2).

Residential aldrin and dieldrin levels exceeded non-residential levels. As such, we calculated exposure doses based on the highest residential levels. Exposure dose estimates are shown in Table D.4.1 below.

**Table D.4.1. Calculated Exposure Doses, Hazard Quotients and Excess Cancer Risk Estimates based on the highest concentration of Aldrin and Dieldrin in soil samples**

| CoC | Sample type, and conc (µg/kg). | Exposed Population | Estimated Ingestion and Dermal Exposure Dose (ED) µg/kg/day | Chronic Hazard Quotient | Int Hazard Quotient | Acute Hazard Quotient | Excess Cancer Risk Estimate[3] |
|---|---|---|---|---|---|---|---|
| Aldrin[1] | Residential 53 (highest) | Adult | 0.000094 | <1 | NA | <1 | 7 in 10,000,000 |
| | | Child (birth to <1y) | 0.0013 | <1 | NA | <1 | 2 in 1,000,000 |
| Dieldrin[2] | Residential 1,300 (highest) | Adult | 0.0023 | <1 | <1 | NA | 2 in 100,000 |
| | | Child (birth to <1y) | 0.031 | <1 | <1 | NA | 5 in 100,000 |

CoC = Chemical of Concern; Conc = Concentration in surface soil; Int = Intermediate; MRL = Minimal Risk Level; NA = Not Applicable; y = year of age
[1]MRLs for Aldrin in µg/kg/day: 0.04 (chronic), 2 (acute). There is no intermediate MRL for aldrin.
[2]MRLs for Dieldrin in µg/kg/day: 0.05 (chronic), 0.1 (intermediate). There is no acute MRL for dieldrin.
[3]Cancer risk estimates were evaluated using EPA's Cancer Slope Factor of $(17mg/kg/day)^{-1}$ for aldrin and $(16mg/kg/day)^{-1}$ for dieldrin. They encompass children (<21 years) and adults (21 years or older).

*Non-cancer health evaluation*

***Aldrin.*** The highest Norwood estimated exposure doses for aldrin were 0.000094 µg/kg/day for adults and 0.0013 µg/kg/day for children. These doses were well below ATSDR's provisional chronic and acute MRLs of 0.04 and 2 µg/kg/day, respectively (Table D.4.1). **Therefore, adults or children exposed to the highest aldrin soil concentrations for chronic (>1 a year) or acute duration (1-14 days) are unlikely to experience adverse non-cancer health effects.**

ATSDR has not derived an intermediate MRL for aldrin, so we compared Norwood exposure doses to the lowest intermediate LSEs of the scientific literature. Few toxicological studies have assessed intermediate-duration aldrin exposures, and no reliable intermediate human data was identified (ATSDR 2021). The lowest effect levels identified in laboratory animal studies ranged from 260 µg/kg/day for developmental effects, to 890 µg/kg/day for neurological and gastrointestinal effects; these effects were mainly found from studies of the 1950s, when aldrin was still in production. Though the studies found effects deemed "serious" by ATSDR, their LSEs (260-890 µg/kg/day) are at least 200,000 times the highest aldrin exposure dose from Norwood soil, of 0.0013 µg/kg/day for children at birth to 1 year of age. **Therefore, we would not expect adults or children exposed to the highest aldrin soil concentrations for an intermediate-duration period (15-364 days) to experience these adverse non-cancer health effects.**

***Dieldrin.*** The highest Norwood estimated exposure doses for dieldrin were 0.0023 µg/kg/day for adults and 0.031 µg/kg/day for children. These doses were below ATSDR's provisional chronic and intermediate MRLs of 0.05 µg/kg/day and 0.1 µg/kg/day, respectively (Table D.4.1). **Therefore, adults or children exposed to the highest dieldrin concentrations for intermediate or chronic duration (15 days or longer) are unlikely to experience adverse non-cancer health effects.**

ATSDR has not derived an acute oral MRL for dieldrin. The highest Norwood dieldrin exposure dose was 0.031 µg/kg/day. This dose is nearly 3,000 times lower than an acute LSE found by Loose et al. 1981, in which mice exposed to 90 µg/kg/day dietary dieldrin for two weeks experienced signs of impaired macrophage antigen processing (an immunological effect; ATSDR 2021). **Based on this study we would not expect these health effects to occur from acute exposures (1-14 days) to the highest detected Norwood dieldrin concentrations.**

*Neurological and Immunological Considerations*

Dieldrin and aldrin can stimulate the human central nervous system (CNS), causing hyperexcitation and generalized seizures. These effects have occurred at very high doses of 25,000-120,000 µg/kg (ATSDR 2021), more than 800,000 times the highest Norwood exposure dose of 0.031 µg/kg for children up to 12 months (Table D.4.1). Longer human studies on the neurological impact of dieldrin have produced conflicting results. One study identified by ATSDR did not find CNS effects in volunteers exposed to 3 µg/kg/day dieldrin for 18 months (ATSDR 2021). This 3 µg/kg/day chronic threshold at which no effects were observed is approximately 100 times' the highest Norwood dieldrin exposure dose of 0.031 µg/kg/day for infants up to 12 months (Table D.4.1). No Norwood exposure doses exceeded chronic aldrin or dieldrin MRLs, which are protective against adverse health effects from long term exposures.

Dieldrin can also affect the CNS of animals. ATSDR's intermediate MRL for dieldrin was derived from a Smith et al. 1976 study finding impaired learning in squirrel monkeys. These neurological effects were observed from exposures to 100 µg/kg/day dieldrin for 55 days (ATSDR 2021). This effect level is more than 3,000 times' the highest Norwood dose of 0.031 µg/kg/day. Aldrin's acute oral MRL of 2 µg/kg/day is derived from a mouse study and is also protective against neurodevelopmental effects, and the highest Norwood exposure dose did not exceed this MRL.

There are a few human studies on the immune system effects from aldrin or dieldrin exposure (ATSDR 2021). In a 2000 study by Dewailly et al. of 98 breastfed and 73 bottle-fed Inuit infants from Nunavik (Arctic Quebec, Canada), risk for experiencing 3 or more episodes of otitis media (middle ear infection) over the first year of life was reportedly increased with prenatal exposure to dieldrin, however no clinically relevant differences were noted between breastfed and bottle-fed infants with regard to immunologic parameters (ATSDR 2021). In animals (as previously discussed), Loose et al.'s 1981 study found evidence of immunosuppression in mice exposed to 90 µg/kg/day dieldrin for 2 weeks, which is 3,000 times' the highest estimated exposure dose at Norwood, of 0.031 µg/kg/day.

*Cancer Health Effects*

Excess cancer risk was calculated using EPA's Cancer Slope Factors of 17 $(mg/kg/day)^{-1}$ for aldrin and 16 $(mg/kg/day)^{-1}$ for dieldrin. The highest excess cancer risk was **2 in 100,000 for adults and 5 in 100,000 for children** based on the maximum dieldrin sample (Table D.4.1).

Our excess cancer estimates assume daily, year-round exposure to Norwood aldrin and dieldrin concentrations for 33 consecutive years of adult and 20 years of child residency. The estimate

would be lower for an adult who did not live in the same home for 33 years consecutively or for a child who did not live at the same household up to 21 years of age.

## D.5 Copper

*Overview*

Copper occurs naturally throughout the environment, including in all plants and animals, and is essential for good health. It is also produced through many industrial practices such as mining, and used in the manufacture of metal products. Copper tends to collect in the upper layers of soil and collect in river, lake, and estuary sediments.

Humans are exposed to copper from breathing air, drinking water, eating food or making skin contact. People consume approximately 1 mg of copper daily through diet, food and beverages. Drinking water is the primary source of excess copper, which can lead to nausea, vomiting, stomach cramps or diarrhea (ATSDR 2004). A very small percentage of infants and children are unusually sensitive to copper. EPA has not classified copper as a carcinogen due to a lack of adequate human or animal cancer studies.

*Health effects evaluation*

Copper was detected in all Norwood samples, with residential soil having the highest concentrations. Two of 21 residential samples (10%), 2 of 17 non-residential samples (12%), and all 8 sediment samples (100%) exceeded soil-pica CVs. *Non-pica* CVs were not exceeded. Creek surface water concentrations did not exceed CVs (Appendix Tables C1-C4).

Estimated exposure doses from the highest Norwood copper concentration, in residential soil, are shown in Table D.5.1.

**Table D.5.1. Calculated Exposure Doses and Hazard Quotients based on the highest Copper soil sample**

| Sample type, and Conc (µg/kg) | Exposed Population and Time Period | Estimated Ingestion and Dermal Exposure Dose (ED) µg/kg/day | Intermediate Hazard Quotient<br><br>MRL: 10 | Acute Hazard Quotient<br><br>MRL: 10 | Excess Cancer Risk Estimate[1] |
|---|---|---|---|---|---|
| Residential 264,000 (highest) | Adult | 0.35 | <1 | <1 | NA |
| | Child (birth to <1y) | 5.3 | <1 | <1 | |
| Conc = Concentration (µg/kg) in surface soil; MRL=Minimal Risk Level; NA = Not Applicable; y = year of age. MRLs are expressed in µg/kg/day. There is no chronic MRL or EPA RfD for copper.<br>[1]EPA has not classified copper as a carcinogen | | | | | |

*Non-cancer health effects*

ATSDR has not derived a chronic oral MRL for copper due to inadequacies in the toxicological database. In its 2004 toxicological profile for copper, ATSDR identified a single chronic duration study. In that study by Massie and Aiello 1984, there was a reported decrease in lifespan (a serious effect) in mice exposed to 42,000 µg/kg/day copper gluconate in drinking water for 2 years (ATSDR 2004). The same study did not find increases in body weight in mice exposed at the 42,000 µg/kg/day threshold. These thresholds are well above (7,900-120,000 times') the highest Norwood copper exposure doses of 5.3 µg/kg/day for children and 0.35 µg/kg/day for adults (Table D.5.1). Norwood exposures also fell below ATSDR's intermediate and acute MRLs for copper, of 10 µg/kg/day. **Therefore, adult and childhood exposures to the highest detected copper concentrations at Norwood are unlikely to result in adverse health effects.**

In April 2022, ATSDR released an updated Toxicological Profile for Copper (Draft for Public Comment), which includes new provisional MRLs. The public comment period for this draft closed on July 26, 2022. PADOH will assess Norwood exposure doses in the context of this updated profile as part of the addendum to this Health Consultation, which will encompass EPA's 2020 expanded sampling data.

*Neurological and Immunological Considerations*

There are limited data on neurological and immunological effects from copper exposure. In its 2004 toxicological profile, ATSDR noted no human studies and four intermediate-duration animal studies that evaluated neurological and/or immunological health endpoints. The animal studies observed evidence for decreased dopamine in the brain at 36,000 µg/kg/day in rats and impaired cellular and humoral immunity at 13,000 µg/kg/day in mice. These effect levels are far higher than the highest Norwood exposure doses. However, the toxicity of copper is highly species-dependent and the relevance of extrapolating copper-induced effects from rats to humans has not been fully evaluated (ATSDR 2004).

## D.6 Iron

Iron is an essential nutrient naturally present in many foods; it is also a dietary supplement (NIH 2021). Iron is an essential component of hemoglobin, a protein that transfers oxygen from the lungs to tissues in the body. Iron is also necessary for muscle metabolism, physical growth, neurological development, cell development, and hormone formation. Iron deficiency is not uncommon in the U.S. and can lead to anemia. Excess iron intake can lead to gastrointestinal effects, such as nausea, abdominal pain, and vomiting. Long-term excessive intake may lead to heart, pancreas, liver, and kidney damage.

Excessive iron accumulation in the body, known as secondary iron overload, can suppress the body's immune system. Secondary iron overload most often occurs in children from taking in too much iron in mineral supplements, in individuals with liver disease who undergo repeated

blood transfusions, and in individuals with the liver disease hemochromatosis that increases the rate of iron absorption (ATSDR 2017; EPA 2006; WHO 1996).

There is a health condition for which too much iron can be dangerous. Iron overload or hemochromatosis occurs when the body absorbs too much iron from foods (and other sources such as vitamins containing iron). Although hemochromatosis can have other causes, in the U.S. the disease is usually caused by a genetic defect. The genetic defect is inherited from both parents and is present at birth, but symptoms rarely appear before adulthood. The iron overload associated with hemochromatosis can be detected through two blood tests. Treatment consists of periodically taking blood from the arm, much like giving blood (CDC 2020).

*Health effects evaluation*

Iron was detected in all Norwood samples with creek sediment having the highest concentrations (Appendix Tables C1-C4). All soil and sediment samples exceeded EPA's residential Regional Screening Level (RSL) for iron in soil (5,500 mg/kg).

We calculated exposure doses for all media sampled, however Table D.6.1 below shows estimates for iron in sediment and residential soil. We calculated sediment exposure doses using the 95$^{th}$ Upper Confidence Limit of the mean (95 UCL) of the 8 samples, and residential exposure doses based on the highest detected concentration. Exposure doses account only for ingestion because there is no dermal absorption factor for iron.

Because there are no MRLs for iron, we compared Norwood doses to EPA's provisional reference dose of 700 µg/kg/day, which protects against GI effects in humans and encompasses subchronic (30-90 days) and chronic exposures (EPA 2006). EPA's provisional RfD is derived from an LSE of 1,000 µg/kg/day for gastrointestinal effects in humans.

We also compared Norwood exposures to Tolerable Upper Intake Levels (ULs) for children and adults. The Food and Nutrition Board of the National Research Council has developed ULs for iron based on reported gastrointestinal effects from ingestion of iron supplements. The ULs for children up to 13 years of age and adults are 40 and 45 mg/day, respectively (IOM 2001). Estimated Norwood exposure doses are shown in Table D.6.1.

**Table D.6.1. Calculated Exposure Doses and Hazard Quotients based on Iron concentrations in creek sediment and highest residential soil sample**

| Sample type, and Conc (mg/kg for soil/sediment) | Exposed Population and Time Period | Estimated Ingestion Exposure Dose (ED) RfD: µg/kg/day<br><br>(Provisional RfD: 700 µg/kg/day) | Hazard Quotient (ED/RfD) | Estimated daily Iron intake (mg) per day[2]<br><br>(Tolerable Upper Intake Level for adults: 45mg<br>For children: 40mg) | Excess Cancer Risk Estimate[3] |
|---|---|---|---|---|---|

| | | | | | |
|---|---|---|---|---|---|
| Residential 25,300 (highest) | Adult | 32 | <1 | 2.6 | NA |
| | Child (birth to <1y) | 490 | <1 | 3.8 | |
| Sediment 34,255 (95 UCL) | Adult | 43 | <1 | 3.4 | |
| | Child (6-10y)[1] | 220 | <1 | 7.0 | |

95 UCL = 95th Upper Confidence Limit of the Mean; Conc = Concentration (µg/kg) in; IOM = Institute of Medicine; NA = Not Applicable; y = year of age. Provisional RfD from EPA (2006) is expressed in µg/kg/day.
[1]The youngest age group we considered for sediment was children ages 6-10y, as we presumed children younger than 6y would not be regularly exposed to sediment.
[2]Calculated by multiplying the Exposure Dose in mg/kg/day by ATSDR's Exposure Dose Guidance for Body Weight (2016a). Example: for child up to 12 months, an estimated exposure dose is 490 µg/kg/day ÷ 1,000 = 0.49 mg/kg/day, × body weight estimate of 7.8 kg = estimated daily iron intake (in mg) of 3.8 mg. Body weight estimates by age can be found in ATSDR (2016a): Exposure Dose Guidance for Body Weight
[3]Iron is not considered a human carcinogen

As shown, the highest exposure doses in adults, of 43 µg/kg/day, and children, of 490 µg/kg/day, were below EPA's provisional RfD for iron of 700 µg/kg/day. Estimated exposures were also below IOM daily ULs (Table D.6.1). **Therefore, adults and children exposed to the highest Norwood iron concentrations are unlikely to experience chronic health effects.**

*Neurological and Immunological Considerations*

A daily estimate in mg/kg/day or µg/kg/day that induced adverse neurological and/or immunological effects was not available in EPA's peer reviewed toxicity profile for iron (EPA 2006).

## D.7 Manganese

Manganese is a naturally occurring substance found in many types of rocks and soil. It is ubiquitous in the environment and normal constituent of air, water, soil, and food. In manufacturing and commerce, it is mainly used to strengthen and harden steel. It is also found in many other products, including paints, fertilizer, cosmetics, and as a nutritional supplement.

Diet is the primary source of manganese intake. Adults consume between 0.7 and 10.9 mg of manganese per day, with higher intake among vegetarians who consume manganese-rich fruits, vegetables and nuts (ATSDR 2012a). Dermal exposure is not a typical pathway for manganese because it does not penetrate the skin readily.

Manganese is an essential nutrient and certain intake levels are necessary for human health. It is found in the brain and all mammalian tissues, with the highest human levels in the liver, pancreas and kidney. At high levels, manganese can be harmful to health, including neurological health. Children are potentially more sensitive to manganese toxicity than adults. There is no evidence that manganese causes cancer in humans (ATSDR 2012a).

*Health Effects Evaluation*

Manganese was detected in all Norwood samples (Appendix Tables C1-C4). Concentrations were similar in residential (105-553 mg/kg) and non-residential soil (165-710 mg/kg) which were comparable to typical U.S. values (Appendix Table H.3.1). Creek sediment had higher manganese concentrations (420-848 mg/kg), all of which were estimated ("J") values. Creek surface water manganese concentrations ranged from 141-297 µg/L.

All but 4 soil samples exceeded EPA's RSL CV of 180 mg/kg. A CV/RSL was not identified for surface water.

*PADOH Evaluation of Agency Health Guidelines for Manganese*

ATSDR has not derived acute, intermediate, or chronic duration oral MRLs for manganese. It established an interim guidance value of 160 µg/kg/day based on a Tolerable UL for adult dietary intake (ATSDR 2012a). However, children's nervous systems are more sensitive to the toxic effects of manganese (OEHHA 2006). ATSDR considered several studies and case reports for an MRL that found that school-aged children exposed to elevated manganese in drinking water over multiple years experienced neurological effects such as reduced performance on intellectual, verbal and perceptual reasoning tests. These studies and case reports had several limitations, such as lack of account for dietary manganese sources and small sample sizes (ATSDR 2012a). As a result, ATSDR recommends use of its 160 µg/kg/day guidance value for health assessments.

In a school-site risk assessment for childhood manganese exposure, California's Office of Environmental Health Hazard Assessment (OEHHA) set a chronic RfD of 30 µg/kg/day (OEHHA 2006). In so doing, OEHHA considered dietary manganese exposures, evidence that neonates absorb more manganese from the GI tract and are less likely to excrete it, and evidence that the developing brain is more sensitive to manganese. Our evaluation references OEHHA's RfD of 30 µg/kg/day for children in addition to ATSDR's interim guidance MRL of 160 µg/kg/day.

*Non-cancer health effects*

We calculated exposure doses for all media sampled, however Table D.7.1 below shows estimated doses for manganese in sediment, which had the highest concentrations, and for manganese in residential soil. Non-residential soil and creek surface water exposure doses were lower than these estimates.

**Table D.7.1. Calculated Exposure Doses and Hazard Quotients based on Manganese concentrations in creek sediment and highest residential soil sample**

| Sample type, and Conc (µg/kg) | Exposed Population and Time Period | Estimated Ingestion and Dermal Exposure Dose (ED), µg/kg/day | Hazard Quotient (ED/Int Guidance Value)<br><br>Int Guidance Value: 160 | Excess Cancer Risk Estimate[2] |
|---|---|---|---|---|
| Residential 553,000 (highest) | Adult | 1.4 | <1 | NA |
| | Child (birth to <1y) | 17 | <1 | |
| Sediment 813,200 (J) (95 UCL) | Adult | 2.1 | <1 | |
| | Child (6-10y)[1] | 10 | <1 | |

Conc = Concentration; 95UCL = 95th Upper Confidence Limit of the Mean; ED = Exposure Dose; NA = Not Applicable; y = years of age. Int Guidance Value = ATSDR's interim guidance value for manganese in µg/kg/day.
[1]The youngest age group we considered for sediment exposure was children ages 6-10y, as we presumed children younger than 6y would not be regularly exposed to sediment.
[2]Manganese is not considered a human carcinogen

The highest Norwood manganese exposure dose was 17 µg/kg/day in children up to 12 months. This is below ATSDR's interim guidance MRL of 160 µg/kg/day and OEHHA's chronic RfD of 30 µg/kg/day (Table D.7.1). **Therefore, exposures to the highest Norwood manganese concentrations for a year or more are unlikely to result in adverse health effects among adults or children.**

As there is no intermediate MRL for manganese, we compared the Norwood dose of 17 µg/kg/day to levels of significant exposure (LSE) in the toxicological literature (ATSDR 2012a). ATSDR's manganese profile identified a single intermediate-duration human study, by Finley et al. 2003 (ATSDR 2012a). In that study, no neurological effects such as decreased steadiness or ability to control muscular tremor were observed in 17 adult women exposed to 10 or 300 µg/kg/day dietary manganese for 8 weeks.

The highest Norwood exposure dose of 17 µg/kg/day is within the 10 or 300 µg/kg/day range of the Finley et al. 2003 study at which no effects were observed. There is uncertainty comparing the Norwood child exposure dose to this single study on adults. **However, based on this study, we would not expect these adverse neurological effects to occur in children.**

The highest Norwood exposure dose of 17 µg/kg/day is also approximately 500 times' below acute-effect levels known to influence neurochemical changes in rats (ATSDR 2012a). Yet the usefulness of the rat model for manganese neurotoxicity is limited because the distribution of manganese in rat brain regions is dissimilar to that of humans. No acute human studies were located for which to compare the 17 µg/kg/day Norwood dose to toxicological LSEs.

*Neurological and Immunological Considerations*

As mentioned, the nervous system is a sensitive target for manganese toxicity, however, Norwood exposure estimates were below OEHHA's chronic RfD, which accounts for neurotoxicity, as well as intermediate neurological LSEs. The principal manner by which manganese neurotoxicity occurs is not clearly established (ATSDR 2012a).

There are scant scientific studies on the human immunological effects from oral exposure to manganese. A few intermediate studies on rats found increases in neutrophil counts at 33,000 µg/kg/day and decreases in lymphocytes at exposures greater than 130,000 µg/kg/day. It is unknown if these changes were associated with significant impairment of immune system function (ATSDR 2012a).

## D.8. Arsenic

Arsenic is a naturally occurring element that is widely distributed in the Earth's crust (ATSDR 2007). It exists in two forms: organic and inorganic. Inorganic arsenic occurs naturally in soil and many kinds of rock; it is also more harmful to human health. The human health effects of arsenic have been widely studied, with most studies evaluating oral exposure, the most common route for many people. Chronic oral exposure to arsenic has led to skin lesions and discoloration of the skin, which is the most sensitive non-cancer endpoint. Chronic exposures have also been associated with effects such as peripheral neuropathy, characterized by numbness and/or a "pins and needles" sensation in the hands and feet. Arsenic is a well-known human carcinogen and classified as such by the IARC and EPA, among other agencies. Some of the more common increased cancer risk from long term arsenic exposure include lung cancer (primarily through inhalation) and skin and bladder cancers (primarily from consuming high levels of inorganic arsenic in drinking water). In the U.S., studies of people have not identified an increased risk of bladder or respiratory tumors following oral exposure to inorganic arsenic (ATSDR 2007).

Small exposures to arsenic can occur from food, drinking water, or inhalation. Arsenic can also be released from ores containing metals, or from coal-fired power plants. If not properly disposed at hazardous waste sites, arsenic enter the surrounding air, water or soil. Ingested arsenic may quickly enter the body, but soil arsenic is absorbed to a lesser extent than arsenic salt solutions; dermal arsenic exposure is not usually a concern. Most inorganic arsenic is excreted within days, though some will remain in the body for several months or longer.

*Health Effects Evaluation*

Arsenic was detected in all Norwood samples. Concentrations were slightly higher in residential soil (4.2-9.7 mg/kg) than non-residential soil (2.6-7.8 mg/kg), with highest concentrations in creek sediment (9.4-26.6 mg/kg) (Appendix Tables C1-C4). Residential and non-residential

arsenic concentrations were similar to U.S. background levels (Appendix Table H.3.1). Arsenic in creek surface water ranged from 1.1-2.2 µg/L. This range is similar to levels of arsenic found in U.S. surface water, of 1 µg/L (median) and 3 µg/L (75$^{th}$ percentile) (ATSDR 2007). Creek sediment arsenic was skewed by a duplicate, maximum sample (SD-12) of 26.6 mg/kg. Sediment arsenic concentrations vary widely around the world, and in the U.S., reported lake, river and stream sediment arsenic ranged from 0.1-4,400 mg/kg (ATSDR 2007).

All detected arsenic exceeded ATSDR's CREG CV. However, arsenic's CREG is below background levels. Two sediment and one surface water sample exceeded a non-CREG CV.

*Non-cancer health effects evaluation*

Table D.8.1 below shows estimated exposure doses for arsenic in sediment, which had the highest concentrations, and arsenic in residential soil. Non-residential soil and creek surface water exposures were below these estimates.

**Table D.8.1. Calculated Exposure Doses, Hazard Quotients, and Excess Cancer Risk Estimates based on Arsenic concentrations in creek sediment and highest residential soil sample**

| Sample type, and Conc (µg/kg) | Exposed Population and Time Period | Estimated Ingestion and Dermal Exposure Dose (ED) µg/kg/day | Chronic Hazard Quotient (ED/MRL) MRL: 0.3 | Acute Hazard Quotient (ED/MRL) MRL: 5 | Excess Cancer Risk Estimate[2] |
|---|---|---|---|---|---|
| Residential 9,700 (highest) | Adult | 0.0088 | <1 | <1 | 6 in 1,000,000 |
| | Child (birth to <1y) | 0.13 | <1 | <1 | 2 in 100,000 |
| Sediment 18,000 (95 UCL) | Adult | 0.016 | <1 | <1 | 1 in 100,000 |
| | Child (6 to 10y)[1] | 0.081 | <1 | <1 | 1 in 100,000 |

Conc = Concentration (µg/kg) in surface soil; 95 UCL = 95th Upper Confidence Limit of the Mean; ED = Exposure Dose; MRL = Minimal Risk Level; y = year of age. MRLs are expressed in µg/kg/day. There is no intermediate MRL for arsenic.
[1]The youngest age group we considered for sediment exposures was children ages 6-10y, as we presumed children younger than 6y would not be regularly exposed to sediment.
[2]Cancer Risk estimates were calculated using EPA's cancer risk slope factors of 1.5 $(mg/kg/day)^{-1}$ for arsenic. For residential soil, they encompass children (birth up to <21 years) and adults (21 years or older). For sediment, they encompass children (6 to <21 years) and adults (21 years and older). We presumed children younger than 6 would not be regularly exposed to sediment.

The highest arsenic exposure doses at Norwood were below ATSDR's chronic and acute MRLs (Table D.8.1). **Therefore, adults or children exposed to the highest arsenic concentrations for acute (up to 14 days) or chronic duration (a year or more) are unlikely to experience adverse non-cancer health effects.**

ATSDR has not derived an intermediate-duration MRL due to inadequacies of the toxicological database, and laboratory animals are not appropriate models to study arsenic-induced health effects in humans. Therefore, we compared the highest Norwood doses to the lowest levels of significant exposure (LSE) from intermediate-duration, human studies.

ATSDR identified an intermediate-duration study by Franzblau and Lilis (1989) that observed dermal and ocular effects in 2 humans exposed to 100 µg/kg/day arsenic in contaminated drinking water for 3 months (ATSDR 2007). These people also experienced severe nausea and other gastrointestinal effects, large changes in liver enzymes, anemia and confusion, paresthesia of hands and feet, and mental sluggishness. The 100 µg/kg/day effect level of this study is >700 times' the highest exposure dose at Norwood, of 0.13 µg/kg/day among infants up to 12 months (Table D.8.1).

ATSDR identified separate intermediate-duration studies by Huang et al. 1985 and Wagner et al. 1979 that reported a variety of effects, including some serious effects such hyperpigmentation with keratosis (possibly pre-cancerous), and anemia, weight loss and bone marrow effects (ATSDR 2007). In the Wagner study, humans were exposed to contaminated drinking water arsenic for 4 months; in the Huang study, humans were exposed to contaminated water from 6 months to 14 years (ATSDR 2007). The reported effect levels of these studies, of 50-60 µg/kg/day, are still many orders of magnitude higher (>380 times') than the highest exposure doses at Norwood, of 0.13 µg/kg/day among young children. Further, because most Norwood residents obtain their drinking water from the Aqua Pennsylvania public water source, these arsenic drinking water exposures are unlikely. **Based on these collective studies, we would not expect these adverse health effects to occur among children or adults from the highest detected arsenic concentrations at Norwood sampling locations.**

*Neurological and Immunological Considerations*

Studies and case reports indicate that ingesting inorganic arsenic can injure the human nervous system. Acute or single high-dose exposures to 2,000 µg/kg arsenic and higher has led to encephalopathy, a disease that affects the brain, with symptoms ranging from headaches to seizures (ATSDR 2007). This 2,000 µg/kg effect level is 15,000 times' the highest Norwood exposure dose, of 0.13 µg/kg/day. Longer duration arsenic studies have found neurological effects such as numbness, muscle weakness, and a pins and needles sensation in humans exposed to 30-100 µg/kg/day (ATSDR 2007). These thresholds are 230-3,500 times' the highest Norwood exposure doses for children, of 0.13 µg/kg/day, and for adults, of 0.016 µg/kg/day.

Evidence suggests an association between arsenic and intellectual deficits and neurodevelopmental effects in children (ATSDR 2007, ATSDR 2016b). These findings occurred from chronic exposures to contaminated drinking water. Exposure doses at Norwood still fell

below these reported health effect levels, and because most Norwood residents obtain their drinking water from the Aqua Pennsylvania public water source, elevated drinking water exposures are unlikely. Arsenic concentrations in Norwood residential and non-residential soil were similar to background U.S. levels (Appendix Table H.3.1). Further, no Norwood exposure estimates exceeded ATSDR's chronic MRL of 0.3 µg/kg/day, which is protective against the most sensitive long term adverse health effects.

A few studies have found increased susceptibility to respiratory infections in infants from prenatal increases in maternal arsenic exposure. A cross-sectional study by Ahmed et al. 2014 in Bangladeshi children (mean age, 4.5 years) found associations between higher urinary arsenic and effects on cell-mediated immunity (ATSDR 2016b). Laboratory animal studies are not appropriate for assessing potential human effects from arsenic exposure (ATSDR 2007).

*Cancer Health Effects*

We calculated excess cancer risks for arsenic using EPA's oral cancer slope factor of 1.5 $(mg/kg/day)^{-1}$. For residential soil, excess cancer risk based on exposure to the highest sample **was 2 in 100,000 for children and 6 in 1 million for adults**. Residential excess cancer risk estimates assume 33 consecutive years' exposure to the levels of arsenic found for adults, and 20 consecutive-years' exposure (from birth up to age 21) for children.

For creek sediment, the highest arsenic excess cancer risk was 1 in 100,000 for adults and 1 in 100,000 for children ages 6-20 years. These estimates assume adults are exposed daily to creek sediment at EPA's 2017-2018 sampling locations for 33 consecutive years and that children are exposed daily for 15 consecutive years, which are unlikely scenarios. A scenario in which children are exposed to arsenic sediment daily from birth to age 21, which is also unlikely, would result in an excess cancer risk estimate of 3 in 100,000.

## D.9. Chromium

*Overview*

Chromium is a naturally-occurring element found in rocks, animals, plants and soil, where it combines with other elements to form compounds (ATSDR 2012b). There are three primary forms – chromium(0), chromium(III), and chromium(VI). Of these forms, chromium(VI) is more toxic and widely studied. Studies evaluating health effects have identified respiratory (e.g., rhinorrhea, bronchitis), gastrointestinal (e.g., diarrhea, abdominal pain), immunological (e.g., allergic sensitization), and reproductive effects at higher chromium(VI) exposures. Most, but not all, of these studies have involved workers exposed to chromium compounds.

The IARC and U.S. Department of Health and Human Services have classified chromium(VI) as carcinogenic to humans, and EPA has classified it as a carcinogen by inhalation. Inhalation exposure to chromium (VI) has been shown to cause lung cancer in occupationally-exposed workers. Workers in chromium industries can be exposed at two orders the magnitude as the general population (ATSDR 2012b). There are limited data on the chronic oral toxicity of chromium in humans.

Chromium can be released into the environment from the combustion of fossil fuels.
It is mainly used in manufacturing to make stainless steel and other metal alloys. It is found in many consumer products such as certain wood and leather products, as well as stainless steel cookware. Food is the most common source of chromium exposure for the general public. A certain amount of chromium(III) is needed for human health, and low amounts occur naturally in a variety of foods including fruits, vegetables, nuts, beverages and meats. Chromium is also found in air and drinking water. Living near a hazardous waste facility that contains chromium can lead to exposure, as can cigarette smoke – in fact, smoking can result in indoor air with 10-400 times the chromium concentration of outdoor air (ATSDR 2012b).

Laboratory animal studies have reported that chromium(VI) may be linked to cancer effects when ingested. The National Toxicology Program (NTP) reported that sodium dichromate dihydrate, a compound containing chromium(VI), was associated with an increase in oral and stomach tumors in laboratory animals following ingestion (NTP 2008). The final release of EPA's IRIS reassessment of the carcinogenic effects of chromium(VI) through oral ingestion is pending. EPA is evaluating the carcinogenic mode of action (MOA) of chromium(VI). This MOA research is based on the hypothesis that ingestion of high concentrations of chromium(VI) results in excessive oxidative stress that exceeds the cellular capacity to reduce it, and points to the occurrence of a threshold for hexavalent chromium carcinogenesis (Health Canada 2018).

MOA weight of evidence (WOE) should be considered for evaluating the potential intestinal carcinogenicity of oral chromium(VI) exposure. The WOE indicates that cytotoxicity-induced regenerative hyperplasia (cell growth that occurs in response to cell damage) is the most scientifically supported MOA at environmentally relevant concentrations. This MOA also points to the occurrence of a threshold for chromium(VI) carcinogenesis. Health Canada (Health Canada 2018) used a cytotoxic MOA (not a mutagenic MOA) to develop a drinking water drinking guideline for total chromium (0.05 mg/L) and concluded that the cytotoxic MOA is protective of both cancer and non-cancer effects. The Texas Commission on Environmental Quality (TCEQ 2016) used the MOA approach to develop a hexavalent chromium oral reference dose of 0.0031 mg/kg-day (3.1 µg/kg/day) and concluded that it is protective of both cancer and non-cancer effects. Several regulatory and health agencies have used targeted studies to develop threshold-based toxicity criteria for chromium(VI) which has in many cases resulted in safe water levels ranging from 30-100 ppb (Chappell et al. 2021).

*Health Effects Evaluation*

Chromium was detected in all residential soil, non-residential soil, and creek sediment samples, and 37.5% (3 of 8) creek surface water samples (Appendix tables C1-C4). Concentrations ranged from 14.1-26.1 mg/kg (residential soil), 13.6-31.0 mg/kg (non-residential soil), 43.1-115.0 mg/kg (creek sediment) and 2.5-24.3 µg/L (creek surface water). Creek sediment had the highest values.

Chromium concentrations did not exceed a comparison value (CV) for chromium(III). However, the valency form for sampled chromium (chromium(VI) or chromium(III)) was unspecified, and thus we deemed chromium a chemical of concern (CoC) warranting further evaluation.

*Approach for assessing Norwood chromium concentrations*

As a health protective approach, we assumed that all detected chromium was in its more toxic chromium(VI) form. This is an unlikely scenario. In most soils, chromium is predominately in its much less toxic chromium(III) state because chromium(VI) tends to be reduced to chromium(III) by organic matter (ATSDR 2012b). In surface water chromium(VI) may react with organic matter or other agents to form chromium(III) (EPA 1998).

Assuming all sampled chromium at Norwood was chromium(VI) rather than total chromium, all soil and sediment samples exceeded a chromium(VI) soil CREG CV of 0.22 mg/kg. Conversely, none of the 21 residential soil samples and 3 of 8 creek sediment samples exceeded ATSDR's chronic Environmental Media Evaluation Guide (EMEG) CV for chromium(VI), of 47 mg/kg.

Table D.9.1 below shows estimated chromium(VI) exposure doses from creek sediment, residential soil, and creek surface water, all of which were higher than non-residential soil estimates.

**Table D.9.1. Calculated Exposure Doses, Hazard Quotients, and Excess Cancer Risk Estimates based on Total Chromium concentrations in creek sediment, surface water, and highest residential soil sample. Calculations assume that detected Total Chromium is 100% chromium(VI)**

| Sample type, and Conc (µg/kg for soil and sediment, µg/L for surface water) | Exposed Population and Time Period | Estimated Ingestion and Dermal Exposure Dose (ED), µg/kg/day | Chronic Hazard Quotient (ED/MRL)<br><br>MRL: 0.9 | Int Hazard Quotient (ED/MRL)<br><br>MRL: 5 | Excess Cancer Risk[5] |
|---|---|---|---|---|---|
| Residential 26,100 (highest) | Adult | 0.088 | <1 | <1 | 2 in 100,000 |
| | Child (birth to <1y) | 0.98 | **1.1** | <1 | 2 in 10,000 |

| | | | | | |
|---|---|---|---|---|---|
| Sediment 80,900 (95 UCL) | Adult | 0.27 | <1 | <1 | 6 in 100,000 |
| | Child (6-10y)[2] | 1.3 | **1.4** | <1 | 2 in 10,000 |
| Surface Water 24.3 µg/L (max) | Adult (swimming)[3] | 0.5 | NA | <1 | NA |
| | Adult (wading)[4] | 0.18 | <1 | <1 | 1 in 100,000 |
| | Child (6-10y), swimming | 0.75 | NA | <1 | NA |
| | Child (6-10y), wading | 0.23 | <1 | <1 | 4 in 100,000 |

**Bold =** Exceedance of a Minimal Risk Level (MRL). Conc = Concentration; ED = Exposure Dose; 95UCL = 95th Upper Confidence Limit of the Mean; Int = Intermediate; y = year of age. MRLs expressed in µg/kg/day.

[1]The youngest age group we considered for sediment exposures was children ages 6-10y, as we presumed children younger than 6y would not be regularly exposed to sediment.

[2]MRLs are expressed in µg/kg/day. There are no acute MRLs for chromium(VI).

[3]Swimming scenario: 1 hour per day, 7 days per week, 12 weeks per year. Cancer risks were not calculated for a swimming scenario due to intermittent exposures for less than a year.

[4]Wading scenario: 1 hour per day, 7 days per week, 52 weeks per year, 10 years.

[5]Cancer Risk estimates were calculated using California EPA's cancer slope factor of 0.5 $(mg/kg/day)^{-1}$ for chromium VI. For residential soil, they encompass children (birth up to <21 years) and adults (21 years or older). For sediment and surface water, they encompass children (6 to <21 years) and adults (21 years and older). We presumed children younger than 6 would not be regularly exposed to sediment or surface water.

*Non-cancer health effects evaluation*

As shown in Table D.9.1, the highest exposure dose for adults was 0.27 µg/kg/day. This dose is below ATSDR's chronic hazard quotient of 0.9 µg/kg/day. By contrast, the exposure dose to residential soil for children up to <1 year was 0.98 µg/kg/day, and the sediment exposure dose for children 6-10 years was 1.3 µg/kg/day. These doses exceeded ATSDR's chronic MRL of 0.9 µg/kg/day.

ATSDR's chronic MRL of 0.9 µg/kg/day is derived from a NTP study on rats and mice exposed to chromium in the form of dichromate dihydrate in drinking water for 2 years. ATSDR modeled and selected the lowest lower bound benchmark dose ($BMDL_{10}$) of 90 µg/chromium(VI)/day (ATSDR 2012b). This $BMDL_{10}$ was selected for an effect of diffuse epithelial hyperplasia of the duodenum (the first part of the small intestine), found in female mice. ATSDR then applied uncertainty factors to derive a chronic oral MRL of 0.9 µg/kg/day. The NTP 2008 study mentioned above represents the lowest level of significant exposure to chromium from chronic-duration studies (whether of humans or animals), based on ATSDR's toxicological profile (ATSDR 2012b).

The highest exposure dose for Norwood was 1.3 µg/kg/day in children. This estimate is approximately 70 times lower than the ATSDR's $BMDL_{10}$ of 90 µg chromium(VI)/day of the NTP study. It is also below TCEQ's oral reference dose value of 3.1 µg/kg/day, which TCEQ deemed protective of both non-cancer and cancer effects.

As mentioned, our estimated exposure doses for chromium assume that all detected total chromium in Norwood soil and creek sediment are 100% chromium(VI), the more toxic form. In most soils, chromium is predominately in its much less toxic chromium(III) state. All detected residential soil concentrations, and 5 of 8 creek sediment concentrations, were below ATSDR's

chronic non-cancer screening value for chromium(VI), of 47 mg/kg. The maximum creek surface water concentration of 24.3 µg/L (24.3 ppb) was lower than concentrations recently deemed safe from some regulatory and health agencies, of 30-100 ppb (Chappell et al. 2021). Mean total chromium concentrations at Norwood in residential and non-residential soil were 19.5 and 20.1 mg/kg, respectively, which are below geometric mean and median levels found in the U.S. of 30-37 mg/kg (Appendix Table H.3.1; ATSDR 2012; USGS 2013).

**Based on the highest estimated Norwood exposure doses and these collective data above, we would not expect chronic exposures to chromium (exposure duration of a year or more) to induce adverse non-cancer health effects in adults or children.**

Regarding intermediate-duration health effects, chromium exposure doses of intermediate duration did not exceed ATSDR's intermediate chromium(VI) oral MRL of 5 µg/kg/day (Table D.9.1). **Therefore, children and adults are unlikely to experience adverse non-cancer health effects from chromium exposures of intermediate duration (15 days or more).**

ATSDR has not derived an acute oral MRL for chromium(VI). Thus we compared the highest chromium (VI) exposure doses at Norwood to acute LSE thresholds (1-14 days) in ATSDR's toxicological profile (ATSDR 2012b).

The lowest identified LSEs have occurred in oral and dermal studies involving humans or animals exposed to chromium once. Exposure doses ranged from 9-90 µg/kg and subjects experienced dermatitis (skin inflammation), contact sensitivity and/or redness. In these cases, the humans or animals had a chromium sensitivity condition (ATSDR 2012b).

Although the highest combined oral and dermal exposure doses to chromium at Norwood approach these low single occasion dermatitis thresholds in scientific studies (between 9-90 µg/kg), these effects were only observed in people and animals that were chromium-sensitive. Acute studies evaluating health effects other than dermatitis have identified LSEs at 2,000-3,000 µg/kg/day and higher, far exceeding the highest Norwood acute chromium(VI) exposure doses (ATSDR 2012b). **Unless there are individuals who are chromium sensitive (further discussed in the next section), we would not expect adults or children exposed to the highest estimated soil chromium for acute-duration (1-14 days) to experience contact dermatitis.**

*Neurological and Immunological Considerations*

In some individuals, chromium(VI) can affect the immune system, leading to allergic sensitization. In these situations, the individual is first sensitized to chromium(VI) and subsequent exposures produce an allergic response, with symptoms such as dermatitis or asthma. U.S. prevalence of chromium sensitivity is estimated between 0.08-7% (ATSDR 2012b). Chromium sensitization can occur by inhalation, oral or dermal exposure and has typically been observed in work settings. For dermal exposures in sensitized individuals, allergic contact dermatitis in the form of skin reddening and/or blisters is typically isolated to the site of contact and can last from a few days to a few weeks.

Broader immune system changes such as histopathological and functional changes (e.g., increased proliferative response) in rats have occurred from intermediate and chronic chromium(VI) exposure doses ranging from 380-20,900 µg/kg/day (ATSDR 2012b). **These reported effect levels are at least 290 times' the highest exposure dose estimates at Norwood for adults, of 0.27 µg/kg/day, and for children, of 1.3 µg/kg/day.**

Very few oral or dermal studies have evaluated chromium's capacity to induce neurological effects (ATSDR 2012b). Rats experienced decreases in motor activity and balance when given 98,000 µg/kg/day chromium(VI) as sodium chromate in drinking water for 28 days; no effects were observed at 10,000 µg/kg/day. In separate studies, researchers did not observe histopathological abnormalities in the brain or central nervous system tissues in rats and mice exposed to 27,900 µg/kg/day and 8,700 µg/kg/day chromium(VI) in drinking water for 3 months and 2 years, respectively (ATSDR 2012b). **These exposures at which no effects were observed (8,700 – 27,900 µg/kg/day) are more than 6,000 times' the highest intermediate and chronic exposure doses at Norwood for chromium(VI), of 1.3 µg/kg/day.**

*Cancer Effects Evaluation*

Currently, ATSDR's recommended oral cancer slope factor for chromium(VI) is a California EPA value of 0.5 $(mg/kg/day)^{-1}$ (ATSDR 2019). Under the assumption that all detected total chromium concentrations were in the form of chromium(VI), the highest excess cancer risk for residential soil **was 2 in 10,000 for children and 2 in 100,000 for adults**. Excess cancer risk would be considerably lower if chromium concentrations at Norwood are in their much less toxic, chromium(III) forms, as they often are for most soils. If we were to apply our 100% chromium(VI) assumption to detected total chromium in soils throughout the U.S., on average excess cancer risk would most likely be higher than the 2 in 10,000 residential estimate at Norwood. This is because U.S. mean and median total chromium levels exceeded levels in Norwood residential and non-residential soil (Appendix Table H.3.1).

The highest excess cancer risk for creek sediment was 2 in 10,000 for children from ages 6-20 years, and 6 in 100,000 in for adults (ages 21 years and over). These risk estimates assume that the levels of total chromium detected in creek sediment are 100% chromium(VI). They also assume that adults are exposed daily to creek sediment at EPA's 2017-2018 sampling locations for 33 consecutive years and that children are exposed daily for 15 consecutive years, which is unlikely.

A scenario in which children were exposed to sediment *daily* from birth to up to age 21 years would result in an excess cancer risk estimate of 7 in 10,000. These exposures are also unlikely.

As mentioned, TCEQ 2016 used the MOA approach to develop a hexavalent chromium oral reference dose of 3.1 µg/kg/day and concluded that it is protective of both cancer and non-cancer effects. The highest Norwood exposure doses among adults (0.27 µg/kg/day) and children (1.3 µg/kg/day) were below this reference dose.

*Assessing a scenario in which total chromium is 75% trivalent, and 25% hexavalent*

We also assessed a scenario in which detected total chromium was primarily in its less toxic, chromium(III) form, a more likely scenario. Under a scenario in which total chromium detected at Norwood is 75% in its chromium(III) form and 25% in its chromium(VI) form, the highest exposure doses for children and adults were below chronic and intermediate MRLs, indicating that non-cancer effects are unlikely to occur. The highest lifetime excess cancer risk based on exposures to residential soil was 6 in 100,000 children and 6 in 1 million adults (Table D.9.2).

**Table D.9.2. Calculated Exposure Doses, Hazard Quotients, and Excess Cancer Risk Estimates based on Total Chromium concentrations in creek sediment, surface water, and highest residential soil sample. Calculations assume a 75% chromium(III) and 25% chromium(VI) scenario based on the Total Chromium concentrations detected.**

| Sample type, and Conc (µg/kg for soil and sediment, µg/L for surface water) | Exposed Population and Time Period | Estimated Ingestion and Dermal Exposure Dose (ED), µg/kg/day | Chronic Hazard Quotient (ED/MRL)<br><br>MRL: 0.9 | Int Hazard Quotient (ED/MRL)<br><br>MRL: 5 | Excess Cancer Risk[5] |
|---|---|---|---|---|---|
| Residential 6,600 | Adult | 0.022 | <1 | <1 | 6 in 1,000,000 |
| | Child (birth to <1y) | 0.25 | <1 | <1 | 6 in 100,000 |
| Sediment 20,225 | Adult | 0.17 | <1 | <1 | 1 in 100,000 |
| | Child (6-10y)[2] | 0.32 | <1 | <1 | 6 in 100,000 |
| Surface Water 6.08 µg/L | Adult (swimming)[3] | 0.12 | NA | <1 | NA |
| | Adult (wading)[4] | 0.045 | <1 | <1 | 1 in 1,000,000 |
| | Child (6-10y), swimming | 0.17 | NA | <1 | NA |
| | Child (6-10y), wading | 0.058 | <1 | <1 | 1 in 100,000 |

**Bold =** Exceedance of a Minimal Risk Level (MRL). Conc = Concentration; ED = Exposure Dose; 95UCL = 95th Upper Confidence Limit of the Mean; Int = Intermediate; NA, Not Applicable; y = year of age. MRLs expressed in µg/kg/day.

[1]The youngest age group we considered for sediment exposures was children ages 6-10, as we presumed children younger than 6 would not be regularly exposed to sediment.

[2]MRLs are expressed in µg/kg/day. There are no acute MRLs for chromium(VI).

[3]Swimming scenario: 1 hour per day, 7 days per week, 12 weeks per year. Cancer risks were not calculated for a swimming scenario due to intermittent exposures for less than a year (ATSDR 2018).

[4]Wading scenario: 1 hour per day, 7 days per week, 52 weeks per year, 10 years

[5]Cancer Risk estimates were calculated using California EPA's cancer risk slope factor of 0.5 $(mg/kg/day)^{-1}$ for chromium VI. For residential soil, they encompass children (birth up to 21 years) and adults (21 years or older). For sediment and surface water, they encompass children (6-21 years) and adults (21 years and older). We presumed children younger than 6 would not be regularly exposed to sediment or surface water.

## D.10. Mercury

*Overview*

Mercury is a naturally-occurring metal that is ubiquitous in the environment from natural and man-made sources (ATSDR 1999). Natural sources include the weathering of rocks and soil and volcanic activity; man-made sources include the burning of fossil fuels, mining, smelting, and

solid waste incineration. Most mercury in the environment is in the form of metallic and inorganic mercury compounds, which can enter the air from the burning of medical waste, emissions of coal-fired power plants, and other industrial processes. Methylmercury forms when environmental microorganisms convert inorganic mercury to organic (methyl) mercury; it is the most toxic form of mercury.

Humans are exposed to low background mercury in air, water and food. The most common additional mercury source is fish consumption; however, commercial fish cannot be sold unless mercury levels are below a Food and Drug Administration (FDA) threshold of 1 ppm, which is below concentrations associated with health effects (ATSDR 1999).

Generally, the nervous system and kidneys are most sensitive to mercury toxicity. Mercury poisoning, usually occurring from consuming extremely high levels of contaminated fish, can induce permanent neurological effects. Once absorbed into the body, metallic and methylmercury can readily move into the brain. Methylmercury can also pass from pregnant mother to child and induce developmental effects (e.g., reduced IQ) on the child. Because methylmercury is easily absorbed and readily bioaccumulates in the aquatic food chain, pregnant mothers are advised to avoid consuming larger fish (e.g., mackerel, swordfish), which often have higher mercury concentrations due to their consuming smaller fish.

The Department of Health and Human Services (DHHS) has not classified mercury as to its human carcinogenicity. Animal studies provide only limited information about whether mercury causes cancer in humans. EPA has determined that mercuric chloride and methylmercury are possible human carcinogens but has not derived a cancer slope factor for these mercury forms (ATSDR 1999).

*Health effects evaluation*

Mercury was detected in all residential (21 of 21), non-residential (17 of 17), and creek sediment (8 of 8) samples; it was not detected in surface water. Mercury concentrations were similar in residential (32-880 µg/kg) and non-residential soil (42-640 µg/kg), and slightly higher in creek sediment (300-1,100 µg/kg; Appendix Tables C1-C4).

Mercury in residential and non-residential soil did not exceed CVs. One sediment sample, the SD-12 duplicate (1,100 µg/kg), met or exceeded EPA's Regional Screening Level CV of 1,100 µg/kg and California's Department of Toxic Substances Control (DTSC's) CV of 1,000 µg/kg.

*Non-cancer health effects evaluation*

Estimated exposure doses from creek sediment mercury are displayed in Table D.10.1. We used the 95th Upper Confidence Limit of the mean (95UCL) for the 8 sediment mercury samples.

**Table D.10.1. Calculated Exposure Doses and Hazard Quotients based on the highest Mercury concentrations, found in creek sediment**

| Sample Type, and Conc (µg /kg) | Exposed Population and Time Period | Ingestion and Dermal Exposure Dose (ED) µg /kg/day | Chronic Hazard Quotient (ED/RfD) MRL: 0.3 | Intermediate Hazard Quotient (ED/MRL) MRL: 2 | Acute Hazard Quotient (ED/MRL) MRL: 7 | Excess Cancer Risk[1] |
|---|---|---|---|---|---|---|
| Sediment 958 (95 UCL) | Adult | 0.0019 | <1 | <1 | <1 | NA |
| | Child (6 to 10y) | 0.0093 | <1 | <1 | <1 | |

Conc = Concentration; ED=Exposure Dose; 95UCL = 95th Upper Confidence Limit of the Mean; NA = Not Applicable; RfD = Reference Dose; y = year of age. Minimal Risk Levels (MRLs) in this table are shown in µg/kg/day and pertain to inorganic mercury

[1]There is no cancer slope factor for mercury.

As shown in Table D.10.1, the highest mercury exposure doses for adults and children, at 0.0019 µg/kg/day and 0.0093 µg/kg/day, respectively, were well below EPA's chronic reference dose (RfD) and ATSDR's intermediate and acute minimal risk levels for inorganic mercury, of 0.3, 2 and 7 µg/kg/day, respectively. **Therefore, adult or child exposures to the highest concentrations of inorganic mercury at Norwood, from creek sediment, are unlikely to result in adverse noncancer health effects.**

We also considered a scenario in which creek sediment mercury was methylmercury as opposed to inorganic mercury. This is unlikely given that nearly all U.S. methylmercury exposures occur from eating fish and shellfish containing high levels (EPA. n.d.). In a methylmercury sediment scenario, if a child up to 12 months was exposed to Norwood mercury sediment concentrations for a year or longer, the child's estimated exposure dose would be 0.062 µg/kg/day (data not shown). This is below ATSDR's chronic methylmercury MRL of 0.3 µg/kg/day. ATSDR's methylmercury MRL is protective against adverse neurodevelopmental effects among populations most sensitive to methylmercury toxicity – the developing fetus and infant (ATSDR 1999).

In April 2022, ATSDR released an updated Toxicological Profile for Mercury (Draft for Public Comment), which includes new provisional MRLs. The public comment period for this draft closed on July 26, 2022. PADOH will assess Norwood exposure doses in the context of this updated profile as part of the addendum to this Health Consultation, which will encompass EPA's 2020 expanded sampling data.

*Neurological and Immunological Considerations*

The nervous system is very sensitive to mercury. Poisonings from methylmercury and other organic mercury compounds can result in permanent damage to the brain. Compared to methylmercury, inorganic mercury (the presumed form of mercury measured at Norwood) does not enter the brain as readily (ATSDR 1999). **No exposure doses to mercury or methylmercury in creek sediment exceeded ATSDR chronic health guidelines, which, for methylmercury, are protective against neurological effects.**

The immune response from oral mercury exposure is complex and dependent in part on the amount of exposure and genetics of the exposed population. ATSDR's 1999 toxicological profile for mercury noted no human studies and very few animal studies evaluating immunological effects from oral exposure to inorganic or organic mercury. The animal studies found varied effects on the immune system following organic or inorganic mercury exposure, with some intermediate studies finding evidence for immunosuppression in mice at effect levels between 500 – 2,900 µg/kg/day (ATSDR 1999). **These immune effect levels are more than 50,000 times' the highest exposure doses at Norwood, of 0.0093 µg/kg/day for children (Table D.10.1).**

## D.11. Dimethyl Phthalate (DMP)

*Overview*

Dimethyl phthalate (DMP) has many uses, including uses in solid rocket propellants, lacquers, plastics, safety glasses, rubber coating agents, molding powders, and insect repellants (EPA 2000). Limited information is available on the long-term health effects from DMP in humans such as reproductive, developmental, or cancer effects (EPA 2000). EPA has not classified DMP for its carcinogenicity.

*Health effects evaluation*

At Norwood DMP was sampled in non-residential soil and creek sediment. It was detected in 16 of 17 non-residential samples (94%) with a maximum concentration of 540 µg/kg. It was detected in 8 of 8 sediment samples (100%) with a maximum concentration of 1,200 µg/kg. We considered DMP as a CoC because a comparison value (CV) could not be located from ATSDR, EPA, or any other agency. We evaluated the 95UCL for DMP in creek sediment, which had higher values than non-residential soil. Sediment results are shown in Table D.11.1 below.

**Table D.11.1. Calculated Exposure Doses and Hazard Quotients based on the highest Dimethyl Phthalate concentrations, found in creek sediment**

| Sample Type, and | Exposed Population and Time Period | Ingestion and Dermal Exposure | Chronic Hazard Quotient | Intermediate Hazard Quotient | Acute Hazard Quotient | Excess Cancer Risk[2] |
|---|---|---|---|---|---|---|

| Conc (µg /kg) | | Dose (ED) (µg /kg/day) | | (EPA Oral Subchronic Reference Screening Value: 100 µg/kg/day)[1] | | |
|---|---|---|---|---|---|---|
| Sediment 862 (95 UCL) | Adult | 0.0015 | NA | <1 | NA | NA |
| | Child (6 to 10y) | 0.0075 | NA | <1 | NA | NA |

[1]Value of 100 µg/kg/day from EPA (2007). Conc = Concentration; 95UCL = 95th Upper Confidence Limit of the Mean; NA = Not Applicable; y = year of age.
[2]EPA has deemed DMP as not classifiable as to its carcinogenicity

There are very few studies on DMP from oral exposures. In EPA's Provisional Peer Reviewed Toxicity Values (PPRTV) document for possible DMP toxicity values, it was unable to locate a human study (EPA 2007). Available animal studies were also very limited. Due to the limited data overall, EPA was unable to derive a provisional toxicity value for DMP. Instead, it derived a "DMP oral subchronic reference screening value" of 100 µg/kg/day. This value is based on an Oishi and Hiraga 1980 study of male weanling rats exposed to DMP for a week at 302,000 µg/kg/day (EPA 2007). At this high effect level exposed rats experienced significant increases in absolute and relative liver weight, and decreased serum and testicular testosterone levels, compared to controls.

As shown in Table D.11.1, estimated adult and child exposure doses to sediment DMP were well below EPA's oral subchronic reference screening value of 100 µg/kg/day. **Based on these data, we would not expect adverse health effects to occur from the highest intermediate-duration (15 days to a year) DMP exposures at Norwood.**

EPA was unable to locate a chronic DMP toxicity study that had suitable reporting methods. Regarding acute-duration studies, the LSE of 302,000 µg/kg/day in the Oishi and Hiraga study discussed above also represents the lowest LSE based on limited available data.

*Neurological and Immune System effects*

There are no studies identified in EPA's PPRTV document that assess neurological or immune system effects from DMP exposure.

## D.12 Lead

Lead (Pb) is an element found in Pb ore deposits that are widely distributed throughout the world. It is released from a variety of anthropogenic sources including mining and smelting of ore, combustion of coal and oil, and waste incineration. Lead or lead mixtures are used in Pb-acid batteries, coverings and cables, building construction materials, and other uses. Once in the environment, lead does not degrade but is transferred between air, water and soil by natural or chemical processes. The general public may be exposed via outdoor air, food, drinking water, soil, and dust; exposure primarily occurs orally, with some contribution from inhalation (ATSDR 2020). Urban exposures are usually higher than rural due to housing

characteristics and proximity to roadways. Additional risk factors that contribute to lead body burden include living in older buildings with deteriorating lead paint, certain occupational exposures (which can then track lead into a home from clothing or tools), socioeconomic status, lead in water service lines, living in areas where lead was produced or disposed, use of lead-contaminated imported consumer products, or second-hand smoke exposures. (Note: section 5.13 of the main report discusses additional lead exposure sources, such as from certain hobbies. Further information on lead exposure sources, including occupation types with higher exposures, can be found from the U.S. Centers for Disease Control and Prevention website: https://www.cdc.gov/nceh/lead/prevention/sources.htm.)

There is no safe blood level of lead, and some of its toxic effects can be irreversible. U.S. public health policy has focused on eliminating the potential for lead poisoning in children, who are especially susceptible. U.S. exposure to lead is measured via blood-lead levels and has declined since the phase out and ban of lead-based paint and leaded gasoline.

Lead induces toxicity in every organ, and for some organs, at low levels. Lead cancer studies have produced inconsistent results and are usually confounded by other factors; however, the Department of Health and Human Services (HHS) has classified lead and lead compounds as "reasonably anticipated to be human carcinogens" (ATSDR 2020). EPA and IARC has deemed lead as a probable human carcinogen based mainly on animal studies, however there is no cancer slope factor for lead. Human studies provide some evidence that lead is a carcinogen, but cancer results are inconsistent and often confounded by other factors (e.g., smoking status, family history of cancer, co-exposure to other carcinogens).

*Norwood environmental lead concentrations*

Lead was detected in all Norwood samples at concentrations ranging from 30-1,800 mg/kg in residential soil, 20.7-358 mg/kg in non-residential soil, 74.5-214 mg/kg in creek sediment, and 2.2-24.8 µg/L in creek surface water. In residential soil, 17 of 21 samples were estimated ("J") values (though, the highest residential concentration of 1,800 mg/kg was an actual value). All samples containing lead at remaining locations were actual (not estimated) values. Table D.12.1 displays the levels of lead at Norwood compared to U.S. background soil levels.

**Table D.12.1** Lead concentrations (mg/kg) in Norwood residential soil, non-residential soil, and creek sediment at sampling locations, 2017-2018

| | Residential Soil | Non-Residential Soil | Creek Sediment | US Background levels (soil)** |
|---|---|---|---|---|
| Samples (% Detected) | 21 (100%) | 17 (100%) | 8 (100%) | 4,841 |
| Range | 30-1800 | 20.7-358 | 74.5-214.0 | <0.5 – 12,400 |
| Median | 54.7 | 145.0 | 73.3 | 18.1 |
| Mean | 162.5 | 147.3 | 102.9 | 25.8 |

| | | | | |
|---|---|---|---|---|
| # of Samples Exceeding 245 mg/kg IEUBK* threshold | 3 | 5 (SS5, SS9, SS11, SS13, SS19) | 0 | NA |

NA = Not applicable
*EPA's Integrated Exposure Uptake of Biokinetic Model (IEUBK) for Lead in Children (version 2.0) https://semspub.epa.gov/work/HQ/400700.pdf that could result in a 6-12 month child blood lead level of ≥ 3.5 µg/dL
**USGS sampled 4,841 soil samples (0-5 cm in depth) and released their results in 2013: https://pubs.usgs.gov/ds/801/pdf/ds801.pdf. Levels are derived from Table 2 of the report.

The concentration of lead in the top layers of soil varies widely due to deposition and accumulation of atmospheric particulates from anthropogenic sources (ATSDR 2020). Lead concentration ranges in Norwood soil were within those typically found in U.S. urban soils, which range from 150-10,000 mg/kg (Penn State University 2010). However, mean and median lead values were higher at Norwood than background U.S. levels (Table D.12.1). They were also above median (46.4 mg/kg) and mean values (60.2 mg/kg) for Pennsylvania based on 2007-2010 United States Geological Survey (USGS) data, although median residential soil lead at Norwood (54.7 mg/kg) was nearer to the Pennsylvania median (46.4 mg/kg) than was median non-residential lead (145.0 mg/kg). Lead levels at Norwood sampling locations were below levels typically found in more urban areas, such as Philadelphia (O'Shea et al. 2021).

Lead in creek surface water ranged from 2.2-24.8 µg/L (Appendix C4). The mean lead for the creeks was 6.0 µg/L (data not shown). While higher than that of typical U.S. surface waters of 3.9-4.0 µg/L (ATSDR 2020), mean creek lead was skewed by one surface water sample (SW11) of 24.8 µg/L. The median creek lead level of 3.9 µg/L (data not shown), was closer to national averages (3.9-4.0 µg/L). As mentioned, 7 of 8 creek samples were taken in Darby as opposed to Muckinipattis Creek.

*Non-cancer Health Effects Evaluation*

No blood lead level (BLL) is considered safe in children. In October 2021 the U.S. Centers of Disease Control and Prevention (CDC) updated the blood lead (BL) reference value to 3.5 µg/dL based on the 97.5$^{th}$ percentile of BL distribution in U.S. children 1-5 years old (Ruckart et al. 2021). This threshold is not a health-based standard or toxicity threshold, as even low BLLs cause harm. Rather it is meant as a policy tool to prioritize prevention efforts and identify children who have higher levels of lead in their blood compared to most U.S. children. Effects from lead exposure on multiple health endpoints (e.g., neurological, renal, cardiovascular, immunological) have been observed ≤ 5 µg/dL BLL (ATSDR 2020).

We estimated a lead threshold for Norwood soil that would result in a 3.5 µg/dL or higher BLL in children. To do so we used EPA's Integrated Exposure Uptake of Biokinetic Model (IEUBK,

version 2.0) for Lead in Children. IEUBK integrates lead soil concentrations in addition to other sources of lead including outdoor air, dietary intake, and drinking water to estimate BLLs in children. We used IEUBK's default values and input incremental increases of soil lead concentrations until surpassing a threshold that induced a BLL estimate of 3.5 µg/dL or higher. As shown in Table D.12.2, this occurred for a soil lead concentration of 245 mg/kg, for children 6 months to 1 year old.

**Table D.12.2. Estimated Blood Lead (µg/dL) in young children from a 245 mg/kg soil concentration, from use of EPA's Integrated Exposure Uptake of Biokinetic Model (IEUBK) for Lead in Children (version 2.0)**

| Age in years | Air (µg/day) | Diet (µg/day) | Alternate (µg/day) | Water (µg/day) | Soil + Dust (µg/day) | Total (µg/day) | Blood (µg/dL) |
|---|---|---|---|---|---|---|---|
| 0.5-1 | 0.034 | 1.236 | 0.00 | 0.167 | 5.036 | 6.473 | **3.5** |
| 1-2 | 0.057 | 2.344 | 0.00 | 0.180 | 5.520 | 8.101 | 3.4 |
| 2-3 | 0.075 | 2.478 | 0.00 | 0.218 | 4.017 | 6.789 | 2.7 |
| 3-4 | 0.093 | 2.579 | 0.00 | 0.233 | 3.807 | 6.713 | 2.4 |
| 4-5 | 0.102 | 2.714 | 0.00 | 0.247 | 3.807 | 6.713 | 2.4 |
| 5-6 | 0.111 | 2.928 | 0.00 | 0.262 | 3.178 | 6.479 | 2.1 |
| 6-7 | 0.118 | 2.890 | 0.00 | 0.275 | 3.367 | 6.651 | 1.9 |
| These estimates were calculated using EPA's IEUBK Model, v. 2.0: https://www.epa.gov/superfund/lead-superfund-sites-software-and-users-manuals#integrated | | | | | | | |

At Norwood, 3 of 21 residential soil samples (at 248 mg/kg, 283 mg/kg, and 1800 mg/kg), 5 of 17 non-residential soil samples, and 0 of 8 sediment samples exceeded a concentration of 245 mg/kg.

*Neurological and Immunological Considerations*

There are consistent associations between lead exposure and decreases in neurological function in children and adults. In children and adults, BLLs at or below 10 µg/dL have been associated with neurological effects, including but not limited to decreased cognitive function (including IQ), altered mood and behavior contributing to learning and attention deficits, and altered neuromotor and neurosensory function. Cognitive effects have been observed at BLLs well below 10 µg/dL (and in children below 5 µg/dL), with no evidence for a threshold in children. Higher BLLs (for example, greater than 10 µg/dL) have also induced neurological impairment (ATSDR 2020).

Lead can also impact the immune system. Laboratory animal studies show that it can alter the humoral and cell-mediated immune systems, leading to decreased resistance to disease, sensitization, autoimmunity and inflammation (ATSDR 2020). These studies support human evidence. At BLLs at or below 10 µg/dL, human health effects have included increased

susceptibility to infection, sensitization to allergies, changes in indicators of humoral and cell-mediated immunity, and changes in inflammatory response (ATSDR 2020).

*Lead Summary*

**There is no safe BLLs and PADOH recommends reducing lead exposure whenever possible.** Based on EPA's 2017-2018 sampling data, lead in Norwood residential soil, non-residential soil, and creek sediment were within U.S. background ranges. Mean and median lead concentrations were higher than average Pennsylvania and U.S. background levels but lower than levels found in more urban areas such as Philadelphia. To estimate a soil threshold at which child blood lead would increase to 3.5 µg/dL, we used EPA's IEUBK model for estimating potential lead levels in children. Three residential samples, five non-residential samples and zero sediment samples exceeded a 245 mg/kg soil threshold that would produce an estimated blood lead level of 3.5 µg/dL among children 6-12 months old. Although lead is considered a probable carcinogen, there is no cancer slope factor from which to estimate excess cancer risk.

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# Appendix E. Pica Health Effects Evaluation for Norwood Chemicals of Concern, based on EPA's 2017-2018 Environmental Sampling Data

**Overview.** Soil-pica involves eating soil. It is most likely to occur in preschool children (ATSDR 2018a). Children 1-2 years old have the greatest tendency to exhibit this behavior, which diminishes as they become older. Soil-pica is uncommon. As a health protective approach, we assumed that pica-behavior could occur at the site.

For residential soil, we evaluated an intermediate (3 days per week for up to a year) and single soil-pica scenario, with the assumption that although uncommon these exposures could still occur. For non-residential soil and creek sediment, we evaluated a single occasion soil-pica scenario. Each scenario assumed 5,000 mg ingestion of soil, which is equivalent to consuming 5 tea/coffee artificial sweetener packets worth of soil. According to ATSDR methodology, as a health protective approach, pica exposures are estimated based on the maximum soil or sediment concentrations at a given location and not the 95 UCL.

Pica-based exposures were compared against intermediate and acute health guidelines, where applicable.

The toxicological evaluation of this Appendix omits chemical of concern (CoC) descriptions and Norwood CoC detection rates, which are found in Appendix D.

## E.1 Pica Health Effect Evaluation Summary for Norwood Chemicals of Concern

Table E.1.1 shows the highest estimated Norwood intermediate and single soil-pica doses compared to Minimal Risk Levels (MRLs) in scientific studies and displays hazard quotients (HQs).

**Table E.1.1 – Highest Intermediate and Single Pica Exposure Dose Estimates and Hazard Quotients for Norwood Chemicals of Concern**

| Chemical | Location | Conc (µg/kg) | Intermediate or Single Pica | Age group, years | Exposure Dose (ED) (µg/kg) | MRL, RfD, or UL (µg/kg/day) | Hazard Quotient (ED/MRL) |
|---|---|---|---|---|---|---|---|
| Benzo[a]pyrene equivalent PAHs (Residential) | Residential | 13,490 | Intermediate | 1 | 2.6 | NA | NA |
| | | | Single | 1 | 0.92 | NA | NA |
| Di(2-ethylhexyl)phthalate (Residential) | Residential | 6,800 | Intermediate | 1 | 1.3 | 0.1 | **13** |
| | | | | 2-5 | 0.86 | 0.1 | **8.6** |
| | | | Single | 1 | 0.45 | 3 | <1 |
| Aroclor 1254 | Non-residential | 450 | Single | 1 | 0.031 | NA | NA |
| Aroclor 1260 | Residential | 310 | Intermediate | 1 | 0.06 | NA | NA |
| Dieldrin | Residential | 1,300 | Intermediate | 1 | 0.25 | 0.1 | **2.5** |

| | | | | | | | |
|---|---|---|---|---|---|---|---|
| | | | Single | 1 | 0.087 | NA | NA |
| Copper | Residential | 264,000 | Intermediate | 1 | 50 | 10 | **5.0** |
| | | | | 2-5 | 33 | 10 | **3.3** |
| | | | Single | 1 | 17 | 10 | 1.7 |
| | | | | 2-5 | 11 | 10 | 1.1 |
| Iron | Residential | 25,300,000 | Intermediate | 1 | 4,800 | 700 | **6.9** |
| | | | | | 3,100 | 700 | **4.4** |
| | | | Mg per day (Intermediate) | 1 | 54.7 | 40 mg[1] | **1.4** |
| | | | | 2-5 | 53.9 | 40 mg[1] | **1.3** |
| | | | Single (µg/kg) | 1 | 1,600 | NA | NA |
| | | | | 2-5 | 1,000 | NA | NA |
| | | | Mg per day (single) | 1 | 18.2 | 40 mg[1] | <1 |
| | | | Mg per day (single) | 2-5 | 17.4 | 40 mg[1] | <1 |
| | Sediment | 37,700,000 | Single (µg/kg) | 1 | 2,400 | NA | NA |
| | | | | 2-5 | 1,500 | NA | NA |
| | | | Mg per day (single) | 1 | 27.3 | 40 mg[1] | <1 |
| | | | Mg per day (single) | 2-5 | 26.1 | 40 mg[1] | <1 |
| Manganese | Residential | 553,000 | Intermediate | 1 | 110 | NA | NA |
| | | | Single | 1 | 38 | NA | NA |
| | Sediment | 848,000 | Single | 1 | 59 | NA | NA |
| Arsenic | Residential | 9,700 | Intermediate | 1 | 1.1 | NA | NA |
| | | | Single | 1 | 0.38 | 5 | <1 |
| | Sediment | 26,600 | Single | 1 | 1 | 5 | <1 |
| Chromium | Residential | 26,100 | Intermediate | 1 | 5.3 | 5 | **1.1** |
| | Sediment | 115,000 | Single | 1 | 9.1 | NA | NA |
| Mercury | Sediment | 1,100 | Single | 1 | 0.075 | 7 | <1 |
| Dimethyl Phthalate | Sediment | 1,200 | Single | 1 | 0.08 | NA | NA |

**Bold** = exceedance of an intermediate or acute health guideline (please see Appendix B3 for more information). Conc = Concentration, HQ = Hazard Quotient, Int = Intermediate, MRL = Minimal Risk Level, NA = Not Applicable; RfD = Reference Dose; UL = Tolerable Upper Intake Level. MRLs and RfDs expressed in µg/kg/day. We considered intermediate and single pica exposures at residential locations and only single pica exposures at non-residential locations. NA applies to occasions where there is no MRL or RfD and no Hazard Quotient.

[1] 40 mg is the tolerable upper limit of iron per day for children. Iron UL exposures were calculated by multiplying the Exposure Dose in mg/kg/day by ATSDR's Exposure Dose Guidance for Body Weight (2016). Example: for an intermediate pica child (age 1 year) exposed to residential soil, an estimated exposure dose is 4,800 µg/kg/day ÷ 1,000 = 4.8 mg/kg/day, × body weight estimate of 11.4 kg = estimated daily iron intake (in mg) of 54.7 mg. Body weight estimates by age can be found in ATSDR's Exposure Dose Guidance for Body Weight Manual (2016)

Although multiple Norwood CoCs lack acute or intermediate health guidelines, in these instances the highest pica exposure dose estimates shown in Table E.1.1 were still multiple orders of magnitude below levels of significant exposure (LSEs) for health effects (whether general, immunological, or neurological effects) of scientific studies. Those studies are

discussed further in Appendix D, the non-pica health effects evaluation. Thus for these CoCs, we would not expect adverse health effects to occur from soil pica behavior.

For 5 chemicals, **Di(2-Ethylhexyl)Phthalate (DEHP), Dieldrin, Copper, Iron, and Chromium**, the **highest intermediate soil-pica doses (3 of 7 days per week)** exceeded intermediate health guidelines. Further evaluation of DEHP and dieldrin revealed that these pica doses (DEHP: 1.3 µg/kg/day; dieldrin: 0.25 µg/kg/day) were 23 and 400 times lower, respectively, than the most sensitive LSEs reported in scientific studies. In the case of DEHP, such effects (which were immunological effects) only occurred in laboratory animals that were sensitized to ovalbumin, or egg allergy (Han et al. 2014; Guo et al. 2012). Further discussion of these studies is in Appendix D. For chromium, the highest intermediate-pica estimate (5.3 µg/kg) was approximately 100 times lower than a 520 µg/kg/day benchmark dose threshold for hematological effects in rats, which ATSDR used to derive its intermediate MRL (ATSDR 2012a).

Being several orders of magnitude lower than these reported effect levels, **we would <u>not expect</u> adverse health effects to occur among children exposed to DEHP, dieldrin or chromium** at Norwood in an intermediate duration pica scenario (3 days per week for up to 1 year). Chromium pica estimates assumed that detected chromium in soil/sediment was 100% chromium(VI).

The highest <u>single</u> <u>(one-time)</u> pica dose for chromium (9.1 µg/kg for creek sediment) approached a reported effect level of dermatitis at 36 µg/kg from a 1977 acute study by Kaaber and Veinen (ATSDR 2012a). The dermatitis finding in Kaaber and Veinen was only in people with chromium sensitivity, which has low prevalence in the U.S. of 0.08-7% and is typically observed from workplace exposures. Chromium sensitivity is thus unlikely in young children and we wouldn't expect these dermatitis effects to occur. Acute duration studies evaluating health effects other than dermatitis have identified LSEs at 2,000-3,000 µg/kg/day and higher (ATSDR 2012a), far exceeding the highest single pica chromium(VI) dose at Norwood.

Intermediate and single-pica doses for Norwood CoCs were also below reported neurological and immunological effect levels. Further discussion on these reported levels is provided in Appendix D.

Of the Norwood CoCs shown in Table E.1.1., **copper and iron** pica-doses exceeded or closely approached reported health effect levels. These two CoCs are discussed further. Additionally, since there is no safe blood lead level, pica exposures to lead in soil at Norwood could induce adverse health effects, including nervous system and/or immunological effects. Our health effects evaluation for lead can be found in Appendix D12.

### E.1.2. Copper

ATSDR intermediate and acute MRLs for copper are 10 µg/kg/day. Based on the highest residential soil concentration for copper (264,000 µg/kg), Norwood pica estimates for children 1-5 years exceeded these MRLs, producing HQs >1 (Table E.1.1).

ATSDR's intermediate MRL of 10 µg/kg/day is derived from a 2003 study by Araya et al. in which adult men and women were exposed to copper sulfate in drinking water for 2 months (ATSDR 2004). No effects were observed at exposures to 42 µg/kg/day; however, at 91 and 170 µg/kg/day, there were significant increases in gastrointestinal (GI) symptoms such as nausea, abdominal pain, and vomiting. Norwood's highest pica dose of 50 µg/kg/day exceeded this study's No Observed Adverse Effect Level (NOAEL) of 42 µg/kg/day and was only 1.8 times below the study Lowest Observed Adverse Effect Level (LOAEL) of 91 µg/kg/day. **Therefore, it is possible that a 1 year old child engaging in intermediate pica behavior (3 days/week), and exposed to the highest residential soil copper concentration, could experience GI symptoms such as nausea, abdominal pain and vomiting.** In other intermediate-duration studies, reported effect levels occurred at far higher levels than in Araya et al. 2003 (ATSDR 2004). Note: the next highest residential soil sample (72,000 µg/kg/day) produced an intermediate pica exposure dose below the NOAEL of the Araya et al. 2003 study (data not shown).

Similarly, based on the highest residential copper soil concentration (264,000 µg/kg), Norwood single-pica doses among children 1 year old (17 µg/kg) and 2-5 years old (11 µg/kg) exceeded ATSDR's acute MRL of 10 µg/kg/day. This acute MRL is derived from a Pizzaro et al. 1999 study. The study found significantly increased GI symptoms in women exposed to 73.1 µg/kg/day copper sulfate in drinking water for 2 weeks, with 1 week between exposure periods (ATSDR 2004). Estimated single-pica doses at Norwood among children 1-5 years old were 4.3-6.6 times lower than this LOAEL**. As a result, a 1-5 year old child engaging in a single soil pica event for the highest copper soil concentrations at Norwood (from the highest residential soil sample) may experience adverse GI effects such as abdominal pain, nausea, and/or vomiting.**

Numerous human and animal studies report that the GI tract is the most sensitive endpoint from acute oral exposure to copper. In humans, single exposures to 11-30 µg/kg copper sulfate in drinking water caused vomiting, nausea and abdominal pain (ATSDR 2004). These exposure ranges are similar to the highest Norwood single-pica estimates of 11-17 µg/kg/day for children 1 to 5 years old. A few studies have suggested that children are more sensitive to copper's GI effects than adults; however, the available data are inconclusive to assess whether there is an age-related difference in the GI toxicity of copper (ATSDR 2004)**.**

ATSDR notes that copper in soil often is bound to organic molecules. Thus, its bioavailability from soil cannot be assessed based on that from drinking water or food studies (ATSDR 2004). Although more information would be helpful to characterize soil-based copper exposures, pica-estimates at Norwood approached levels that induced gastrointestinal effects from drinking water. **Therefore, children 1-5 years old, if engaging in single or regular (3 days/week) soil-pica behavior and exposed to the highest residential Norwood copper concentration, could experience GI symptoms such as nausea, abdominal pain and vomiting.** Single pica exposure doses for non-residential soil and creek sediment copper were below ATSDR's acute MRL of 10 µg/kg/day.

In April 2022, ATSDR released an updated Toxicological Profile for Copper (Draft for Public Comment), which includes new provisional MRLs. The public comment period for this draft closed on July 26, 2022. PADOH will assess Norwood pica exposure doses in the context of this updated profile as part of the addendum to this Health Consultation, which will encompass EPA's 2020 expanded sampling data.

**E.1.3. Iron**

Pica-based estimates for the highest detected iron concentrations are shown in Table E.1.1. As there are no MRLs for iron, we compared pica doses to EPA's provisional RfD of 700 µg/kg/day, which is protective against gastrointestinal effects found at 1,000 µg/kg/day (EPA 2006). We also compared pica doses to the Institute of Medicine's (IOMs) Tolerable Upper Intake Levels (ULs) for children, of 40mg (IOM 2001).

As shown in Table E.1.1, intermediate pica doses to the highest residential soil sample exceeds EPA's provisional RfD of 700 µg/kg/day, and the LOAEL for gastrointestinal effects, at 1,000 µg/kg/day. However, a single pica exposure estimate for children, of 18.2 mg/per day (highest; residential soil) and 27.3 mg/per day (highest, sediment) were below IOM ULs, of 40 mg. **Therefore, children who engage in regular pica behavior (3 days per week) and ingest iron at the maximum surface soil concentration could experience nausea, vomiting, stomach cramps, or diarrhea.**

**Adults who are on a reduced iron diet to treat intake to treat hemochromatosis should also avoid the consumption of soil.** Further information on hemochromatosis is provided in Appendix D6.

## E2. Additional Chemicals Evaluated for Intermediate Soil-pica Exceedances and High Non-Detect Quantitation Limits

Several chemicals only exceeded an ATSDR screening health-based comparison value (CV) for intermediate-duration soil pica behavior; CVs for non-pica behavior were not exceeded. For these chemicals **(aluminum, antimony, lindane and cadmium),** we evaluated an intermediate and single soil-pica scenario for children 1 year old.

We also evaluated thallium, which was not detected at Norwood but whose maximum non-detect threshold (e.g., the laboratory limit of detection) exceeded EPA's Regional Screening Level (RSL) CV comparison value of 0.078 mg/kg.

Table E.2.1 displays the soil pica intermediate and single-pica estimates for aluminum, antimony, lindane and cadmium based on the highest concentrations detected at Norwood for each of these chemicals. As mentioned, we evaluated an intermediate and single soil-pica scenario for residential soil, and a single soil-pica scenario for creek sediment.

**Table E.2.1. Calculated Pica Exposure Doses and Hazard Quotients for children 1 year old from with pica consumption of the highest aluminum, antimony, lindane, and cadmium concentrations**

| Chemical | Sample location and concentration (µg/kg) | Highest Estimated Intermediate Soil Pica Exposure Dose (µg/kg/day) | Intermediate HQ | Highest Estimated Single Soil Pica Exposure Dose (µg/kg/day) | Acute HQ |
|---|---|---|---|---|---|
| Aluminum | Sediment 25,200,000 | NA | NA | 1,600 Acute MRL: NA | NA |
| | Residential soil 13,600,000 | 2,600 Int MRL: 1,000 | **2.6** | 860 Acute MRL: NA | NA |
| Antimony | Residential Soil 4,200 | 0.8 Int MRL: 0.6 | **1.3** | 0.27 Acute MRL: 1,000 | <1 |
| Lindane | Residential Soil 65 | 0.012 Int MRL: 0.1 | **1.2** | 0.0042 Acute MRL: 3 | <1 |
| Cadmium | Sediment 4,100 | NA | NA | 0.26 Acute MRL: NA | NA |
| | Residential Soil 3,400 | 0.64 Int MRL: 0.5 | **1.3** | 0.22 Acute MRL: NA | NA |

**Bold values** = exceedance of an MRL. HQ = Hazard Quotient; Int = Intermediate; MRL = Minimal Risk Level, expressed in µg/kg/day; NA = Not applicable. NA denotes that intermediate pica was not considered for sediment concentrations. It also denotes occasions where there is no Hazard Quotient due to lack of health guideline.

### E.2.1 Aluminum

Aluminum is the most abundant metal and third most abundant element found in the earth's crust (ATSDR 2008). It is ubiquitous in air, water, soil, and food. Higher aluminum exposures are found in workers involved in aluminum processing, in patients in some medical settings (e.g., patients requiring dialysis or intravenous fluids), in individuals taking large amounts of certain medications, or in people living near industrial plants or hazardous waste sites (ATSDR 2008). The respiratory and nervous systems are most sensitive to chronic aluminum exposure.

Aluminum was detected in all residential (21 of 21), non-residential (17 of 17) and creek sediment samples (8 of 8; Appendix Table C1-C4). All detected concentrations exceeded ATSDR's intermediate soil pica comparison value of 5,300 mg/kg, but not its Chronic Evaluation Media Guide (EMEG) CV of 52,000 mg/kg. Non soil-pica CVs were not exceeded.

The highest intermediate-duration soil pica dose for residential soil was 2,600 µg/kg/day, which exceeded ATSDR's intermediate MRL of 1,000 µg/kg (Table E2).

ATSDR's intermediate MRL is based on two studies finding neurobehavioral and developmental effects in the offspring of Sprague-Dawley rats and Swiss Webster mice. The offspring were exposed to aluminum during gestation and postnatally via diet or drinking water. Developmental and neurobehavioral effects occurred at 53,000 µg/kg/day and 103,000

µg/kg/day thresholds, respectively (ATSDR 2008). ATSDR's intermediate MRL is derived from the studies' No Observed Adverse Effect Level (NOAEL) of 26,000 µg/kg/day.

At Norwood, the highest estimated dose from intermediate-duration soil pica behavior was 2,600 µg/kg/day. This is 10 times lower than the studies' NOAEL of 26,000 µg/kg/day. It is also at least 20 times lower than the studies' developmental (53,000 µg/kg/day) and neurobehavioral (103,000 µg/kg/day) effect levels. Norwood's estimated exposure doses are more than an order of magnitude from these reported effect levels. **As a result, intermediate-duration (3 days/week) soil pica behavior among 1 year old children consuming the highest soil aluminum concentrations is unlikely to result in adverse neurobehavioral or developmental effects.**

ATSDR has not established an acute MRL for aluminum due to inadequate data. ATSDR's toxicological profile identified two acute oral studies in which rat pups exposed during gestation to 100,000-141,000 µg/kg/day aluminum *did not* experience changes in viability/lethality, body weight, or malformation incidence (ATSDR 2008). The highest estimated single soil pica doses at Norwood of 1,600 µg/kg (Table E.2.1) are below these 100,000 – 141,000 µg/kg/day NOAELs.

### E.2.2 Antimony

Antimony is a naturally occurring element present in the earth's crust. General population exposure to antimony, as measured by urinary levels, declined between 1999 and 2006 and has since mostly remained stable. Small amounts of exposure occur from ingestion of food and drinking water. Occupational workers in industries that process or release antimony, such as smelters, coal-fired plants or refuse incinerators, are more highly exposed (ATSDR 2019).

Antimony was detected in 1 of 21 residential samples (4.8%), all 17 non-residential samples (100%) and 5 of 8 sediment samples (62.5%; Appendix Tables C1-C3). The single detected residential concentration of 4,200 µg/kg exceeded an ATSDR intermediate soil-pica CV of 3,200 µg/kg. This sample was an estimated ("J") concentration, indicating that the actual antimony concentration could be higher or lower than the listed value.

As shown in Table E.2.1, an intermediate duration soil-pica scenario produced an exposure dose estimate of 0.8 µg/kg/day. This dose slightly exceeded ATSDR's intermediate MRL for antimony of 0.6 µg/kg/day. Antimony's intermediate MRL is derived from a study by Poon et al. 1987 finding decreases in serum glucose levels in female rats exposed to antimony potassium tartrate in drinking water for 13 weeks (ATSDR 2019). In the Poon et al. 1987 study, no effects were observed at 60 µg/kg/day. This 60 µg/kg/day NOAEL is 75 times' the highest intermediate Norwood soil pica estimate of 0.8 µg/kg/day.

Additionally, a single Norwood soil pica dose scenario did not exceed ATSDR's acute MRL for antimony of 1,000 µg/kg/day (Table E.2.1). **Therefore, intermediate (3 days/week) or single soil-pica behavior among 1 year old children consuming the highest antimony soil concentrations is unlikely to result in adverse health effects.**

### E.2.3. Lindane

Gamma BHC/Lindane is one of several forms of a synthetic chemical called Hexachlorocyclohexane (HCH). It is used as an insecticide on fruit, vegetables, and forest crops, and animals and animal premises (ATSDR 2005). Lindane has not been produced in the U.S. since 1976; however, it is available as an imported insecticide and, in small quantities, as a prescription medicine to treat head lice or scabies. Ingesting lindane residues in contaminated food is the most common route of exposure (ATSDR 2005).

Lindane was only sampled in residential soil and was detected in 6 of 21 (29%) samples. One of these samples (65 µg/kg) exceeded an intermediate-duration soil pica CV of 53 µg/kg. Non soil-pica CVs were not exceeded.

As shown in Table E.2.1, an intermediate soil-pica scenario produced a dose estimate of 0.012 µg/kg/day. This estimate slightly exceeded ATSDR's intermediate MRL for lindane of 0.01 µg/kg/day. ATSDR's intermediate MRL is derived from a 24 week study by Meera et al. 1992 on female Swiss mice, in which mice exposed to 12 µg/kg/day dietary lindane experienced initial stimulation followed by suppression of the cell-mediated and humoral immune systems (ATSDR 2005). The 12 µg/kg/day LOAEL of the Meera et al. study is 1,000 times' the highest Norwood soil-pica estimate of 0.012 µg/kg/day.

Further, a Norwood single soil-pica exposure scenario did not exceed ATSDR's acute lindane MRL of 3 µg/kg/day (Table E.2.1). **Therefore, intermediate (3 days/week) or single soil-pica behavior among 1 year old children consuming the highest lindane soil concentrations is unlikely to result in adverse health effects.**

### E.2.4. Cadmium

Cadmium is a metal found in the earth's crust and associated with zinc, lead, and copper ores (ATSDR 2012b). Batteries are the most common consumer product in which cadmium is used. In the U.S. the primary source of cadmium exposure is the food supply, where leafy vegetables such as lettuce and spinach as well as potatoes and grains, peanuts, and soybeans and sunflower seeds contain approximately 0.05-0.12 mg cadmium. Smoking roughly doubles cadmium body burden compared to not smoking (ATSDR 2012b).

Cadmium was detected in 20 of 21 (95%) residential samples, 17 of 17 (100%) non-residential samples, and 8 of 8 (100%) sediment samples. One residential, 0 non-residential, and 6 sediment samples exceeded ATSDR's intermediate duration soil-pica comparison value of 2.7 mg/kg. Non soil-pica comparison values were not exceeded.

Table E.2.1 displays the highest residential soil and sediment pica estimates for cadmium. As shown in the table, the intermediate residential pica estimate for children 1 year old, of 0.64 µg/kg/day, slightly exceeded ATSDR's intermediate MRL for cadmium of 0.5 µg/kg/day.

ATSDR's intermediate MRL of 0.5 µg/kg/day is derived from Brzóska and colleagues' 2005 study in which 3-week old female rats experienced decreases in bone mineral density and other effects at ≥ 200 µg/kg/day (ATSDR 2012b). The rats were exposed to cadmium chloride in drinking water for 3 months. From this effect level ATSDR set a point of departure lower benchmark (BMDL) dose of 50 µg/kg/day, associated with 1 standard deviation change in 9-month lumbar spine bone density in exposed rats compared to unexposed rats. Norwood's highest pica estimates are several orders of magnitude (78 times') lower than ATSDR's BMDL of 50 µg/kg/day. **Therefore, a 1 year old child consuming residential soil with the highest cadmium concentrations for 3 days/week is unlikely to experience lower bone density.**

ATSDR has not derived an acute oral MRL for cadmium. There are no reliable human studies on the acute toxicity of cadmium (ATSDR 2012b). The highest single pica cadmium estimate for Norwood was 0.26 µg/kg. Among animal studies, effects on bone development (delayed ossification of the sternum and ribs) were observed in rat pups whose mothers were exposed to 2,000 µg/kg/day during gestation, which was the lowest LOAEL identified for possible MRL derivation (ATSDR 2012b). ATSDR did not derive an MRL from this study (by Baranski et al. 1985) due to limitations in the study's reporting (ATSDR 2012b). The 2,000 µg/kg/day threshold identified in the study is approximately 7,500 times' the highest estimated single pica dose estimate at Norwood, of 0.26 µg/kg from creek sediment (Table E.2.1).

**E.2.5. Thallium**

Pure thallium is a metal distributed widely in trace amounts throughout the earth's crust. It exists in air, water and soil and is also found in food. Though no longer produced the United States, thallium is imported and used in small amounts to make electronic device and switches (ATSDR 1992). It's also used in the manufacture of optic lenses (EPA 2009). The most common population-based exposures to thallium occur through food, particularly in home-grown fruits and vegetables. Small amounts of thallium are released into the atmosphere from coal-fired power plants, cement factories and smelting operations, and can deposit onto nearby gardens.

**Thallium Norwood Results.** Thallium was **not** present above the laboratory limit of detection at Norwood (Appendix Tables C1-C4). However, the maximum non-detect quantitation limits

for residential soil (0.76 mg/kg U), non-residential soil (2.9 mg/kg U), and creek sediment (5.9 mg/kg U) exceeded EPA's RSL comparison value for thallium of 0.078 mg/kg.

ATSDR has not derived oral or dermal acute, intermediate, or chronic MRLs for thallium. We therefore conducted a toxicological evaluation based on the maximum quantitation limits of the non-detect values. Estimates are shown in Table E.2.2.

**Table E.2.2. Calculated Exposure Doses and Hazard Quotients based on the highest non-detect Thallium quantitation limits at Norwood**

| Sampling Location | Highest quantitation non-detect limit | Highest exposure estimate (µg/kg/day) | Acute, Intermediate, and Chronic Hazard Quotients |
|---|---|---|---|
| Residential Soil | 0.76 mg/kg U<br>(760 µg/kg U) | 0.14 (int pica, age 1y)<br>0.048 (single pica, age 1y) | NA |
| Creek Sediment | 5.9 mg/kg U<br>(5,900 µg/kg U) | 0.039 (non-pica, child age 6-10 y)<br>0.37 (single pica, age 1y) | |
| U = not detected quantitation limit, Int Pica = Intermediate-duration pica; NA = not applicable; y=years | | | |

EPA has not set a chronic RfD for thallium. It considered a 1988 study that observed hair follicle atrophy in female rats exposed to 200 µg/kg/day thallium salts (EPA 2009). Based on benchmark dose (BMD) modeling, EPA set a potential 95% lower bound BMDL of 10 µg/kg/day. From this modeling EPA considered "candidate" RfDs of 0.01 µg/kg/day for hair follicle atrophy, and 0.003 µg/kg/day for clinical changes in the rats. EPA noted that the thallium database was of poor quality and the principal study suffered multiple limitations (EPA 2009); therefore, it did not derive an RfD from this study.

The highest non-pica exposure dose at Norwood was 0.039 µg/kg/day. This dose exceeds EPA's "candidate" RfDs of 0.01 and 0.003 µg/kg/day but is 5,000 times below the 200 µg/kg/day effect level of hair follicle atrophy reported in the principal 1988 study. It is also 255 times lower than EPA's potential ("candidate") BMDL of 10 µg/kg/day.

Due to the lack of reliable data ATSDR has not established acute, intermediate, or chronic MRLs for thallium. However, its toxicological profile lists several animal studies of acute and intermediate duration. Among acute studies (1-14 days), the lowest identified LOAEL was 80 µg/kg/day for performance deficit in rats exposed to 4 days' thallium via gestation (ATSDR 1992). Although a NOAEL wasn't identified, this 80 µg/kg/day LOAEL is approximately 216 times' the highest "non-detect" thallium single acute pica dose estimate of 0.37 µg/kg/day.

Among intermediate studies (15 days to a year), several found that rats exposed orally to thallium experienced hair loss at thresholds of 1,200 and 1,800 µg/kg/day (ATSDR 1992). No effects (hepatic, renal, cardiovascular, gastrointestinal, respiratory, neurological) were

observed at 200 and 400 µg/kg/day. The lowest observed effects for hair loss at 1,200 µg/kg/day is approximately 8,500 times' Norwood's intermediate pica dose estimate of 0.14 µg/kg/day, among children age 1 year consuming residential soil (Table E.2.2). **Based on these available studies, we would not expect adverse health effects from thallium, under the assumption that the highest thallium quantitation limits represented Norwood site concentrations.**

**Appendix E References**

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# Appendix F. Discussion of Exposures to Chemical of Concern (CoC) Mixtures, based on EPA's 2017-2018 Sampling Data

## F1. Chemical Mixtures

A substantial number of scientific studies have evaluated the health effects from individual environmental chemicals. However, humans are often exposed to multiple chemicals simultaneously. These exposures are called "chemical mixtures," which are influenced by chemical interactions.

Chemical interactions can occur in several ways. In an **additive** reaction, the harmful effects of the mixture would amount to the sum of the effects from each individual chemical. Mathematically, such a reaction could be represented as 2+3=5. In a **synergistic** reaction, the presence of two chemicals produces a greater-than-additive effect than each individually. Mathematically, a synergistic reaction could be represented as 2+3=8 (or 10, or 12, etc.). In an **antagonistic** mixture, one chemical diminishes the effect of the other. Mathematically, an antagonistic effect could be represented as 2+3=3 (ATSDR 2017).

Compared to studies on individual chemicals, there are fewer scientific studies on the impact of chemical mixtures on health. Despite the additive toxicity for some mixtures, there is no evidence for this toxicity when the chemicals are administered simultaneously, but well below their individual thresholds (ATSDR 2005). Of the chemicals of concern (CoC) at Norwood, **benzo[a]pyrene-equivalent polycyclic aromatic hydrocarbons (PAHs)** and **di(2-ethylhexyl) phthalate (DEHP),** exceeded a chronic RfD or intermediate Minimal Risk Level (MRL). Each of these exceedances was for a single residential sample based on the highest childhood estimate. A third CoC, **chromium**, also exceeded a chronic MRL for children based on the highest residential and sediment samples, under the health protective assumption that detected total chromium at Norwood was in its more toxic (chromium VI) form.

As discussed in Appendix D1, we evaluated PAHs as a mixture using California Office of Environmental Health Hazard (OEHHA), as well as ATSDR's potency and toxic equivalency factors for PAHs relative to benzo(a)pyrene (ATSDR 1995). Our toxicological evaluation revealed that adults and children were unlikely to experience adverse non-cancer health effects from the highest estimated combined ingestion and dermal soil PAH exposures. Our toxicological evaluations for DEHP and chromium(VI) also came to these conclusions (Appendices D2 and D9). A brief discussion of these compounds in mixtures is below in section F2.

## F2. Interaction Discussion of PAHs, DEHP and Chromium

***PAHs.*** Humans are usually exposed to PAHs as complex mixtures instead of as individual compounds, from automobile emissions, combustion products of tobacco, or other sources. In occupational settings use of tobacco products in combination with workplace PAH exposure

(e.g., from shale oils, roofing tar emissions, etc.) has been associated with adverse health outcomes. Animal studies show that the interaction of carcinogenic and noncarcinogenic PAHs in a mixture can affect the overall toxicity, including the carcinogenicity, of the mixture, or an individual compound within the mixture (ATSDR 1995). We evaluated both carcinogenic and noncarcinogenic PAHs as a mixture relative to benzo[a]pyrene (e.g., a "benzo[a]pyrene-equivalent" mixture) for residential and non-residential soil PAHs that exceeded benzo[a]pyrene CREG screening levels. **Based on our assessment, exposures to PAH mixtures at Norwood are unlikely to result in adverse non-cancer health effects,** and the highest excess cancer risk estimate from the mixture was **3 in 10,000, for children based on benzo[a]pyrene-equivalent PAH soil concentrations at a single residence.**

***DEHP.*** In animal studies DEHP has been evaluated for its interaction with multiple compounds, including other phthalates (e.g., butlybenzyl phthalate, Di-n-butylphthalate), acetone, and heptachlor (ATSDR 2022). Concentrations of these other compounds at Norwood were well below screening levels (Appendix C). DEHP was evaluated as a mixture with benzo[a]pyrene in a study by Xu et al. 2010 on female mouse reproductive activity, which produced no qualitative evidence of an interaction (ATSDR 2022). In intermediate-duration oral studies, doses of very high DEHP (≥ 500,000 µg/kg/day) with Aroclor 1254 (a polychlorinated biphenyl mixture) led to an additive effect that changed thyroid cell structure and reduced serum thyroid hormones. These additive effects weren't found when exposures were reduced to 50,000 and 100,000 µg/kg/day (ATSDR 2022). **Based on these data and Norwood concentrations, it is unlikely that a DEHP mixture would produce adverse non-cancer health effects.**

***Chromium.*** As discussed in Appendix D, we assumed detected total chromium at Norwood was chromium(VI), an unlikely scenario given that chromium is often in its much less toxic, chromium(III) form in soils. Chromium(VI) has been evaluated for its interaction with ethanol and selenium. Ethanol was detected at low concentration in a single sample at Norwood, and detected selenium was well below comparison values. One study by Myers and Myers 1998 on human cells from 5 participants suggested that agents that increased intracellular iron might lead to increased risk of chromium(VI) toxicity (ATSDR 2012a). Such a study on cells is difficult to conceptualize in the context of Norwood exposures, and chronic iron exposure estimates at Norwood were below health thresholds. Another study by Chou et al. 2008 among cement workers found that the addition of ferrous sulfate (an iron supplement) to the cement reduced total chromium body burden (ATSDR 2012a). **Based on these data and Norwood total chromium concentrations, it is unlikely that a chromium mixture would produce adverse non-cancer health effects.** Further discussion on chromium's interaction with metals is below in section F3.

**F3. Mixtures pertaining to CoC metals**

As discussed in Appendices D and E, we evaluated multiple Norwood metals as CoCs: copper, iron, manganese, arsenic, chromium, mercury, lead, antimony, aluminum and cadmium. Metal exposure doses were low overall, and aside from our assumption of a chromium(VI) scenario,

none exceeded non-pica health guidelines. Further, most CoC metals in soil were similar to typical U.S. concentrations (Appendix Table H.3.1) and only lead had notably higher mean and median concentrations than U.S. background levels. (Mean Norwood soil levels for copper, mercury and cadmium were also higher at Norwood, but below non-pica screening levels.)

Of the Norwood CoC metals, manganese, arsenic, chromium, lead, and cadmium have been most studied for their capacity to cause neurological health effects as mixtures. Arsenic and manganese are thought to have a synergistic effect on lead (ATSDR 2004a, ATSDR 2004b). Laboratory animal studies indicate that manganese, in particular, increases the distribution and/or retention of lead in the brain (ATSDR 2004b). Scientific confidence in these synergistic effects is moderate (ATSDR 2004a, ATSDR 2004b).

The neurological interaction effects for the remaining Norwood metals (e.g., additive, synergistic, antagonistic), whether evaluated with lead or otherwise, are inconsistent, have low confidence ratings, or lack enough evidence to make a scientific determination. In addition, Norwood CoCs copper and iron have been shown to impede the gastrointestinal (GI) absorption of lead in scientific studies, which may reduce the toxicity of lead co-exposure (ATSDR 2017). Animal studies show that gastrointestinal absorption (and the toxicity) of manganese is inversely related to dietary iron (ATSDR 2012b).

**Given the summary information presented in this section, it is possible that the risk for neurological effects for residents (particularly children) exposed to a mixture of metals at Norwood would be greater than for each chemical individually; however, the degree to which this would occur is uncertain. Estimated exposure doses from Norwood metals were low overall and average soil concentrations of manganese, arsenic and chromium were similar to/lower than background U.S. levels (please see Appendix H1). Lead may influence the neurological effect of a chemical mixture; however, two additional CoCs (copper and iron) are known to impede lead GI absorption. Still, there is no safe blood level of lead, particularly in children.**

**Based on existing scientific studies on chemical mixtures and concentrations at Norwood, we would not expect adverse interaction effects for chemicals of highest exposure concern for child non-cancer effects: benzo[a]pyrene-equivalent PAHs, DEHP, and chromium.**

## Appendix F References

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# Appendix G. Pennsylvania Department of Health Cancer Registry Results for Norwood and Surrounding Boroughs, 1985-2019

**Overview of Age-adjusted Standardized Incidence Ratios (SIRs).** SIRs account for age and sex and are obtained by dividing the observed versus expected number of cases of a particular cancer for a defined location and time period. SIRs greater than 1.0 indicate that more cancer cases were observed than expected over a defined time period; SIRs less than 1.0 indicate fewer cases were observed than expected.

The 95% confidence interval (CI) surrounding an SIR determines the precision of the SIR estimate. The narrower the CI the more precise the cancer estimate. A CI that does not include 1.0 is considered statistically significant. Statistically significant SIRs are less likely to have occurred by chance, though chance or other factors cannot be ruled out. For example:

- An SIR of 1.40 (95% CI 0.80 – 4.00) indicates that the observed cancer cases are 40% higher than expected, and this increase is not statistically higher. The 95% CI is wide (not precise) and indicates that we are 95% certain that the true SIR falls somewhere between 0.80 (which would indicate less cancer risk) and 4.00 (which would indicate greater cancer risk).
- An SIR of 1.10 (95% CI 1.05-1.15) indicates that the observed number of cancer cases are 10% higher than expected, and this increase is statistically higher. The 95% CI is precise and indicates that we are 95% certain that the true SIR falls between 1.05 and 1.15 (which would indicate greater cancer risk).

Below are the PA 1985-2019 cancer registry results for 22 specific cancers by sex, comparing SIRs at Norwood, Prospect Park and Folcroft combined (Appendix Table G1) and at Norwood compared to Pennsylvania (Appendix Table G2). The Table for Norwood, Prospect Park and Folcroft combined compared to Delaware County is listed and discussed in Section 7.1 of the main report.

**Appendix Table G1.** Age-adjusted Standardized Incidence Ratios (SIR) and 95% Confidence Intervals (CI) for various cancers among Male and Females at **Norwood, Folcroft, and Prospect Park Boroughs combined compared to the Commonwealth of Pennsylvania** (1985-1994, 1995-2004, 2005-2014, 2015-2019). 2019 is the most recent year of Pennsylvania Cancer Registry data based on the date of this report.

**Bold** = observed were cases than expected and statistically significant for the specified time period. *Underlined Italics* = observed cases were lower than expected and statistically significant for the specified time period.

| **Time Period** | | **1985 – 1994** | | **1995-2004** | | **2005-2014** | | **2015-2019** | |
|---|---|---|---|---|---|---|---|---|---|
| **Gender** | | Male | Female | Male | Female | Male | Female | Male | Female |
| **Bladder** | **Exp.** | 31.7 | 11.7 | 36.4 | 13.2 | 36.7 | 12.6 | 16.4 | 5.8 |
| | **Obs.** | 39 | 17 | 46 | 15 | 43 | 17 | 20 | 5 |
| | **SIR** | 1.23 | 1.45 | 1.26 | 1.14 | 1.17 | 1.35 | 1.22 | 0.86 |
| | **95% CI** | 0.88 - 1.68 | 0.85 - 2.33 | 0.92 - 1.69 | 0.64 - 1.88 | 0.85 - 1.58 | 0.79 - 2.16 | 0.75- 1.88 | 0.28 – 2.0 |
| | | | | | | | | | |
| **Brain** | **Exp.** | 7.4 | 6.2 | 8.0 | 7.8 | 16.4 | 25.2 | 8.7 | 12.6 |
| | **Obs.** | 4 | 5 | 7 | 10 | 21 | 26 | 14 | 8 |
| | **SIR** | 0.54 | 0.81 | 0.87 | 1.28 | 1.28 | 1.03 | 1.62 | 0.63 |
| | **95% CI** | 0.15 - 1.38 | 0.26 - 1.88 | 0.35 - 1.8 | 0.62 - 2.36 | 0.79 - 1.96 | 0.67 - 1.51 | 0.88 - 2.71 | 0.27 - 1.25 |
| | | | | | | | | | |
| **Breast (Female Pop. Only)** | **Exp.** | N/A | 145.3 | N/A | 171.3 | N/A | 178.1 | N/A | 85.9 |
| | **Obs.** | N/A | 168 | N/A | 185 | N/A | 198 | N/A | 82 |
| | **SIR** | N/A | 1.16 | N/A | 1.08 | N/A | 1.11 | N/A | 0.96 |
| | **95% CI** | N/A | 0.99 - 1.35 | N/A | 0.93 - 1.25 | N/A | 0.96 - 1.28 | N/A | 0.76 - 1.19 |
| | | | | | | | | | |
| **Cervix (Female Pop. Only)** | **Exp.** | N/A | 43.9 | N/A | 14.6 | N/A | 7.9 | N/A | 3.6 |
| | **Obs.** | N/A | 42 | N/A | 16 | N/A | 8 | N/A | 4 |
| | **SIR** | N/A | 0.96 | N/A | 1.10 | N/A | 1.02 | N/A | 1.11 |
| | **95% CI** | N/A | 0.69 - 1.29 | N/A | 0.63 - 1.78 | N/A | 0.44 - 2.01 | N/A | 0.30 - 2.84 |
| | | | | | | | | | |
| **Colon** | **Exp.** | 66.0 | 65.2 | 66.4 | 64.9 | 52.6 | 51.0 | 22.0 | 20.9 |
| | **Obs.** | 75 | 69 | 82 | 77 | 73 | 51 | 37 | 23 |
| | **SIR** | 1.14 | 1.06 | 1.23 | 1.19 | **1.39** | 1.00 | **1.68** | 1.10 |
| | **95% CI** | 0.89 - 1.43 | 0.82 - 1.34 | 0.98 - 1.53 | 0.94 - 1.48 | **1.09 - 1.74** | 0.74 - 1.31 | **1.19 - 2.32** | 0.70 - 1.65 |
| | | | | | | | | | |
| **Esophagus** | **Exp.** | 6.9 | 2.4 | 8.3 | 2.4 | 8.6 | 2.4 | 4.4 | 1.2 |
| | **Obs.** | 7 | 3 | 5 | 2 | 7 | 4 | 2 | 2 |
| | **SIR** | 1.02 | 1.26 | 0.61 | 0.82 | 0.82 | 1.67 | 0.46 | 1.71 |

| | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| | **95% CI** | 0.41 - 2.10 | 0.26 - 3.69 | 0.20 - 1.41 | 0.10 - 2.96 | 0.33 - 1.68 | 0.46 - 4.28 | 0.06 - 1.64 | 0.21 - 6.16 |
| | | | | | | | | | |
| **Hodgkin's Lymphoma** | **Exp.** | 3.8 | 3.2 | 3.6 | 3.0 | 3.4 | 3.0 | 1.7 | 1.3 |
| | **Obs.** | 1 | 2 | 2 | 1 | 3 | 3 | 0 | 5 |
| | **SIR** | 0.27 | 0.62 | 0.55 | 0.34 | 0.87 | 1.01 | - | **3.73** |
| | **95% CI** | 0.01 - 1.48 | 0.08 - 2.26 | 0.07 - 1.99 | 0.01 - 1.87 | 0.18 - 2.55 | 0.21 - 2.96 | - | **1.21 - 8.71** |
| | | | | | | | | | |
| **Kidney** | **Exp.** | 12.1 | 7.9 | 16.5 | 10.7 | 21.3 | 13.4 | 11.5 | 6.5 |
| | **Obs.** | 6 | 5 | 18 | 16 | 22 | 14 | 11 | 1 |
| | **SIR** | 0.49 | 0.63 | 1.09 | 1.49 | 1.03 | 1.04 | 0.96 | *0.15* |
| | **95% CI** | 0.18 - 1.08 | 0.20 - 1.47 | 0.64 - 1.72 | 0.85 - 2.42 | 0.65 - 1.57 | 0.57 - 1.75 | 0.48 - 1.71 | *0 - 0.86* |
| | | | | | | | | | |
| **Laryngeal** | **Exp.** | 9.9 | 2.4 | 8.0 | 2.2 | 6.8 | 1.9 | 2.9 | 0.8 |
| | **Obs.** | 15 | 3 | 13 | 6 | 9 | 3 | 3 | 1 |
| | **SIR** | 1.52 | 1.23 | 1.63 | 2.73 | 1.32 | 1.58 | 1.05 | 1.21 |
| | **95% CI** | 0.85-2.51 | 0.25 - 3.60 | 0.87-2.79 | 1.0 - 5.95 | 0.60-2.51 | 0.33-4.60 | 0.22-3.06 | 0.03 - 6.73 |
| | | | | | | | | | |
| **Leukemia** | **Exp.** | 12.0 | 9.6 | 14.7 | 11.4 | 16.4 | 12.5 | 8.4 | 6.3 |
| | **Obs.** | 11 | 6 | 10 | 13 | 15 | 13 | 6 | 8 |
| | **SIR** | 0.91 | 0.62 | 0.68 | 1.14 | 0.92 | 1.04 | 0.71 | 1.28 |
| | **95% CI** | 0.46 - 1.63 | 0.23 - 1.36 | 0.33 - 1.25 | 0.61 - 1.95 | 0.51 - 1.51 | 0.55 - 1.78 | 0.26 - 1.56 | 0.55 - 2.52 |
| | | | | | | | | | |
| **Liver** | **Exp.** | 3.2 | 1.8 | 6.4 | 2.9 | 10.9 | 4.0 | 6.4 | 2.5 |
| | **Obs.** | 5 | 1 | 10 | 4 | 15 | 9 | 8 | 2 |
| | **SIR** | 1.54 | 0.55 | 1.56 | 1.40 | 1.38 | **2.27** | 1.24 | 0.79 |
| | **95% CI** | 0.50 - 3.60 | 0.01 - 3.08 | 0.75 - 2.86 | 0.38 - 3.59 | 0.77 - 2.27 | **1.04 - 4.32** | 0.54 - 2.45 | 0.10 - 2.86 |
| | | | | | | | | | |
| **Lung** | **Exp.** | 80.8 | 45.8 | 80.5 | 60.4 | 70.7 | 64.3 | 30.5 | 31.2 |
| | **Obs.** | 110 | 72 | 109 | 102 | 119 | 104 | 56 | 56 |
| | **SIR** | **1.36** | **1.57** | **1.35** | **1.69** | **1.68** | **1.62** | **1.83** | **1.79** |
| | **95% CI** | **1.12 - 1.64** | **1.23 - 1.98** | **1.11-1.63** | **1.38 - 2.05** | **1.4 - 2.02** | **1.32 - 1.96** | **1.38 - 2.38** | **1.36 - 2.33** |
| | | | | | | | | | |
| **Melanoma** | **Exp.** | 10.6 | 8.5 | 21.9 | 17.8 | 36.8 | 31.0 | 22 | 17.8 |
| | **Obs.** | 17 | 5 | 27 | 22 | 44 | 36 | 27 | 12 |
| | **SIR** | 1.60 | 0.59 | 1.23 | 1.24 | 1.20 | 1.16 | 1.23 | 0.67 |
| | **95% CI** | 0.93-2.56 | 0.19 - 1.37 | 0.81-1.79 | 0.77 - 1.87 | 0.87-1.60 | 0.81 - 1.61 | 0.81-1.79 | 0.35 - 1.18 |
| | | | | | | | | | |
| **Myeloma** | **Exp.** | 4.6 | 4.6 | 5.7 | 5.5 | 6.9 | 5.9 | 3.8 | 3.1 |

| | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| | **Obs.** | 3 | 5 | 3 | 5 | 10 | 6 | 1 | 2 |
| | **SIR** | 0.65 | 1.09 | 0.52 | 0.90 | 1.44 | 1.02 | 0.26 | 0.64 |
| | **95% CI** | 0.13 - 1.90 | 0.35 - 2.53 | 0.11 - 1.53 | 0.29 - 2.11 | 0.69 - 2.65 | 0.37 - 2.21 | 0.01 - 1.47 | 0.08 - 2.32 |
| | | | | | | | | | |
| **Non-Hodgkin's Lymphoma** | **Exp.** | 15.9 | 14.8 | 21.1 | 19.3 | 22.7 | 20.1 | 10.7 | 9.4 |
| | **Obs.** | 16 | 18 | 19 | 26 | 24 | 13 | 19 | 10 |
| | **SIR** | 1.01 | 1.22 | 0.90 | 1.35 | 1.06 | 0.65 | **1.78** | 1.06 |
| | **95% CI** | 0.58 - 1.64 | 0.72 - 1.92 | 0.54 - 1.41 | 0.88 - 1.97 | 0.68 - 1.57 | 0.35 - 1.11 | **1.07 - 2.78** | 0.51 - 1.95 |
| | | | | | | | | | |
| **Oral** | **Exp.** | 13.6 | 6.5 | 13.7 | 6.5 | 16.6 | 7.5 | 9.2 | 3.9 |
| | **Obs.** | 23 | 6 | 15 | 8 | 17 | 4 | 14 | 5 |
| | **SIR** | **1.69** | 0.93 | 1.10 | 1.23 | 1.03 | 0.53 | 1.51 | 1.27 |
| | **95% CI** | **1.07 - 2.53** | 0.34 - 2.02 | 0.61 - 1.81 | 0.53 - 2.41 | 0.60 - 1.64 | 0.15 - 1.37 | 0.83 - 2.54 | 0.41- 2.97 |
| | | | | | | | | | |
| **Ovary (Female Pop. Only)** | **Exp.** | N/A | 17.4 | N/A | 18.0 | N/A | 14.5 | N/A | 6.2 |
| | **Obs.** | N/A | 14 | N/A | 18 | N/A | 14 | N/A | 11 |
| | **SIR** | N/A | 0.81 | N/A | 1.00 | N/A | 0.96 | N/A | 1.78 |
| | **95% CI** | N/A | 0.44 - 1.35 | N/A | 0.59 - 1.58 | N/A | 0.53 - 1.62 | N/A | 0.89 - 3.18 |
| | | | | | | | | | |
| **Pancreas** | **Exp.** | 8.4 | 9.0 | 11.0 | 11.2 | 13.1 | 13.3 | 7.5 | 7.3 |
| | **Obs.** | 12 | 10 | 8 | 7 | 12 | 15 | 9 | 9 |
| | **SIR** | 1.43 | 1.11 | 0.73 | 0.63 | 0.92 | 1.13 | 1.20 | 1.23 |
| | **95% CI** | 0.74 - 2.5 | 0.53 - 2.04 | 0.31 - 1.43 | 0.25 - 1.29 | 0.47 - 1.6 | 0.63 - 1.86 | 0.55 - 2.28 | 0.56 - 2.33 |
| | | | | | | | | | |
| **Prostate (Male Pop. Only)** | **Exp.** | 105.2 | N/A | 148.9 | N/A | 124.9 | N/A | 52.4 | N/A |
| | **Obs.** | 115 | N/A | 129 | N/A | 130 | N/A | 49 | N/A |
| | **SIR** | 1.09 | N/A | 0.87 | N/A | 1.04 | N/A | 0.93 | N/A |
| | **95% CI** | 0.90 - 1.31 | N/A | 0.72 - 1.03 | N/A | 0.87 - 1.24 | N/A | 0.69 - 1.24 | N/A |
| | | | | | | | | | |
| **Stomach** | **Exp.** | 10.2 | 6.4 | 9.5 | 5.9 | 8.7 | 5.0 | 3.8 | 2.2 |
| | **Obs.** | 9 | 4 | 13 | 4 | 18 | 3 | 3 | 4 |
| | **SIR** | 0.88 | 0.62 | 1.37 | 0.68 | **2.08** | 0.60 | 0.78 | 1.78 |
| | **95% CI** | 0.40 - 1.67 | 0.17 - 1.60 | 0.73 - 2.34 | 0.19 - 1.74 | **1.23 - 3.28** | 0.12 - 1.75 | 0.16 - 2.28 | 0.49 - 4.56 |
| | | | | | | | | | |
| **Testis (Male Pop. Only)** | **Exp.** | 5.8 | N/A | 5.9 | N/A | 6.1 | N/A | 3.3 | N/A |
| | **Obs.** | 4 | N/A | 8 | N/A | 2 | N/A | 3 | N/A |
| | **SIR** | 0.69 | N/A | 1.36 | N/A | 0.33 | N/A | 0.92 | N/A |
| | **95% CI** | 0.19- 1.77 | N/A | 0.59- 2.68 | N/A | 0.04- 1.18 | N/A | 0.19- 2.70 | N/A |

| | | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| **Thyroid** | **Exp.** | 2.4 | 6.4 | 4.4 | 14.9 | 8.8 | 28.8 | 4.7 | 13.2 |
| | **Obs.** | 2 | 6 | 0 | 10 | 7 | 20 | 6 | 16 |
| | **SIR** | 0.83 | 0.93 | - | 0.67 | 0.79 | 0.70 | 1.27 | 1.21 |
| | **95% CI** | 0.10 - 3.01 | 0.34 - 2.03 | - | 0.32 - 1.23 | 0.32 - 1.63 | 0.42 - 1.07 | 0.47 - 2.77 | 0.69 - 1.97 |
| | | | | | | | | | |
| **Uterus (Female Pop. Only)** | **Exp.** | N/A | 29.8 | N/A | 33.1 | N/A | 35.5 | N/A | 17.9 |
| | **Obs.** | N/A | 28 | N/A | 30 | N/A | 36 | N/A | 24 |
| | **SIR** | N/A | 0.94 | N/A | 0.91 | N/A | 1.01 | N/A | 1.34 |
| | **95% CI** | N/A | 0.62 - 1.36 | N/A | 0.61 - 1.29 | N/A | 0.71 - 1.40 | N/A | 0.86 - 1.99 |

Exp = Expected, Obs = Observed, SIR = Standardized Incidence Ratio, CI = Confidence Interval, N/A = Not Applicable

**Appendix Table G2.** Age-adjusted Standardized Incidence Ratios (SIR) and 95% Confidence Intervals (CI) for various cancers among Male and Females at **Norwood Borough compared to the Commonwealth of Pennsylvania** (1985-1994, 1995-2004, 2005-2014, 2015-2019). 2019 is the most recent year of Pennsylvania Cancer Registry data based on the date of this report.

**Bold** = observed were cases than expected and statistically significant for the specified time period.
*Underlined Italics* = observed cases were lower than expected and statistically significant for the specified time period.

| **Time Period** | | **1985 – 1994** | | **1995-2004** | | **2005-2014** | | **2015-2019** | |
|---|---|---|---|---|---|---|---|---|---|
| **Gender** | | **Male** | **Female** | **Male** | **Female** | **Male** | **Female** | **Male** | **Female** |
| **Bladder** | **Exp.** | 9.7 | 3.4 | 10.5 | 3.8 | 11.1 | 3.9 | 4.6 | 1.8 |
| | **Obs.** | 14 | 2 | 11 | 5 | 12 | 5 | 7 | 3 |
| | **SIR** | 1.45 | 0.58 | 1.05 | 1.33 | 1.08 | 1.30 | 1.54 | 1.62 |
| | **95% CI** | 0.79 - 2.43 | 0.07 - 2.10 | 0.52 - 1.88 | 0.43 - 3.09 | 0.56 - 1.88 | 0.42 - 3.02 | 0.62 - 3.17 | 0.33 - 4.74 |
| | | | | | | | | | |
| **Brain** | **Exp.** | 2.3 | 1.9 | 2.4 | 2.3 | 5.1 | 7.9 | 2.6 | 4.1 |
| | **Obs.** | 1 | 2 | 0 | 3 | 7 | 6 | 9 | 5 |
| | **SIR** | 0.44 | 1.08 | - | 1.29 | 1.36 | 0.76 | **3.43** | 1.21 |
| | **95% CI** | 0.01 - 2.46 | 0.13 - 3.89 | - | 0.27 - 3.76 | 0.55 - 2.81 | 0.28 - 1.65 | **1.57 - 6.50** | 0.39 - 2.83 |
| | | | | | | | | | |
| **Breast (Female Pop. Only)** | **Exp.** | N/A | 43.7 | N/A | 51.3 | N/A | 57.2 | N/A | 30.5 |
| | **Obs.** | N/A | 51 | N/A | 65 | N/A | 70 | N/A | 29 |
| | **SIR** | N/A | 1.17 | N/A | 1.27 | N/A | 1.22 | N/A | 0.95 |
| | **95% CI** | N/A | 0.87 - 1.53 | N/A | 0.98 - 1.61 | N/A | 0.95 - 1.55 | N/A | 0.64 - 1.37 |
| | | | | | | | | | |
| **Cervix (Female Pop. Only)** | **Exp.** | N/A | 12.9 | N/A | 4.5 | N/A | 2.5 | N/A | 1.2 |
| | **Obs.** | N/A | 12 | N/A | 3 | N/A | 1 | N/A | 2 |
| | **SIR** | N/A | 0.93 | N/A | 0.67 | N/A | 0.40 | N/A | 1.73 |
| | **95% CI** | N/A | 0.48 - 1.62 | N/A | 0.14 - 1.95 | N/A | 0.01 - 2.23 | N/A | 0.21 - 6.26 |
| | | | | | | | | | |
| **Colon** | **Exp.** | 20.1 | 19.2 | 19.2 | 18.5 | 16.4 | 15.7 | 6.7 | 6.7 |
| | **Obs.** | 20 | 20 | 20 | 26 | 19 | 15 | 14 | 10 |
| | **SIR** | 0.99 | 1.04 | 1.04 | 1.40 | 1.16 | 0.96 | **2.10** | 1.49 |
| | **95% CI** | 0.61 - 1.53 | 0.64 - 1.61 | 0.64 - 1.61 | 0.92 - 2.06 | 0.7 - 1.81 | 0.54 - 1.58 | **1.15 - 3.52** | 0.72 - 2.75 |
| | | | | | | | | | |
| **Esophagus** | **Exp.** | 2.1 | 0.7 | 2.4 | 0.7 | 2.7 | 0.7 | 1.4 | 0.4 |
| | **Obs.** | 5 | 1 | 3 | 1 | 3 | 2 | 1 | 0 |
| | **SIR** | 2.38 | 1.42 | 1.24 | 1.43 | 1.11 | 2.70 | 0.72 | - |
| | **95% CI** | 0.77 - 5.56 | 0.04 - 7.91 | 0.26 - 3.64 | 0.04 - 7.96 | 0.23 - 3.25 | 0.33 - 9.77 | 0.02 - 3.99 | - |

| | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| **Hodgkin's Lymphoma** | **Exp.** | 1.2 | 1.0 | 1.1 | 0.9 | 1.1 | 0.9 | 0.5 | 0.4 |
| | **Obs.** | 0 | 1 | 1 | 0 | 0 | 2 | 0 | 2 |
| | **SIR** | - | 1.04 | 0.91 | - | - | 2.21 | - | 4.78 |
| | **95% CI** | - | 0.03 - 5.82 | 0.02 - 5.06 | - | - | 0.27 - 8 | - | 0.58 - 17.26 |
| | | | | | | | | | |
| **Kidney** | **Exp.** | 3.7 | 2.4 | 4.9 | 3.2 | 6.7 | 4.2 | 3.7 | 2.2 |
| | **Obs.** | 0 | 2 | 6 | 6 | 11 | 9 | 2 | 0 |
| | **SIR** | - | 0.84 | 1.23 | 1.90 | 1.63 | 2.13 | 0.54 | - |
| | **95% CI** | - | 0.1 - 3.04 | 0.45 - 2.68 | 0.7 - 4.14 | 0.82 - 2.93 | 0.97 - 4.04 | 0.07 - 1.96 | - |
| | | | | | | | | | |
| **Laryngeal** | **Exp.** | 3.0 | 0.7 | 2.3 | 0.7 | 2.2 | 0.6 | 0.9 | 0.3 |
| | **Obs.** | 5 | 1 | 7 | 0 | 4 | 1 | 0 | 0 |
| | **SIR** | 1.66 | 1.37 | **2.99** | - | 1.85 | 1.64 | - | - |
| | **95% CI** | 0.54 - 3.88 | 0.03 - 7.62 | **1.2 - 6.16** | - | 0.50 - 4.74 | 0.04 - 9.15 | - | - |
| | | | | | | | | | |
| **Leukemia** | **Exp.** | 3.6 | 2.8 | 4.3 | 3.3 | 5.0 | 3.8 | 2.5 | 2.0 |
| | **Obs.** | 5 | 1 | 2 | 3 | 2 | 3 | 1 | 0 |
| | **SIR** | 1.37 | 0.35 | 0.47 | 0.90 | 0.40 | 0.78 | 0.40 | - |
| | **95% CI** | 0.44 - 3.20 | 0.01 - 1.96 | 0.06 - 1.68 | 0.19 - 2.64 | 0.05 - 1.44 | 0.16 - 2.28 | 0.01 - 2.26 | - |
| | | | | | | | | | |
| **Liver** | **Exp.** | 1.0 | 0.5 | 1.9 | 0.8 | 3.5 | 1.2 | 2.2 | 0.9 |
| | **Obs.** | 3 | 0 | 3 | 2 | 1 | 4 | 4 | 1 |
| | **SIR** | 3.03 | - | 1.58 | 2.42 | 0.29 | 3.24 | 1.84 | 1.17 |
| | **95% CI** | 0.62 - 8.85 | - | 0.33 - 4.61 | 0.29 - 8.73 | 0.01 - 1.60 | 0.88 - 8.31 | 0.50 - 4.70 | 0.03 - 6.53 |
| | | | | | | | | | |
| **Lung** | **Exp.** | 24.7 | 13.9 | 23.3 | 17.7 | 21.8 | 20.0 | 9.3 | 10.7 |
| | **Obs.** | 36 | 26 | 22 | 28 | 32 | 39 | 26 | 21 |
| | **SIR** | **1.46** | **1.87** | 0.95 | **1.59** | 1.47 | **1.95** | **2.79** | **1.96** |
| | **95% CI** | **1.02 - 2.02** | **1.22 - 2.75** | 0.59 - 1.43 | **1.05 - 2.29** | 1 - 2.07 | **1.39 - 2.66** | **1.82 - 4.09** | **1.21 - 2.99** |
| | | | | | | | | | |
| **Melanoma** | **Exp.** | 3.3 | 2.6 | 6.5 | 5.4 | 11.5 | 9.8 | 6.7 | 6.0 |
| | **Obs.** | 4 | 2 | 9 | 11 | 14 | 13 | 14 | 4 |
| | **SIR** | 1.23 | 0.78 | 1.38 | **2.04** | 1.22 | 1.32 | **2.10** | 0.67 |
| | **95% CI** | 0.34 - 3.15 | 0.09 - 2.82 | 0.63 - 2.62 | **1.02 - 3.66** | 0.67 - 2.05 | 0.7 - 2.26 | **1.15 - 3.52** | 0.18 - 1.71 |
| | | | | | | | | | |
| **Myeloma** | **Exp.** | 1.4 | 1.4 | 1.7 | 1.6 | 2.2 | 1.8 | 1.2 | 1.1 |
| | **Obs.** | 1 | 1 | 2 | 3 | 3 | 1 | 1 | 2 |
| | **SIR** | 0.71 | 0.73 | 1.20 | 1.88 | 1.39 | 0.55 | 0.86 | 1.89 |

| | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| | **95% CI** | 0.02 - 3.96 | 0.02 - 4.08 | 0.15 - 4.34 | 0.39 - 5.49 | 0.29 - 4.08 | 0.01 - 3.05 | 0.02 - 4.77 | 0.23 - 6.82 |
| | | | | | | | | | |
| **Non-Hodgkin's Lymphoma** | **Exp.** | 4.8 | 4.4 | 6.2 | 5.6 | 7.1 | 6.2 | 3.2 | 3.1 |
| | **Obs.** | 5 | 6 | 10 | 9 | 9 | 3 | 10 | 3 |
| | **SIR** | 1.03 | 1.36 | 1.61 | 1.59 | 1.28 | 0.48 | **3.11** | 0.96 |
| | **95% CI** | 0.34 - 2.41 | 0.50 - 2.95 | 0.77 - 2.95 | 0.73 - 3.03 | 0.58 - 2.42 | 0.10 - 1.41 | **1.49 - 5.72** | 0.20 - 2.81 |
| | | | | | | | | | |
| **Oral** | **Exp.** | 4.1 | 1.9 | 4.1 | 1.9 | 5.3 | 2.4 | 3.1 | 1.3 |
| | **Obs.** | 8 | 2 | 5 | 3 | 6 | 0 | 6 | 1 |
| | **SIR** | 1.93 | 1.04 | 1.22 | 1.56 | 1.13 | - | 1.96 | 0.74 |
| | **95% CI** | 0.83 - 3.80 | 0.13 - 3.76 | 0.40 - 2.85 | 0.32 - 4.56 | 0.41 - 2.46 | - | 0.72 - 4.27 | 0.02 - 4.15 |
| | | | | | | | | | |
| **Ovary** | **Exp.** | N/A | 5.2 | N/A | 5.4 | N/A | 4.6 | N/A | 2.1 |
| | **Obs.** | N/A | 3 | N/A | 1 | N/A | 1 | N/A | 0 |
| | **SIR** | N/A | 0.58 | N/A | 0.19 | N/A | 0.22 | N/A | - |
| | **95% CI** | N/A | 0.12 - 1.68 | N/A | 0 - 1.04 | N/A | 0.01 - 1.21 | N/A | - |
| | | | | | | | | | |
| **Pancreas** | **Exp.** | 2.6 | 2.7 | 3.2 | 3.2 | 4.0 | 4.1 | 2.3 | 2.4 |
| | **Obs.** | 6 | 4 | 2 | 3 | 3 | 8 | 2 | 3 |
| | **SIR** | 2.35 | 1.50 | 0.62 | 0.94 | 0.74 | 1.97 | 0.87 | 1.27 |
| | **95% CI** | 0.86 - 5.11 | 0.41 - 3.83 | 0.08 - 2.26 | 0.19 - 2.75 | 0.15 - 2.17 | 0.85 - 3.88 | 0.11 - 3.16 | 0.26 - 3.71 |
| | | | | | | | | | |
| **Prostate (Male Pop. only)** | **Exp.** | 32.3 | N/A | 42.8 | N/A | 39.6 | N/A | 18.0 | N/A |
| | **Obs.** | 29 | N/A | 45 | N/A | 44 | N/A | 12 | N/A |
| | **SIR** | 0.90 | N/A | 1.05 | N/A | 1.11 | N/A | 0.67 | N/A |
| | **95% CI** | 0.60 - 1.29 | N/A | 0.77 - 1.41 | N/A | 0.81 - 1.49 | N/A | 0.34 - 1.17 | N/A |
| | | | | | | | | | |
| **Stomach** | **Exp.** | 3.1 | 1.9 | 2.7 | 1.7 | 2.7 | 1.5 | 1.2 | 0.7 |
| | **Obs.** | 4 | 1 | 7 | 2 | 4 | 0 | 0 | 0 |
| | **SIR** | 1.28 | 0.54 | **2.55** | 1.21 | 1.49 | - | - | - |
| | **95% CI** | 0.35 - 3.28 | 0.01 - 2.99 | **1.02 - 5.25** | 0.15 - 4.36 | 0.41 - 3.82 | - | - | - |
| | | | | | | | | | |
| **Testis (Male Pop. Only)** | **Exp.** | 1.7 | N/A | 1.8 | N/A | 1.9 | N/A | 1.0 | *N/A* |
| | **Obs.** | 3 | N/A | 3 | N/A | 0 | N/A | 2 | *N/A* |
| | **SIR** | 1.72 | N/A | 1.65 | N/A | - | N/A | 2.04 | *N/A* |
| | **95% CI** | 0.36 - 5.03 | N/A | 0.34 - 4.83 | N/A | - | N/A | 0.25 - 7.36 | *N/A* |
| | | | | | | | | | |
| **Thyroid** | **Exp.** | 0.7 | 1.9 | 1.3 | 4.6 | 2.8 | 9.1 | 1.5 | 4.3 |

| | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| | **Obs.** | 0 | 3 | 0 | 1 | 1 | 9 | 0 | 6 |
| | **SIR** | - | 1.55 | - | 0.22 | 0.35 | 0.98 | - | 1.39 |
| | **95% CI** | - | 0.32 - 4.54 | - | 0.01 - 1.20 | 0.01 - 1.97 | 0.45 - 1.87 | - | 0.51 - 3.02 |
| | | | | | | | | | |
| **Uterus (Female Pop. Only)** | **Exp.** | N/A | 8.9 | N/A | 9.9 | N/A | 11.5 | N/A | 6.6 |
| | **Obs.** | N/A | 12 | N/A | 10 | N/A | 5 | N/A | 12 |
| | **SIR** | N/A | 1.34 | N/A | 1.01 | N/A | 0.44 | N/A | 1.81 |
| | **95% CI** | N/A | 0.69 - 2.34 | N/A | 0.48 - 1.86 | N/A | 0.14 - 1.02 | N/A | 0.93 - 3.15 |
| Exp = Expected, Obs = Observed, SIR = Standardized Incidence Ratio, CI = Confidence Interval, N/A = Not Applicable | | | | | | | | | |

# Appendix H. Evaluation of Community Concerns and Timeline of Agency Activities

**H1. Cancer**

Winona residents identified cancer from their proximity to the former Norwood Landfill as a concern. As discussed in section 7, we evaluated cancer in two ways:

1. By calculating excess cancer risk based on exposures to chemical levels found from 2017-2018 concentrations at Norwood (please see HC section 5 or Appendix D).

2. By evaluating Norwood and surrounding borough age-adjusted cancer incidence rates by sex compared to the rest of Delaware County and Pennsylvania (please see HC section 7).

As for item 1, the highest excess cancer risk estimates for residential exposure were 3 in 10,000 for children and 2 in 100,000 based on the maximum detected benzo[a]pyrene-equivalent polycyclic aromatic hydrocarbon (PAHs) exposures at Norwood. The residence with the next highest PAH concentrations produced an excess cancer risk estimate of 7 in 100,000 for children and 5 in 1 million for adults.

Aside from benzo[a]pyrene-equivalent PAHs, the chemicals producing the highest cancer risk estimates were chromium(VI) (2 in 10,000 for children and 2 in 100,000 for adults) and dieldrin (5 in 100,000 for children and 2 in 100,000 for adults). Our chromium(VI) exposure estimate assumed that all detected total chromium concentrations were chromium(VI). In most soils, total chromium is in its much less toxic, chromium(III) form.

Further details on these chemicals are discussed in section 5 (summary) and Appendix D (in more detail) of this HC. Appendix B4 discusses how excess cancer risk is derived and provides further detail on its interpretation.

As for item 2, lung cancer was consistently higher and statistically significant for both sexes for 4 time periods assessed (1985-1994; 1995-2004; 2005-2014; 2015-2019) between 1985-2019. Several other cancer types were higher and statistically significant, though showed no clear pattern.

The consistent lung cancer pattern at Norwood does not establish causality between the rates observed and environmental exposures. There are many factors that influence cancer rates, and while the registry accounts for age (an important factor), it does not account for many other factors known to influence cancer rates, such as behaviors (such as smoking or diet), family history, etc. Smoking is by far the biggest risk factor for lung cancer. Regarding environmental contributors to cancers, exposure to a cancer-causing chemical does *not* mean

someone will get cancer; the risk increases by how much, how often, and how long the exposures have been taking place (ATSDR 2015). In scientific studies, several Norwood CoCs – PAHs/benzo[a]pyrene, lead, PCBs, arsenic or chromium(VI) – have been associated with increased risk for lung cancer. Most often these risks have been found in certain workers exposed to these compounds regularly (e.g., PAHs/benzo[a]pyrene, lead, chromium(VI) or PCBs) or in populations exposed to contaminated drinking water (e.g., to arsenic). Given that most residents are and have historically been on a public water system, these exposures are unlikely for most residents, or for workers who do not use these compounds. Our full cancer outcome data discussion is found in section 7.1 of the main report.

**H2. Multiple Sclerosis**

Our discussion of multiple sclerosis is provided in part 7.2 of the main report.

**H3. Metals and PCB contamination**

Another concern was that Winona community homes may have been constructed on contaminated soil containing excavated materials from the construction of Walt Whitman bridge of the 1950s, or from another unknown source (Weston 2018; Tetra Tech 2020). Among the concerns was that soil on which the houses were built contained elevated levels of polychlorinated biphenyls (PCBs) and/or heavy metals (Weston 2018). As summarized in Section 5 of the main report, based on EPA's 2017-2018 sampling, PCBs and heavy metals were detected at low concentrations overall. Although metals and PCBs can remain in the environment for extended periods, EPA's 2017-2018 sampling results may not necessarily represent past concentrations. As a result, we cannot fully address this community concern.

Based on EPA's 2017-2018 sampling data, of the 4 sampling areas (residential soil, non-residential soil, sediment, surface water), creek sediment contained the highest levels of metals. Metal exposure estimates were unlikely to pose non-cancer health effects but for intermediate-duration (rare) and/or single pica scenarios for iron, copper, and lead.

Table H.3.1 below shows metal concentrations found in Norwood residential and non-residential soil (the arithmetic mean and ranges) compared to background U.S. levels:

**Table H.3.1. Metal concentrations in Norwood residential and non-residential soil compared to background U.S. levels[1]**

| Metal Chemical of Concern (CoC) | Metric | Residential Soil (mg/kg) | Non-residential Soil (mg/kg) | Background levels in soils (mg/kg) |
|---|---|---|---|---|
| Arsenic | Range | 4.2 – 9.7 | 2.6 – 7.8 | <0.6 – 830 |
| | Mean | 5.6 | 4.9 | 6.4 |
| Copper | Range | 10.2 – 264 | 1.9 – 64.2 | <0.5 - 996 |
| | Mean | 34.3 | 32.2 | 17.9 |
| Manganese | Range | 105 – 553 | 165 – 710 | <5 – 7,780 |

| | | | | |
|---|---|---|---|---|
| | Mean | 318.1 | 311.2 | 612 |
| Iron | Range | 12,300 – 25,300 | 11,800 – 35,100 | <100 – 133,000 |
| | Mean | 16,452.38 | 19,864.7 | 21,400[2] |
| Chromium | Range | 14.1 – 26.1 | 12.4 – 31.0 | <1 – 4,120 |
| | Mean | 19.5 | 20.1 | 36.0 |
| Mercury | Range | 0.032 – 0.88 | 0.042 – 0.64 | <0.01 – 56.4 |
| | Mean | 0.14 | 0.2 | 0.05 |
| Lead | Range | 30.6 – 1,800 | 20.7 – 358.0 | <0.5 – 12,400 |
| | Mean | 168.3 | 147.3 | 25.8 |
| Aluminum | Range | 8,990 – 13,600 | 5,700 – 16,800 | 200 – 153,000[2] |
| | Mean | 11,861.4 | 8,687.6 | 45,900 |
| Antimony | Range | 4.2 J (1 detected) | 0.52 – 1.8 J | <0.05 – 482 |
| | Mean | 4.2 | 0.9 | 0.83 |
| Cadmium | Range | 0.1 – 3.4 | 0.59 – 2.3 | <0.1 – 76.8 |
| | Mean | 0.57 | 1.3 | 0.3 |

[1]Based on US Geological Survey Data (published in 2013) of 4,841 soil samples (0-5 cm in depth): https://pubs.usgs.gov/ds/801/pdf/ds801.pdf. Levels are derived from Table 2 of the report.
[2]Extrapolated from listed weight percent concentrations from USGS's report, Table 2, under the assumption that 1% weight = 10,000 mg/kg.

As shown in Table H.3.1, Norwood metal concentrations were within U.S. ranges, however mean **lead** was higher than background U.S. values. Geometric mean and median lead in Norwood residential soil (78.9 and 54.7 mg/kg, respectively) and non-residential soil (102.6 mg/kg and 145 mg/kg) were lower than the arithmetic mean values listed in Table H.3.1 but remained higher than mean U.S. levels (25.8 mg/kg). Mean and median lead at the 2017-2018 Norwood sampling locations were also above Pennsylvania values, of 60.2 and 46.4 mg/kg, respectively (EPA, n.d.), though they were below levels typically found in more urban areas, such as Philadelphia. Copper, mercury and cadmium had slightly higher averages than background U.S. values, though their concentrations at Norwood from 2017-2018 did not exceed non-pica comparison values. Antimony in residential soil was a fair bit higher than the U.S. average, but it was only detected once (4.2 mg/kg) and its concentration was an estimated, "J" value; antimony concentrations in non-residential soil were similar to U.S. averages. (Note: the U.S. Geological Survey Data values listed in Table H.3.1 represent shallower depths, at 0-5 centimeters, than sampled depths Norwood, in which residential soil was sampled at 0-12 inches, and non-residential soil was sampled at 0-6 inches.)

Although lead was higher at Norwood than U.S. averages, 3 of 21 residential samples exceeded a 245 mg/kg threshold that, according to use of EPA Integrated Exposure Update Biokinetic (IEUBK) modeling, could lead to childhood blood levels at or exceeding 3.5 µg/dL. (This is discussed further in section 5 and Appendix D12.) Of note, 17 of 21 residential samples for lead were estimated ("J") values, indicating that the true lead concentrations could be higher or lower than each listing.

Lead is naturally present in the earth's crust at 15-20 mg/kg (ATSDR 2020). Its concentration in soil varies widely due to deposition and accumulation of atmospheric particulates from manmade sources. Soil lead concentrations beside or near roadways are often much higher than natural levels, as are lead concentrations near homes in more urban settings (Penn State University 2010). The use of lead in paints prior to its 1978 ban, particularly in older structures, is also a source of lead in soil and within homes. The age of a house is often used as a surrogate for the amount of lead in paints (Mielke et al. 2008; ATSDR 2020).

It is possible that the source(s) of higher lead levels at Norwood compared to U.S. soils is related to older housing of the Winona community, which was originally constructed during the 1950s, or proximity to nearby roadways, including from I-95 across from Darby Creek or Lincoln Avenue to its west. Based on the percentage of pre-1960s housing, lead paint indicators within a mile radius of the site were in the 71$^{st}$ percentile compared to the rest of the state, 82$^{nd}$ percentile compared to EPA Region 3, and 87$^{th}$ percentile compared to the rest of the USA (Appendix A). Of note, the lead paint indicator does not account for home renovations or other efforts undertaken to remediate lead paint. The recommendations section of the main report has information on ways to reduce exposures to lead.

**H4. Chemical Exposure from Gardening**

Some community members expressed concern of consuming homegrown crops in contaminated soil. Crop samples were not collected but exposure to soil chemicals from vegetable gardens is negligible (EPA 2021a). To reduce potential exposure, EPA recommends several practices for urban-area gardening including a) using raised beds and pots filled with clean soil, b) mixing additional compost into existing in-ground gardens, c) washing all produce and removing outer leaves of vegetables before eating, and d) cleaning tools gloves and shoes before bringing them indoors, or else leave them outside (EPA 2021a).

Our Norwood health effects evaluation encompassed a gardening scenario for adults. This assumption did not consider consumption of homegrown crops but did consider default incidental soil intake rates (100 mg/day), soil's adherence to skin, and default adult skin surface areas; in essence, assumptions for tending to one's garden. No CoCs were expected to cause non-cancer health effects for adults at Norwood, including gardeners, and the highest residential excess cancer risk estimate for adults was 2 in 100,000, from soil exposure to the maximum benzo[a]pyrene-equivalent PAH sample, or to chromium(VI) under the unlikely scenario that detected total chromium in residential soil was 100% chromium(VI).

**H5. Proximity to Lower Darby Creek Superfund Site and other Locations**

***Lower Darby Creek Area.*** Residents also expressed concern of lower Norwood's Darby Creek being downstream from the Lower Darby Creek Area (LDCA) Superfund site in which two nearby landfills, Clearview and Folcroft, have undergone remediation (Kummer 2019). The Clearview and Folcroft landfills operated from the 1950s to the 1970s and closed during the 1970s. EPA placed the LDCA site on the National Priorities List in 2001 and began cleanup at the

Clearview Landfill in 2017. Investigations at Folcroft Landfill are ongoing. Federal, state and potentially-responsible parties are responsible for the cleanup (EPA 2021b).

Darby Creek in lower Norwood is indeed downstream from the Lower Darby Creek Superfund site. Only inorganic metals were sampled in Darby and Muckinipattis Creeks, and their concentrations from 2017-2018 EPA sampling were low overall. As shown in Appendix Table C4, only arsenic exceeded comparison values for water. In our exposure estimates for creek surface water we considered a seasonal swimming (12 weeks per year) and year-round wading scenario. The swimming scenario accounts for incidental ingestion and dermal contact of surface water. The wading scenario accounts for dermal contact of surface water. No non-cancer health guidelines were exceeded for these exposure scenarios to inorganic metals.

Muckinipattis Creek flows into Darby Creek and has a public fishing dock near the convergence of the two creeks (Figure 2 of the main report). Most recreationally caught fish in Pennsylvania are safe to eat, and fish are nutritious and provide substantial human health benefits. Certain chemicals, however, such as polychlorinated biphenyls (PCBs) and mercury, build up in the human body and also in fish. Larger and older fish in particular, such as channel catfish, carp and eels tend to collect PCBs and other organic chemicals that can affect human health. Levels of these unavoidable chemicals are generally low but could be of concern to pregnant and breast-feeding women, women of childbearing age, children, and individuals with a high fish diet (Pennsylvania Fish and Boat Commission 2022).

The Pennsylvania Fish and Boating Commission's 2022 Public Health Advisory for Fish Consumption recommends limiting sport caught fish in the commonwealth's waterways to a maximum of 1 meal (1/2 pound) per week. At time of this report's release, for the basin of Darby Creek, a Pennsylvania Fish Consumption advisory is in effect to limit sport caught channel catfish to 1 meal a month, due to PCBs (Pennsylvania Fish and Boat Commission 2022). Darby Creek is part of the Delaware River watershed and both Darby and Muckinipattis Creeks are tidally influenced. For the tidal portions of all Pennsylvania water bodies that feed the Delaware River Estuary, a 1 meal per month consumption advisory is also in effect for several other species of fish, including white perch, channel catfish, flathead catfish, and striped bass (Pennsylvania Fish and Boat Commission 2022). Fish consumption advisory updates can be found on the Pennsylvania Department of Environmental Protection's website.

***Other Former Industry.*** Community members also expressed concern of Norwood landfill contamination from the former Glenolden laboratories and Muckinipattis Wastewater Treatment Plant (WWTP), the latter of which operated from some time prior to 1957 until it was demolished in the early 1980s. EPA considers the former Glenolden laboratory property located on South Avenue as a separate site. The site's previous owners conducted a voluntary cleanup pursuant to Pennsylvania Department of Environmental Protection (PADEP's) Cleanup Program (EPA 2021c). EPA's expanded 2020 sampling included locations along the

Muckinipattis Creek area of the former WWTP. EPA released its expanded sampling results in December 2021.

**H6. Contamination at Norwood Elementary School.** Residents, teachers and staff expressed concern of contamination leading to adverse health outcomes at Norwood Elementary School, which borders the lower Norwood community to the north. Currently, there is no sampling data for the school for PADOH to assess. Based on historical information, there is currently no indication that the school site is contaminated from historical landfill activities (EPA 2021d).

**H7. Timeline of Agency Activities.** A timeline of agency activities for the Norwood Landfill (EPA, ATSDR, PADOH), including site visits, is below:

- **February 2017:** The Environmental Protection Agency (EPA) initiated a site assessment through the Superfund Program to determine if there was a threat posed to human health and the environment by actual or potential releases of hazardous substances and if there was need for additional action.

- **September 2017**: EPA collected surface and subsurface soil samples within the landfill area and adjacent to residential properties, as well as surface water and sediment samples in the Muckinipattis and Darby Creeks. Samples were analyzed for volatile organic compounds, semi-volatile organic compounds, polycyclic aromatic hydrocarbons, pesticides, polychlorinated biphenyls, and metals.

- **May 2018:** EPA performed additional surface soil sampling at 21 residential properties adjacent to the landfill. ATSDR was present and screened the yards with an X-Ray Fluorescence (XRF) instrument for soil lead concentrations.

- **July 2019:** EPA met with representatives from the Norwood Borough and interested stakeholders to present the findings of the site assessment and discuss plans for a larger public meeting. Following the meeting, new information was presented to EPA about the existence of the Old Norwood Dump and a 10-acre area along Muckinipattis Creek, at the eastern end of Norwood Borough Park, which may also have been used for waste disposal.

- **November 2019**: ATSDR and PADOH held a meeting at the Norwood Elementary School for teachers and staff with health concerns regarding environmental exposures at the school.

  EPA held a public meeting at the Norwood Fire Company to present the current findings of the site assessment and discuss additional plans for sampling. ATSDR also presented.

EPA discussed its plan to sample the Old Norwood Dump and additional residential properties in the Lower Norwood area, to gain a broader representative sample of residential soil in the community.

- **January 2020:** EPA conducted a site scoping visit and walked areas of the Old Norwood Dump, the former Muckinipattis Wastewater Treatment Plant (now DELCORA pumping station), and the Norwood Landfill. This site visit was used to determine future sampling locations and identify any accessibility issues.

- **Fall 2020:** EPA conducted additional environmental sampling (known as a Site Investigation) in the lower Norwood area. The goals were to determine 1) if any chemicals from the landfill site posed a risk to the human health and the environment in the Norwood community, and 2) if any longer-term investigation or immediate action is needed to address chemicals. The sampling was performed in two phases – non-residential sampling (completed in September/October 2020) and residential sampling (completed November 2020). EPA expanded sampling areas for both sampling phases and included the site of the Old Norwood Dump, Norwood Lower Park, Muckinipattis Creek, and 70 total homes in the Winona neighborhood. Non-residential sampling including groundwater, surface water, wetland and sediment, and residential and non-residential soil sampling included subsurface soil (up to 4 feet below surface) and deep soil (up to 15 feet below surface).

- **December 2021.** EPA held a virtual public meeting to inform residents that based on their 2017-2020 sampling results, that the community is not exposed to contaminants at levels of concern. Thus the Norwood site did not warrant inclusion on the National Priorities List. EPA discussed plans to further evaluate wetland areas in Darby and Muckinipattis Creeks to determine whether chemicals from the Old Norwood Dump or the former Norwood landfill were contributing to wetland contamination. ATSDR and PADOH attended to provide an update on the status of the Health Consultation document. EPA released its expanded sampling (2020) results to its website on December 9, 2021.

Information on EPA site activities for the former landfill can be found here: https://www.epa.gov/norwood.

A frequently asked questions page for the site can be found here: https://www.epa.gov/norwood/norwood-pa-frequently-asked-questions

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