# Public Health Assessment

Public Health Implications from Exposures to Contaminants Associated with

Fair Lawn Well Field Superfund Site

Fair Lawn, Bergen County, New Jersey

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**Public Comment Release**

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Prepared by: New Jersey Department of Health

Under a Cooperative Agreement with the
U.S. Department of Health and Human Services
Agency for Toxic Substances and Disease Registry (ATSDR)
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# List of Acronyms

ATSDR = Agency for Toxic Substances and Disease Registry

CDC = Centers for Disease Control and Prevention

COC = Contaminant of concern

CREG = Cancer Risk Evaluation Guide

CTE = Central tendency exposure

CV = Comparison Value

EMEG = Environmental Media Evaluation Guide

EPA = United States Environmental Protection Agency

EPC = Exposure Point Concentration

GAC = Granular Activated Carbon

HEC = Human equivalent concentration

HQ = Hazard quotient

LECR = Lifetime excess cancer risk

LOAEL = Lowest observed adverse effect level

MCL = Maximum Contaminant Level

MRL = Minimal Risk Level

NJDEP = New Jersey Department of Environmental Protection

NJDOH = New Jersey Department of Health

NJPDES = New Jersey Pollutant Discharge Elimination System

NOAEL = No observed adverse effect level

NPL = National Priorities List

NSF = National Sanitation Foundation

PBPK = physiologically based pharmacokinetic modeling

PCE = Tetrachloroethylene

PFAS = per- and polyfluoroalkyl substances

PFBS = Perfluorobutane sulfonic acid

PFHxS = perfluorohexane sulfonic acid

PFHpA = perfluoroheptanoic acid

PFNA = perfluorononanoic acid

PFOA = perfluorooctanoic acid

PFOS = perfluorooctane sulfonate

PHAST = Public health assessment site tool

PRP = Potentially Responsible Party

RI/FS = Remedial Investigation/Feasibility Study

RfD = Reference Dose

RfC = Reference Concentration

RME = Reasonable maximum exposure

RMEG = Reference Media Evaluation Guide

RSL = EPA Regional Screening Level

SSDS = Sub-slab depressurization system

TCE = Trichloroethylene

UCL = Upper Confidence Limit

UCMR = Unregulated contaminant monitoring rule

VISL = Vapor Intrusion Screening Level

VOC = Volatile Organic Compound

WMWF = Westmoreland Well Field

# Summary

**Introduction** In summer 2023, the New Jersey Department of Health (NJDOH) became aware of health concerns and possible environmental exposures from the Fair Lawn Well Field Superfund site. The United States Environmental Protection Agency (EPA) placed the site on the National Priorities List (NPL) in September 1983. The NPL is the list of sites throughout the United States and its territories with known releases or threatened releases of hazardous substances, pollutants, or contaminants. The NPL is intended to guide EPA in determining which sites warrant further investigation. Sites listed on the NPL are also known as Superfund sites.

The Fair Lawn Well Field Superfund Site includes the groundwater that impacts four municipal wells located on or around Westmoreland Avenue in the Borough of Fair Lawn, Bergen County, NJ. These wells are part of the Westmoreland Well Field (WMWF).

In 1978, volatile organic compounds (VOCs), including tetrachloroethylene (PCE) and trichloroethylene (TCE), were detected in the WMWF wells. To determine the origin of the contamination, the New Jersey Department of Environmental Protection (NJDEP) investigated all industrial and commercial facilities within a 3,000-foot radius of the contaminated municipal wells. The investigation concluded that the primary source of the contamination is within the Fair Lawn Industrial Park. In 1986, the borough of Fair Lawn installed a treatment system on the WMWF wells to remove the VOCs from the community water supply.

EPA took over the lead for the groundwater cleanup in 1992, while the NJDEP continued addressing the source area properties within the Fair Lawn Industrial Park. EPA and the United States Geological Survey conducted an area-wide groundwater study between 1999 and 2005 to characterize the nature and extent of the groundwater contamination.

Several Potentially Responsible Parties (PRPs) conducted a Remedial Investigation and Feasibility Study (RI/FS) under EPA's oversight to evaluate the extent of contamination and potential environmental and public health threats. This work was conducted between September 2009 and June 2016. The WMWF was disconnected from the public water supply in May 2016 due to the presence of 1,4 dioxane at levels above the NJDEP groundwater standard. The RI/FS was completed in July 2018.

EPA selected a final cleanup plan in September 2018. The plan involves pumping and treating groundwater outside the source area properties and included the WMWF, Little Diamond (formally Henderson) Brook, and the surrounding residential areas. It also included construction of a new water treatment system to replace the existing WMWF water treatment plant that

treats VOCs, 1,4-dioxane, and per- and polyfluoroalkyl substances (PFAS), which were found in the WMWF wells. The construction was completed in spring 2025. This new system is monitored to ensure the treated water meets federal and state drinking water standards. Once the new water treatment system is operating effectively, the Borough of Fair Lawn expects to take over its operation and maintenance by the end of 2026. If the borough decides not to do that, the PRPs will continue to operate and maintain the system until the remedial action objectives are achieved under the 2018 cleanup plan.

On August 30, 2023, NJDOH held two public availability sessions at the Fair Lawn Borough Hall. NJDOH met with community members and gathered information on specific exposure concerns from the site.

To address the community concerns expressed during the availability sessions, NJDOH prepared this public health assessment under its cooperative agreement with the federal Agency for Toxic Substances and Disease Registry (ATSDR). It evaluates the past, present and future potential public health implications for residents and workers from exposures to contaminants in drinking water, surface water, and indoor air from the Fair Lawn Well Field Superfund Site.

It should be noted that currently, the residents of Fair Lawn are not drinking the water from the WMWF. Groundwater from the WMWF is treated with the existing system using granulated activated carbon (GAC) and discharged to Little Diamond Brook under a New Jersey Pollutant Discharge Elimination System (NJPDES) Discharge to Surface Water Permit.

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| **Conclusions** | Based on the available data (2006-2024) and information provided by EPA, NJDOH and ATSDR reached the following conclusions for the Fair Lawn Well Field site: |
| **Conclusion 1** | Past, current and future exposures to PFAS (PFOA and PFOS) in drinking water delivered by the Fair Lawn blended municipal water system (combination of well fields in Fair Lawn and purchased water with two water utilities) may harm people's health. |

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| **Basis for Conclusion** | Based on the detected PFOA water concentration in the Fair Lawn municipal water system, doses for all age groups with average and above-average tap water ingestion rates have exposures that exceed doses reported to be associated with reduced antibody response and elevated cholesterol levels in the blood of humans. Another potential health effect is decreased birth weight from *in utero* exposures in the first and second trimesters. Mild liver damage might also be possible in residents with above-average tap water ingestion rates.<br><br>Based on the detected PFOS water concentration in the Fair Lawn municipal water system, doses in children birth to < 2 years with above-average tap water ingestion rates have exposures that approach doses associated with reduced antibody response, increased cholesterol levels, and mild liver damage. Doses in older children, teenagers, and adults are below levels thought to cause harmful effects.<br><br>The reported health effects are very similar for the two PFAS (PFOA and PFOS) that target the cardiovascular, immune, developmental, and hepatic systems. PFOA and PFOS were present in drinking water in the Fair Lawn municipal water system. All age groups with average and above-average tap water ingestion rates exceed doses thought to cause elevated cholesterol levels, reduced antibody response, decreased infant birth weight, and elevated liver enzymes in adults.<br><br>The lifetime increased cancer risk for children and adults with above-average tap water ingestion rates represent an increased theoretical cancer risk from exposures to PFOA in drinking water from the Fair Lawn municipal water system. For drinking water exposures to PFOS, the lifetime increased cancer risk for children and adults represent no concern for an increased theoretical cancer risk. |
| **Conclusion 2** | Exposure to 1,4-dioxane through ingestion of or showering and bathing in water delivered by the Fair Lawn municipal water system is not likely to harm people's health. |
| **Basis for Conclusion** | Calculated exposure doses and air vapor concentrations for 1,4-dioxane were below the ATSDR health guideline for noncancer health effects. Additionally, there is no concern for an increased theoretical cancer risk. |
| **Conclusion 3** | Past exposures to TCE in the Columbia Savings Bank may harm people's health. |
| **Basis for Conclusion** | Based on the March 2011 air sampling results, an increased risk for kidney effects from chronic exposures to TCE might exist for longtime workers at the Columbia Savings Bank. Estimated exposures exceed health guidelines and approach levels that might harm the kidneys of longtime workers who worked at the bank before 2011. Some uncertainty exists in this conclusion because we don't know TCE concentrations in air prior to March 2011. Because of uncertainty in the science, we don't know if TCE exposure in |

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| | pregnant women before 2011 might result in fetal heart malformation in newborns [ATSDR 2025].<br><br>Based on a January 2012 air sampling event at the bank, health effects would not be expected in workers or visitors. TCE levels decreased after the March 2011 sampling event and estimated exposures are below health guidelines, so there is no concern for noncancer effects after the most recent sampling event in January 2012.<br><br>Based on NJDOH's discussions with EPA, EPA's evaluation of soil gas data showed that elevated TCE levels in the bank were caused by background sources within the building, not vapor intrusion from groundwater beneath the bank. |
| **Conclusion 4** | Except for Columbia Savings Bank, exposures to indoor air contaminants in the remaining commercial and residential properties are not likely to harm people's health. |
| **Basis for Conclusion** | Harmful noncancer health effects are not expected from exposure to indoor air contaminants in the eight residential properties where indoor air data were collected. Harmful noncancer health effects are not expected for workers in the Sandvik, Hampshire, Kushner, Mikia, or Biomet buildings. The levels of detected contaminants were below health guideline values or below levels where harmful health effects were observed in toxicological studies. Indoor air contaminants in the eight homes were from background sources within the homes and not vapor intrusion from groundwater beneath the structures. There is also no concern for an increased theoretical cancer risk from exposures to indoor air contaminants for occupants in the residential and commercial properties. |
| **Conclusion 5** | Harmful health effects are not expected for children and adults wading in Little Diamond Brook. |
| **Basis for Conclusion** | Calculated exposure doses for dermal (skin) exposures were below health guideline values for noncancer health effects. Additionally, there is no concern for an increased theoretical cancer risk. |
| **Next Steps** | **PFAS in Drinking Water**<br><br>It is important to note that the residents of Fair Lawn have not been drinking water from the WMWF since 2016. Groundwater from the WMWF is treated using the existing system with granulated activated carbon (GAC) and discharged into Little Diamond Brook under a New Jersey Pollutant Discharge Elimination System (NJPDES) Discharge to Surface Water Permit.<br><br>• It is recommended that community members refer to NJDOH's **PFAS fact sheet** which provides up-to-date information on potential health effects. This fact sheet includes resources for health care providers and other information for communities with concerns about PFAS in |

drinking water. As PFAS science evolves, the fact sheet is updated to include new information on regulations and responses to frequently asked questions (nj.gov/health/ceohs/documents/pfas_drinking%20water.pdf).

- ATSDR funded a **multisite study** to learn more about the relationship between PFAS exposure and health outcomes across seven U.S. communities exposed to PFAS-contaminated drinking water. Work is ongoing, and results are pending (atsdr.cdc.gov/pfas/activities/studies/multi-site.html).
- Specific health concerns regarding PFAS exposures may be discussed with a health care provider. ATSDR has developed **PFAS information for clinicians** that can be shared with health care providers to determine the best path forward based on an individual's unique circumstances (atsdr.cdc.gov/pfas/resources/pfas-information-for-clinicians.html).
- Residents may consider using bottled water for drinking and cooking to reduce exposures to PFAS. Bottled water sold in New Jersey is required to meet the New Jersey Maximum Contaminant Levels (MCLs.)
- Residents may consider using home water treatment devices that can reduce levels of PFAS, but they do not ensure PFAS concentrations will be lower than the New Jersey MCLs. NSF International, an independent and accredited organization, certifies products proven effective for reducing PFOA and PFOS below 20 parts-per-trillion (ppt). PPT also equals nanograms per liter (ng/L). When purchasing a filter:

    - Visit the National Sanitation Foundation (NSF) International website, (nsf.org/consumer-resources/drinking-water/water-treatment-systems) to identify a product that is NSF/ANSI Standard 53 certified and check the standard version to know if the filter is certified to remove total PFAS (PFOA, PFOS, PFNA, PFHxS, and PFHpA).

**Vapor Intrusion (Indoor Air Impacts)**

NJDOH and ATSDR recommend that NJDEP and EPA continue to ensure vapor mitigation systems at Sandvik, Hampshire, and Mikia operate effectively to prevent sub-surface vapors from entering the buildings. Based on the information provided to NJDOH, vapor intrusion was not occurring in the remaining commercial and residential properties evaluated in this document. Therefore, no further actions are recommended for these properties.

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| **For More Information** | **Public Comment Period:** This document will have a 30-day public comment period which provides an opportunity for the public to comment on the findings contained in this document.<br><br>Questions and comments about this public health assessment should be directed to the NJDOH at (609) 826-4984 or by email at nj.gov/health/workplacehealthandsafety/occupational-health-surveillance/contact.shtml |

# Background and Purpose

In summer 2023, the New Jersey Department of Health (NJDOH) became aware of health concerns due to possible environmental exposures from various contaminated properties including the Fair Lawn Well Field Superfund Site and demolition work at the nearby former Nabisco facility in the Borough of Fair Lawn, Bergen County New Jersey. To address these concerns, NJDOH held two public availability sessions at the Fair Lawn Borough Hall on August 30, 2023, to meet individually with community members and gather more information on specific exposure concerns from the site.

This public health assessment was prepared under NJDOH's cooperative agreement with the federal Agency for Toxic Substances and Disease Registry (ATSDR). The assessment evaluates public health implications from exposures to contaminants from the Fair Lawn Well Field Superfund Site (Site). Groundwater from the Westmoreland Well Field (WMWF) is currently treated and discharged to surface water. The U.S. Environmental Protection Agency (EPA) has overseen the design and construction of a new treatment system that addresses the presence of volatile organic compounds (VOCs), 1,4-dioxane, and per- and polyfluoroalkyl substances (PFAS) in the well field groundwater. This treatment system is expected to become operational by the end of 2026. A separate report that addresses potential exposures to other environmental contaminants that are not part of the Fair Lawn Well Field Superfund Site is available at https://www.nj.gov/health/ceohs/documents/eohap/haz_sites/bergen/fairlawn/FairLawnNCFinalJuly2025.pdf

# Site Description and Timeline

## Drinking Water and Groundwater

The Site includes the groundwater that impacts four municipal wells located on or around Westmoreland Avenue in the Borough of Fair Lawn, Bergen County, New Jersey. These wells are part of the WMWF. **Figure 1 in Appendix A** shows the location of the site and surrounding towns.

The WMWF was developed in 1948, beginning with the installation of well FL-10. Between 1948 and 1950, wells FL-11, FL-12, and FL-14 were installed in this well field, and wells FL-11 and FL-14 were brought on-line. Since well FL-12 produced little groundwater, it was used only as an observation well. Between 1952 and 1969, the borough installed two non-potable industrial wells. FL-23 is located across Pollitt Drive to the east of the former Eastman Kodak Company (Kodak) property. FL-24 is located midway along the northeastern boundary of the property. The location of the well field and the Fair Lawn Industrial Park source area are shown in **Figure 2, Appendix A**.

In 1978, VOCs, including tetrachloroethylene (PCE) and trichloroethylene (TCE), were detected in the WMWF wells. The New Jersey Department of Environmental Protection (NJDEP) investigated all industrial and commercial facilities within a 3,000-foot radius of the contaminated municipal wells to determine the origin of the contamination. NJDEP concluded that the primary source of the contamination was within the Fair Lawn Industrial Park.

In 1979, after discovering VOCs, including PCE, TCE and other compounds, in wells FL-23 and FL-24, the borough took them offline and collected groundwater samples. The site was placed on the Superfund National Priorities List in September 1983.

An air stripper system was installed in 1986 to treat the municipal potable production wells FL-10, FL-11, and FL-14. Between 1988 and 1990, FL-24 was sealed and FL-23 was capped. FL-23 is still used for observation. In 1996, FL-11 was taken out of service and is currently used as an observation well.

EPA took over the groundwater cleanup in 1992, while NJDEP continued addressing the source area properties within the Fair Lawn Industrial Park. Between 1999 and 2005, EPA and the United States Geological Survey conducted an area-wide groundwater study to characterize the nature and extent of the groundwater contamination at the WMWF.

In 2008, several Potentially Responsible Parties (PRPs) collectively entered into legal agreements with EPA to conduct a Remedial Investigation and Feasibility Study (RI/FS) at the site. The RI/FS would evaluate the extent of contamination and potential environmental and public health threats. These PRPs included facilities owned by Thermo Fisher Scientific International Inc. (Fisher), Sandvik Inc. (Sandvik), and Kodak. An additional property, Hampshire, was identified as a source of hazardous substances [Langan 2018a]. These four PRPs conducted investigations and remedial actions under the NJDEP's site remediation program.

Groundwater investigations, including surface water sampling and the installation and sampling of numerous monitoring wells, were conducted between September 2009 and June 2016. Vapor intrusion investigations were also conducted during this time to determine the potential indoor air impacts from contaminated groundwater.

**Figure 2 in Appendix A** shows the location of the WMWF wells and the Fair Lawn Industrial Park source area. The site includes the groundwater that impacts four municipal wells located on or around Westmoreland Avenue, which are a part of the WMWF. **Figure 3 in Appendix A** shows the extent of the groundwater plume.

The site encompasses the groundwater below the source area properties in the Fair Lawn Industrial Park, the area northeast of the Fair Lawn Industrial Park, and the WMWF to the southwest, as well as the surface water impacted by the groundwater contamination. Little Diamond Brook (the former North Branch of Henderson Brook) flows west along the southern property line of several source area facilities and southwest along the south side of Route 208 near the WMWF until it reaches the Passaic River. FL-10 and FL-14 have been taken out of service, and the treated water (using Liquid Granular Activated Carbon filtration units in addition to the existing air stripper) is discharged to Little Diamond Brook.

Potable water for residences and commercial properties within the site boundary is served by the Fair Lawn Borough Water Department. The water department obtains potable water from several well fields it operates. It augments the supply with purchased or treated water from two other water utilities. The well fields and public water systems supplying water to Fair Lawn are

- WMWF
- Dorothy Street Treatment Plant
- Well 28 Treatment Plant
- Cadmus Place Treatment Plant

The WMWF was disconnected from the public water supply in May 2016 due to the presence of 1,4-dioxane above the NJDEP groundwater quality standard. The Dorothy Street and Well 28 treatment plants were disconnected in November 2017 due to the presence of PFAS. Based on an EPA review of well records in the area, private wells are not utilized for drinking water.

Potable water is currently provided by the Cadmus Place well field, which has a treatment system to remove PFAS. The Fair Lawn Water Department also purchases water from two water utilities (Passaic Valley Water Commission and Veolia Water New Jersey Hackensack) to supplement water in place of the WMWF treatment plant. The water delivered to consumers is blended from these well fields that feed into the Cadmus treatment plant and from water purchased from the two water utilities.

In September 2018, EPA selected a final cleanup plan. It called for pumping and treating groundwater outside the source area properties, including the WMWF, Little Diamond Brook, and the surrounding residential areas. The plan also included an upgrade of the WMWF treatment plant, which was completed in the spring of 2025.

**Vapor Intrusion: Residential and Commercial Properties**

Vapor intrusion is the migration of vapors from any subsurface contamination source, such as soil or groundwater, into an overlying building. Vapors released from contaminated groundwater or soil can seep through cracks in basements, foundations, sewer lines, and other openings into overlying structures or buildings.

Vapor intrusion is a concern at this site because of the presence of volatile organic compounds in groundwater near occupied buildings. PCE, TCE, vinyl chloride, carbon tetrachloride, and chloroform are the primary contaminants presenting a vapor intrusion concern [Langan 2018a]. Additional contaminants identified as a potential vapor intrusion concern in groundwater included 1,1-dichloroethylene, 1,4-dichlorobenzene, benzene, chlorobenzene, ethylbenzene, hexachlorobutadiene, hexachloroethane, xylenes, cyanide, and mercury.

In accordance with the January 2009 EPA RI/FS, the PRPs conducted vapor intrusion investigations at 17 residential properties and six commercial properties for soil gas and indoor air sampling. Access was requested for each property, and sampling was completed at 10 residential properties and four commercial properties. Access was denied at the remaining seven residential properties and two commercial properties.

Since that time, EPA or individual property owners have investigated additional properties as requested through informal verbal and electronic communications. The PRPs, under

NJDEP and/or EPA oversight, have contacted the owners of 99 properties regarding access for sampling of soil gas and, in select cases, indoor air.

The PRPs and other parties have also completed vapor intrusion investigations at or near four additional commercial and three additional residential properties, under their respective NJDEP-led cases.

Between 2006 and 2015, soil gas and/or indoor air sampling activities were completed for 51 residential properties, nine commercial properties, one childcare center, and one school (Westmoreland Elementary). The EPA determined no further vapor intrusion investigations are warranted at this time for properties sampled as part of the Fair Lawn Well Field Superfund Site [Langan 2018a].

**Vapor Intrusion Screening Levels**

The NJDOH supports the use of EPA and NJDEP regulations regarding vapor intrusion investigations at the Fair Lawn Well Field site. The regulations that determine the need for soil gas and indoor air sampling and follow-up remedial actions are intended to protect public health. NJDOH understands that vapor intrusion screening levels and standards for some contaminants may have changed since the time of the vapor intrusion investigations. NJDOH supports EPA and NJDEP's decisions about further sampling and remediation if groundwater conditions change at the site. The EPA and PRPs under NJDEP oversight used the most health-protective vapor intrusion screening levels available at the time, which are described below:

**EPA Screening Levels**

Groundwater Screening:

- EPA's June 2017 default Residential Groundwater Vapor Intrusion Screening Levels (VISLs) were used to evaluate contaminants for the potential for vapor intrusion.

Sub-Slab and Indoor Air Screening:

- EPA published default Sub-Slab and Indoor Air VISLs for both residential and commercial buildings.
- The June 2017 Residential VISLs were used to screen soil gas and indoor air data for residential buildings and the Westmoreland Elementary School.
- The June 2017 Commercial VISLs were used to screen soil gas and indoor air data for commercial buildings under EPA oversight.

**NJDEP Guidance and Screening Levels**

Vapor Intrusion Investigation:

- NJDEP's August 2016 Vapor Intrusion Technical Guidance was used for the vapor intrusion investigations at the site.

Groundwater Screening:

- NJDEP's March 2013 generic groundwater screening levels for vapor intrusion were used as VISLs for groundwater if they were more stringent than EPA's June 2017 residential groundwater VISLs.

Sub-Slab and Indoor Air Screening:

- NJDEP published sub-slab and indoor air screening levels for both residential and commercial buildings.
- NJDEP's March 2013 residential soil gas and indoor air screening levels were used as VISLs for residential soil gas and indoor air if they were more stringent than EPA's residential soil gas and indoor air VISLs.
- NJDEP's VISLs were used to evaluate available soil gas and indoor air data collected for properties under NJDEP's oversight.

ATSDR's comparison values were also used to screen indoor air contaminants to determine which would be selected for further evaluation.

**Vapor Intrusion Sampling Activities**

In March and April 2009, the PRPs collected two rounds of indoor air and sub-slab soil gas samples. The first round of sampling in March 2009 included sub-slab samples from 10 residential properties and four commercial buildings near Route 208. Based on the first round of results, the PRPs collected a second round of sub-slab and indoor air samples in April 2009 from those same properties

In August 2013, EPA collected sub-slab vapor samples from the Westmoreland Elementary School. Between September and December 2013, EPA collected sub-slab samples from 12 additional residential properties.

Since that time, at the request of EPA, the PRPs sampled two residential properties between March and April 2014 and one residential property between November and December 2015. The PRPs and other parties performed additional vapor intrusion investigations at nine commercial and three residential properties. Several of the commercial buildings required the installation of vapor mitigation systems under NJDEP's authority.

Of the properties described above, indoor air samples were collected at eight residential properties and six commercial properties. The residential properties are identified as R11, R18, R19, R31, R32, R33, R45, and R50. The six commercial properties are Sandvik, Hampshire, Kushner, Brizzolara/Brioschi/Mikia LLC (Mikia), Columbia Savings Bank, and Zimmer Biomet Inc. (Biomet).

Overall, the sample results from the EPA-led investigations showed that the residential properties are not at risk for vapor intrusion, so no additional sampling was planned. However, if the site conditions change, EPA will determine if additional vapor intrusion sampling is

necessary. Vapor intrusion was occurring in the Sandvik, Hampshire, and Mika commercial businesses. Therefore, sub-slab depressurization systems (SSDS) were installed to prevent sub-surface vapors from entering these buildings.

Finally, the former Kodak property, which is currently owned by Fair Lawn Promenade, consists of five individual commercial/residential buildings. Four of the five buildings are equipped with SSDS. The fifth building has an unoccupied open-air parking lot on the ground floor. In February 2020, EPA collected sub-slab soil gas samples from locations within the five buildings to ensure the SSDS was effectively removing vapors from beneath the building's contaminated groundwater. Sampling results indicated the SSDS was working properly.

**Surface Water**

Little Diamond Brook flows east to west along the southern property boundaries of Fisher and Sandvik, then turns southwest as it continues beyond Sandvik's western property boundary (see **Figure 2, Appendix A**).

A former branch of Little Diamond Brook is mapped along the western side of McBride Avenue, west of the Sandvik property (see **Figure 2, Appendix A**). It is now a partially covered drainage ditch and no longer functions as a natural surface water feature. The NJDEP classifies the main branch of Little Diamond Brook as a Freshwater 2 – Non-Trout (FW2 NT) water body, while the Former North Branch of Little Diamond Brook is not listed as a classified surface water feature. **Figure 4 in Appendix A** shows locations of monitoring wells, WMWF wells, surface water sampling locations, and vapor intrusion sampling locations.

**Geology and Hydrogeology**

Unconsolidated surface materials (i.e. glacial and postglacial deposits) are in the vicinity of Fair Lawn. Glacial sediments are primarily glaciolacustrine, glaciofluvial, and till, while the postglacial sediments are primarily modern channel and floodplain deposits [Langan 2018b].

The site is underlain by Glacial Lake Paramus Deposits, which can be up to 70 feet thick, and Rahway Till, which can be up to 20 feet beneath the lake deposits. Localized alluvial deposits are also present along Little Diamond Brook, as identified by the New Jersey Geological Society. These deposits are typically composed of sands, cobbles and pebbles, silty sands, sandy silts, and some local occurrence of clays. The permeability of the fine sand, sandy silt, alluvial, and clay portions of these deposits is moderate to low, with hydraulic conductivities averaging 0.1 to 0.001 feet per day [Langan 2018a]. This means that groundwater flow is low, moving only 0.1 to 0.001 feet each day.

The bedrock directly beneath Fair Lawn is the Passaic Formation, which consists of layers of conglomerate, sandstone, and siltstone. The Passaic Formation is a primary source of groundwater for municipal, industrial, and other uses in Fair Lawn and surrounding communities. Groundwater well pumping rates of several hundred gallons per minute have been achieved and sustained in the Passaic Formation.

The groundwater contaminant plumes at the site have generally exhibited a diving configuration, meaning the depth of the plumes increases with distance from the sources. In areas farther from the sources, the affected groundwater, where present, is typically confined to the deeper bedrock aquifer and may have clean groundwater lenses above the impacted depths.

Water table depth varies throughout the Site from approximately five to 30 feet below ground surface (ft. bgs). The water table is deepest in the southeastern portion of the site, where water is generally found in bedrock, and shallowest in the vicinity of Little Diamond Brook [Langan 2018b].

Groundwater movement primarily occurs through secondary porosity (fractures, joints, bedding plane partings, etc.) rather than primary porosity (rock matrix). The Passaic Formation exhibits directional hydraulic properties, with wells aligned along bedding strike tend to be hydraulically connected [Langan 2018a].

**Demographics**

According to the 2020 U.S. Census, 53,538 people live within one mile of the site (a 5% increase since 2010). The area around the site is densely populated, indicating a larger population may have been within the potential exposure area of the Fair Lawn Well Field site. **Appendix C** provides more detailed information about the people in the area.

**Community Concerns**

As mentioned previously, NJDOH held two public availability sessions at the Fair Lawn Borough Hall on August 30, 2023. NJDOH representatives met with community members individually and gathered information on specific exposure concerns from the Fair Lawn Well Field site.

During these public availability sessions, residents specifically raised concerns about birth defects and cancer incidence, including pediatric cancer. They also expressed concerns about possible exposures to drinking water containing PFAS and 1,4 dioxane, as well as potential indoor air impacts from the groundwater contamination.

During our meetings with residents, NJDOH explained how we determine whether people are at risk of harmful effects by first looking at their exposure levels. We compare those exposure levels to levels from human and animal studies to decide if people's health might be harmed. NJDOH provided residents with a fact sheet on the Process to Evaluate Community Environmental Exposures. (nj.gov/health/ceohs/documents/Evaluating_Potential_Public_Health_Implications_from_Environmental_Exposures_FAQs.pdf).

### Prior NJDOH/ATSDR Involvement

NJDOH prepared several documents after the site was placed on the NPL in September 1983:

- A public health assessment (Jan 1989)
- A site review and update (December 1995)
- A public health consultation (September 1996) evaluating possible exposures to contaminants from the Fair Lawn Well Field site

These documents were released between January 1989 and September 1996 and can be found at nj.gov/health/ceohs/environmental-occupational/hazardous-waste-sites/bergen/index.shtml#4. Between the release of these documents and the availability sessions in August 2023, NJDOH was not aware of any community concerns about this site.

## Scientific Evaluation

NJDOH used ATSDR's Public Health Assessment Guidance Manual for assessing whether a community is at risk for a health hazard [ATSDR PHAGM 2022; Ulirsch et al., 2022]. The scientific evaluation has the following steps:

1. Exposure pathway evaluation
2. Screening analysis
3. Exposure Point Concentrations (EPCs) and exposure calculations
4. In-depth toxicological effects analysis – noncancer health effects
5. In-depth toxicological effects analysis – cancer health effects

### 1. Exposure Pathway Evaluation

The first step in the assessment process is to determine how people may come into contact with hazardous substances (exposure pathway) and whether there is a completed exposure pathway. An exposure pathway is the link between an environmental release, or source of contamination, and the point where a population might be exposed to the contaminant. Exposure pathways are used to evaluate specific ways in which people were, are, or will be exposed to environmental contamination in the past, present, and future (see Table 1).

A completed exposure pathway has the following five elements:

1) Source of contamination (Fair Lawn Well Field)
2) Environmental media and transport mechanisms (indoor air, drinking water, surface water)
3) Point of exposure (residential and commercial properties)
4) Route of exposure (inhalation, ingestion, and dermal)
5) Receptor population (residents and workers)

A completed exposure pathway does not necessarily mean that harmful health effects will occur. It indicates that all five elements are present, that at least some people are exposed, and that further evaluation and screening of contaminants is necessary.

Generally, ATSDR considers the following three exposure pathway categories:

1) Completed: All five elements of a pathway are present.
2) Potential: One or more of the elements is absent, but information is insufficient to eliminate or exclude the element.
3) Eliminated: One or more of the elements is absent and will never be present.

**Table 1. Exposure Pathways – Fair Lawn Well Field Superfund Site**

| Pathway | Environmental Medium | Exposure Route | Exposure Point | Exposed Population | Pathway Classification |
|---|---|---|---|---|---|
| Drinking water | Community water supply | Ingestion/dermal contact (showering) | Commercial and residential properties | Workers and residents | Past, current and future – **complete** |
| Vapor intrusion | Indoor air | Inhalation | Commercial properties | Workers | Past – **complete** Current and future – **eliminated*** |
| Vapor intrusion | Indoor air | Inhalation | Residential properties; Westmoreland Elementary School | Residents | Past, current and future – **eliminated** ** |
| Surface water | Water | Dermal contact (wading) | Little Diamond Brook | Residents | Past, current and future – **complete** |

*Pathway classification for vapor intrusion at some commercial properties is complete for past exposures, but impacted buildings have vapor mitigation systems that remove vapors. Therefore, current and future exposures are eliminated.
**Pathway classification for residential vapor intrusion is eliminated based on EPA and NJDEP vapor intrusion investigations showing no completed vapor intrusion pathway for residences or the Westmoreland Elementary School.

## Completed Exposure Pathways

- *Inhalation of indoor air contaminants from contaminated groundwater entering commercial buildings (past)*

  In the past, there was a completed exposure pathway for workers breathing contaminated air from vapor intrusion in some commercial buildings (Sandvik, Hampshire, Fair Lawn Promenade (former Kodak property), and Mikia). These buildings have vapor-mitigation systems to prevent contaminated groundwater vapor from entering. Therefore, current and future exposures to contaminated groundwater vapor have been eliminated.

The vapor intrusion pathway was determined to be incomplete for Kushner, Columbia Savings Bank, and Biomet. Indoor air contaminants detected in these spaces were caused by background sources and not vapor intrusion. According to EPA, the vapor mitigation system for the Fair Lawn Promenade (former Kodak property) is operating properly, so the vapor intrusion pathway for this property is also incomplete.

- *Ingestion of and dermal (skin) contact with contaminated drinking water (past, current, future)*

  In the past, there was a completed exposure pathway for residents from drinking water containing PFAS and 1,4-dioxane from the WMWF and other sources. Currently, the water delivered to consumers is blended from well fields that feed into the Cadmus treatment plant and from water purchased from two water utilities. The Cadmus treatment plant removes these contaminants before it is distributed to customers, but the purchased water sources show the presence PFAS and 1,4-dioxane. This represents a current and future completed exposure pathway.

- *Inhalation of contaminated water vapors in air while showering or bathing*

  For the past, there was a completed exposure pathway for residents breathing air contaminated with 1,4-dioxane while bathing or showering. Some of the 1,4-dioxane in the water supplying the homes can volatilize into the air while showering and be inhaled by residents.

  In the past, there was a completed exposure pathway for residents from showering in the water containing 1,4-dioxane from the WMWF and other sources. Currently, the water delivered to consumers is blended from well fields that feed into the Cadmus treatment plant and from water purchased from two water utilities. The Cadmus treatment plant removes these contaminants before being distributed to customers, but the purchased water sources show the presence of 1,4-dioxane. This represents a current and future completed exposure pathway.

- *Dermal (skin) contact with contaminated surface water from Little Diamond Brook (past, current, future)*

  Residents, particularly children, who may wade and play in accessible areas of the Little Diamond Brook may be exposed to contaminants present in surface water. Based on information from EPA, the Little Diamond Brook is not used for fishing or swimming, so dermal contact would be the only exposure pathway.

**Eliminated Exposure Pathways**

- *Inhalation of indoor air contaminants from contaminated groundwater vapor entering residential properties and the Westmoreland Elementary School*

As stated earlier, based on soil gas and indoor air results for the residential properties and the Westmoreland Elementary School, the EPA and NJDEP determined vapor intrusion was not occurring. Out of an abundance of caution, NJDOH chose to evaluate indoor air data for the eight homes where indoor air samples were collected.

The likelihood of health effects depends on specific exposure conditions, including exposure duration, contaminant toxicity and concentration, and exposure frequency. In other words, it considers how long the exposure occurs, how toxic the contaminants are, how much contamination is present, and how often people come into contact with the contaminant. To determine whether adverse health effects are possible, NJDOH evaluates all completed exposure pathways.

## 2. Screening Analysis

A screening analysis involves comparing maximum concentrations of detected substances to media-specific screening levels. These levels help us understand which exposure levels of contaminants are considered safe. Screening levels may include ATSDR-comparison values (CVs) or other non-ATSDR values established by NJDEP or EPA. If concentrations meet or exceed the CV, these substances — potential contaminants of concern (COCs) — are selected for further evaluation. Concentrations that meet or exceed ATSDR-CVs or non-ATSDR screening levels do not necessarily indicate adverse health effects are likely. They assist health assessors with prioritizing which contaminants to evaluate further [ATSDR 2022].

### Comparison Values

Many CVs are available for screening contaminants and identifying potential COCs. CVs include ATSDR environmental media evaluation guides (EMEGs) and reference media evaluation guides (RMEGs). EMEGs represent estimated contaminant concentrations below which humans exposed during a specific timeframe (acute: up to 14 days; intermediate: between 15 and 364 days; or chronic: 365 days or more) are not expected to experience harmful noncancer health effects. RMEGs are based on EPA's reference doses. They indicate the concentration in water or soil at which daily human exposure over a lifetime is unlikely to result in harmful noncancer health effects.

If the substance is a known or a probable carcinogen and has cancer toxicity values, health assessors consider ATSDR's cancer risk evaluation guides (CREGs) as comparison values. CREGs represent estimated contaminant concentrations in soil or water that are expected to cause no more than one excess cancer in a million ($10^{-6}$) people exposed during their lifetime.

If ATSDR CVs are not available, EPA's regional screening levels (RSLs) or maximum contaminant levels (MCLs) are used. Other non-ATSDR screening levels include EPA and NJDEP's MCLs and surface water quality standards, although these must go through an approval process at ATSDR. As noted above, for vapor intrusion, NJDEP and EPA used their vapor intrusion screening levels, which are levels that are set to be protective of public health. We also included ATSDR's CVs as part of the screening process for vapor intrusion.

Contaminants exceeding applicable CVs and other screening levels will be evaluated further to determine the potential for health effects. These contaminants are identified as potential COCs.

**Drinking Water Data**

Data used for evaluation included the following:

- Sampling collected by EPA as part of the Unregulated Contaminant Monitoring Rule (UCMR) in 2013 and 2014
- Sampling collected by NJDEP in 2006
- Data from NJDEP's Drinking Water Watch (from 2018 to 2024)

Table 2 summarizes the detected contaminants and compares them to the applicable screening level. As noted in Table 2, perfluorobutane sulfonic acid (PFBS), perfluoroheptanoic acid (PFHpA), and perfluorohexane sulfonic acid (PFHxS) were not detected and therefore were not evaluated further. The maximum concentrations for perfluorononanoic acid (PFNA) were below applicable screening levels and were not evaluated further. No harmful effects are likely from contaminants that did not exceed screening levels.

Perfluorooctanesulfonic acid (PFOS), perfluorooctanoic acid (PFOA), and 1,4-dioxane exceeded screening levels. These contaminants were selected for further evaluation to determine their potential for harmful health effects.

**Table 2. Summary of Detected Drinking Water Contaminants from the Fair Lawn municipal water system (June 2006 to July 2024)**

| Contaminant | Chemical class | Number of samples | Number of detections | Minimum concentration (ng/L or ppt) | Maximum concentration (ng/L or ppt) | Screening level (ng/L or ppt) | Source | Exceed screening level |
|---|---|---|---|---|---|---|---|---|
| 1,4-Dioxane | SVOC | 21 | 6 | ND | 3,240 | 240 | CREG | **Yes** |
| Perfluorooctanesulfonic acid (PFOS) | PFAS | 303 | 264 | ND | 66 | 4 | EPA MCL | **Yes** |
| Perfluorooctanoic acid (PFOA) | PFAS | 299 | 268 | ND | 37 | 4 | EPA MCL | **Yes** |
| Perfluorononanoic acid (PFNA) | PFAS | 270 | 18 | ND | 2.9 | 10 | EPA MCL | No |
| Perfluorobutane sulfonic acid (PFBS) | PFAS | 20 | 0 | ND | ND | 2,000 | RMEG | No |
| Perfluorohexane sulfonic acid (PFHxS) | PFAS | 20 | 0 | ND | ND | 10 | EPA MCL | No |
| Perfluoroheptanoic acid (PFHpA) | PFAS | 20 | 0 | ND | ND | Not Available | Not Available | No |

Abbreviations: SVOC = semi-volatile organic compounds; PFAS = per- and polyfluoroalkyl substances; CREG = ATSDR's cancer risk evaluation guide; EPA MCL = US Environmental Protection Agency Maximum Contaminant Level; RMEG = ATSDR's reference media evaluation guide; ND = not detected; ng/L = nanograms per liter of water = parts per trillion or ppt.

### Vapor Intrusion Data: Residential Properties

EPA and the PRPs under NJDEP oversight conducted vapor intrusion investigations for the residential properties between 2009 and 2015. Most residential properties only have sub-slab soil gas data, as results did not warrant indoor air testing. Of the 51 homes evaluated for vapor intrusion, eight were sampled for indoor air. EPA and NJDEP determined that the vapor intrusion pathway was not complete for any residential properties based on the soil gas data. No further actions are planned for the residential properties.

**Table B1 in Appendix B** summarizes the indoor air contaminants found in the eight residences where indoor air was sampled by EPA or by the PRPs based on sub-slab soil gas results. A total of 19 indoor air samples were collected from these homes between February 2010 and May 2015. The eight homes where indoor air samples were collected are identified as properties R11, R18, R19, R31, R32, R33, R45, and R50. Eight contaminants exceeded applicable CVs in these homes. **Tables B2 to B9 in Appendix B** summarize the potential COCs for each residence.

### Vapor Intrusion Data: Commercial Properties

Indoor air samples were collected by EPA or by the PRPs under NJDEP oversight between November 2006 and February 2015 for eight commercial and industrial properties. **Tables B10 through B15 in Appendix B** summarize the potential COCs found in the six commercial properties where indoor air samples were collected. The remaining two commercial properties did not have indoor air data and were not included in this evaluation. The commercial properties are Sandvik, Hampshire, Kushner, Brizzolara/Brioschi/Mikia LLC (Mikia), Columbia Savings Bank (Columbia Bank), and Zimmer Biomet Inc. (Biomet).

### Daycare Center (currently Kiddie Academy)

There is a childcare center in the Fair Lawn Industrial Park. NJDOH reviewed indoor air data collected in December 2012 and determined there were no impacts from vapor intrusion. Per NJDOH's childcare safe siting unit, this center has a "Safe Building Interior Certificate," which was issued in November 2021 and expires in November 2024. No additional indoor air sampling was required at that time.

### Surface Water Data: Little Diamond Brook

As part of the sitewide sampling programs, surface water samples were collected from Little Diamond Brook and from outfalls located along the brook. **Table B16 in Appendix B** summarizes the contaminants detected in Little Diamond Brook, including surface water data for 17 locations along the brook that may be accessed by area residents. These samples were collected between May 2006 and June 2016.

As shown in **Table B16 in Appendix B**, the maximum concentrations of 1,4-dioxane, benzene, benzo[a]anthracene, benzo[a]pyrene, benzo[b]fluoranthene, benzo[k]fluoranthene, bromodichloromethane, carbon tetrachloride, dibenz[a,h]anthracene, dibromochloromethane,

hexachlorobenzene, indeno[1,2,3-cd]pyrene, pentachlorophenol, PCE, TCE, arsenic, dieldrin, chlordane, and heptachlor epoxide exceeded their respective CVs. These contaminants were identified as potential COCs in the Little Diamond Brook and thus were selected for further evaluation.

It should be noted that, with the exception of 1,4-dioxane (21 detections out of 169 samples), carbon tetrachloride (33 detections out of 169 samples) and PCE (68 detections out of 169 samples), the number of detections of the remaining potential COCs ranged from 1 to 4 out of 169 samples. The low frequency of detections for these remaining potential COCs may be considered outliers and not representative of exposures; therefore, these contaminants were not evaluated further.

## 3. Exposure Point Concentrations for Potential Contaminants of Concern

The next step in the scientific evaluation is to determine exposure point concentrations (EPCs) for contaminants exceeding CVs. EPCs refer to the estimated concentration of a contaminant in soil, water, or air at a specific location where individuals may be exposed. Contaminants detected below applicable CVs were not evaluated further because harmful health effects are not likely.

When assessing the public health implications of exposure to a contaminant of concern, ATSDR recommends using the 95% upper confidence limit (UCL) of the arithmetic mean as the exposure point concentration (EPC) when sufficient data are available. [ATSDR 2019]. The 95% UCL is considered a health-protective estimate of the average contaminant concentrations in an environmental medium.

Using ATSDR guidance, the 95% UCL is used for contaminants with at least eight samples and for samples with at least 20% detections [ATSDR 2019]. Maximum concentrations are used as EPCs for contaminants with fewer than eight samples and less than 20% detections.

Table 3 presents the EPCs for potential COCs in drinking water that exceed contaminants' CVs. Tables 4 and 5 show the EPCs used for each contaminant of potential concern in indoor air for residential and commercial properties. Table 6 shows the potential COCs in Little Diamond Brook.

These EPCs will be used to evaluate the potential for adverse health effects. In Tables 3 to 6, the drinking water concentrations are expressed as nanograms of contaminant per liter of water (ng/L), which is the same as parts per trillion (ppt). Surface water contaminant concentrations are expressed as micrograms of contaminant per liter of water (µg/L). Indoor air concentrations are expressed as micrograms of contaminant per cubic meter of air ($\mu g/m^3$).

**Table 3. EPCs for Drinking Water Contaminants from the Fair Lawn municipal water system**

| Contaminant | Number of samples | Number of detections | EPC (ng/L or ppt) | EPC type |
|---|---|---|---|---|
| 1,4-Dioxane | 21 | 6 | 0.740 | 95% UCL of the mean |
| Perfluorooctanesulfonic acid (PFOS) | 303 | 264 | 6.52 | 95% UCL of the mean |
| Perfluorooctanoic acid (PFOA) | 299 | 268 | 9.89 | 95% UCL of the mean |

Abbreviations: ng/L = nanograms of contaminant per liter of water = parts per trillion (ppt); UCL = upper confidence limit; EPC = exposure point concentration.

**Table 4. Residential Properties – EPCs – Indoor Air**

| Property ID | 1,2-dichloro-ethane (µg/m$^3$) | Benzene (µg/m$^3$) | Bromodichloro-methane (µg/m$^3$) | Carbon tetrachloride (µg/m$^3$) | Chloroform (µg/m$^3$) | Ethylbenzene (µg/m$^3$) | PCE (µg/m$^3$) | TCE (µg/m$^3$) |
|---|---|---|---|---|---|---|---|---|
| R11 | 2 | 1 | X | X | 1 | 2 | X | X |
| R18 | X | X | X | X | X | X | X | 0.24 |
| R19 | X | 4.5 | X | X | X | 2.7 | 4.3 | 0.3 |
| R31 | 0.191 | 1.45 | 2 | 0.391 | 5 | X | X | X |
| R32 | X | 1.34 | X | 0.348 | 0.325 | X | X | X |
| R33 | X | 1.48 | X | 0.357 | 0.549 | X | X | X |
| R45 | X | 10 | X | 0.399 | 1.37 | 8.15 | X | 0.197 |
| R50 | 2.26 | 1.89 | X | 0.391 | 1.59 | 1.54 | 4 | X |

Abbreviations: EPC = exposure point concentration
Note: The EPC represents the maximum concentration of contaminants detected in the indoor air samples.
µg/m$^3$ = micrograms of contaminant per cubic meter of air.
PCE = tetrachloroethylene; TCE = trichloroethylene.
X = EPC not calculated because contaminant was either not detected or was detected below applicable comparison value.

**Table 5. Commercial Properties – EPCs – Indoor Air**

| Property ID | 1,3-butadiene (µg/m$^3$) | Benzene (µg/m$^3$) | Carbon tetrachloride (µg/m$^3$) | Chloroform (µg/m$^3$) | Ethylbenzene (µg/m$^3$) | PCE (µg/m$^3$) | TCE (µg/m$^3$) | Xylenes (µg/m$^3$) |
|---|---|---|---|---|---|---|---|---|
| Sandvik | X | 1.1 | 1.4 | 1 | X | 5.4 | 0.4 | X |
| Hampshire | X | 0.8 | X | X | X | X | 1.1 | X |
| Kushner | X | 37.5 | X | X | 5 | X | 3 | X |
| Mikia | X | 3 | X | X | 36 | 53 | X | 188 |
| Columbia Bank | X | 1.2 | X | 1.7 | X | 210 | 38 | X |
| Biomet | 0.5 | 1.3 | X | X | 3.5 | 7.5 | X | X |

Abbreviations: EPC = exposure point concentration

UCL = upper confidence limit
Note: The EPC represents the 95% UCL of the mean or the maximum concentration of contaminants in the indoor air samples
µg/$m^3$ = micrograms of contaminant per cubic meter of air; PCE = tetrachloroethylene; TCE = trichloroethylene
X = EPC not calculated because contaminant was either not detected or was detected below applicable comparison value

**Table 6. EPCs for Surface Water Contaminants from Little Diamond Brook**

| Contaminant | Number of samples | Number of detections | EPC (µg/L) | EPC type |
|---|---|---|---|---|
| 1,4-Dioxane | 169 | 21 | 1.02 | 95% UCL of the mean |
| Carbon Tetrachloride | 169 | 33 | 1.44 | 95% UCL of the mean |
| PCE | 169 | 68 | 2.11 | 95% UCL of the mean |

Abbreviations: µg/L = micrograms of contaminant per liter of water
EPC = exposure point concentration
UCL = upper confidence limit
PCE = tetrachloroethylene

# Health Evaluation

## Exposure Dose Assumptions and Scenarios

**Drinking Water:** ATSDR's exposure dose guidance for water ingestion was used to calculate exposure doses [ATSDR 2023]. Exposure doses can be calculated for two water ingestion scenarios using the ATSDR Public Health Assessment Site Tool (PHAST). For individuals with typical (average) drinking water ingestion rates, a central tendency exposure (CTE) scenario can be applied. For those with above-average ingestion rates, a reasonable maximum exposure (RME) scenario is used. The RME refers to individuals with above-average exposures that are still within a realistic daily water intake.

To estimate the risk from showering and bathing in the water, ATSDR's SHOWER Model was used to estimate the amount of 1,4-dioxane released into the air during showering and bathing. The model also includes dermal uptake from skin contact with contaminated drinking water [ATSDR 2022a].

**Indoor Air:** For residential exposures, NJDOH assumed individuals are breathing potentially contaminated indoor air for 24 hours per day, seven days per week. for 52 weeks per year. Occupational exposures assume a worker scenario (RME) of 8.5 hours per day, five days per week, for 50 weeks per year, over a span of 20 years. The PHAST tool exposure worker scenario accounts for both full-time and part-time workers.

**Surface Water:** For exposures to surface water in Little Diamond Brook, NJDOH assumed adults and children ages 6 years and older may wade in the brook during the summer months. The brook is not used for swimming or fishing [Langan 2018c].

For drinking water and indoor air exposures, NJDOH used the most conservative RME scenario in this evaluation, representing the "worst-case" scenario for assessing potential harmful health effects. For surface water exposures, there is no difference between CTE and RME

scenarios for wading in Little Diamond Brook. **The scenarios, exposure parameters, and exposure dose equations used in this evaluation can be found in Appendix D – PHAST Results.**

### 4. In-Depth Analysis: Noncancer Health Effects

For health effects other than cancer, exposure doses and health guidelines are used to calculate hazard quotients (HQs). The hazard quotient is defined as the estimated exposure dose divided by the appropriate health guideline value. When the HQ exceeds 1.0 and approaches effect levels seen in toxicological literature, the potential for harmful effects increases. Noncancer health effects are not expected for hazard quotients below 1.0.

ATSDR developed health guidelines known as minimal risk levels (MRLs) for contaminants that are commonly found at hazardous waste sites. An MRL represents an estimate of the daily human exposure to a hazardous substance that is unlikely to result in adverse noncancer health effects. MRLs are developed for a specific route of exposure, such as ingestion (swallowing) and inhalation (breathing), and are defined for various specified exposure periods. Exposure periods are classified as follows:

- acute (less than 14 days)
- intermediate (15–364 days)
- chronic (365 days or more)

MRLs are primarily based on toxicological studies conducted in animals and reports of human workplace exposures. MRLs are typically derived from extrapolated doses (with safety factors applied) from effect levels reported in animal toxicological studies or occupational studies. In the toxicological literature, effect levels are usually reported as follows:

- no observed adverse effect level (NOAEL)
- lowest observed adverse effect level (LOAEL)

A NOAEL is the highest dose of a substance in a study that has been reported to have no harmful health effects on humans or animals. A LOAEL is the lowest dose of a substance in a study that has been reported to cause harmful health effects in humans or animals. Based on current ATSDR guidance, calculated exposure doses are compared to effect levels (LOAELs) when determining the potential for health effects. As the exposure dose increases beyond the MRL to the level of the LOAEL, the likelihood of adverse health effects also increases.

For contaminants without an MRL, the EPA's reference dose (RfD) or reference concentration (RfC) is used as a health guideline. An RfD is an estimate of a daily oral exposure to the human population (including sensitive subgroups such as children, pregnant women and the elderly) that is unlikely to pose appreciable risk of deleterious effects over a lifetime. An RfC is an estimate of continuous inhalation exposure concentration for people (including sensitive subgroups) that is unlikely to pose a risk of harmful effects over a lifetime. NJDOH used the most conservative chronic exposure scenarios for this evaluation.

## Exposure Dose Assumptions and Scenarios

*Exposures to 1,4-dioxane:*

To evaluate exposures to 1,4-dioxane in drinking water, ATSDR's SHOWER Model was used. This model predicts how much 1,4-dioxane would be released into the air that could be breathed in while bathing or showering. It also accounts for dermal uptake from skin contact with contaminated water.

Table 7a shows the combined doses for water ingestion and skin contact with 1,4-dioxane in drinking water at a concentration of 0.740 µg/L (0.00074 mg/L). Exposure doses for children ages birth through 20 years were calculated using ATSDR's PHAST tool. The highest calculated dose is for children ages birth to younger than 1 year and is presented in Table 7a.

Based on the maximum calculated exposure doses for chronic exposures to 1,4-dioxane, the hazard quotients for children and adults were below 1.0. Therefore, harmful noncancer health effects are not expected for children and adults ingesting contaminated drinking water and having skin contact with contaminated water while showering and bathing.

**Table 7a. Noncancer Health Effects associated with drinking water containing 1,4-dioxane**

| Contaminant | EPC [a] (mg/L) | RME dose [b] (mg/kg/day) | MRL [c] (mg/kg/day) | Hazard quotient (HQ) [d] | Potential for health effects |
|---|---|---|---|---|---|
| Child (birth to < 1 year) | 0.00074 | 0.00010 | 0.1 | 0.001 | No (HQ < 1) |
| Adult | 0.00074 | 0.00003 | 0.1 | 0.0003 | No (HQ < 1) |

[a] EPC = exposure point concentration;
Note: The EPC represents the maximum concentration of post-treatment samples.
[b] RME dose = reasonable maximum exposure dose representing above-average water ingestion rates for children ages birth to one year of age
[c] MRL = ATSDR's minimal risk level
[d] Hazard quotient = RME dose/MRL
mg/L = milligrams of contaminant per liter
mg/kg/day = milligrams of contaminant per kilogram of body weight per day

*Exposures to 1,4-dioxane from showering and bathing:*

The EPC for 1,4-dioxane in the water is 0.740 µg/L and was used as the input concentration in the shower model for breathing in 1,4-dioxane vapors. The model is based upon four people, including children, living in a residence and simulates exposure for the person with the highest exposure to 1,4-dioxane while showering.

Table 7b summarizes the predicted air concentrations for 1,4-dioxane from showering for the RME scenario. This scenario assumes the first three people take 10-minute morning showers and the fourth person takes a 15-minute morning shower, each followed by five-minute bathroom stays. Due to the accumulation of 1,4-dioxane in the air from three consecutive showers, the fourth person is exposed to the highest concentration of 1,4-dioxane in air.

The adjusted 1,4-dioxane concentration in Table 7b is based on a time-weighted average air concentration calculated for the fourth person as they spend their time in various parts of the house following their morning shower. This daily exposure concentration applies to children and adults who take showers. Younger children may not take showers but are exposed by taking baths. Their inhalation and skin contact exposures from a 20-minute bath scenario are comparable to the RME shower scenario. Therefore, the adjusted 1,4-dioxane air concentration for the 4th person taking a shower is the same for both adults and children and is similar to the exposure for young children taking a bath.

**Table 7b. ATSDR SHOWER Model Results**

| Exposure group | Adjusted 1,4-dioxane concentration (RME) µg/m$^3$ | MRL (µg/m$^3$) | Hazard quotient (RME) | Further evaluation needed |
|---|---|---|---|---|
| Child or adult resident | 0.016 | 110 | 0.0001 | No (HQ<1) |

Abbreviations: RME = reasonable maximum (above average) exposure; µg/m$^3$ = micrograms of 1,4-dioxane per cubic meter of air; MRL = chronic duration minimal risk level; Hazard Quotient (HQ) = Adjusted 1,4-dioxane concentration RME/MRL = 0.016/110 = 0.00014

As shown above in Table 7b, the RME hazard quotient is below 1.0. Therefore, adverse health effects are not expected for residents showering or bathing in water containing 1,4-dioxane at 0.740 µg/L.

*Exposures to PFAS in drinking water*

Tables 8 and 9 summarize the calculated exposure doses and hazard quotients for children and adults for exposures to PFOA and PFOS in drinking water. The maximum exposure dose for children using the RME scenario is for children age birth to younger than one year.

**Table 8. CTE and RME doses and hazard quotients for PFOA in drinking water**

| Age Group (years) | EPC (mg/L) | CTE dose [b] (mg/kg/day) | RME dose [c] (mg/kg/day) | RfD (mg/kg/day) | Hazard Quotient (HQ) Range for CTE and RME scenarios [e] | Requires Further Toxicological Evaluation |
|---|---|---|---|---|---|---|
| Birth to < 1 | $9.9 \times 10^{-6}$ | $7.5 \times 10^{-7}$ | $1.4 \times 10^{-6}$ | $3.0 \times 10^{-8}$ | 25 - 47 | **Yes** (HQ > 1) |
| 1 to < 2 | $9.9 \times 10^{-6}$ | $2.1 \times 10^{-7}$ | $5.7 \times 10^{-7}$ | $3.0 \times 10^{-8}$ | 7 - 19 | **Yes** (HQ > 1) |
| 2 to < 6 | $9.9 \times 10^{-6}$ | $1.9 \times 10^{-7}$ | $4.8 \times 10^{-7}$ | $3.0 \times 10^{-8}$ | 6 - 16 | **Yes** (HQ > 1) |
| 6 to < 11 | $9.9 \times 10^{-6}$ | $1.4 \times 10^{-7}$ | $3.9 \times 10^{-7}$ | $3.0 \times 10^{-8}$ | 5 - 13 | **Yes** (HQ > 1) |
| 11 to <16 | $9.9 \times 10^{-6}$ | $9.8 \times 10^{-8}$ | $3.1 \times 10^{-7}$ | $3.0 \times 10^{-8}$ | 3 - 10 | **Yes** (HQ > 1) |
| 16 to <21 | $9.9 \times 10^{-6}$ | $1.0 \times 10^{-7}$ | $3.1 \times 10^{-7}$ | $3.0 \times 10^{-8}$ | 3 - 10 | **Yes** (HQ > 1) |
| Adult | $9.9 \times 10^{-6}$ | $1.6 \times 10^{-7}$ | $4.0 \times 10^{-7}$ | $3.0 \times 10^{-8}$ | 5 - 13 | **Yes** (HQ > 1) |

Note: The EPC represents the maximum concentration of post-treatment samples

[a] EPC = exposure point concentration

[b] CTE dose = central tendency exposure dose representing average water ingestion rates for children age birth to one year of age

[c] RME dose = reasonable maximum exposure dose representing above-average water ingestion rates for children ages birth to one year of age

[d] RfD = EPA Reference Dose

[e] Hazard quotient = RME dose/RfD
mg/L = milligrams of contaminant per liter
mg/kg/day = milligrams of contaminant per kilogram of body weight per day

**Table 9. CTE and RME doses and hazard quotients for PFOS in drinking water**

| Age Group (years) | EPC [a] (mg/L) | RME dose [b] (mg/kg/day) | RfD [c] (mg/kg/day) | Hazard Quotient (HQ) [d] | Requires Further Toxicological Evaluation |
|---|---|---|---|---|---|
| Child (birth to < 1) | $6.5 \times 10^{-6}$ | $9.2 \times 10^{-7}$ | $1.0 \times 10^{-7}$ | 9 | **Yes** (HQ > 1) |
| 1 to < 2 | $6.5 \times 10^{-6}$ | $3.8 \times 10^{-7}$ | $1.0 \times 10^{-7}$ | 4 | **Yes** (HQ > 1) |
| 2 to < 6 | $6.5 \times 10^{-6}$ | $3.2 \times 10^{-7}$ | $1.0 \times 10^{-7}$ | 3 | **Yes** (HQ > 1) |
| 6 to < 11 | $6.5 \times 10^{-6}$ | $2.6 \times 10^{-7}$ | $1.0 \times 10^{-7}$ | 3 | **Yes** (HQ > 1) |
| 11 to <16 | $6.5 \times 10^{-6}$ | $2.0 \times 10^{-7}$ | $1.0 \times 10^{-7}$ | 2 | **Yes** (HQ > 1) |
| 16 to <21 | $6.5 \times 10^{-6}$ | $2.0 \times 10^{-7}$ | $1.0 \times 10^{-7}$ | 2 | **Yes** (HQ > 1) |
| Adult | $6.5 \times 10^{-6}$ | $2.6 \times 10^{-7}$ | $1.0 \times 10^{-7}$ | 3 | **Yes** (HQ > 1) |

[a] EPC = exposure point concentration
Note: The EPC represents the maximum concentration of post-treatment samples
[b] RME dose = reasonable maximum exposure dose representing above-average water ingestion rates for children age birth to one year of age
[c] RfD = EPA Reference Dose
[d] Hazard quotient = RME dose/RfD
mg/L = milligrams of contaminant per liter
mg/kg/day = milligrams of contaminant per kilogram of body weight per day

### Potential for Noncancer Health Effects: PFOA and PFOS in Drinking Water

As shown in Tables 8 and 9 above, both PFOA and PFOS exceeded the Hazard Quotient of 1.0, which means that further toxicological evaluation is needed to determine if harmful effects might be possible. The exposure parameters and equations used to calculate the hazard quotients can be found in **Appendix D – PHAST Results.**

PFOA:

The oral RfD of $3 \times 10^{-8}$ mg/kg/day is based on the following three health effects:

- cardiovascular (increased total cholesterol in adults)
- immune (decreased serum anti-tetanus and anti-diphtheria antibody concentrations in children)
- developmental effects (decreased infant birth weight)

These co-critical effects for the oral RfD were based on decreased serum anti-tetanus and anti-diphtheria antibody concentrations in children, decreased infant birth weight, and increased total cholesterol in adults. EPA identified these co-critical effects in their peer-reviewed human health toxicity assessment document for PFOA (U.S. EPA, 2024a).

The RfD was derived by using a total uncertainty factor of 10 to account for human variability. Notably, the RfD is protective of effects that may occur in sensitive populations (e.g.., the developing fetus, infants, and young children), as well as hepatic (liver) effects in adults that may result from PFOA exposure. As two of the co-critical effects identified for PFOA are developmental endpoints and can potentially result from a short-term exposure during critical periods of development, EPA concludes that the overall RfD for PFOA is applicable to both short-term and chronic risk assessment scenarios [U.S. EPA 2024a].

Based on the detected PFOA water concentration, doses for all age groups with average (CTE scenario) and above-average ingestion rates (RME scenario) have exposures that exceed doses identified in EPA's toxicity assessment document that might cause reduced antibody response and elevated cholesterol levels in the blood of humans. Additionally decreased birth weights in newborn infants from in utero exposures during the first and second trimesters is another potential adverse health effect. Mild liver damage (indicated by increased liver enzymes in the blood) might be possible in residents with above-average ingestions rates (RME scenario).

PFOS:

The oral RfD of $1 \times 10^{-7}$ mg/kg/day is based on the following adverse health effects in humans:

- cardiovascular (increased total cholesterol in adults); and
- developmental effects (decreased infant birth weight).

These co-critical effects for the oral RfD were based on decreased infant birth weight and increased total cholesterol in adults. EPA identified these co-critical effects in their peer-reviewed human health toxicity assessment document for PFOS [U.S. EPA, 2024b].

The RfD was derived by using a total uncertainty factor of 10 to account for human variability. Notably, the RfD is protective of effects that may occur in sensitive populations (e.g. the developing fetus, infants, and young children), as well as hepatic effects in adults that may result from PFOS exposure. As one of the co-critical effects identified for PFOS is a developmental endpoint and can potentially result from a short-term exposure during critical periods of development, EPA concludes that the overall RfD for PFOS is applicable to both short-term and chronic risk assessment scenarios [U.S. EPA 2024b].

Based on the detected PFOS water concentration, doses in children birth to age < 2 years with above-average ingestion rates (RME scenario) have exposures that approach doses associated with reduced antibody response, increased cholesterol levels, and mild liver damage. Doses in other age groups (older children, teenagers, and adults) are below levels thought to cause harmful effects.

The effects are very similar between PFOS and PFOA, which target the cardiovascular, immune, developmental, and hepatic systems. As these two PFAS are present as a mixture in drinking water, doses for all age groups with average and above-average ingestion rates have exposures that exceed doses thought to cause elevated cholesterol levels in blood, reduced antibody response, decreased infant birth weight, and elevated liver enzymes in adults.

**Potential for Noncancer Health Effects: Vapor Intrusion (Indoor Air Impacts)**

ATSDR's approach for evaluating exposures via inhalation in residential settings used the measured air concentrations to calculate EPCs of the contaminants and compared them to the MRL via a ratio known as a "hazard quotient." The hazard quotient is defined as the measured air concentration divided by ATSDR's MRL or EPA's RfC. For occupational exposures, the measured air concentration is adjusted for a worker scenario using 8.5 hours/day, five days/week, for 50 weeks. This adjusted concentration is then compared to ATSDR's inhalation MRL or EPA's RfC to obtain a hazard quotient.

The exposure factor (EF) for noncancer health effects in workers is calculated as follows:

$$EF = \frac{\text{number of hours worked}}{\text{24 hours per day}} \times \frac{\text{number of days}}{\text{7 days per week}} \times \frac{\text{50 working weeks}}{\text{52 weeks per year}}$$

This EF is then multiplied by the measured air concentration to get an adjusted air concentration to compare with ATSDR's inhalation MRL or EPA's RfC to obtain a hazard quotient.

When a hazard quotient is above 1.0, further evaluation is warranted to determine if harmful effects might be possible. Harmful noncancer health effects are not expected for hazard quotients below 1.0.

**Tables B17 through B24 in Appendix B** summarize the hazard quotients for potential COCs in each residence where indoor air data were collected. The exposure parameters and equations used to calculate the hazard quotients can be found in **Appendix D – PHAST Results.**

As shown in the tables, the indoor air contaminants found in all eight residences where indoor air data was collected did not exceed a HQ of 1.0, except for benzene in residence R45 (**Table B23, Appendix B**) and chloroform in residence R31 (**Table B20, Appendix B**). Therefore, benzene and chloroform will be evaluated further for possible noncancer health effects as described below. According to the EPA, the benzene and chloroform detected in these homes were due to background sources within the homes and not from site-related vapor intrusion.

A chronic inhalation MRL (6.4 µg/$m^3$) was derived for benzene was based on an occupational study that collected blood and urine samples from workers at two shoe manufacturing plants that used benzene. Workers were exposed for eight hours per day for six days per week [Lan et al. 2004].

The study noted reduced numbers of platelets and white blood cells in workers exposed to increasing levels of benzene. These types of cells help fight off infection and help the blood to clot. The LOAEL from this study was 0.57 parts per million (ppm), or approximately 1,820 µg/$m^3$. This concentration was adjusted from a worker exposure to a continuous exposure of 0.16 ppm (rounded to 0.2 ppm). An uncertainty factor of 100 was then used to account for human variability and the use of a LOAEL to derive the MRL of 0.002 ppm (6.4 µg/$m^3$).

The EPC for benzene in residence R45 was 10 µg/m$^3$ which is well below the level where health effects were observed in the occupational study used to derive the MRL. Therefore, harmful noncancer health effects are not likely from chronic indoor air exposures to benzene at this residence.

A chronic duration MRL (2 µg/m$^3$) was derived for chloroform based on nasal damage in mice exposed to chloroform concentrations of 24,400 µg/m$^3$ and higher for five days per week for six hours per day for 104 weeks [Yamamoto et al. 2002].

The MRL is based on a LOAEL of 24,400 µg/m$^3$ that was adjusted from an intermittent to a continuous exposure of 24 hours per day, 7 days per week and then converted to a human equivalent concentration of 537 µg/m$^3$ using EPA models. This concentration was then divided by an uncertainty factor of 300 to account for the use of a LOAEL, extrapolating from animals to humans, and for human variability.

The maximum concentration of chloroform detected in residence R31 was 5 µg/m$^3$. This concentration is approximately 100 times lower than the LOAEL human equivalent concentration of 537 µg/m$^3$ used to derive the MRL. Therefore, harmful noncancer health effects are not expected from exposure to chloroform in this residence. The remaining contaminants in the eight residences did not exceed an HQ of 1.0, thus noncancer health effects are not expected from exposures to indoor air contaminants in these homes.

**Potential for Noncancer Health Effects: Indoor Air in Commercial Properties**

As mentioned earlier, the concentrations of contaminants detected in the commercial properties were adjusted for a worker scenario to calculate hazard quotients. Occupational exposures assume a full-time worker scenario of 8.5 hours per day, five days per week, for 50 weeks each year. **(See Appendix D – PHAST Results)**. Although part-time workers were included, their data are not presented in the following tables for simplicity. Because they work part-time, their exposures were significantly lower than full time workers. **Tables B25 through B30 in Appendix B** summarize the hazard quotients for potential COCs in the commercial properties.

As shown in the tables in **Appendix B**, PCE and TCE had elevated hazard quotients in Columbia Bank (**Table B29, Appendix B**) and benzene had an elevated hazard quotient in the Kushner facility (**Table B27, Appendix B**). Based on the toxicological evaluation described above for benzene in the residential scenario, noncancer health effects also are not expected for workers at the Kushner facility. This is because the adjusted air concentration for workers at the Kushner facility (9.1µg/m$^3$) is lower than the residential indoor air concentration (10 µg/m$^3$). Because the toxicological evaluation indicated no adverse health effects expected for residents, adverse health effects would also not be expected for workers at the Kushner facility because their exposure was lower.

All other hazard quotients for the commercial properties had hazard quotients that were below 1.0, which means that noncancer health effects are not expected.

### Columbia Bank: Health Effects of Tetrachloroethylene (PCE) in Indoor Air

As described earlier, the LOAEL is the lowest tested amount of a substance, that has been reported to cause harmful health effects. At a LOAEL of 11,530 µg/m$^3$, an epidemiological study of PCE exposure in dry cleaner workers showed a significant decrease in blue-yellow color vision compared to controls, and workers who experienced continued exposure demonstrated a further deterioration in blue-yellow color vision when evaluated two years after the initial measurements [Cavalleri A et al. 1994]. A chronic MRL of 41 µg/m$^3$ has been derived from this human study by applying uncertainty factors to account for human variability and a lack of information about immune system effects.

Other occupational studies showed workers exposed to PCE concentrations ranging from approximately 76,000 to 277,000 µg/m$^3$ performed below expectation on tasks assessing memory, motor skills (reaction times), visual and executive function deficits following low-level exposure for one year or more [Echeverria, 1995]. Another human study showed mild kidney damage at a LOAEL of 68,000 µg/m$^3$ [Franchini et al. 1983]. Table 10 summarizes these health effects.

**Table 10. Health Effect Levels – PCE**

| Study | ATSDR MRL Derivation Study (human) | Other Studies (Human) | Other Studies (Human) |
|---|---|---|---|
| **LOAEL (µg/m$^3$)** | 11,530 * | 68,000 | 76,000 -277,000 |
| **Health Effect** | Decreased color vision | Mild kidney damage | Decreased neurological functions |

Source: ATSDR Toxicological Profile for PCE available at: atsdr.cdc.gov/toxprofiles/tp18.pdf; µg/m$^3$ = micrograms of PCE per cubic meter of air; MRL = ATSDR Minimal risk level; PCE = Tetrachloroethylene; * Represents LOAEL adjusted from occupational exposures to obtain an equivalent continuous exposure concentration.

The exposure point concentration for PCE detected in Columbia Bank was 210 µg/m$^3$ (Table B29, Appendix B). This level was adjusted for a full-time worker scenario to obtain 51 µg/m$^3$. The adjusted worker exposure to PCE at Columbia Bank (51 µg/m$^3$) is slightly above the MRL (41 µg/m$^3$) and is well below the LOAEL of 11,530 µg/m$^3$ for decreased color vision and other harmful effects. Therefore, harmful noncancer health effects from PCE exposures are not expected for full-time or part-time workers in the Columbia Bank.

### Columbia Bank: Health Effects of Trichloroethylene (TCE) in Indoor Air

ATSDR adopted EPA's RfC as the chronic, inhalation MRL (2.1 µg/m$^3$). The RfC for TCE is based on two oral rodent studies [Keil DE et al. 2009, Johnson PD et al. 2003] and one supporting study. In these studies, animals were exposed to TCE orally via drinking water. The most sensitive adverse effects involved the immune system and the developing fetus. Effects to the immune system were decreased thymus weight and increased serum levels of lgG and selected autoantibodies. Effects to the developing fetus were cardiac malformations. The EPA used physiologically based pharmacokinetic (PBPK) modeling to convert the oral dose in animals to a human equivalent concentration (HEC) of TCE in air. Based on these studies, EPA identified the effect levels for TCE exposures in air as follows:

- Mouse Study - Immunological effects = 180 µg/m$^3$
- Rat Study – Developmental effects = 20 µg/m$^3$

The EPA also cites a third study conducted in 1988 by the National Toxicology Program (of lower confidence) in support of the RfC where female rats were exposed to TCE in corn oil by gavage for a 104-week period [NTP, 1988]. The EPA used PBPK modeling to convert the oral dose in animals to a HEC of 30 µg/m$^3$ TCE in air for kidney damage (See Table 11).

**Table 11. Health effect levels - TCE**

| Study | EPA RfC Basis | EPA RfC Basis | EPA Support Study (National Toxicology Program) |
|---|---|---|---|
| **Effect Level (µg/m$^3$) *** | 20 | 180 | 30 |
| **Health Effect** | Developmental Effects (Rat Study) | Immune System Effects (Mouse Study) | Kidney Effects (Rat Study) |

*The effect levels for these studies were derived using EPA models to derive "human equivalent concentrations (HECs): EPA Toxicological Review of TCE (iris.epa.gov/static/pdfs/0199tr.pdf).

It should be noted that ATSDR has recently determined the Johnson study referenced above where fetal cardiac malformations were observed has insufficient confidence to determine an exposure level at which cardiac effects may or may not occur. Additionally, other studies showed very low to low potential for fetal cardiac effects [ATSDR 2025]. Therefore, there's some uncertainty about whether TCE exposure in pregnant women might result in fetal heart malformation in newborns.

### Columbia Bank: Evaluating the Risk of Harmful Effects from TCE Exposure

The measured air concentration in the Columbia Bank (38 µg/m$^3$) was adjusted to a worker exposure concentration (9.2 µg/m$^3$). Table 12 shows the chronic inhalation RfC and MRL, the adjusted TCE air concentrations for workers, the air concentrations where kidney and immune effects might occur, and whether the adjusted air concentration is approaching effect levels. EPA identified the effect level for kidney damage (30 µg/m$^3$) in its derivation of the RfC [U.S. EPA 2011] and EPA and ATSDR identified the immune effect level of 177 µg/m$^3$ [U.S. EPA 2011, ATSDR 2019b].

**Table 12. Comparing the Adjusted Air Concentration for TCE to the RfC, MRL, and Effect Levels**

| Commercial space | Chronic RfC and MRL (µg/m$^3$) | Adjusted worker air concentration* (µg/m$^3$) | Kidney Effect Level (µg/m$^3$) | Immune Effect Level (µg/m$^3$) | Adjusted Air Concentration Exceeds MRL/RfC | Adjusted Air Concentration Approaches Kidney Effect Level | Adjusted Air Concentration Approaches Immune Effect Level |
|---|---|---|---|---|---|---|---|
| Columbia Bank | 2.1 | 9.2 | 30 | 177 | Yes | Yes | No |

Abbreviations: TCE = trichloroethylene; RfC = Reference Concentration; MRL = Minimal Risk Level; µg/m$^3$ = micrograms of TCE per cubic meter of air

* Adjusted air concentration for worker scenario= 38 µg/m$^3$ x 8.5hrs/24hrs x 5days/7days x 50weeks/52weeks = 9.2 µg/m$^3$

As shown in Table 12 above, the adjusted TCE level in Columbia Bank approaches the kidney effect level but not the immune effect level. Therefore, workers in the bank are at risk of harmful effects to the kidneys from TCE exposure.

The conclusions listed above for workers exposed to TCE in Columbia Bank were based on data collected in March 2011. Another air sample from the bank was collected in January 2012, which showed lower TCE levels. The maximum concentration of TCE detected during the January 2012 sampling event was also evaluated to determine the potential for health impacts since levels decreased.

Using the January 2012 TCE concentration of 5.9 µg/m$^3$, the adjusted concentration for workers is 1.4 µg/m$^3$. The adjusted TCE concentration is below the RfC and inhalation MRL and results in a hazard quotient of 0.68 (see Table 13). Therefore, based on the most recent sampling event from January 2012, harmful noncancer health impacts would not be expected. No additional indoor air sampling has been conducted since that time.

**Table 13. Hazard Quotient for TCE – January 2012 Sampling Event**

| Commercial space | Chronic RfC and MRL (µg/m$^3$) | Adjusted worker air concentration* (µg/m$^3$) | Hazard Quotient | Potential for Noncancer Health Effects |
|---|---|---|---|---|
| Columbia Bank | 2.1 | 1.4 | 0.68 | No (HQ < 1) |

Abbreviations: TCE = trichloroethylene; µg/m$^3$ = micrograms of TCE per cubic meter of air; RfC = Reference Concentration; MRL = Minimal Risk Level;
* Adjusted air concentration using January 2012 TCE data = 5.9 µg/m$^3$ x 8.5hrs/24hrs x 5days/7days x 50 weeks/52weeks = 1.4 µg/m$^3$; Hazard quotient = Adjusted air concentration / TCE MRL = 1.4/2.1=0.68.

**Potential for Noncancer Health Effects: Surface Water (Wading in Little Diamond Brook)**

Based on the maximum calculated exposure doses for chronic exposures to 1,4-dioxane, carbon tetrachloride, and PCE in the Little Diamond Brook, the hazard quotients for all three contaminants were below 1.0 (see Table 14). Therefore, harmful noncancer health effects are not expected for children and adults wading in Little Diamond Brook.

**Table 14. Hazard Quotients – Little Diamond Brook**

| Contaminant | EPC (mg/L) | Exposure Dose * (mg/kg/day) | Health Guideline (mg/kg/day) | Hazard Quotient (HQ) | Potential for noncancer health effects |
|---|---|---|---|---|---|
| 1,4-dioxane | 0.00102 | $4.4 \times 10^{-9}$ | 0.1 (MRL) | $4.4 \times 10^{-8}$ | No (HQ < 1) |
| Carbon Tetrachloride | 0.00144 | $4.5 \times 10^{-7}$ | 0.004 (RfD) | $1.1 \times 10^{-4}$ | No (HQ <1) |
| PCE | 0.00211 | $1.5 \times 10^{-6}$ | 0.008 (MRL) | $1.8 \times 10^{-4}$ | No (HQ <1) |

Abbreviations: EPC = exposure point concentration representing the 95% UCL of the mean; PCE = tetrachloroethylene; mg/L = milligrams of contaminant per liter of water; mg/kg/day = milligrams of contaminant per kilogram of body weight per day; MRL = ATSDR chronic minimal risk level; RfD = EPA Reference Dose;

*Exposure dose represents the maximum dermal dose for children ages 6 to < 11 years; Hazard Quotient example calculation for 1,4-dioxane = exposure dose / health guideline = $4.4 \times 10^{-9} / 0.1 = 4.4 \times 10^{-8}$

## 5. In-Depth Analysis: Cancer Health Effects

NJDOH evaluates the potential for cancer health effects by assessing the excess cancer risk from exposure to site-related contaminants that exceed the background cancer risk. In New Jersey, approximately 45% of women and 47% of men (about 46% overall) will be diagnosed with cancer in their lifetime [NJDOH 2023]. This is referred to as the "background cancer risk."

The term "excess cancer risk" represents the risk on top of the background cancer risk and is referred to as the Lifetime Excess Cancer Risk, or LECR. An LECR of "one-in-a-million" (1 excess case per 1,000,000 similarly exposed individuals or $1x10^{-6}$ cancer risk) means that if 1,000,000 people are similarly exposed to a cancer-causing substance at a certain level for a specified period, then one cancer above the background number of cancers may develop in those one million people over the course of their lifetime (approximately 78 years).

To put the LECR of $1x10^{-6}$ in the context of New Jersey's background cancer risk, the number of cancers expected in one million people over their lifetime is 460,000 (46%) in New Jersey. If these one million people are all exposed similarly to a cancer-causing substance for a specific duration, then 460,001 individuals might develop cancer instead of the expected 460,000 over the course of their lifetime (approximately 78 years).

The NJDOH follows ATSDR's guidelines to evaluate theoretical cancer risks from environmental exposures (ATSDR 2022). A **concern for an increased risk** is categorized as an excess of one or more additional cancer cases per 10,000 people (expressed as risk in "the $1x10^{-4}$ range" or higher). When cancer risk estimates are less than $1 \times 10^{-4}$, a determination is needed as to whether a concern exists or does not exist for an increased risk of cancer. Several factors are considered in determining whether cancer risks less than $1 \times 10^{-4}$ are a health concern. Site specific factors may be considered in addition to the default factors used in the risk assessment model (which include the length of exposure, sensitive populations who may already have an elevated risk due to exposure to other carcinogens, and exposure to mutagenic carcinogens at a young age). Additionally, if maximum contaminant concentrations are used in the risk assessment model due to limited environmental data, the cancer risk might be overestimated.

It should be noted that the estimated cancer risks are a theoretical estimate of risk that NJDOH and ATSDR use as a tool for deciding whether public health actions are needed to protect health. It is not an actual estimate of cancer cases in a community and is not a prediction that cancer will occur. It is also important to note that while cancer rates are readily available at an aggregate level, individual exposures cannot be captured in these analyses, and other risk factors associated with cancer are not taken into account.

Health guidelines are typically developed for carcinogens based on one excess cancer case per 1,000,000 individuals similarly exposed. According to the U.S. Department of Health and Human Services (DHHS) National Toxicology Program (NTP), the International Agency for Research on Cancer (IARC) and the EPA, possible cancer classes for contaminants detected at a site are as follows:

- Known to be carcinogenic to humans
- Reasonably anticipated to be carcinogenic to humans
- Not classified

The information below regarding the potential carcinogenicity of contaminants evaluated in this document was obtained from ATSDR's Toxicological Profiles.

**Benzene:** Long-term exposure to high levels of benzene in the air can cause leukemia, particularly acute myelogenous leukemia, often referred to as AML. This is cancer of the blood forming cells. The DHHS has determined that benzene is known to be a human carcinogen. The IARC and the EPA have determined that benzene is carcinogenic to humans [ATSDR 2024a].

**Bromodichloromethane:** Large intestine, kidney, and/or liver tumors were found in lab animals exposed to high levels of bromodichloromethane. These levels are higher than levels usually found in the environment. The DHHS considers bromodichloromethane to be reasonably anticipated to be a human carcinogen, and the EPA considers it to be a probable human carcinogen [ATSDR 2020a].

**1,3-Butadiene:** The DHHS, IARC, and EPA have determined that 1,3-butadiene is a human carcinogen. Studies have shown that workers exposed to 1,3-butadiene may have an increased risk of cancers of the stomach, blood, and lymphatic system. Animal studies found increases in a variety of tumor types from exposure to 1,3-butadiene [ATSDR 2012a].

**Carbon Tetrachloride:** Studies in humans have not been able to determine whether carbon tetrachloride can cause cancer because usually there has been exposure to other chemicals at the same time. Swallowing or breathing carbon tetrachloride for years caused liver tumors in animals. Mice that breathed carbon tetrachloride also developed tumors of the adrenal gland. The DHHS has determined that carbon tetrachloride may reasonably be anticipated to be a carcinogen. The IARC has determined that carbon tetrachloride is possibly carcinogenic to humans, whereas the EPA determined that carbon tetrachloride is a probable human carcinogen [ATSDR 2005].

**Chloroform:** The ability of chloroform to cause cancer in people has not been well studied. Tumors in the kidneys were seen in mice that breathed chloroform for a long period of time. Animals that drank chloroform in water for long periods developed cancer of the liver and kidneys. The DHHS determined that chloroform is reasonably anticipated to be a human carcinogen based on sufficient evidence of carcinogenicity in experimental animals. The EPA classified chloroform as likely to be carcinogenic to humans by all routes of exposure at high dose levels that result in cell damage; EPA determined chloroform is not likely to be carcinogenic by any route at lower dose levels that do not cause cell damage. The IARC determined that chloroform is possibly carcinogenic to humans based on inadequate evidence in humans and sufficient evidence in experimental animals [ATSDR 2024b].

**1,2-Dichloroethane:** The DHHS has determined that 1,2-dichloroethane may reasonably be anticipated to be a human carcinogen. The EPA has classified 1,2-dichloroethane as a probable

human carcinogen. The IARC has determined that 1,2-dichloroethane is possibly carcinogenic to humans. Cancers, including hemangiosarcomas and mammary gland tumors in both rats and mice; subcutaneous fibromas and forestomach carcinomas in rats; and liver, lung, and endometrial tumors in mice have been observed in long-term animal studies of exposure to 1,2-dichloroethane via oral and inhalation routes [ATSDR 2024c].

**1,4-Dioxane:** The limited number of studies available do not show whether 1,4-dioxane causes cancer in humans. Laboratory rats that breathed 1,4-dioxane during most of their lives developed cancer inside the nose and abdominal cavity. Laboratory rats and mice that drank water containing 1,4-dioxane during most of their lives developed liver cancer; the rats also developed cancer inside the nose. Scientists are debating the degree to which the findings in rats and mice apply to exposure situations commonly encountered by humans. The DHHS considers 1,4-dioxane as reasonably anticipated to be a human carcinogen [ATSDR 2012b].

**Ethylbenzene:** The IARC has determined that ethylbenzene is a possible human carcinogen. However, EPA has not classified the carcinogenicity of ethylbenzene in humans. Animal studies have reported effects on the blood, liver, and kidneys from chronic exposures to ethylbenzene [ATSDR 2010].

**PCE:** Studies in humans suggest that exposure to PCE might lead to a higher risk of developing bladder cancer, multiple myeloma, or non-Hodgkin's lymphoma. In animals, PCE has been shown to cause cancers of the liver, kidney, and blood system. The DHHS considers PCE to be reasonably anticipated to be a human carcinogen. The EPA considers PCE likely to be carcinogenic to humans by all routes of exposure. The IARC considers PCE to be a probable human carcinogen [ATSDR 2019a].

**PFOA:** EPA concluded that PFOA is likely to be carcinogenic to humans. (U.S. EPA, 2005a). EPA selected the critical effect of renal cell carcinomas in human males as the basis of the overall cancer slope factor (CSF) for PFOA [USEPA 2024a].

**PFOS:** EPA concluded that PFOS is likely to be carcinogenic to humans (U.S. EPA, 2005a). EPA selected hepatocellular adenomas and carcinomas in female rats as the basis of the overall CSF for PFOS [USEPA 2024b].

**TCE:** There is strong evidence that TCE can cause kidney cancer in people and some evidence for TCE-induced liver cancer and malignant lymphoma. Lifetime exposure to TCE resulted in increased liver cancer in mice and increased kidney cancer and testicular cancer in rats. The DHHS considers TCE to be a known human carcinogen. The IARC classified TCE as carcinogenic to humans. The EPA has characterized TCE as carcinogenic to humans by all routes of exposure [ATSDR 2019b].

### Calculating Cancer Risk: Drinking Water Ingestion

Cancer exposure doses were calculated using the following formula:

$$\textit{Cancer Exposure Dose (mg/kg-day)} = \frac{C \times IR}{BW} \times \frac{ED}{AT}$$

where, C = exposure point concentration in mg/L
IR = ingestion rate in liters per day (L/day)
BW = age specific body weight for children, 80 kg for adults
ED = exposure duration in years (varies with exposure scenario)
AT= averaging time = 78 years (approximate lifetime expectancy)

The site-specific assumptions and recommended exposure factors used to calculate the lifetime excess cancer risk for children and adults are the same as those used to assess noncancer health effects. Using the ATSDR water exposure dose guidance, the LECR was calculated by multiplying the cancer exposure dose by EPA's oral cancer slope factor (CSF).

The CSF is a numerical value to estimate the risk of cancer associated with exposure to carcinogenic substances. It is defined as the slope of the dose-response curve obtained from animal or human cancer studies and represents the upper-bound estimate of the probability of developing cancer over a lifetime from exposure to a specific chemical at a given dose for a specified duration. The CSF is expressed as the inverse of the daily exposure dose, i.e., $(mg/kg/day)^{-1}$, which means that as the daily dose increases, so will the cancer risk estimate. The LECRs for drinking water exposures were calculated using the following formula [ATSDR 2005]:

$$LECR_{drinking\ water} = \textit{Cancer Exposure Dose from Drinking Water x oral CSF}$$

where,
LECR = Lifetime Excess Cancer Risk
*CSF = Cancer Slope Factor* $(mg/kg\text{-}day)^{-1}$

Table 15a shows the calculated LECRs for 1,4-dioxane in drinking water. As shown in Table 15a, the LECR for children is less than one in one million individuals exposed ($9 \times 10^{-7}$). The LECR for adults is approximately one in one million people. NJDOH and ATSDR considered the factors described above in deciding whether these calculated theoretical cancer risks represent a health concern for people exposed to 1,4-dioxane in drinking water. NJDOH and ATSDR determined that both LECRs represent no concern for increased cancer risk due to the LECR being at or below one in a million or $1 \times 10^{-6}$.

**Table 15a. Above-Average Ingestion Rates (RME Scenario) – Lifetime Excess Cancer Risk for 1,4-Dioxane in Drinking Water**

| Exposed Population | EPC (mg/L) | Exposure Duration (years) | CSF $(mg/kg/day)^{-1}$ | LECR |
|---|---|---|---|---|
| Child | 0.00074 | 21 | 0.1 | $9 \times 10^{-7}$ |
| Adult | 0.00074 | 33 | 0.1 | $1 \times 10^{-6}$ |

Abbreviations: EPC = exposure point concentration represents the maximum concentration of post-treatment drinking water samples; CSF = cancer slope factor; LECR = lifetime excess cancer risk; mg/L = milligrams of contaminant per liter of water; mg/kg/day = milligrams of contaminant per kilogram of body weight per day.

The cancer risk evaluation from exposures to 1,4-dioxane vapors in air while showering and bathing are presented in Table 15b. As shown in the table, the LECR is less than one extra cancer case in one million (<1 x $10^{-6}$) similarly exposed people. This indicates no concern for an increased theoretical cancer risk.

**Table 15b. Cancer Risk (LECR) from 1,4-dioxane vapors while showering or bathing***

| Exposure Pathway | 1,4-dioxane predicted air concentration (µg/m$^3$) | Exposure duration (years) | Average lifetime (years) | LECR |
|---|---|---|---|---|
| Inhalation of air while showering or bathing ** | 0.016 | 33 | 78 | 3 x $10^{-8}$ |

Abbreviations: µg/m$^3$ = micrograms of 1,4-dioxane per cubic meter of air; mg/kg/day = milligrams of contaminant per kilogram per day

*Example LECR calculation = 1,4-dioxane concentration of 0.016 µg/m$^3$ x 33 years/78-year lifetime = LECR of 3 x$10^{-8}$

** Represents age category of birth to < 21 years plus 12 years during adulthood.

Table 16 shows the calculated LECRs for exposures to PFOA in drinking water. The LECR for children and adults exceeds one in 1,000 exposed individuals. When estimated cancer risks for PFOA exceed 1 in 1,000, ATSDR does not report the precise estimate because of uncertainty in the estimated cancer risk. Both adult and children LECRs for PFOA, represent an increased theoretical cancer risk and thus a concern for increased cancer risk in both groups.

**Table 16. Above-Average Ingestion Rates (RME Scenario) – Lifetime Excess Cancer Risk for PFOA in Drinking Water**

| Exposed Population | EPC* (mg/L) | Exposure Duration (years) | CSF (ng/kg/day) $^{-1}$ | LECR |
|---|---|---|---|---|
| Child | 9.9 x $10^{-6}$ | 21 | 0.0293 | >1 x $10^{-3}$ |
| Adult | 9.9 x $10^{-6}$ | 33 | 0.0293 | >1 x $10^{-3}$ |

Abbreviations: CSF = cancer slope factor; LECR = lifetime excess cancer risk; mg/L = milligrams of contaminant per liter of water; ng/kg/day = nanograms of contaminant per kilogram body weight per day.

* EPC = exposure point concentration and represents the maximum concentration of post-treatment drinking water samples

**It should be noted that meeting the currently established EPA MCL for PFOA (4 x $10^{-6}$ mg/L or 4 parts per trillion) would also result in LECRs in the same range as shown above in Table 16.**

Table 17 shows the calculated LECRs for exposures to PFOS in drinking water. The LECR for children is approximately three in 1,000,000 people (3 x $10^{-6}$). The LECR for adults is approximately four in 1,000,000 people (4 x $10^{-6}$). NJDOH and ATSDR considered the factors described above in deciding whether these calculated theoretical cancer risks present a health concern for people exposed to PFOS in drinking water. These factors include site specific factors in addition to the default factors used in the risk assessment model, including the length of exposure, sensitive populations who may already have an elevated risk due to exposure to other carcinogens, and exposure to mutagenic carcinogens at a young age. NJDOH and ATSDR

determined that both LECRs for PFOS represent no concern for an increased theoretical cancer risk.

**Table 17. Above-Average Ingestion Rates (RME Scenario) – Lifetime Excess Cancer Risk for PFOS in Drinking Water**

| Exposed Population | EPC* (mg/L) | Exposure Duration (years) | CSF (mg/kg/day) $^{-1}$ | LECR |
|---|---|---|---|---|
| Child | $6.5 \times 10^{-6}$ | 21 | 39.5 | $3 \times 10^{-6}$ |
| Adult | $6.5 \times 10^{-6}$ | 33 | 39.5 | $4 \times 10^{-6}$ |

Abbreviations: CSF = cancer slope factor; LECR = lifetime excess cancer risk; mg/L = milligrams of contaminant per liter of water; mg/kg/day = milligrams of contaminant per kilogram body weight per day.
* EPC = exposure point concentration and represents the maximum concentration of post-treatment samples

### Calculating Cancer Risk: Wading in Little Diamond Brook

Tables 18 and 19 summarize the LECRs for children and adults wading in Little Diamond Brook. The LECRs associated with 1,4-dioxane, carbon tetrachloride, and PCE are less than one excess cancer case in one million exposed individuals.

NJDOH and ATSDR considered the factors described earlier in deciding whether these calculated theoretical cancer risks represent a health concern for people exposed to 1,4-dioxane, carbon tetrachloride, and PCE in Little Diamond Brook. These factors include site-specific factors in addition to the default factors used in the risk assessment model, including the length of exposure, sensitive populations who may already have an elevated risk due to exposure to other carcinogens, and exposure to mutagenic carcinogens at a young age. NJDOH and ATSDR determined that the LECRs for children and adults represent no concern for an increased theoretical cancer risk.

**Table 18. LECR for Children Wading in Little Diamond Brook**

| Contaminant | EPC (mg/L) | Exposure Duration (years) | CSF (mg/kg/day) $^{-1}$ | LECR |
|---|---|---|---|---|
| 1,4-dioxane | 0.00102 | 15 | 0.1 | $7 \times 10^{-11}$ |
| Carbon Tetrachloride | 0.00144 | 15 | 0.07 | $5 \times 10^{-9}$ |
| PCE | 0.00211 | 15 | 0.0021 | $5 \times 10^{-10}$ |
| **Total LECR** | ---------- | ---------- | ---------- | **$6 \times 10^{-9}$** |

Abbreviations: EPC = exposure point concentration representing the 95% UCL of the mean; mg/L = milligrams of contaminant per liter of water; CSF = cancer slope factor; mg/kg/day = milligrams of contaminant per kilogram of body weight per day; LECR = lifetime excess cancer risk rounded to one significant figure; PCE = tetrachloroethylene

**Table 19. LECR for Adults Wading in Little Diamond Brook**

| Contaminant | EPC (mg/L) | Exposure Duration (years) | CSF (mg/kg/day) $^{-1}$ | LECR |
|---|---|---|---|---|
| 1,4-dioxane | 0.00102 | 33 | 0.1 | $1 \times 10^{-10}$ |
| Carbon Tetrachloride | 0.00144 | 33 | 0.07 | $1 \times 10^{-8}$ |
| PCE | 0.00211 | 33 | 0.0021 | $1 \times 10^{-9}$ |

| | | | | |
|---|---|---|---|---|
| **Total LECR** | **----------** | **----------** | **----------** | **1 x $10^{-8}$** |

Abbreviations: EPC = exposure point concentration representing the 95% UCL of the mean; mg/L = milligrams of contaminant per liter of water; CSF = cancer slope factor; mg/kg/day = milligrams of contaminant per kilogram of body weight per day; LECR = lifetime excess cancer risk rounded to one significant figure; PCE = tetrachloroethylene

The cancer risk equations and results for drinking water and surface water exposures can be found in **Appendix D (PHAST Results**).

**Calculating Cancer Risk: Vapor Intrusion (Indoor Air Impacts)**

The LECR for indoor air exposures is calculated for residential and occupational exposures by multiplying the air concentration and exposure duration by the EPA's inhalation unit risk (IUR) for cancer. The IUR is the incremental risk posed by a specific concentration unit in air (usually expressed as micrograms of a pollutant in air per cubic meter ($\mu g/m^3$) of ambient air. This LECR calculation yields the relative increase in cancer risk (above the background rate) from indoor air exposure to pollutants.

The LECRs for each residence and commercial property were calculated using ATSDR's guidance for inhalation exposures [ATSDR 2020b]. Using the most conservative scenario, per ATSDR's guidance, LECRs were calculated using the same exposure parameters used for noncancer health effects and includes an exposure duration of 21 years for children and 33 years for adults over a 78-year average lifetime. The exposure duration for workers was assumed to be 20 years based on ATSDR's PHAST. **Appendix D – PHAST Results** shows an example of the exposure parameters and calculated LECRs for adults and children in residential areas and for workers in commercial properties.

**Lifetime Excess Cancer Risks – Residential Properties**

**Tables B31 through B38** in **Appendix B** show the calculated LECRs for the eight residential properties where indoor air samples were collected. The LECRs for each contaminant at a residence were added to obtain a total LECR for that residence. As shown in the tables, the total LECRs for each residence ranged from less than one extra cancer case in 1,000,000 ($<1 \times 10^{-6}$) similarly exposed people to nine extra cancer cases in 100,000 ($9 \times 10^{-5}$) similarly exposed people. NJDOH and ATSDR considered the factors described previously in deciding whether these calculated theoretical cancer risks represent a health concern. NJDOH and ATSDR determined that these LECRs represent no concern for an increased theoretical cancer risk due to exposure to indoor air contaminants.

**Lifetime Excess Cancer Risks – Commercial Properties**

**Tables B39 through B44** in **Appendix B** show the calculated LECRs for the six commercial properties where indoor air samples were collected. The LECRs for contaminants at each commercial property were added to obtain a total LECR for that property. The total LECRs for the commercial properties ranged from less than one extra cancer case in one million ($<1 \times 10^{-6}$) similarly exposed individuals to two extra cancer cases in 100,000 ($2 \times 10^{-5}$) similarly exposed individuals. NJDOH and ATSDR considered the factors described previously in

deciding whether these calculated theoretical cancer risks represent a health concern. NJDOH and ATSDR determined that these LECRs represent no concern for an increased theoretical cancer risk from inhalation of air contaminants in indoor air.

# Conclusions

Based on the information available and analysis present in this document, NJDOH and ATSDR conclude the following:

## Drinking Water

*PFAS*

1. Past, current and future exposures to PFAS (PFOA and PFOS) in drinking water delivered by the Fair Lawn blended municipal water system (combination of well fields in Fair Lawn and purchased water with two water utilities) may harm people's health.

    - Based on the detected PFOA water concentration, doses for all age groups with average and above-average tap water ingestion rates have exposures that exceed doses reported to be associated with reduced antibody response and elevated cholesterol levels in the blood of humans. Another potential health effect is decreased birth weight from *in utero* exposures in the first and second trimesters. Mild liver damage might also be possible in residents with above-average tap water ingestion rates.
    - Based on the detected PFOS water concentration, doses in children birth to < 2 years with above-average tap water ingestion rates have exposures that approach doses reported to be associated with reduced antibody response, increased cholesterol levels, and mild liver damage. Doses in older children, teenagers, and adults are below levels thought to cause harmful effects.
    - The reported health effects are very similar for the two PFAS (PFOA and PFOS), which target the cardiovascular, immune, developmental, and hepatic systems. PFOA and PFOS were present in drinking water. All age groups with average and above-average tap water ingestion rates exceed doses thought to cause elevated cholesterol levels, reduced antibody response, decreased infant birth weight, and elevated liver enzymes in adults.
    - The lifetime excess cancer risk for children and adults with above-average water ingestion rates represents an increased theoretical cancer risk from exposures to PFOA in drinking water from the Fair Lawn municipal water system. There is no concern for an increased theoretical cancer risk for people exposed to PFOS in drinking water.

ATSDR and the National Institute of Environmental Health Sciences funded the National Academies of Science, Engineering, and Medicine (NASEM) to provide an objective review of the current evidence regarding human health effects of PFAS. Based on their review of epidemiological studies that have evaluated health impacts from population-level PFAS exposures, the NASEM committee found strong or moderate evidence for a connection between PFAS exposure and a number of health conditions or biomarkers. However, because of

uncertainty about how PFAS exposure translates to individual health risk, the NASEM committee recommended providers offer and discuss the risks and benefits of certain medical screening practices with patients exposed to PFAS, through a process of shared decision making.

PFAS have been widely used in many consumer products since the 1940s, including but not limited to: nonstick cookware; food packaging; stain-repellant and water-repellant treatments for clothing, carpets, and furniture; and certain firefighting foams (e.g., aqueous film forming foam).

- Most people have been exposed to some PFAS, but people who may have the highest levels of exposure include those who
    - Drink contaminated water, beverages made with contaminated water (e.g., baby formula, coffee), or food prepared with contaminated drinking water (boiling water does not remove PFAS)
    - Eat food grown or raised in PFAS-contaminated areas (e.g., produce, fish, wild game)
    - Eat food packaged in materials containing PFAS (e.g., microwave popcorn bags, fast food containers, pizza boxes)[1]
    - Put hands and objects in their mouth that contain PFAS (e.g., certain toys, household dust, treated fabrics) or that have been contaminated with PFAS
    - Work in industries that manufacture, handle, or use PFAS
    - Live in a community near industrial or waste-related sources of PFAS

*1,4-Dioxane*

2. Exposures to 1,4-dioxane through ingestion of or showering and bathing in water delivered by the Fair Lawn municipal water system is not likely to harm people's health. This is because calculated exposure doses and air vapor concentrations for 1,4-dioxane were below the ATSDR health guideline for noncancer health effects. Additionally, there is no concern for an increased theoretical cancer risk.

**Vapor Intrusion (Indoor Air Impacts)**

3. Past exposures to TCE in the Columbia Savings Bank may harm people's health.

[1] On January 3, 2025 the U.S. Food and Drug Administration (FDA) issued a notice in the Federal Register announcing its determination that 35 food contact notifications related to per- and polyfluoroalkyl substances (PFAS) are no longer effective because the manufacturers or suppliers have ceased production, supply, or use of the food contact substances. The 35 food contact notifications had previously authorized food contact substances used for grease-proofing coatings applied to paper and paperboard packaging to prevent leaking of oil and water.

In July 2020, manufacturers or suppliers of the food contact substances voluntarily agreed to phase out their sales of the grease-proofing substances that contained PFAS. In February 2024, the FDA announced that all grease-proofing substances containing PFAS are no longer being sold by manufacturers for food contact use in the U.S. market.

- Based on the March 2011 sample results, an increased risk for kidney effects from chronic exposures to TCE might exist for longtime workers at the Columbia Savings Bank. Estimated exposures exceed health guidelines and approach levels that might harm the kidneys for longtime workers who worked at the bank before 2011. Some uncertainty exists in this conclusion because we don't know TCE concentrations in air prior to March 2011. Because of uncertainty in the science, there's uncertainty in whether TCE exposure in pregnant women before 2011 might result in fetal heart malformation in newborns.
- Based on a subsequent sampling event in January 2012 for Columbia Savings Bank, health effects would not be expected in workers or visitors because the TCE levels have decreased compared to the March 2011 sampling event. Estimated exposures are below health guidelines so there is no concern for noncancer effects after January 2012.
- Based on NJDOH's discussions with EPA, EPA's evaluation of soil gas data showed that elevated TCE levels in the bank were caused by background sources within the building and not vapor intrusion from groundwater beneath the bank.

4. Except for Columbia Savings Bank, exposures to indoor air contaminants in the residential and commercial properties are not likely to harm people's health.

   - Harmful noncancer health effects are not expected from exposures to indoor air contaminants in the eight residential properties where indoor air data were collected. Harmful noncancer health effects are not expected for workers in the Sandvik, Hampshire, Kushner, Mikia, or Biomet buildings.
   - The levels of detected contaminants were below health guideline values or below levels where harmful health effects were observed in toxicological studies. Indoor air contaminants in the eight homes were from background sources within the homes and not vapor intrusion from groundwater beneath the structures. There is also no concern for an increased theoretical cancer risk from exposures to indoor air contaminants for both residential and commercial properties.

**Surface Water: Little Diamond Brook**

5. Harmful health effects are not expected for children and adults wading in Little Diamond Brook. This is because calculated exposure doses were below health guideline values for noncancer health effects. Additionally, there is no concern for an increased theoretical cancer risk.

## Recommendations

**PFAS in Drinking Water**

1. It is recommended that community members refer to NJDOH's per- and polyfluoroalkyl substances (PFAS) fact sheet that provides up-to-date information on potential health effects as the science evolves. The fact sheet includes resources for health care providers

and other information for communities with concerns about PFAS in drinking water (nj.gov/health/ceohs/documents/pfas_drinking%20water.pdf). ATSDR has funded a multi-site study to learn more about the relationship between PFAS exposure and health outcomes across seven U.S. communities exposed to PFAS-contaminated drinking water. Preliminary results are available at: Multi-site study of communities with PFAS-contaminated drinking water: Methods, demographics, and PFAS serum concentrations - ScienceDirect.

2. Specific health concerns regarding PFAS exposures may be discussed with a health care provider. ATSDR has developed PFAS information for clinicians that can be shared with health care providers to determine the best path forward based on an individual's unique circumstances (atsdr.cdc.gov/pfas/hcp/clinical-overview/index.html).

3. Residents may consider using bottled water for drinking and cooking to reduce exposures to PFAS. Bottled water sold in New Jersey is required to meet the New Jersey Maximum Contaminant Levels (MCLs).

4. Residents may consider using home water treatment devices that can reduce levels of PFAS; however, they are not guaranteed to achieve PFAS concentrations lower than the New Jersey's MCLs. The National Sanitation Foundation (NSF International), an independent and accredited organization, certifies products to have PFOA and PFOS concentrations below 20 parts per trillion (PFAS in Drinking Water NSF).
    a. When purchasing a filter, verify that the product is NSF/ANSI Standard 53 and check for the standard version to know at which level the filter is certified to remove PFAS (e.g. PFOA, PFOS, PFNA, PFHxS, PFHpA).

    b. For more specific information regarding the effectiveness of home water filters for reducing PFAS, visit the NSF International website, PFAS in Drinking Water NSF.

**Vapor Intrusion (Indoor Air Impacts)**

1. NJDEP and EPA continue to ensure that vapor mitigation systems at Sandvik, Hampshire, and Mikia continue to operate effectively to prevent sub-surface vapors from entering the buildings. Based on the information provided to NJDOH, vapor intrusion was not occurring in the remaining commercial and residential properties evaluated in this document and therefore, no further actions are recommended for these properties.

## Public Health Action Plan

The purpose of a public health action plan is to ensure that this provides a plan of action designed to reduce and prevent adverse human health effects resulting from exposure to hazardous substances in the environment. Included in this public health assessment is a commitment from NJDOH to follow up on this plan to ensure that it is implemented.

## Public Health Actions Taken

NJDOH has taken the following actions:

1. Reviewed information provided by EPA to evaluate the potential health implications of exposures to contaminants from the Fair Lawn Well Field site.
2. Hosted two public availability sessions in Fair Lawn in August 2023 to gather information about community health concerns.

## Public Health Actions Planned

NJDOH plans to take the following actions:

1. Assist community members in understanding the findings of this report upon request.
2. Present the findings of this evaluation to the community.
3. Any public comments received will be addressed in the Final version.

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# Report Preparation

NJDOH prepared this public health assessment for the Fair Lawn Well Field NPL site located in Fair Lawn, Bergen County, NJ. This publication was made possible by a cooperative agreement [program #CDC-RFA-TS-23-0001] with ATSDR. NJDOH evaluated data of known quality using approved methods, policies, and procedures existing at the date of publication. ATSDR reviewed this document and concurs with its findings based on the information presented by NJDOH.

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# APPENDIX A – Figures

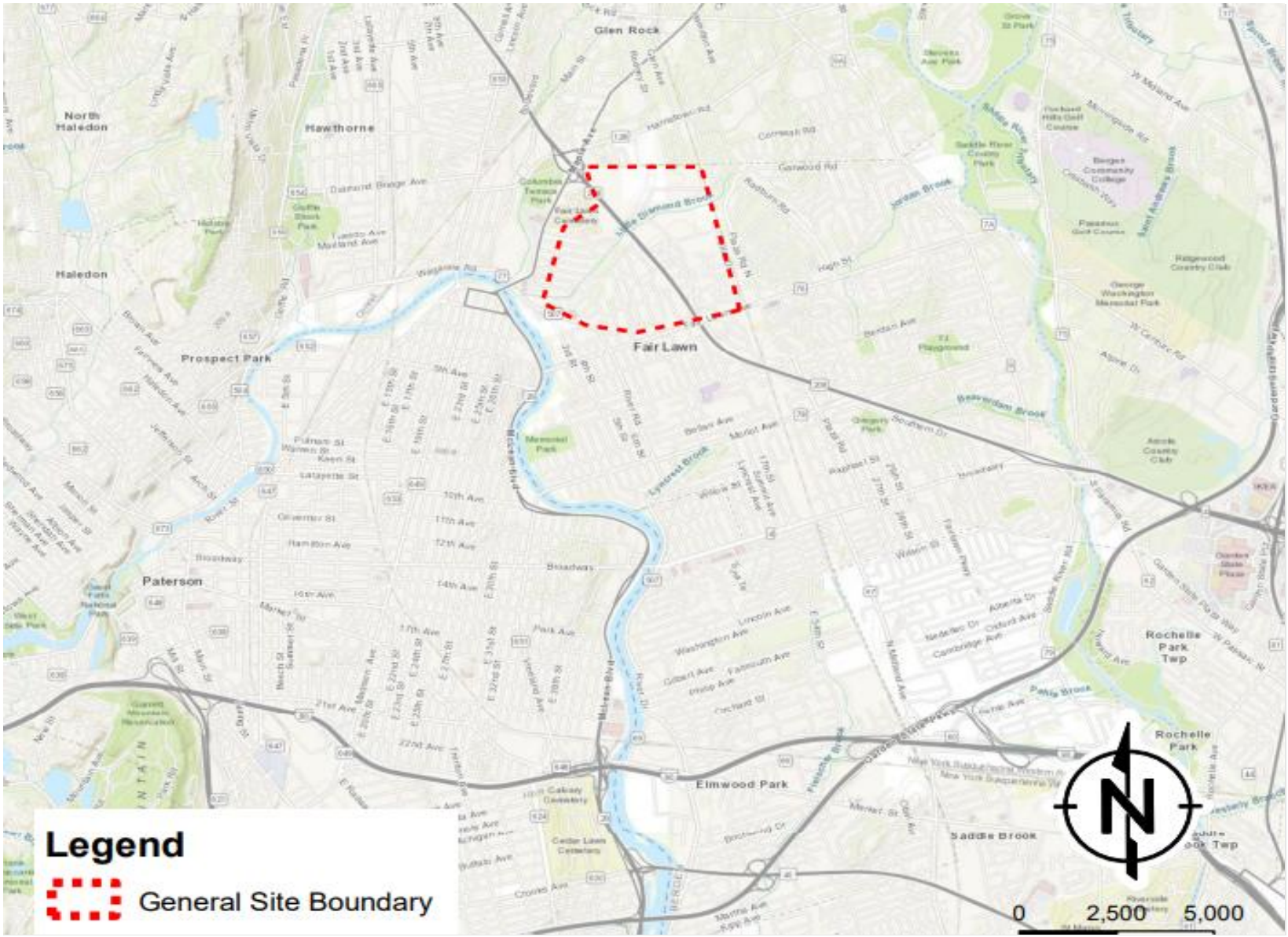



**Figure 1. Fair Lawn Well Field Site Location Map (source – Final Remedial Investigation Report - Fair Lawn Well Field Superfund Site)**

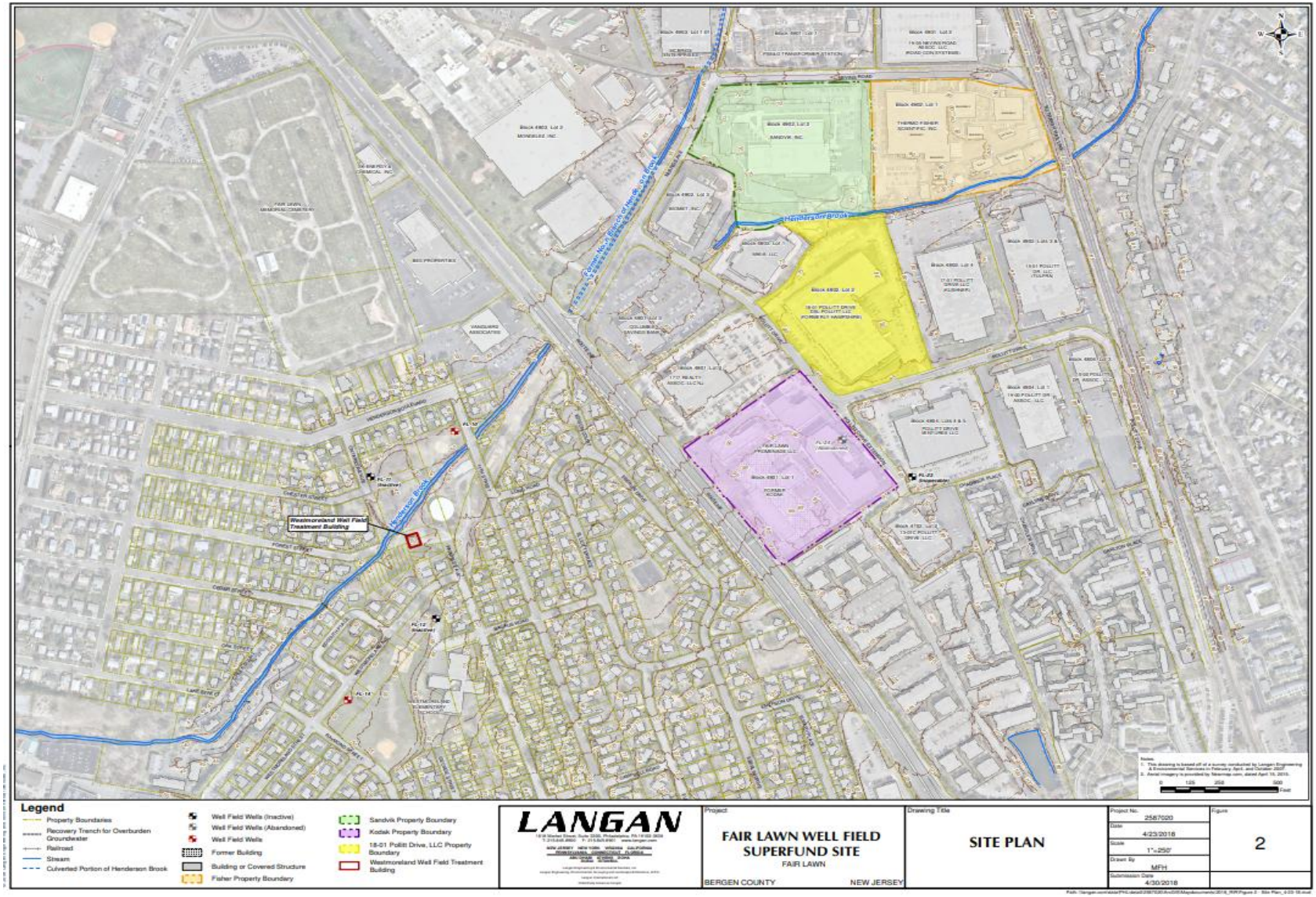



**Figure 2. Fair Lawn Well Field Site Area Map (source – Final Remedial Investigation Report - Fair Lawn Well Field Superfund Site)**

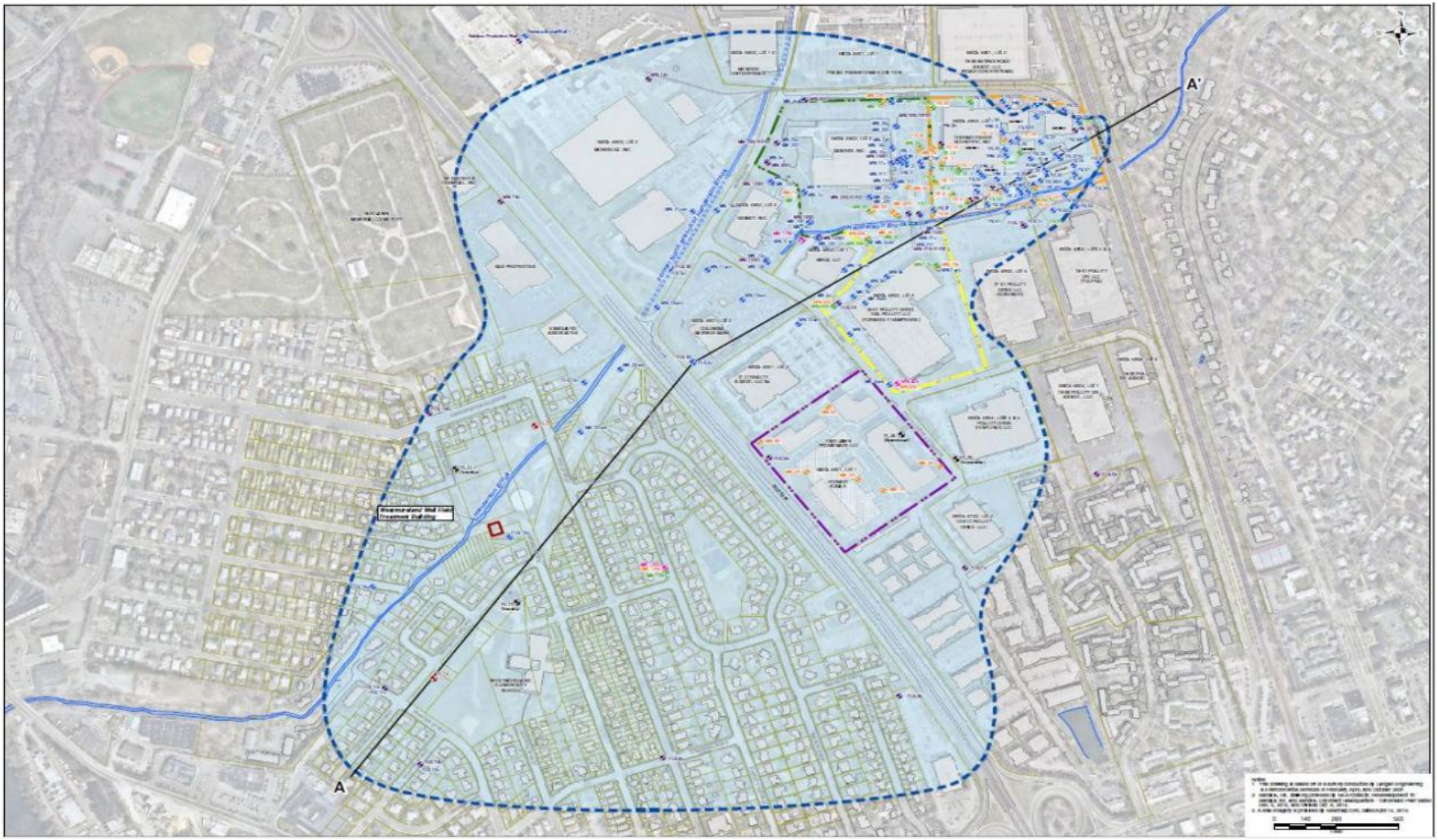



**Figure 3. Fair Lawn Well Field Groundwater Plume Map (source – Final Remedial Investigation Report - Fair Lawn Well Field Superfund Site)**

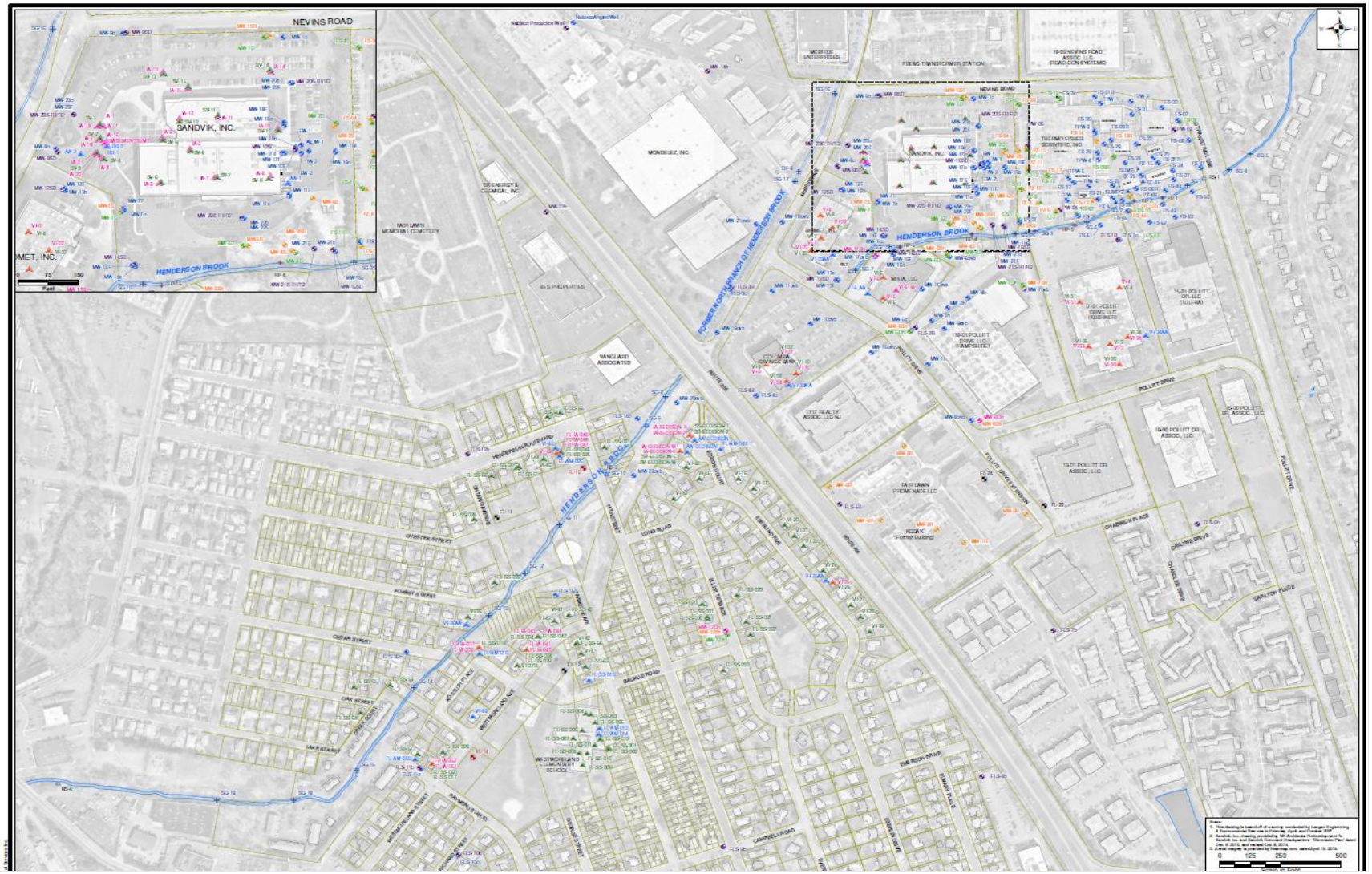



**Figure 4. Fair Lawn Well Field Sample Location Map (source – Final Remedial Investigation Report - Fair Lawn Well Field Superfund Site)**

# APPENDIX B – Tables

**Table B1. Residential Indoor Air Data – Sample Dates: February 2010 – May 2015**

| Contaminant | Number of Samples | Number of Detections | Minimum (µg/m$^3$) | Maximum (µg/m$^3$) | Comparison Value (CV) (µg/m$^3$) | Maximum Exceed CV |
|---|---|---|---|---|---|---|
| 1,1,1-trichloroethane | 19 | 2 | ND | 2.0 | 3,800 (Intermediate EMEG) | No |
| 1,2-dichloroethane | 19 | 5 | ND | 2.3 | 0.038 (CREG) | **Yes** |
| 1,2,4-trimethylbenzene | 19 | 13 | ND | 6.4 | 60 (RMEG) | No |
| 1,3,5-trimethylbenzene | 19 | 11 | ND | 1.7 | 60 (RMEG) | No |
| 1,4-dichlorobenzene | 19 | 8 | ND | 6.1 | 10 (EMEG) | No |
| 2-hexanone | 19 | 7 | ND | 0.4 | 30 (RMEG) | No |
| Acetone | 19 | 19 | 14.0 | 110.0 | 19,000 (acute EMEG) | No |
| Benzene | 19 | 16 | ND | 10.0 | 0.13 (CREG) | **Yes** |
| Bromodichloromethane | 19 | 1 | ND | 2.0 | 0.076 (RSL) | **Yes** |
| Bromoform | 19 | 1 | ND | 0.4 | 0.91 (CREG) | No |
| Carbon Tetrachloride | 19 | 11 | ND | 0.4 | 0.17 (CREG) | **Yes** |
| Chloroform | 19 | 15 | ND | 5.0 | 0.043 (CREG) | **Yes** |
| Chloromethane | 19 | 17 | ND | 5.1 | 62 (EMEG) | No |
| Cyclohexane | 19 | 13 | ND | 2.0 | 6,000 (RMEG) | No |
| Dichlorodifluoromethane | 19 | 18 | ND | 3.0 | 100 (RSL) | No |
| Ethyl Acetate | 19 | 14 | ND | 38.5 | 73 (RSL) | No |
| Ethylbenzene | 19 | 16 | ND | 8.2 | 1.1 (RSL) | **Yes** |
| 2-butanone | 19 | 19 | ND | 47.0 | 5,000 (RMEG) | No |
| Methyl Isobutyl Ketone | 19 | 9 | ND | 1.6 | 3,000 (RMEG) | No |
| Methyl methacrylate | 19 | 2 | ND | 4.0 | 700 (RMEG) | No |
| Methylene Chloride | 19 | 18 | ND | 53.5 | 63 (CREG) | No |
| n-heptane | 19 | 16 | ND | 15.4 | 420 (RSL) | No |
| n-hexane | 19 | 19 | 1.0 | 19.0 | 700 (RMEG) | No |
| Propylene | 19 | 11 | ND | 28.0 | 3100 (RSL) | No |
| Styrene | 19 | 12 | ND | 1.4 | 850 (EMEG) | No |
| PCE | 19 | 18 | ND | 4.3 | 3.8 (CREG) | **Yes** |
| Tetrahydrofuran | 19 | 15 | ND | 5.0 | 2,000 (RMEG) | No |
| Toluene | 19 | 19 | 1.7 | 48.9 | 3,800 (EMEG) | No |
| Total Xylenes | 19 | 18 | ND | 33.4 | 100 (RMEG) | No |
| TCE | 19 | 3 | ND | 0.3 | 0.040 (CREG) | **Yes** |
| Vinyl Acetate | 19 | 11 | ND | 10.5 | 200 (RMEG) | No |

Abbreviations: µg/m$^3$ = micrograms of contaminant per cubic meter of air; CREG = ATSDR cancer risk evaluation guide; EMEG = ATSDR environmental media evaluation guide; RMEG = ATSDR reference media evaluation guide; RSL = EPA regional screening level; PCE = tetrachloroethylene; TCE = trichloroethylene; ND = contaminant not detected; Data represent eight residential properties where indoor air data was collected based on soil gas results; comparison values represent chronic exposures unless noted otherwise.

**Table B2. Residence R11 – Potential Contaminants of Concern – Indoor Air**

| Contaminant | Number of samples | Number of detections | Minimum (µg/m$^3$) | Maximum (µg/m$^3$) | Comparison value (CV) µg/m$^3$ | Maximum Exceed CV |
|---|---|---|---|---|---|---|
| 1,2-dichloroethane | 2 | 2 | 2 | 2 | 0.038 (CREG) | **Yes** |
| Benzene | 2 | 2 | 1 | 1 | 0.13 (CREG) | **Yes** |
| Chloroform | 2 | 2 | 1 | 1 | 0.043 (CREG) | **Yes** |
| Ethylbenzene | 2 | 2 | 1 | 2 | 1.1 (RSL) | **Yes** |
| PCE | 2 | 2 | 2 | 3 | 3.8 (CREG) | No |

Abbreviations: µg/m$^3$ = micrograms of contaminant per cubic meter of air; CREG = ATSDR cancer risk evaluation guide; RSL = EPA regional screening level for residential exposures; PCE = tetrachloroethylene

**Table B3. Residence R18 – Potential Contaminants of Concern – Indoor Air**

| Contaminant | Number of samples | Number of detections | Minimum (µg/m$^3$) | Maximum (µg/m$^3$) | Comparison value (CV) µg/m$^3$ | Maximum Exceed CV |
|---|---|---|---|---|---|---|
| PCE | 2 | 2 | 0.33 | 0.88 | 3.8 (CREG) | No |
| TCE | 2 | 1 | Not Detected | 0.24 | 0.040 (CREG) | **Yes** |

Abbreviations: µg/m$^3$ = micrograms of contaminant per cubic meter of air; CREG = ATSDR cancer risk evaluation guide; PCE = tetrachloroethylene; TCE = trichloroethylene

**Table B4. Residence R19 – Potential Contaminants of Concern – Indoor Air**

| Contaminant | Number of samples | Number of detections | Minimum (µg/m$^3$) | Maximum (µg/m$^3$) | Comparison value (CV) µg/m$^3$ | Maximum Exceed CV |
|---|---|---|---|---|---|---|
| Benzene | 2 | 2 | 2.4 | 4.5 | 0.13 (CREG) | **Yes** |
| Ethylbenzene | 2 | 2 | 1.5 | 2.7 | 1.1 (RSL) | **Yes** |
| PCE | 2 | 2 | 2.5 | 4.3 | 3.8 (CREG) | **Yes** |
| TCE | 2 | 1 | Not Detected | 0.3 | 0.040 (CREG) | **Yes** |

Abbreviations: µg/m$^3$ = micrograms of contaminant per cubic meter of air; CREG = ATSDR cancer risk evaluation guide; RSL = EPA regional screening level for residential exposures; PCE = tetrachloroethylene; TCE = trichloroethylene

**Table B5. Residence R31 – Potential Contaminants of Concern – Indoor Air**

| Contaminant | Number of samples | Number of detections | Minimum (µg/m$^3$) | Maximum (µg/m$^3$) | Comparison value (CV) µg/m$^3$ | Maximum Exceed CV |
|---|---|---|---|---|---|---|
| 1,2 dichloroethane | 5 | 1 | Not Detected | 0.191 | 0.038 (CREG) | **Yes** |
| Benzene | 5 | 4 | Not Detected | 1.45 | 0.13 (CREG) | **Yes** |
| Bromodichloromethane | 5 | 1 | Not Detected | 2 | 0.076 (RSL) | **Yes** |
| Carbon Tetrachloride | 5 | 3 | Not Detected | 0.391 | 0.17 (CREG) | **Yes** |
| Chloroform | 5 | 5 | 0.256 | 5 | 0.043 (CREG) | **Yes** |
| Ethylbenzene | 5 | 4 | Not Detected | 1 | 1.1 (RSL) | No |
| PCE | 5 | 4 | Not Detected | 1 | 3.8 (CREG) | No |

Abbreviations: µg/m$^3$ = micrograms of contaminant per cubic meter of air; CREG = ATSDR cancer risk evaluation guide; RSL = EPA regional screening level for residential exposures; PCE = tetrachloroethylene.

**Table B6. Residence R32 – Potential Contaminants of Concern – Indoor Air**

| Contaminant | Number of samples | Number of detections | Minimum (µg/m$^3$) | Maximum (µg/m$^3$) | Comparison value (CV) µg/m$^3$ | Maximum Exceed CV |
|---|---|---|---|---|---|---|
| Benzene | 2 | 2 | 1.32 | 1.34 | 0.13 (CREG) | **Yes** |
| Carbon Tetrachloride | 2 | 2 | 0.347 | 0.348 | 0.17 (CREG) | **Yes** |
| Chloroform | 2 | 2 | 0.24 | 0.325 | 0.043 (CREG) | **Yes** |
| Ethylbenzene | 2 | 2 | 0.717 | 0.797 | 1.1 (RSL) | No |
| PCE | 2 | 2 | 1.32 | 1.41 | 3.8 (CREG) | No |

Abbreviations: µg/m$^3$ = micrograms of contaminant per cubic meter of air; CREG = ATSDR cancer risk evaluation guide; RSL = EPA regional screening level for residential exposures; PCE = tetrachloroethylene

**Table B7. Residence R33 – Potential Contaminants of Concern – Indoor Air**

| Contaminant | Number of samples | Number of detections | Minimum (µg/m$^3$) | Maximum (µg/m$^3$) | Comparison value (CV) µg/m$^3$ | Maximum Exceed CV |
|---|---|---|---|---|---|---|
| Benzene | 2 | 2 | 1.48 | 1.48 | 0.13 (CREG) | **Yes** |
| Carbon Tetrachloride | 2 | 2 | 0.34 | 0.357 | 0.17 (CREG) | **Yes** |
| Chloroform | 2 | 2 | 0.533 | 0.549 | 0.043 (CREG) | **Yes** |
| Ethylbenzene | 2 | 2 | 0.751 | 0.752 | 1.1 (RSL) | No |
| PCE | 2 | 2 | 1.61 | 1.63 | 3.8 (CREG) | No |

Abbreviations: µg/m$^3$ = micrograms of contaminant per cubic meter of air; CREG = ATSDR cancer risk evaluation guide; RSL = EPA regional screening level for residential exposures; PCE = tetrachloroethylene

**Table B8. Residence R45 – Potential Contaminants of Concern – Indoor Air**

| Contaminant | Number of samples | Number of detections | Minimum (µg/m$^3$) | Maximum (µg/m$^3$) | Comparison value (CV) µg/m$^3$ | Maximum Exceed CV |
|---|---|---|---|---|---|---|
| Benzene | 2 | 2 | 6.49 | 10 | 0.13 (CREG) | **Yes** |
| Carbon Tetrachloride | 2 | 2 | 0.392 | 0.399 | 0.17 (CREG) | **Yes** |
| Chloroform | 2 | 2 | 0.57 | 1.37 | 0.043 (CREG) | **Yes** |
| Ethylbenzene | 2 | 2 | 5.49 | 8.15 | 1.1 (RSL) | **Yes** |
| PCE | 2 | 2 | 1.28 | 1.35 | 3.8 (CREG) | No |
| TCE | 2 | 1 | Not Detected | 0.197 | 0.040(CREG) | **Yes** |

Abbreviations: µg/m$^3$ = micrograms of contaminant per cubic meter of air; CREG = ATSDR cancer risk evaluation guide; RSL = EPA regional screening level for residential exposures; PCE = tetrachloroethylene; TCE = trichloroethylene

**Table B9. Residence R50 – Potential Contaminants of Concern – Indoor Air**

| Contaminant | Number of samples | Number of detections | Minimum (µg/m$^3$) | Maximum (µg/m$^3$) | Comparison value (CV) µg/m$^3$ | Maximum Exceed CV |
|---|---|---|---|---|---|---|
| 1,2 dichloroethane | 2 | 2 | 1.01 | 2.26 | 0.038 (CREG) | **Yes** |
| Benzene | 2 | 2 | 1.61 | 1.89 | 0.13 (CREG) | **Yes** |
| Carbon Tetrachloride | 2 | 2 | 0.386 | 0.391 | 0.17 (CREG) | **Yes** |
| Chloroform | 2 | 2 | 1.22 | 1.59 | 0.043 (CREG) | **Yes** |
| Ethylbenzene | 2 | 2 | 1.38 | 1.54 | 1.1 (RSL) | **Yes** |
| PCE | 2 | 2 | 3.76 | 4 | 3.8 (CREG) | **Yes** |

Abbreviations: µg/m$^3$ = micrograms of contaminant per cubic meter of air; CREG = ATSDR cancer risk evaluation guide; RSL = EPA regional screening level for residential exposures; PCE = tetrachloroethylene

**Table B10. Sandvik Inc. Indoor Air Data – Sample Dates: November 2006 – February 2010**

| Contaminant | Number of Samples | Number of Detections | Minimum (µg/m$^3$) | Maximum (µg/m$^3$) | Comparison Value (CV) (µg/m$^3$) | Maximum Exceed CV |
|---|---|---|---|---|---|---|
| 1,1,1-Trichloroethane | 66 | 2 | ND | 1.2 | 3,800 (Intermediate EMEG) | No |
| 1,1,2-Trichloro-1,2,2-Trifluoroethane | 66 | 32 | ND | 16 | 22,000 (RSL) | No |
| 1,1-Dichloroethene | 66 | 2 | ND | 1.6 | 4 (EMEG) | No |
| 1,2,4-Trimethylbenzene | 66 | 46 | ND | 4.9 | 60 (RMEG) | No |
| 1,3,5-Trimethylbenzene | 66 | 20 | ND | 1.8 | 60 (RMEG) | No |
| 1,4-Dichlorobenzene | 66 | 18 | ND | 6.6 | 10 (EMEG) | No |
| 2-Hexanone | 66 | 1 | ND | 1.8 | 30 (RMEG) | No |
| Acetone | 66 | 65 | ND | 58.9 | 19,000 (acute EMEG) | No |

| | | | | | | |
|---|---|---|---|---|---|---|
| Benzene | 66 | 65 | ND | 5.1 | 0.13 (CREG) | **Yes** |
| Carbon Disulfide | 66 | 6 | ND | 1.5 | 700 (RMEG) | No |
| Carbon Tetrachloride | 66 | 3 | ND | 1.4 | 0.17 (CREG) | **Yes** |
| Chloroform | 66 | 5 | ND | 1 | 0.043 (CREG) | **Yes** |
| Chloromethane | 66 | 63 | ND | 1.6 | 62 (EMEG) | No |
| Cyclohexane | 66 | 25 | ND | 25 | 6,000 (RMEG) | No |
| Dichlorodifluoromethane | 66 | 66 | 2.1 | 4.4 | 440 (RSL) | No |
| Ethyl Acetate | 66 | 58 | ND | 247 | 310 (RSL) | No |
| Ethylbenzene | 66 | 38 | ND | 2.6 | 4.9 (RSL) | No |
| 2-Butanone | 66 | 62 | ND | 12 | 5,000 (RMEG) | No |
| Methyl Isobutyl Ketone | 66 | 15 | ND | 1.6 | 3,000 (RMEG) | No |
| Methylene Chloride | 66 | 66 | 0.83 | 44.5 | 63 (CREG) | No |
| n-heptane | 66 | 39 | ND | 5.7 | 1,800 (RSL) | No |
| n-hexane | 66 | 63 | ND | 49.7 | 700 (RMEG) | No |
| Propylene | 66 | 2 | ND | 2.4 | 13,000 (RSL) | No |
| Styrene | 66 | 2 | ND | 1.5 | 850 (EMEG) | No |
| PCE | 66 | 57 | ND | 50 | 3.8 (CREG) | **Yes** |
| Tetrahydrofuran | 66 | 32 | ND | 15 | 2,000 (RMEG) | No |
| Toluene | 66 | 66 | 1.1 | 17 | 3,800 (EMEG) | No |
| Total Heptanes | 66 | 21 | ND | 3.9 | 1,800 (RSL) | No |
| Total Xylenes | 66 | 61 | ND | 9.6 | 100 (RMEG) | No |
| TCE | 66 | 25 | ND | 1.9 | 0.040 (CREG) | **Yes** |
| Vinyl Acetate | 66 | 1 | ND | 3 | 200 (RMEG) | No |

Abbreviations: µg/m$^3$ = micrograms of contaminant per cubic meter of air; CREG = ATSDR cancer risk evaluation guide; EMEG = ATSDR environmental media evaluation guide; RMEG = ATSDR reference media evaluation guide; RSL = EPA industrial indoor air regional screening level; PCE = tetrachloroethylene; TCE = trichloroethylene; ND = contaminant not detected; Comparison values represent chronic exposures unless noted otherwise.

**Table B11. Hampshire Property Indoor Air Data – Sample Date: November 2009**

| Contaminant | Number of Samples | Number of Detections | Minimum (µg/m$^3$) | Maximum (µg/m$^3$) | Comparison Value (CV) (µg/m$^3$) | Maximum Exceed CV |
|---|---|---|---|---|---|---|
| 1,2,4 trimethylbenzene | 8 | 1 | ND | 1.9 | 60 (RMEG) | No |
| Acetone | 8 | 8 | 5.7 | 59.4 | 19,000 (acute EMEG) | No |
| Benzene | 8 | 2 | ND | 0.8 | 0.13 (CREG) | **Yes** |
| Carbon disulfide | 8 | 1 | ND | 0.7 | 700 (RMEG) | No |
| Chloromethane | 8 | 8 | 0.6 | 1.1 | 62 (EMEG) | No |
| Dichlorodifluoromethane | 8 | 8 | 1.8 | 2.5 | 440 (RSL) | No |
| Ethyl Acetate | 8 | 2 | ND | 1.3 | 310 (EPA) | No |
| Heptane | 8 | 4 | ND | 2.3 | 1,800 (RSL) | No |
| Hexane | 8 | 2 | ND | 3.9 | 700 (RMEG) | No |
| 2·Hexanone | 8 | 1 | ND | 0.9 | 30 (RMEG) | No |
| Methylene Chloride | 8 | 8 | 1.8 | 4.9 | 63 (CREG) | No |
| 2-Butanone | 8 | 8 | 0.7 | 7.4 | 5,000 (RMEG) | No |
| Methyl Isobutyl Ketone | 8 | 1 | ND | 1.2 | 3,000 (RMEG) | No |
| Propylene | 8 | 1 | ND | 1.9 | 13,000 (RSL) | No |
| PCE | 8 | 7 | ND | 1.5 | 3.8 (CREG) | No |

| | | | | | | |
|---|---|---|---|---|---|---|
| Tetrahydrofuran | 8 | 3 | ND | 2.5 | 2,000 (RMEG) | No |
| Toluene | 8 | 8 | 1.5 | 15.0 | 3,800 (EMEG) | No |
| TCE | 8 | 2 | ND | 1.1 | 0.040 (CREG) | **Yes** |
| Xylenes (total) | 8 | 7 | ND | 3.7 | 100 (RMEG) | No |

Abbreviations: µg/m$^3$ = micrograms of contaminant per cubic meter of air; CREG = ATSDR cancer risk evaluation guide; EMEG = ATSDR environmental media evaluation guide; RMEG = ATSDR reference media evaluation guide; RSL = EPA industrial indoor air regional screening level; PCE = tetrachloroethylene; TCE = trichloroethylene; ND = contaminant not detected; Comparison values represent chronic exposures unless noted otherwise.

**Table B12. Kushner Property Indoor Air Data – Sample Dates: March 2010 and April 2011**

| Contaminant | Number of Samples | Number of Detections | Minimum (µg/m$^3$) | Maximum (µg/m$^3$) | Comparison Value (CV) (µg/m$^3$) | Maximum Exceed CV |
|---|---|---|---|---|---|---|
| 1,2,4-Trimethylbenzene | 10 | 3 | ND | 4 | 60 (RMEG) | No |
| Acetone | 10 | 3 | ND | 45 | 19,000 (acute EMEG) | No |
| Benzene | 10 | 10 | 0.9 | 80 | 0.13 (CREG) | **Yes** |
| Bromomethane | 10 | 6 | ND | 3 | 3.9 (EMEG) | No |
| Carbon Disulfide | 10 | 4 | ND | 29 | 700 (RMEG) | No |
| Chlorobenzene | 10 | 1 | ND | 1 | 220 (RSL) | No |
| Chloromethane | 10 | 8 | ND | 2 | 62 (EMEG) | No |
| Cyclohexane | 10 | 6 | ND | 120 | 6,000 (RMEG) | No |
| Dichlorodifluoromethane | 10 | 9 | ND | 3 | 440 (RSL) | No |
| Ethylbenzene | 10 | 4 | ND | 5 | 4.9 (RSL) | **Yes** |
| 2-Butanone | 10 | 10 | 2 | 86 | 5,000 (RMEG) | No |
| Methyl Isobutyl Ketone | 10 | 5 | ND | 78 | 3,000 (RMEG) | No |
| Methylene chloride | 10 | 10 | 3 | 11 | 63 (CREG) | No |
| N-heptane | 10 | 10 | 6 | 9000 | 1,800 (RSL) | **Yes *** |
| N-hexane | 10 | 10 | 1 | 76 | 700 (RMEG) | No |
| Styrene | 10 | 8 | ND | 77 | 850 (EMEG) | No |
| Toluene | 10 | 9 | ND | 1600 | 3,800 (EMEG) | No |
| Total Xylenes | 10 | 6 | ND | 20 | 100 (RMEG) | No |
| TCE | 10 | 4 | ND | 3 | 0.040 (CREG) | **Yes** |

Abbreviations: µg/m$^3$ = micrograms of contaminant per cubic meter of air; CREG = ATSDR cancer risk evaluation guide; EMEG = ATSDR environmental media evaluation guide; RMEG = ATSDR reference media evaluation guide; RSL = EPA industrial indoor air regional screening level; TCE = trichloroethylene; ND = contaminant not detected; Comparison values represent chronic exposures unless noted otherwise;* According to information provided by EPA, this contaminant is used by a tenant in the building as part of their business operations and is therefore regulated under the Occupational Safety and Health Administration (OSHA).

**Table B13. Mikia LLC. Property Indoor Air Data – Sample Date: March 2010**

| Contaminant | Number of Samples | Number of Detections | Minimum (µg/m$^3$) | Maximum (µg/m$^3$) | Comparison Value (CV) (µg/m$^3$) | Maximum Exceed CV |
|---|---|---|---|---|---|---|
| 1,1,1 trichloroethane | 2 | 2 | 3 | 5 | 3800 (Intermediate EMEG) | No |
| 1,2,4 trimethylbenzene | 2 | 2 | 1 | 5 | 60 (RMEG) | No |
| 1,3,5 trimethylbenzene | 2 | 1 | ND | 2 | 60 (RMEG) | No |
| Acetone | 2 | 2 | 12 | 19 | 19000 (acute EMEG) | No |
| Benzene | 2 | 2 | 1 | 3 | 0.13 (CREG) | **Yes** |

| | | | | | | |
|---|---|---|---|---|---|---|
| Chloromethane | 2 | 1 | ND | 1 | 62 (EMEG) | No |
| Cyclohexane | 2 | 2 | 0.9 | 2 | 6000 (RMEG) | No |
| Dichlorodifluoromethane | 2 | 1 | ND | 2 | 440 (RSL) | No |
| Ethylbenzene | 2 | 2 | 6 | 36 | 4.9 (RSL) | **Yes** |
| 2-Butanone | 2 | 2 | 10 | 47 | 5000 (RMEG) | No |
| Methyl Isobutyl Ketone | 2 | 2 | 7 | 49 | 3000 (RMEG) | No |
| Methylene Chloride | 2 | 2 | 2 | 2 | 63 (CREG) | No |
| N-heptane | 2 | 2 | 3 | 3 | 1800 (RSL) | No |
| N-hexane | 2 | 2 | 1 | 8 | 700 (RMEG) | No |
| Ter-Butyl Methyl Ether | 2 | 2 | 1 | 4 | 3000 (RMEG) | No |
| PCE | 2 | 2 | 9 | 53 | 3.8 (CREG) | **Yes** |
| Toluene | 2 | 2 | 6 | 23 | 3800 (EMEG) | No |
| Total Xylenes | 2 | 2 | 29 | 188 | 100 (RMEG) | **Yes** |

Abbreviations: µg/m$^3$ = micrograms of contaminant per cubic meter of air; CREG = ATSDR cancer risk evaluation guide; EMEG = ATSDR environmental media evaluation guide; RMEG = ATSDR reference media evaluation guide; RSL = EPA industrial indoor air regional screening level; PCE = tetrachloroethylene; ND = contaminant not detected; Comparison values represent chronic exposures unless noted otherwise.

**Table B14. Columbia Bank Indoor Air Data – Sample Dates: March 2011 and January 2012**

| Contaminant | Number of Samples | Number of Detections | Minimum (µg/m$^3$) | Maximum (µg/m$^3$) | Comparison Value (CV) (µg/m$^3$) | Maximum Exceed CV |
|---|---|---|---|---|---|---|
| 1,2,4-Trimethylbenzene | 10 | 3 | ND | 2.7 | 60 (RMEG) | No |
| 1,3,5-Trimethylbenzene | 10 | 1 | ND | 2 | 60 (RMEG) | No |
| Acetone | 10 | 9 | ND | 28 | 19,000 (acute EMEG) | No |
| Benzene | 10 | 10 | 0.7 | 2 | 0.13 (CREG) | **Yes** |
| Chloroform | 10 | 2 | ND | 1.7 | 0.043 (CREG) | **Yes** |
| Chloromethane | 10 | 10 | 0.85 | 1.2 | 62 (EMEG) | No |
| Cyclohexane | 10 | 5 | ND | 2 | 6,000 (RMEG) | No |
| Dichlorodifluoromethane | 10 | 10 | 2 | 3 | 440 (RSL) | No |
| Ethylbenzene | 10 | 2 | ND | 1.7 | 4.9 (RSL) | No |
| Methylene chloride | 10 | 6 | ND | 1.7 | 63 (CREG) | No |
| 2-Butanone | 10 | 8 | ND | 3 | 5,000 (RMEG) | No |
| n-heptane | 10 | 9 | ND | 5 | 1,800 (RSL) | No |
| n-hexane | 10 | 10 | 1.7 | 42 | 700 (RMEG) | No |
| PCE | 10 | 10 | 1 | 210 | 3.8 (CREG) | **Yes** |
| Toluene | 10 | 10 | 1.1 | 12 | 3,800 (EMEG) | No |
| Total Xylenes | 10 | 6 | ND | 5.4 | 100 (RMEG) | No |
| TCE | 10 | 10 | 0.28 | 73 | 0.040 (CREG) | **Yes** |

Abbreviations: µg/m$^3$ = micrograms of contaminant per cubic meter of air; CREG = ATSDR cancer risk evaluation guide; EMEG = ATSDR environmental media evaluation guide; RMEG = ATSDR reference media evaluation guide; RSL = EPA industrial indoor air regional screening level; PCE = tetrachloroethylene; TCE = trichloroethylene; ND = contaminant not detected; Comparison values represent chronic exposures unless noted otherwise.

**Table B15. Biomet Inc. Indoor Air Data – Sample Dates: February 2010 – February 2015**

| Contaminant | Number of Samples | Number of Detections | Minimum (µg/m$^3$) | Maximum (µg/m$^3$) | Comparison Value (CV) (µg/m$^3$) | Maximum Exceed CV |
|---|---|---|---|---|---|---|
| 1,1,1-Trichloroethane | 28 | 8 | ND | 22 | 3,800 (Intermediate EMEG) | No |
| 1,2,4-Trimethylbenzene | 28 | 14 | ND | 14 | 60 (RMEG) | No |
| 1,3,5-Trimethylbenzene | 28 | 7 | ND | 4 | 60 (RMEG) | No |
| 1,3-Butadiene | 28 | 2 | ND | 1 | 0.033 (CREG) | **Yes** |
| Acetone | 28 | 27 | ND | 66 | 19,000 (acute EMEG) | No |
| Benzene | 28 | 22 | ND | 2 | 0.13 (CREG) | **Yes** |
| Chloromethane | 28 | 26 | ND | 3 | 62 (EMEG) | No |
| Cyclohexane | 28 | 9 | ND | 7 | 6,000 (RMEG) | No |
| Dichlorodifluoromethane | 28 | 26 | ND | 3 | 440 (RSL) | No |
| Ethylbenzene | 28 | 14 | ND | 15 | 4.9 (RSL) | **Yes** |
| Ethyl Acetate | 28 | 18 | ND | 54 | 310 (RSL) | No |
| Methylene Chloride | 28 | 23 | ND | 6 | 63 (CREG) | No |
| 2-Butanone | 28 | 26 | ND | 8 | 5,000 (RMEG) | No |
| N-Heptane | 28 | 12 | ND | 5 | 1,800 (RSL) | No |
| N-Hexane | 28 | 23 | ND | 4 | 700 (RMEG) | No |
| Propylene | 28 | 2 | ND | 4 | 13,000 (RSL) | No |
| Styrene | 28 | 4 | ND | 1 | 850 (EMEG) | No |
| PCE | 28 | 19 | ND | 62 | 3.8 (CREG) | **Yes** |
| Tetrahydrofuran | 28 | 8 | ND | 3 | 2,000 (RMEG) | No |
| Toluene | 28 | 27 | ND | 7 | 3,800 (EMEG) | No |
| Total Xylenes | 28 | 24 | ND | 68 | 100 (RMEG) | No |

Abbreviations: µg/m$^3$ = micrograms of contaminant per cubic meter of air; CREG = ATSDR cancer risk evaluation guide; EMEG = ATSDR environmental media evaluation guide; RMEG = ATSDR reference media evaluation guide; RSL = EPA industrial indoor air regional screening level; PCE = tetrachloroethylene; ND = contaminant not detected; Comparison values represent chronic exposures unless noted otherwise.

**Table B16. Potential Contaminants of Concern in Surface Water**

| Contaminant | Number of samples | Number of detections | Minimum (µg/L) | Maximum (µg/L) | Comparison Value (CV) (µg/L) | Maximum Exceed CV |
|---|---|---|---|---|---|---|
| 1,4-Dioxane | 169 | 21 | ND | 11.9 | 0.24 (CREG) | **Yes** |
| Benzene | 169 | 2 | ND | 1.2 | 0.44 (CREG) | **Yes** |
| Benzo(a)anthracene | 169 | 4 | ND | 0.574 | 0.038 (NJDEP) | **Yes** |
| Benzo(a)Pyrene | 169 | 4 | ND | 0.563 | 0.0075 (CREG) | **Yes** |
| Benzo(b)fluoranthene | 169 | 4 | ND | 0.877 | 0.038 (DEP) | **Yes** |
| Benzo(k)fluoranthene | 169 | 3 | ND | 0.328 | 0.38 (NJDEP) | No |
| Bromodichloromethane | 169 | 1 | ND | 3.3 | 0.39 (CREG) | **Yes** |
| Carbon tetrachloride | 169 | 33 | ND | 6.1 | 0.35(CREG) | **Yes** |
| Chloroform | 169 | 3 | ND | 3.1 | 71 (Child RMEG) | No |
| Cis-1,2-dichloroethylene | 169 | 24 | ND | 5 | 14 (Child RMEG) | No |
| Dibenz (a,h)anthracene | 169 | 1 | ND | 0.124 | 0.0038 (NJDEP) | **Yes** |

| Contaminant | Number of samples | Number of detections | Minimum (µg/L) | Maximum (µg/L) | Comparison Value (CV) (µg/L) | Maximum Exceed CV |
|---|---|---|---|---|---|---|
| Dibromochloromethane | 169 | 1 | ND | 1.5 | 0.29 (CREG) | **Yes** |
| Hexachlorobenzene | 169 | 1 | ND | 0.0855 | 0.015(CREG) | **Yes** |
| Indeno (1,2,3-c,d)pyrene | 169 | 4 | ND | 0.43 | 0.038 (NJDEP) | **Yes** |
| Pentachlorophenol | 169 | 1 | ND | 0.24 | 0.061 (CREG) | **Yes** |
| PCE | 169 | 68 | ND | 32.5 | 12 (CREG) | **Yes** |
| TCE | 169 | 4 | ND | 1.8 | 0.44 (CREG) | **Yes** |
| Xylene (total) | 169 | 11 | ND | 3.9 | 1,400 (EMEG) | No |
| **Metals - Total** | **Number of samples** | **Number of detections** | **Minimum (µg/L)** | **Maximum (µg/L)** | **Comparison Value (CV) (µg/L)** | **Maximum Exceed CV** |
| Aluminum | 169 | 5 | ND | 445 | 7,100 (EMEG) | No |
| Antimony | 169 | 3 | ND | 2.1 | 2.8 (RMEG) | No |
| Arsenic | 169 | 4 | ND | 3.2 | 0.016 (CREG) | **Yes** |
| Barium | 169 | 36 | 2.7 | 347 | 1,400 (EMEG) | No |
| Copper | 169 | 15 | ND | 7.2 | 140 (Intermediate EMEG) | No |
| Lead | 169 | 3 | ND | 1.3 | 5 (NJDEP) | No |
| Zinc | 169 | 17 | ND | 64.9 | 2,100 (EMEG) | No |
| **Pesticides** | **Number of samples** | **Number of detections** | **Minimum (µg/L)** | **Maximum (µg/L)** | **Comparison Value (CV) (µg/L)** | **Maximum Exceed CV** |
| Dieldrin | 169 | 1 | ND | 0.0028 | 0.0015 (CREG) | **Yes** |
| Chlordane | 169 | 1 | ND | 0.016 | 0.0001 (NJDEP) | **Yes** |
| Heptachlor Epoxide | 169 | 1 | ND | 0.015 | 0.0027 (CREG) | **Yes** |

Abbreviations: µg/L = micrograms of contaminant per liter of water; CREG = ATSDR cancer risk evaluation guide; EMEG = ATSDR environmental media evaluation guide; RMEG = ATSDR reference media evaluation guide; NJ = NJDEP surface water quality standards for fresh water; PCE = tetrachloroethylene; TCE = trichloroethylene; CVs represent chronic exposures unless otherwise noted.

## Hazard quotients – Residential Properties

### Table B17. Residence R11: Hazard Quotients for Potential Contaminants of Concern

| Contaminant | EPC ($\mu g/m^3$) | Health Guideline ($\mu g/m^3$) | HQ | Is further evaluation needed? |
|---|---|---|---|---|
| 1,2-dichloroethane | 2 | 7 (RfC) | 0.29 | No (HQ < 1) |
| Benzene | 1 | 6.4 (MRL) | 0.16 | No (HQ < 1) |
| Chloroform | 1 | 2 (MRL) | 0.50 | No (HQ < 1) |
| Ethylbenzene | 2 | 260 (MRL) | 0.01 | No (HQ < 1) |

Abbreviations: EPC = exposure point concentration representing the maximum concentration; $\mu g/m^3$ = micrograms of contaminant per cubic meter of air; RfC = EPA reference concentration; MRL = ATSDR chronic minimal risk level; HQ = hazard quotient; example HQ calculation for benzene = EPC/MRL = 1/6.4 = 0.16.

**Table B18. Residence R18: Hazard Quotients for Potential Contaminants of Concern**

| Contaminant | EPC ($\mu g/m^3$) | Health Guideline ($\mu g/m^3$) | HQ | Is further evaluation needed? |
|---|---|---|---|---|
| TCE | 0.24 | 2.1 (MRL) | 0.11 | No (HQ < 1) |

Abbreviations: EPC = exposure point concentration representing the maximum concentration; $\mu g/m^3$ = micrograms of contaminant per cubic meter of air; TCE = trichloroethylene; MRL = ATSDR chronic minimal risk level; HQ = hazard quotient; example HQ calculation for TCE = EPC/MRL = 0.24/2.1 = 0.11.

**Table B19. Residence R19: Hazard Quotients for Potential Contaminants of Concern**

| Contaminant | EPC ($\mu g/m^3$) | Health Guideline ($\mu g/m^3$) | HQ | Is further evaluation needed? |
|---|---|---|---|---|
| Benzene | 4.5 | 6.4 (MRL) | 0.70 | No (HQ < 1) |
| Ethylbenzene | 2.7 | 260 (MRL) | 0.01 | No (HQ < 1) |
| PCE | 4.3 | 41 (MRL) | 0.1 | No (HQ < 1) |
| TCE | 0.3 | 2.1 (MRL) | 0.14 | No (HQ < 1) |

Abbreviations: EPC = exposure point concentration representing the maximum concentration; $\mu g/m^3$ = micrograms of contaminant per cubic meter of air; PCE = tetrachloroethylene; TCE = trichloroethylene; MRL = ATSDR chronic minimal risk level; HQ = hazard quotient; example HQ calculation for benzene = EPC/MRL = 4.5/6.4 = 0.70.

**Table B20. Residence R31: Hazard Quotients for Potential Contaminants of Concern**

| Contaminant | EPC ($\mu g/m^3$) | Health Guideline ($\mu g/m^3$) | HQ | Is further evaluation needed? |
|---|---|---|---|---|
| 1,2 dichloroethane | 0.19 | 7 (RfC) | 0.03 | No (HQ < 1) |
| Benzene | 1.5 | 6.4 (MRL) | 0.23 | No (HQ < 1) |
| Carbon Tetrachloride | 0.39 | 100 (RfC) | 0.0039 | No (HQ < 1) |
| Chloroform | 5.0 | 2 (MRL) | **2.5** | **Yes (HQ > 1)** |

Abbreviations: EPC = exposure point concentration representing the maximum concentration; $\mu g/m^3$ = micrograms of contaminant per cubic meter of air; MRL = ATSDR chronic minimal risk level; RfC = EPA reference concentration; HQ = hazard quotient; example HQ calculation for benzene = EPC/MRL = 1.5/6.4 = 0.23.

**Table B21. Residence R32: Hazard Quotients for Potential Contaminants of Concern**

| Contaminant | EPC ($\mu g/m^3$) | Health Guideline ($\mu g/m^3$) | HQ | Is further evaluation needed? |
|---|---|---|---|---|
| Benzene | 1.3 | 6.4 (MRL) | 0.20 | No (HQ < 1) |
| Carbon Tetrachloride | 0.35 | 100 (RfC) | 0.0035 | No (HQ < 1) |
| Chloroform | 0.33 | 2 (MRL) | 0.16 | No (HQ < 1) |

Abbreviations: EPC = exposure point concentration representing the maximum concentration; $\mu g/m^3$ = micrograms of contaminant per cubic meter of air; MRL = ATSDR chronic minimal risk level; RfC = EPA reference concentration; HQ = hazard quotient; example HQ calculation for benzene = EPC/MRL = 1.3/6.4 = 0.20.

**Table B22. Residence R33: Hazard Quotients for Potential Contaminants of Concern**

| Contaminant | EPC ($\mu g/m^3$) | Health Guideline ($\mu g/m^3$) | HQ | Is further evaluation needed? |
|---|---|---|---|---|
| Benzene | 1.5 | 6.4 (MRL) | 0.23 | No (HQ < 1) |
| Carbon Tetrachloride | 0.36 | 100 (RfC) | 0.0036 | No (HQ < 1) |
| Chloroform | 0.55 | 2 (MRL) | 0.27 | No (HQ < 1) |

Abbreviations: EPC = exposure point concentration representing the maximum concentration; $\mu g/m^3$ = micrograms of contaminant per cubic meter of air; MRL = ATSDR chronic minimal risk level; RfC = EPA reference concentration; HQ = hazard quotient; example HQ calculation for benzene = EPC/MRL = 1.5/6.4 = 0.23.

**Table B23. Residence R45: Hazard Quotients for Potential Contaminants of Concern**

| Contaminant | EPC ($\mu g/m^3$) | Health Guideline ($\mu g/m^3$) | HQ | Is further evaluation needed? |
|---|---|---|---|---|
| Benzene | 10 | 6.4 (MRL) | **1.6** | **Yes (HQ > 1)** |
| Carbon Tetrachloride | 0.4 | 100 (RfC) | 0.004 | No (HQ < 1) |
| Chloroform | 1.4 | 2 (MRL) | 0.69 | No (HQ < 1) |
| Ethylbenzene | 8.2 | 260 (MRL) | 0.031 | No (HQ < 1) |
| TCE | 0.2 | 2.1 (MRL) | 0.094 | No (HQ < 1) |

Abbreviations: EPC = exposure point concentration representing the maximum concentration; $\mu g/m^3$ = micrograms of contaminant per cubic meter of air; TCE = trichloroethylene; MRL = ATSDR chronic minimal risk level; RfC = EPA reference concentration; HQ = hazard quotient; example HQ calculation for benzene = EPC/MRL = 10/6.4 = 1.6.

**Table B24. Residence R50: Hazard Quotients for Potential Contaminants of Concern**

| Contaminant | EPC ($\mu g/m^3$) | Health Guideline ($\mu g/m^3$) | HQ | Is further evaluation needed? |
|---|---|---|---|---|
| 1,2 dichloroethane | 2.3 | 7 (RfC) | 0.32 | No (HQ < 1) |
| Benzene | 1.9 | 6.4 (MRL) | 0.3 | No (HQ < 1) |
| Carbon Tetrachloride | 0.4 | 100 (RfC) | 0.0039 | No (HQ < 1) |
| Chloroform | 1.6 | 2 (MRL) | 0.8 | No (HQ < 1) |
| Ethylbenzene | 1.5 | 260 (MRL) | 0.0059 | No (HQ < 1) |
| PCE | 4.0 | 41 (MRL) | 0.098 | No (HQ < 1) |

Abbreviations: EPC = exposure point concentration representing the maximum concentration; $\mu g/m^3$ = micrograms of contaminant per cubic meter of air; PCE = tetrachloroethylene; MRL = ATSDR chronic minimal risk level; RfC = EPA reference concentration; HQ = hazard quotient; example HQ calculation for benzene = EPC/MRL = 1.9/6.4 = 0.3.

## Hazard Quotients – Commercial Properties

### Table B25. Sandvik Inc.: Hazard Quotients for Potential Contaminants of Concern

| Contaminant | EPC ($\mu g/m^3$) | Adjusted worker EPC ($\mu g/m^3$) | Health Guideline ($\mu g/m^3$) | HQ | Is further evaluation needed? |
|---|---|---|---|---|---|
| Benzene | 1.1 | 0.26 | 6.4 (MRL) | 0.04 | No (HQ = 1) |
| Carbon Tetrachloride | 1.4 | 0.34 | 100 (RfC) | 0.003 | No (HQ < 1) |
| Chloroform | 1 | 0.24 | 2 (MRL) | 0.12 | No (HQ < 1) |
| PCE | 5.4 | 1.3 | 41 (MRL) | 0.032 | No (HQ < 1) |
| TCE | 0.4 | 0.096 | 2.1 (MRL) | 0.046 | No (HQ < 1) |

Abbreviations: EPC = exposure point concentration representing the 95% UCL of the mean or the maximum concentration; Adjusted worker EPC = EPC x 8.5/24 x 5/7 x 50/52; $\mu g/m^3$ = micrograms of contaminant per cubic meter of air; PCE = tetrachloroethylene; TCE = trichloroethylene; MRL = ATSDR chronic minimal risk level; RfC = EPA reference concentration; HQ = hazard quotient; example HQ calculation for benzene = adjusted worker EPC/MRL = 0.26/6.4 = 0.04.

### Table B26. Hampshire: Hazard Quotients for Potential Contaminants of Concern

| Contaminant | EPC ($\mu g/m^3$) | Adjusted worker EPC ($\mu g/m^3$) | Health Guideline ($\mu g/m^3$) | HQ | Is further evaluation needed? |
|---|---|---|---|---|---|
| Benzene | 0.8 | 0.19 | 6.4 (MRL) | 0.03 | No (HQ = 1) |
| TCE | 1.1 | 0.27 | 2.1 (MRL) | 0.13 | No (HQ < 1) |

Abbreviations: EPC = exposure point concentration representing the maximum concentration; Adjusted worker EPC = EPC x 8.5/24 x 5/7 x 50/52; $\mu g/m^3$ = micrograms of contaminant per cubic meter of air; TCE = trichloroethylene; MRL = ATSDR chronic minimal risk level; HQ = hazard quotient; example HQ calculation for benzene = adjusted worker EPC/MRL = 0.19/6.4 = 0.03.

### Table B27. Kushner: Hazard Quotients for Potential Contaminants of Concern

| Contaminant | EPC ($\mu g/m^3$) | Adjusted worker EPC ($\mu g/m^3$) | Health Guideline ($\mu g/m^3$) | HQ | Is further evaluation needed? |
|---|---|---|---|---|---|
| Benzene | 37.5 | 9.1 | 6.4 (MRL) | **1.4** | **Yes (HQ > 1)** |
| Ethylbenzene | 5 | 1.2 | 260 (MRL) | 0.005 | No (HQ = 1) |
| TCE | 3 | 0.73 | 2.1 (MRL) | 0.35 | No (HQ < 1) |

Abbreviations: EPC = exposure point concentration representing the 95% UCL of the mean or the maximum concentration; Adjusted worker EPC = EPC x 8.5/24 x 5/7 x 50/52; $\mu g/m^3$ = micrograms of contaminant per cubic meter of air; TCE = trichloroethylene; MRL = ATSDR chronic minimal risk level; RfC = EPA reference concentration; HQ = hazard quotient; example HQ calculation for benzene = adjusted worker EPC/MRL = 9.1/6.4 = 1.4.

**Table B28. Mikia Inc.: Hazard Quotients for Potential Contaminants of Concern**

| Contaminant | EPC ($\mu g/m^3$) | Adjusted worker EPC ($\mu g/m^3$) | Health Guideline ($\mu g/m^3$) | HQ | Is further evaluation needed? |
|---|---|---|---|---|---|
| Benzene | 3 | 0.73 | 6.4 (MRL) | 0.11 | No (HQ = 1) |
| Ethylbenzene | 36 | 8.7 | 260 (MRL) | 0.03 | No (HQ < 1) |
| PCE | 53 | 13 | 41 (MRL) | 0.31 | No (HQ < 1) |
| Xylenes | 190 | 190 * | 220 (MRL) | 0.85 | No (HQ < 1) |

Abbreviations: EPC = exposure point concentration representing the maximum concentration; Adjusted worker EPC = EPC x 8.5/24 x 5/7 x 50/52; $\mu g/m^3$ = micrograms of contaminant per cubic meter of air; PCE = tetrachloroethylene; MRL = ATSDR chronic minimal risk level; HQ = hazard quotient; example HQ calculation for benzene = adjusted worker EPC/MRL = 0.73/6.4 = 0.11; *The worker scenario adjustment for xylenes does not apply due to its rapid clearance from the body upon exposure.

**Table B29. Columbia Bank: Hazard Quotients for Potential Contaminants of Concern**

| Contaminant | EPC ($\mu g/m^3$) | Adjusted worker EPC ($\mu g/m^3$) | Health Guideline ($\mu g/m^3$) | HQ | Is further evaluation needed? |
|---|---|---|---|---|---|
| Benzene | 1.2 | 0.3 | 6.4 (MRL) | 0.05 | No (HQ < 1) |
| Chloroform | 1.7 | 0.41 | 2 (MRL) | 0.21 | No (HQ < 1) |
| PCE | 210 | 51 | 41 (MRL) | **1.2** | **Yes (HQ > 1)** |
| TCE | 38 | 9.3 | 2.1 (MRL) | **4.4** | **Yes (HQ > 1)** |

Abbreviations: EPC = exposure point concentration representing the 95% UCL of the mean or the maximum concentration; Adjusted worker EPC = EPC x 8.5/24 x 5/7 x 50/52; $\mu g/m^3$ = micrograms of contaminant per cubic meter of air; PCE = tetrachloroethylene; TCE = trichloroethylene; MRL = ATSDR chronic minimal risk level; HQ = hazard quotient; example HQ calculation for benzene = adjusted worker EPC/MRL = 0.3/6.4 = 0.05.

**Table B30. Biomet: Hazard Quotients for Potential Contaminants of Concern**

| Contaminant | EPC ($\mu g/m^3$) | Adjusted worker EPC ($\mu g/m^3$) | Health Guideline ($\mu g/m^3$) | HQ | Is further evaluation needed? |
|---|---|---|---|---|---|
| 1,3-Butadiene | 0.5 | 0.12 | 2 (RfC) | 0.061 | No (HQ < 1) |
| Benzene | 1.3 | 0.31 | 6.4 (MRL) | 0.05 | No (HQ < 1) |
| Ethylbenzene | 3.5 | 0.85 | 260 (MRL) | 0.003 | No (HQ < 1) |
| PCE | 7.5 | 1.8 | 41 (MRL) | 0.044 | No (HQ < 1) |

Abbreviations: EPC = exposure point concentration representing the 95% UCL of the mean or the maximum concentration; Adjusted worker EPC = EPC x 8.5/24 x 5/7 x 50/52; $\mu g/m^3$ = micrograms of contaminant per cubic meter of air; PCE = tetrachloroethylene; MRL = ATSDR chronic minimal risk level; RfC = EPA reference concentration; HQ = hazard quotient; example HQ calculation for benzene = adjusted worker EPC/MRL = 0.31/ 6.4= 0.05.

## Lifetime Excess Cancer Risk – Residential Properties

### Table B31. Residence R11: Lifetime Excess Cancer Risk (LECR)

| Contaminant | EPC ($\mu g/m^3$) | Exposure duration (years) | Average lifetime (years) | IUR $(\mu g/m^3)^{-1}$ | Maximum LECR * |
|---|---|---|---|---|---|
| 1,2-dichloroethane | 2 | 33 | 78 | $2.6 \times 10^{-5}$ | $2 \times 10^{-5}$ |
| Benzene | 1 | 33 | 78 | $7.8 \times 10^{-6}$ | $3 \times 10^{-6}$ |
| Chloroform | 1 | 33 | 78 | $2.3 \times 10^{-5}$ | $1 \times 10^{-5}$ |
| Ethylbenzene | 2 | 33 | 78 | $2.5 \times 10^{-6}$ | $2. \times 10^{-6}$ |
| **Total LECR** | --- | --- | --- | --- | **$4 \times 10^{-5}$** |

Abbreviations: EPC = exposure point concentration representing the maximum concentration; $\mu g/m^3$ = micrograms of contaminant per cubic meter of air; IUR = EPA inhalation unit risk; LECR = lifetime excess cancer risk represents the highest calculated risk for children or adults; LECR calculation for benzene = EPC x exposure duration/average lifetime x IUR = 1 x 33/78 x 7.8 $x10^{-6}$ = 3 $x10^{-6}$; * LECR rounded to one significant figure.

### Table B32. Residence R18: Lifetime Excess Cancer Risk (LECR)

| Contaminant | EPC ($\mu g/m^3$) | Exposure duration (years) | Average lifetime (years) | IUR $(\mu g/m^3)^{-1}$ | Maximum LECR * |
|---|---|---|---|---|---|
| TCE | 0.24 | 33 | 78 | $4.1 \times 10^{-6}$ | **$4 x10^{-7}$** |

Abbreviations: EPC = exposure point concentration representing the maximum concentration; $\mu g/m^3$ = micrograms of contaminant per cubic meter of air; TCE = trichloroethylene; IUR = EPA inhalation unit risk; LECR = lifetime excess cancer risk represents the highest calculated risk for children or adults; LECR calculation for TCE = EPC x exposure duration/average lifetime x IUR = 0.24 x 33/78 x 4.1 $x10^{-6}$ = 4 $x10^{-7}$ * LECR rounded to one significant figure.

### Table B33. Residence R19: Lifetime Excess Cancer Risk (LECR)

| Contaminant | EPC ($\mu g/m^3$) | Exposure duration (years) | Average lifetime (years) | IUR $(\mu g/m^3)^{-1}$ | Maximum LECR * |
|---|---|---|---|---|---|
| Benzene | 4.5 | 33 | 78 | $7.8 \times 10^{-6}$ | $2 \times 10^{-5}$ |
| Ethylbenzene | 2.7 | 33 | 78 | $2.5 \times 10^{-6}$ | $3 \times 10^{-6}$ |
| PCE | 4.3 | 33 | 78 | $2.6 \times 10^{-7}$ | $5 \times 10^{-7}$ |
| TCE | 0.3 | 33 | 78 | $4.1 \times 10^{-6}$ | $5 \times 10^{-7}$ |
| **Total LECR** | --- | --- | --- | --- | **$2 \times 10^{-5}$** |

Abbreviations: EPC = exposure point concentration representing the maximum concentration; $\mu g/m^3$ = micrograms of contaminant per cubic meter of air; PCE = tetrachloroethylene; TCE = trichloroethylene; IUR = inhalation unit risk; LECR = lifetime excess cancer risk represents the highest calculated risk for children or adults; LECR calculation for benzene = EPC x exposure duration/average lifetime x IUR = 4.5 x 33/78 x 7.8 $x10^{-6}$ = 2 $x10^{-5}$; * LECR rounded to one significant figure.

**Table B34. Residence R31: Lifetime Excess Cancer Risk (LECR)**

| Contaminant | EPC ($\mu g/m^3$) | Exposure duration (years) | Average lifetime (years) | IUR $(\mu g/m^3)^{-1}$ | Maximum LECR * |
|---|---|---|---|---|---|
| 1,2 dichloroethane | 0.19 | 33 | 78 | $2.6 \times 10^{-5}$ | $2 \times 10^{-6}$ |
| Benzene | 1.5 | 33 | 78 | $7.8 \times 10^{-6}$ | $5 \times 10^{-6}$ |
| Bromodichloromethane | 2.0 | 33 | 78 | $3.7 \times 10^{-5}$ | $3 \times 10^{-5}$ |
| Carbon Tetrachloride | 0.39 | 33 | 78 | $6.0 \times 10^{-6}$ | $1 \times 10^{-6}$ |
| Chloroform | 5.0 | 33 | 78 | $2.3 \times 10^{-5}$ | $5 \times 10^{-5}$ |
| **Total LECR** | --- | --- | --- | --- | **$9 \times 10^{-5}$** |

Abbreviations: EPC = exposure point concentration representing the maximum concentration; $\mu g/m^3$ = micrograms of contaminant per cubic meter of air; IUR = inhalation unit risk; LECR = lifetime excess cancer risk represents the highest calculated risk for children or adults; LECR calculation for benzene = EPC x exposure duration/average lifetime x IUR = 1.5 x 33/78 x 7.8 $x10^{-6}$ = 5 $x10^{-6}$; * LECR rounded to one significant figure.

**Table B35. Residence R32: Lifetime Excess Cancer Risk (LECR)**

| Contaminant | EPC ($\mu g/m^3$) | Exposure duration (years) | Average lifetime (years) | IUR $(\mu g/m^3)^{-1}$ | Maximum LECR * |
|---|---|---|---|---|---|
| Benzene | 1.3 | 33 | 78 | $7.8 \times 10^{-6}$ | $4 \times 10^{-6}$ |
| Carbon Tetrachloride | 0.35 | 33 | 78 | $6.0 \times 10^{-6}$ | $9 \times 10^{-7}$ |
| Chloroform | 0.33 | 33 | 78 | $2.3 \times 10^{-5}$ | $3 \times 10^{-6}$ |
| **Total LECR** | --- | --- | --- | --- | **$8 \times 10^{-6}$** |

Abbreviations: EPC = exposure point concentration representing the maximum concentration; $\mu g/m^3$ = micrograms of contaminant per cubic meter of air; IUR = inhalation unit risk; LECR = lifetime excess cancer risk represents the highest calculated risk for children or adults; LECR calculation for benzene = EPC x exposure duration/average lifetime x IUR = 1.3 x 33/78 x 7.8 $x10^{-6}$ = 4 x $10^{-6}$; * LECR rounded to one significant figure.

**Table B36. Residence R33: Lifetime Excess Cancer Risk (LECR)**

| Contaminant | EPC ($\mu g/m^3$) | Exposure duration (years) | Average lifetime (years) | IUR $(\mu g/m^3)^{-1}$ | Maximum LECR * |
|---|---|---|---|---|---|
| Benzene | 1.5 | 33 | 78 | $7.8 \times 10^{-6}$ | $5 \times 10^{-6}$ |
| Carbon Tetrachloride | 0.36 | 33 | 78 | $6.0 \times 10^{-6}$ | $9 \times 10^{-7}$ |
| Chloroform | 0.55 | 33 | 78 | $2.3 \times 10^{-5}$ | $5 \times 10^{-6}$ |
| **Total LECR** | --- | --- | --- | --- | **$1 \times 10^{-5}$** |

Abbreviations: EPC = exposure point concentration representing the maximum concentration; $\mu g/m^3$ = micrograms of contaminant per cubic meter of air; IUR = inhalation unit risk; LECR = lifetime excess cancer risk represents the

highest calculated risk for children or adults; LECR calculation for benzene = EPC x exposure duration/average lifetime x IUR = 1.5 x 33/78 x 7.8 $x10^{-6}$ = 5 x $10^{-6}$; * LECR rounded to one significant figure.

**Table B37. Residence R45: Lifetime Excess Cancer Risk (LECR)**

| Contaminant | EPC ($\mu g/m^3$) | Exposure duration (years) | Average lifetime (years) | IUR ($\mu g/m^3$) $^{-1}$ | Maximum LECR * |
|---|---|---|---|---|---|
| Benzene | 10 | 33 | 78 | 7.8 $x10^{-6}$ | 3 $x10^{-5}$ |
| Carbon Tetrachloride | 0.4 | 33 | 78 | 6.0 $x10^{-6}$ | 1 $x10^{-6}$ |
| Chloroform | 1.4 | 33 | 78 | 2.3 $x10^{-5}$ | 1 $x10^{-5}$ |
| Ethylbenzene | 8.2 | 33 | 78 | 2.5 $x10^{-6}$ | 9 $x10^{-6}$ |
| TCE | 0.2 | 33 | 78 | 4.1 $x10^{-6}$ | 3 $x10^{-7}$ |
| **Total LECR** | --- | --- | --- | --- | **6 x $10^{-5}$** |

Abbreviations: EPC = exposure point concentration representing the maximum concentration; $\mu g/m^3$ = micrograms of contaminant per cubic meter of air; TCE = trichloroethylene; IUR = inhalation unit risk; LECR = lifetime excess cancer risk represents the highest calculated risk for children or adults; LECR calculation for benzene = EPC x exposure duration/average lifetime x IUR = 10 x 33/78 x 7.8 $x10^{-6}$ = 3 $x10^{-5}$; * LECR rounded to one significant figure.

**Table B38. Residence R50: Lifetime Excess Cancer Risk (LECR)**

| Contaminant | EPC ($\mu g/m^3$) | Exposure duration (years) | Average lifetime (years) | IUR ($\mu g/m^3$) $^{-1}$ | Maximum LECR * |
|---|---|---|---|---|---|
| 1,2 dichloroethane | 2.3 | 33 | 78 | 2.6 $x10^{-5}$ | 3 x $10^{-5}$ |
| Benzene | 1.9 | 33 | 78 | 7.8 $x10^{-6}$ | 6 x $10^{-6}$ |
| Carbon Tetrachloride | 0.39 | 33 | 78 | 6.0 $x10^{-6}$ | 1 x $10^{-6}$ |
| Chloroform | 1.6 | 33 | 78 | 2.3 x $10^{-5}$ | 2 x $10^{-5}$ |
| Ethylbenzene | 1.5 | 33 | 78 | 2.5 $x10^{-6}$ | 2 $x10^{-6}$ |
| PCE | 4.0 | 33 | 78 | 2.6 $x10^{-7}$ | 4 $x10^{-7}$ |
| **Total LECR** | --- | --- | --- | --- | **5 x $10^{-5}$** |

Abbreviations: EPC = exposure point concentration representing the maximum concentration; $\mu g/m^3$ = micrograms of contaminant per cubic meter of air; PCE = tetrachloroethylene; IUR = inhalation unit risk; LECR = lifetime excess cancer risk represents the highest calculated risk for children or adults; LECR calculation for benzene = EPC x exposure duration/average lifetime x IUR = 1.9 x 33/78 x 7.8 $x10^{-6}$ = 6 $x10^{-6}$; * LECR rounded to one significant figure.

## Lifetime Excess Cancer Risk – Commercial Properties

### Table B39. Sandvik Inc.: Lifetime Excess Cancer Risk

| Contaminant | EPC ($\mu g/m^3$) | Adjusted worker EPC ($\mu g/m^3$) | Exposure duration (years) | Average lifetime (years) | IUR $(\mu g/m^3)^{-1}$ | LECR * |
|---|---|---|---|---|---|---|
| Benzene | 1.1 | 0.26 | 20 | 78 | $7.8x10^{-6}$ | $5 \times 10^{-7}$ |
| Carbon Tetrachloride | 1.4 | 0.34 | 20 | 78 | $6.0 x10^{-6}$ | $5 \times 10^{-7}$ |
| Chloroform | 1 | 0.24 | 20 | 78 | $2.3 x10^{-5}$ | $1 \times 10^{-6}$ |
| PCE | 5.4 | 1.3 | 20 | 78 | $2.6 x10^{-7}$ | $9 \times 10^{-6}$ |
| TCE | 0.4 | 0.096 | 20 | 78 | $4.1 x10^{-6}$ | $1 \times 10^{-7}$ |
| **Total LECR** | --- | --- | --- | --- | --- | **$1 \times 10^{-5}$** |

Abbreviations: EPC = exposure point concentration representing the 95% UCL of the mean or the maximum concentration; Adjusted worker EPC = EPC x 8.5/24 x 5/7 x 50/52; $\mu g/m^3$ = micrograms of contaminant per cubic meter of air; PCE = tetrachloroethylene; TCE = trichloroethylene; IUR = inhalation unit risk; LECR =lifetime excess cancer risk for workers; LECR calculation for benzene = adjusted worker EPC x exposure duration/average lifetime x IUR = 0.26 x 20/78 x 7.8 $x10^{-6}$ = 5 $x10^{-7}$; * LECR rounded to one significant figure.

### Table B40. Hampshire: Lifetime Excess Cancer Risk

| Contaminant | EPC ($\mu g/m^3$) | Adjusted worker EPC ($\mu g/m^3$) | Exposure duration (years) | Average lifetime (years) | IUR $(\mu g/m^3)^{-1}$ | LECR * |
|---|---|---|---|---|---|---|
| Benzene | 0.8 | 0.19 | 20 | 78 | $7.8 \times 10^{-6}$ | $4 x10^{-7}$ |
| TCE | 1.1 | 0.27 | 20 | 78 | $4.1 \times 10^{-6}$ | $3 \times 10^{-7}$ |
| **Total LECR** | --- | --- | --- | --- | --- | **$7 \times 10^{-7}$** |

Abbreviations: EPC = exposure point concentration representing the maximum concentration; Adjusted worker EPC = EPC x 8.5/24 x 5/7 x 50/52; $\mu g/m^3$ = micrograms of contaminant per cubic meter of air; TCE = trichloroethylene; IUR = inhalation unit risk; LECR =lifetime excess cancer risk for workers; LECR calculation for benzene = adjusted worker EPC x exposure duration/average lifetime x IUR = 0.19 x 20/78 x 7.8 $x10^{-6}$ = 4 $x10^{-7}$; * LECR rounded to one significant figure.

### Table B41. Kushner: Lifetime Excess Cancer Risk

| Contaminant | EPC ($\mu g/m^3$) | Adjusted worker EPC ($\mu g/m^3$) | Exposure duration (years) | Average lifetime (years) | IUR $(\mu g/m^3)^{-1}$ | LECR * |
|---|---|---|---|---|---|---|
| Benzene | 37.5 | 9.1 | 20 | 78 | $7.8 \times 10^{-6}$ | $2 \times 10^{-5}$ |
| Ethylbenzene | 5 | 1.2 | 20 | 78 | $2.5 \times 10^{-6}$ | $8 \times 10^{-7}$ |
| TCE | 3 | 0.73 | 20 | 78 | $4.1 \times 10^{-6}$ | $8 \times 10^{-7}$ |
| **Total LECR** | --- | --- | --- | --- | --- | **$2 \times 10^{-5}$** |

Abbreviations: EPC = exposure point concentration representing the 95% UCL of the mean or the maximum concentration; Adjusted worker EPC = EPC x 8.5/24 x 5/7 x 50/52; $\mu g/m^3$ = micrograms of contaminant per cubic meter of air; TCE = trichloroethylene; IUR = inhalation unit risk; LECR =lifetime excess cancer risk for workers; LECR calculation for benzene =

adjusted worker EPC x exposure duration/average lifetime x IUR = 9.1 x 20/78 x 7.8 x$10^{-6}$ = 2 x$10^{-5}$; * LECR rounded to one significant figure.

**Table B42. Mikia Inc.: Lifetime Excess Cancer Risk**

| Contaminant | EPC ($\mu g/m^3$) | Adjusted worker EPC ($\mu g/m^3$) | Exposure duration (years) | Average lifetime (years) | IUR ($\mu g/m^3$) $^{-1}$ | LECR * |
|---|---|---|---|---|---|---|
| Benzene | 3 | 0.73 | 20 | 78 | 7.8 x $10^{-6}$ | 2 x $10^{-6}$ |
| Ethylbenzene | 36 | 8.7 | 20 | 78 | 2.5 x $10^{-6}$ | 6 x $10^{-6}$ |
| PCE | 53 | 13 | 20 | 78 | 2.6 x $10^{-7}$ | 9 x $10^{-7}$ |
| **Total LECR** | --- | --- | --- | --- | --- | **8 x $10^{-6}$** |

Abbreviations: EPC = exposure point concentration representing the maximum concentration; Adjusted worker EPC = EPC x 8.5/24 x 5/7 x 50/52; $\mu g/m^3$ = micrograms of contaminant per cubic meter of air; PCE = tetrachloroethylene; IUR = inhalation unit risk; LECR =lifetime excess cancer risk for workers; LECR calculation for benzene = adjusted worker EPC x exposure duration/average lifetime x IUR = 0.73 x 20/78 x 7.8 x$10^{-6}$ = 2 x$10^{-6}$; * LECR rounded to one significant figure.

**Table B43. Columbia Bank: Lifetime Excess Cancer Risk**

| Contaminant | EPC ($\mu g/m^3$) | Adjusted worker EPC ($\mu g/m^3$) | Exposure duration (years) | Average lifetime (years) | IUR ($\mu g/m^3$) $^{-1}$ | LECR * |
|---|---|---|---|---|---|---|
| Benzene | 1.2 | 0.3 | 20 | 78 | 7.8 x $10^{-6}$ | 6 x $10^{-7}$ |
| Chloroform | 1.7 | 0.41 | 20 | 78 | 2.3 x $10^{-5}$ | 2 x $10^{-6}$ |
| PCE | 210 | 51 | 20 | 78 | 2.6 x $10^{-7}$ | 3 x $10^{-6}$ |
| TCE | 38 | 9.3 | 20 | 78 | 4.1 x $10^{-6}$ | 1 x $10^{-5}$ |
| **Total LECR** | --- | --- | --- | --- | --- | **2 x $10^{-5}$** |

Abbreviations: EPC = exposure point concentration representing the 95% UCL of the mean or the maximum concentration; Adjusted worker EPC = EPC x 8.5/24 x 5/7 x 50/52; $\mu g/m^3$ = micrograms of contaminant per cubic meter of air; PCE = tetrachloroethylene; TCE = trichloroethylene; IUR = inhalation unit risk; LECR =lifetime excess cancer risk for workers; LECR calculation for benzene = adjusted worker EPC x exposure duration/average lifetime x IUR = 0.3 x 20/78 x 7.8 x$10^{-6}$ = 6 x$10^{-7}$; * LECR rounded to one significant figure.

**Table B44. Biomet: Lifetime Excess Cancer Risk**

| Contaminant | EPC ($\mu g/m^3$) | Adjusted worker EPC ($\mu g/m^3$) | Exposure duration (years) | Average lifetime (years) | IUR $(\mu g/m^3)^{-1}$ | LECR * |
|---|---|---|---|---|---|---|
| 1,3-Butadiene | 0.5 | 0.12 | 20 | 78 | $3.0 \times 10^{-5}$ | $9 \times 10^{-7}$ |
| Benzene | 1.3 | 0.31 | 20 | 78 | $7.8 \times 10^{-6}$ | $6 \times 10^{-7}$ |
| Ethylbenzene | 3.5 | 0.85 | 20 | 78 | $2.5 \times 10^{-6}$ | $5 \times 10^{-7}$ |
| PCE | 7.5 | 1.8 | 20 | 78 | $2.6 \times 10^{-7}$ | $1 \times 10^{-7}$ |
| **Total LECR** | --- | --- | --- | --- | **---** | **$2 \times 10^{-6}$** |

Abbreviations: EPC = exposure point concentration representing the 95% UCL of the mean or the maximum concentration; Adjusted worker EPC = EPC x 8.5/24 x 5/7 x 50/52; $\mu g/m^3$ = micrograms of contaminant per cubic meter of air; PCE = tetrachloroethylene; IUR = inhalation unit risk; LECR =lifetime excess cancer risk for workers; LECR calculation for benzene = adjusted worker EPC x exposure duration/average lifetime x IUR = 0.3 x 20/78 x $7.8 \times 10^{-6} = 6 \times 10^{-7}$; * LECR rounded to one significant figure.

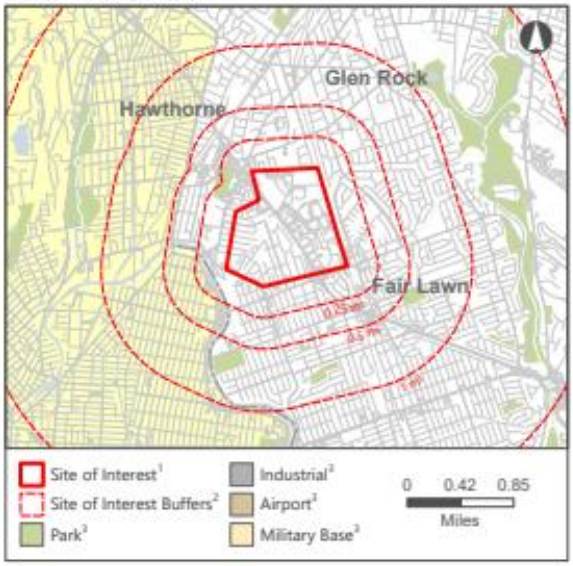
# APPENDIX C – Demographic Maps

# Fair Lawn Well Field Superfund Site

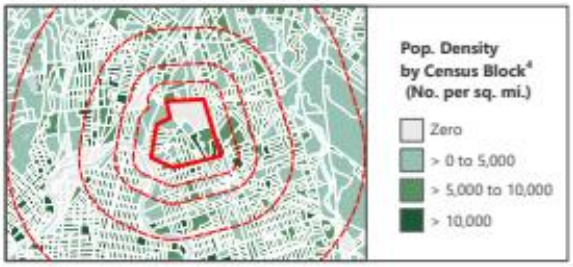
Fair Lawn, Bergen County, NJ

INTRODUCTORY MAP SERIES
GENERAL SITE PROFILE
EPA FACILITY ID NJD980654107

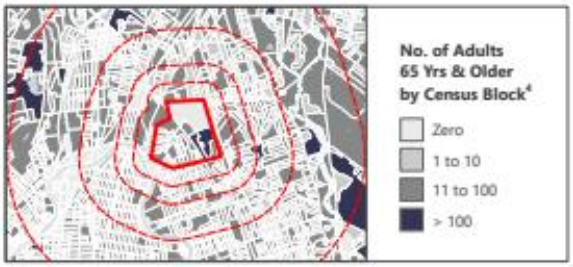
## Site Vicinity Map




## General Population Density




## Sensitive Populations



The **General Site Profile Map** depicts the hazardous waste site of interest, along with any airport, industrial, military, or park land uses. It also provides community demographic and housing statistics.

**Demographic Statistics[4,5]**
Within 1 Miles buffer of site boundary

| Measure | 2010 | 2020 | Change |
|---|---|---|---|
| Total Population | 50,807 | 53,538 | +5% |
| White Alone | 36,640 | 29,662 | -19% |
| Black Alone | 4,410 | 3,572 | -19% |
| Am. Indian/Alaska Native Alone | 203 | 894 | +340% |
| Asian Alone | 3,005 | 4,470 | +48% |
| Native Hawaiian & Other Pacific Islander Alone | 13 | 16 | +23% |
| Some Other Race Alone | 5,271 | 9,064 | +71% |
| Two or More Races | 1,257 | 5,874 | +367% |
| Hispanic or Latino[6] | 13,189 | 16,678 | +26% |
| Children Aged 6 and Younger | 4,577 | 4,696 | +2% |
| Adults Aged 65 and Older | 6,642 | 8,298 | +24% |
| Females Aged 15 to 44 | 9,950 | 10,289 | +3% |
| Housing Units | 18,590 | 19,085 | +2% |
| Housing Units Pre 1950 | 7,823 | 7,557 | -3% |

**Data Sources:** [1]Fair Lawn Well Field General Site Boundary, [2]ATSDR GRASP, [3]TomTom 2021Q3, [4]US Census 2020 Demographic and Housing Characteristics. **Notes:** [5]Calculated using area-proportion spatial analysis method. [6]Individuals identifying origin as Hispanic or Latino may be of any race. **Coordinate System:** Coordinate System used for all map panels is NAD 1983 StatePlane New Jersey FIPS 2900 Feet

ATSDR Agency for Toxic Substances and Disease Registry

GRASP Geospatial Research, Analysis, and Services Program

PRJ ID 13684 | AUTHOR Bethany Foust

FINAL - FOR PUBLIC RELEASE

10/2/2024

**Figure 5. Fair Lawn Well Field Superfund Site Demographics**

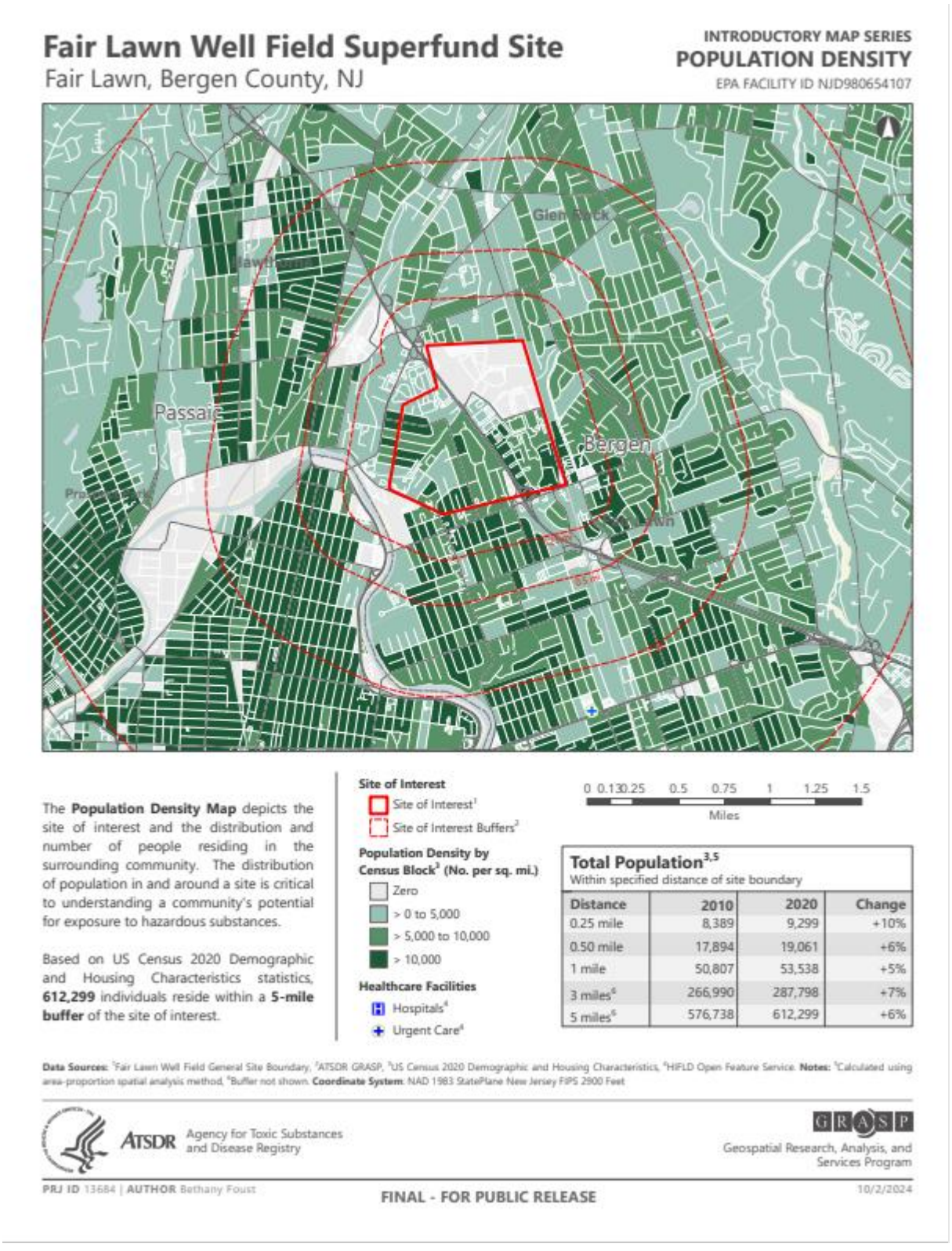



**Figure 6. Fair Lawn Well Field Superfund Site Population Density**

# APPENDIX D – PHAST Results

**Table D1. PHAST Default Input Parameters and Equations – Residential Indoor Air Exposures**

PUBLIC HEALTH ASSESSMENT SITE TOOL

PHAST

## Default Input Parameters and Equations – Residential Indoor Air Exposures

### Equations

**Air Inhalation Exposure Equation**

**Adjusted EPC = EPC x $EF_{noncancer}$** **Equation 1**

EPC = exposure point concentration, $EF_{noncancer}$ = exposure factor (unitless)

**Hazard Quotient**

**HQ = Adjusted EPC ÷ HG** **Equation 2**

HQ = hazard quotient, EPC = exposure point concentration (µg/$m^3$ or ppb), HG = health guideline (e.g., inhalation MRL, RfC)

**Cancer Risk Equations**

**CR = (Adjusted EPC x IUR) x (ED ÷ LY) for each exposure group** **Equation 3**

**ADAF-adjusted CR = (Adjusted EPC x IUR) x (ED ÷ LY) x ADAF for each exposure group** **Equation 4**

**Total CR = Sum of the CR for all exposure groups** **Equation 5**

CR = cancer risk (unitless), EPC = exposure point concentration (µg/$m^3$ or ppb), IUR = inhalation unit risk ((µg/$m^3$ or ppb)$^{-1}$), ED = exposure duration (years), LY = lifetime years (78 years), ADAF = age-dependent adjustment factor (unitless), EF (cancer) = exposure factor (cancer) calculated as follows: EF (noncancer; unitless) x exposure group specific exposure duration (years) ÷ lifetime of 78 years

## Default Exposure Factors

| Duration Category | Hours per Day | Days per Week | Weeks per Year | Years | Exposure Group Specific EF $_{noncancer}$ | Exposure Group Specific* EF $_{cancer}$ |
|---|---|---|---|---|---|---|
| **Acute** | 24 | - | - | - | 1 | - |
| **Intermediate** | 24 | 7 | - | - | 1 | - |
| **Chronic** | 24 | 7 | 52.14 | See exposure group specific exposure durations | 1 | = $EF_{noncancer}$ x ($ED_{age\text{-}specific\ (yrs)}$ ÷ 78 years) |

Abbreviations: EF = exposure factor; NC = not calculated

Cancer EFs are not shown in the table because they are calculated using age-specific durations. The general formula is $EF_{cancer} = EF_{noncancer}$ x ($ED_{age\text{-}specific\ (yrs)}$ ÷ 78 years).

## Contaminant Information

| Contaminant Name | Entered Concentration | EPC Type | Converted Concentration (µg/m³) | Converted Concentration (ppb) |
|---|---|---|---|---|
| 1,2-dichloroethane | 2 µg/m³ | Maximum | 2 | 0.49 |
| Benzene | 1 µg/m³ | Maximum | 1 | 0.31 |
| Chloroform | 1 µg/m³ | Maximum | 1 | 0.2 |
| Ethylbenzene | 2 µg/m³ | Maximum | 2 | 0.46 |
| Tetrachloroethylene | 3 µg/m³ | Maximum | 3 | 0.44 |

Abbreviations: µg/m³ = micrograms per meter cubed; EPC = exposure point concentration

## Air Inhalation Chronic (Default)

### 1,2-dichloroethane

**Table D2. Residential: Default exposure point concentrations for chronic exposure to 1,2-dichloroethane in air at 2 µg/m³ (0.49 ppb) along with cancer risk estimates***

| Exposure Group | CTE Adjusted EPC (µg/m³) | CTE Adjusted EPC (ppb) | CTE Noncancer Hazard Quotient | CTE Cancer Risk | CTE Exposure Duration (yrs) | RME Adjusted EPC (µg/m³) | RME Adjusted EPC (ppb) | RME Noncancer Hazard Quotient | RME Cancer Risk | RME Exposure Duration (yrs) |
|---|---|---|---|---|---|---|---|---|---|---|
| Birth to < 1 year | 2.0 | 0.49 | - | - | 1 | 2.0 | 0.49 | - | - | 1 |
| 1 to < 2 years | 2.0 | 0.49 | - | - | 1 | 2.0 | 0.49 | - | - | 1 |
| 2 to < 6 years | 2.0 | 0.49 | - | - | 4 | 2.0 | 0.49 | - | - | 4 |
| 6 to < 11 years | 2.0 | 0.49 | - | - | 5 | 2.0 | 0.49 | - | - | 5 |
| 11 to < 16 years | 2.0 | 0.49 | - | - | 1 | 2.0 | 0.49 | - | - | 5 |
| 16 to < 21 years | 2.0 | 0.49 | - | - | 0 | 2.0 | 0.49 | - | - | 5 |
| Total Child | - | - | - | 8.0E-6 ‡ | 12 | - | - | - | 1.4E-5 ‡ | 21 |
| Adult | 2.0 | 0.49 | - | 8.0E-6 ‡ | 12 | 2.0 | 0.49 | - | 2.2E-5 ‡ | 33 |
| Birth to < 21 years plus 12 years during adulthood § | - | - | - | - | - | - | - | - | 2.2E-5 ‡ | 33 |

Source: [Final Vapor Intrusion Investigation Report – Fair Lawn Well Field Superfund Site – Langan Engineering and Environmental Services Inc. June 2018]
Abbreviations: adjusted EPC = the exposure point concentration (EPC) times the appropriate exposure factors; µg/m³ = micrograms per meter cubed; ppb = parts per billion; CTE = central tendency exposure (typical); RME = reasonable maximum exposure (higher); yrs = years
* The calculations in this table were generated using ATSDR's PHAST v2.5.0.0. The cancer risks were calculated using the inhalation unit risk of 2.6E-5 $(\mu g/m^3)^{-1}$.
‡ Indicates that the cancer risk exceeds one extra case in a million people similarly exposed, which ATSDR evaluates further.
§ This cancer risk represents a scenario where children are likely to continue to live in their childhood home as adults.

**Benzene**

**Table D3. Residential: Default exposure point concentrations for chronic exposure to benzene in air at 1 µg/m³ (0.31 ppb) along with noncancer hazard quotients and cancer risk estimates***

| Exposure Group | CTE Adjusted EPC (µg/m³) | CTE Adjusted EPC (ppb) | CTE Noncancer Hazard Quotient | CTE Cancer Risk | CTE Exposure Duration (yrs) | RME Adjusted EPC (µg/m³) | RME Adjusted EPC (ppb) | RME Noncancer Hazard Quotient | RME Cancer Risk | RME Exposure Duration (yrs) |
|---|---|---|---|---|---|---|---|---|---|---|
| Birth to < 1 year | 1.0 | 0.31 | 0.16 | - | 1 | 1.0 | 0.31 | 0.16 | - | 1 |
| 1 to < 2 years | 1.0 | 0.31 | 0.16 | - | 1 | 1.0 | 0.31 | 0.16 | - | 1 |
| 2 to < 6 years | 1.0 | 0.31 | 0.16 | - | 4 | 1.0 | 0.31 | 0.16 | - | 4 |
| 6 to < 11 years | 1.0 | 0.31 | 0.16 | - | 5 | 1.0 | 0.31 | 0.16 | - | 5 |
| 11 to < 16 years | 1.0 | 0.31 | 0.16 | - | 1 | 1.0 | 0.31 | 0.16 | - | 5 |
| 16 to < 21 years | 1.0 | 0.31 | 0.16 | - | 0 | 1.0 | 0.31 | 0.16 | - | 5 |
| Total Child | - | - | - | 1.2E-6 ‡ | 12 | - | - | - | 2.1E-6 ‡ | 21 |
| Adult | 1.0 | 0.31 | 0.16 | 1.2E-6 ‡ | 12 | 1.0 | 0.31 | 0.16 | 3.3E-6 ‡ | 33 |
| Birth to < 21 years plus 12 years during adulthood § | - | - | - | - | - | - | - | - | 3.3E-6 ‡ | 33 |

Source: [[Final Vapor Intrusion Investigation Report – Fair Lawn Well Field Superfund Site – Langan Engineering and Environmental Services Inc. June 2018]

Abbreviations: adjusted EPC = the exposure point concentration (EPC) times the appropriate exposure factors; µg/m³ = micrograms per meter cubed; ppb = parts per billion; CTE = central tendency exposure (typical); RME = reasonable maximum exposure (higher); yrs = years

* The calculations in this table were generated using ATSDR's PHAST v2.5.0.0. The noncancer hazard quotients were calculated using the chronic (greater than 1 year) minimal risk level of 6.4 µg/m³ and the cancer risks were calculated using the inhalation unit risk of 7.8E-6 $(\mu g/m^3)^{-1}$.

‡ Indicates that the cancer risk exceeds one extra case in a million people similarly exposed, which ATSDR evaluates further.

§ This cancer risk represents a scenario where children are likely to continue to live in their childhood home as adults.

**Chloroform**

**Table D4. Residential: Default exposure point concentrations for chronic exposure to chloroform in air at 1 µg/m³ (0.20 ppb) along with noncancer hazard quotients and cancer risk estimates***

| Exposure Group | CTE Adjusted EPC (µg/m³) | CTE Adjusted EPC (ppb) | CTE Noncancer Hazard Quotient | CTE Cancer Risk | CTE Exposure Duration (yrs) | RME Adjusted EPC (µg/m³) | RME Adjusted EPC (ppb) | RME Noncancer Hazard Quotient | RME Cancer Risk | RME Exposure Duration (yrs) |
|---|---|---|---|---|---|---|---|---|---|---|
| Birth to < 1 year | 1.0 | 0.20 | 0.50 | - | 1 | 1.0 | 0.20 | 0.50 | - | 1 |
| 1 to < 2 years | 1.0 | 0.20 | 0.50 | - | 1 | 1.0 | 0.20 | 0.50 | - | 1 |
| 2 to < 6 years | 1.0 | 0.20 | 0.50 | - | 4 | 1.0 | 0.20 | 0.50 | - | 4 |
| 6 to < 11 years | 1.0 | 0.20 | 0.50 | - | 5 | 1.0 | 0.20 | 0.50 | - | 5 |
| 11 to < 16 years | 1.0 | 0.20 | 0.50 | - | 1 | 1.0 | 0.20 | 0.50 | - | 5 |
| 16 to < 21 years | 1.0 | 0.20 | 0.50 | - | 0 | 1.0 | 0.20 | 0.50 | - | 5 |
| Total Child | - | - | - | 3.5E-6 ‡ | 12 | - | - | - | 6.2E-6 ‡ | 21 |
| Adult | 1.0 | 0.20 | 0.50 | 3.5E-6 ‡ | 12 | 1.0 | 0.20 | 0.50 | 9.7E-6 ‡ | 33 |
| Birth to < 21 years plus 12 years during adulthood § | - | - | - | - | - | - | - | - | 9.7E-6 ‡ | 33 |

Source: [Final Vapor Intrusion Investigation Report – Fair Lawn Well Field Superfund Site – Langan Engineering and Environmental Services Inc. June 2018]
Abbreviations: adjusted EPC = the exposure point concentration (EPC) times the appropriate exposure factors; µg/m³ = micrograms per meter cubed; ppb = parts per billion; CTE = central tendency exposure (typical); RME = reasonable maximum exposure (higher); yrs = years
* The calculations in this table were generated using ATSDR's PHAST v2.5.0.0. The noncancer hazard quotients were calculated using the chronic (greater than 1 year) minimal risk level of 2 µg/m³ and the cancer risks were calculated using the inhalation unit risk of 2.3E-5 $(\mu g/m^3)^{-1}$.
‡ Indicates that the cancer risk exceeds one extra case in a million people similarly exposed, which ATSDR evaluates further.
§ This cancer risk represents a scenario where children are likely to continue to live in their childhood home as adults.

**Ethylbenzene**

**Table D5. Residential: Default exposure point concentrations for chronic exposure to ethylbenzene in air at 2 µg/m³ (0.46 ppb) along with noncancer hazard quotients***

| Exposure Group | CTE Adjusted EPC (µg/m³) | CTE Adjusted EPC (ppb) | CTE Noncancer Hazard Quotient | CTE Cancer Risk | CTE Exposure Duration (yrs) | RME Adjusted EPC (µg/m³) | RME Adjusted EPC (ppb) | RME Noncancer Hazard Quotient | RME Cancer Risk | RME Exposure Duration (yrs) |
|---|---|---|---|---|---|---|---|---|---|---|
| Birth to < 1 year | 2.0 | 0.46 | 0.0077 | - | 1 | 2.0 | 0.46 | 0.0077 | - | 1 |
| 1 to < 2 years | 2.0 | 0.46 | 0.0077 | - | 1 | 2.0 | 0.46 | 0.0077 | - | 1 |
| 2 to < 6 years | 2.0 | 0.46 | 0.0077 | - | 4 | 2.0 | 0.46 | 0.0077 | - | 4 |
| 6 to < 11 years | 2.0 | 0.46 | 0.0077 | - | 5 | 2.0 | 0.46 | 0.0077 | - | 5 |
| 11 to < 16 years | 2.0 | 0.46 | 0.0077 | - | 1 | 2.0 | 0.46 | 0.0077 | - | 5 |
| 16 to < 21 years | 2.0 | 0.46 | 0.0077 | - | 0 | 2.0 | 0.46 | 0.0077 | - | 5 |
| Total Child | - | - | - | - | 12 | - | - | - | - | 21 |
| Adult | 2.0 | 0.46 | 0.0077 | - | 12 | 2.0 | 0.46 | 0.0077 | - | 33 |

Source: [Final Vapor Intrusion Investigation Report – Fair Lawn Well Field Superfund Site – Langan Engineering and Environmental Services Inc. June 2018]
Abbreviations: adjusted EPC = the exposure point concentration (EPC) times the appropriate exposure factors; µg/m³ = micrograms per meter cubed; ppb = parts per billion; CTE = central tendency exposure (typical); RME = reasonable maximum exposure (higher); yrs = years
* The calculations in this table were generated using ATSDR's PHAST v2.5.0.0. The noncancer hazard quotients were calculated using the chronic (greater than 1 year) minimal risk level of 260 µg/m³.

**Tetrachloroethylene**

**Table D6. Residential: Default exposure point concentrations for chronic exposure to tetrachloroethylene in air at 3 µg/m³ (0.44 ppb) along with noncancer hazard quotients and cancer risk estimates***

| Exposure Group | CTE Adjusted EPC (µg/m³) | CTE Adjusted EPC (ppb) | CTE Noncancer Hazard Quotient | CTE Cancer Risk | CTE Exposure Duration (yrs) | RME Adjusted EPC (µg/m³) | RME Adjusted EPC (ppb) | RME Noncancer Hazard Quotient | RME Cancer Risk | RME Exposure Duration (yrs) |
|---|---|---|---|---|---|---|---|---|---|---|
| Birth to < 1 year | 3.0 | 0.44 | 0.073 | - | 1 | 3.0 | 0.44 | 0.073 | - | 1 |
| 1 to < 2 years | 3.0 | 0.44 | 0.073 | - | 1 | 3.0 | 0.44 | 0.073 | - | 1 |
| 2 to < 6 years | 3.0 | 0.44 | 0.073 | - | 4 | 3.0 | 0.44 | 0.073 | - | 4 |
| 6 to < 11 years | 3.0 | 0.44 | 0.073 | - | 5 | 3.0 | 0.44 | 0.073 | - | 5 |
| 11 to < 16 years | 3.0 | 0.44 | 0.073 | - | 1 | 3.0 | 0.44 | 0.073 | - | 5 |
| 16 to < 21 years | 3.0 | 0.44 | 0.073 | - | 0 | 3.0 | 0.44 | 0.073 | - | 5 |
| Total Child | - | - | - | 1.2E-7 | 12 | - | - | - | 2.1E-7 | 21 |
| Adult | 3.0 | 0.44 | 0.073 | 1.2E-7 | 12 | 3.0 | 0.44 | 0.073 | 3.3E-7 | 33 |
| Birth to < 21 years plus 12 years during adulthood § | - | - | - | - | - | - | - | - | 3.3E-7 | 33 |

Source: [Final Vapor Intrusion Investigation Report – Fair Lawn Well Field Superfund Site – Langan Engineering and Environmental Services Inc. June 2018]
Abbreviations: adjusted EPC = the exposure point concentration (EPC) times the appropriate exposure factors; µg/m$^3$ = micrograms per meter cubed; ppb = parts per billion; CTE = central tendency exposure (typical); RME = reasonable maximum exposure (higher); yrs = years
* The calculations in this table were generated using ATSDR's PHAST v2.5.0.0. The noncancer hazard quotients were calculated using the chronic (greater than 1 year) minimal risk level of 41 µg/m$^3$ and the cancer risks were calculated using the inhalation unit risk of 2.6E-7 (µg/m$^3$)$^{-1}$.
§ This cancer risk represents a scenario where children are likely to continue to live in their childhood home as adults.

**Table D7. PHAST Default Input Parameters and Equations – Occupational Indoor Air Exposures**

PUBLIC HEALTH ASSESSMENT SITE TOOL

## Default Input Parameters and Equations – Occupational Indoor Air Exposures

### Equations

**Air Inhalation Exposure Equation**

**Adjusted EPC = EPC x $EF_{noncancer}$** **Equation 1**

EPC = exposure point concentration, $EF_{noncancer}$ = exposure factor (unitless)

**Hazard Quotient**

**HQ = Adjusted EPC ÷ HG** **Equation 2**

HQ = hazard quotient, EPC = exposure point concentration (µg/$m^3$ or ppb), HG = health guideline (e.g., inhalation MRL, RfC)

**Cancer Risk Equations**

**CR = (Adjusted EPC x IUR) x (ED ÷ LY) for each exposure group** **Equation 3**

**ADAF-adjusted CR = (Adjusted EPC x IUR) x (ED ÷ LY) x ADAF for each exposure group** **Equation 4**

**Total CR = Sum of the CR for all exposure groups** **Equation 5**

CR = cancer risk (unitless), EPC = exposure point concentration (µg/$m^3$ or ppb), IUR = inhalation unit risk ((µg/$m^3$ or ppb)$^{-1}$), ED = exposure duration (years), LY = lifetime years (78 years), ADAF = age-dependent adjustment factor (unitless), EF (cancer) = exposure factor (cancer) calculated as follows: EF (noncancer; unitless) x exposure group specific exposure duration (years) ÷ lifetime of 78 years

## Default Exposure Factors

| Exposure Group | Noncancer Exposure Factor Chronic CTE | Noncancer Exposure Factor Chronic RME | Noncancer Exposure Factor Intermediate CTE | Noncancer Exposure Factor Intermediate RME | Noncancer Exposure Factor Acute CTE | Noncancer Exposure Factor Acute RME |
|---|---|---|---|---|---|---|
| Full-time worker | 0.24 | 0.24 | 0.25 | 0.25 | 0.35 | 0.35 |
| Part-time worker | 0.15 | NA | 0.15 | NA | 0.21 | NA |

Abbreviations: CTE = central tendency exposure (typical); NA = not applicable; RME = reasonable maximum exposure (higher)
Cancer EFs are not shown in the table because they are calculated using age-specific durations. The general formula is $EF_{cancer} = EF_{noncancer} \times (ED_{age\text{-}specific\ (yrs)} \div 78\ years)$.

## Default Exposure Parameters

| Exposure Group | Daily (hours/day) CTE | Daily (hours/day) RME | Weekly (days/week) CTE | Weekly (days/week) RME | Annually (weeks/year) CTE | Annually (weeks/year) RME | Age-Specific Exposure Duration (years) CTE | Age-Specific Exposure Duration (years) RME |
|---|---|---|---|---|---|---|---|---|
| Full-time worker | 8.5 | 8.5 | 5 | 5 | 50 | 50 | 5 | 20 |
| Part-time worker | 5.1 | NA | 5 | NA | 50 | NA | 3.1 | NA |

Abbreviations: CTE = central tendency exposure (typical); RME = reasonable maximum exposure (higher)

## Contaminant Information

| Contaminant Name | Entered Concentration | EPC Type | Converted Concentration (µg/m³) | Converted Concentration (ppb) |
|---|---|---|---|---|
| Benzene | 1.089 µg/m³ | 95% UCL of the mean | 1.1 | 0.34 |
| Carbon tetrachloride | 1.4 µg/m³ | Maximum | 1.4 | 0.22 |
| Chloroform | 1 µg/m³ | Maximum | 1.0 | 0.20 |
| Tetrachloroethylene | 5.447 µg/m³ | 95% UCL of the mean | 5.4 | 0.80 |
| Trichloroethylene | 0.3965 µg/m³ | 95% UCL of the mean | 0.40 | 0.07 |

Abbreviations: µg/m³ = micrograms per meter cubed; EPC = exposure point concentration; UCL = upper confidence limit

## Air Inhalation Chronic (Default)

### Benzene

**Table D8. Occupational: Default exposure point concentrations for chronic exposure to benzene in air at 1.1 µg/m³ (0.34 ppb) along with noncancer hazard quotients and cancer risk estimates***

| Exposure Group | CTE Adjusted EPC (µg/m³) | CTE Adjusted EPC (ppb) | CTE Noncancer Hazard Quotient | CTE Cancer Risk | CTE Exposure Duration (yrs) | RME Adjusted EPC (µg/m³) | RME Adjusted EPC (ppb) | RME Noncancer Hazard Quotient | RME Cancer Risk | RME Exposure Duration (yrs) |
|---|---|---|---|---|---|---|---|---|---|---|
| Full-time worker | 0.26 | 0.083 | 0.041 | 1.3E-7 | 5 | 0.26 | 0.083 | 0.041 | 5.3E-7 | 20 |
| Part-time worker | 0.16 | 0.050 | 0.025 | 4.9E-8 | 3.1 | - | - | - | - | - |

Source: [Final Vapor Intrusion Investigation Report – Fair Lawn Well Field Superfund Site – Langan Engineering and Environmental Services Inc. June 2018]
Abbreviations: adjusted EPC = the exposure point concentration (EPC) times the appropriate exposure factors; µg/m³ = micrograms per meter cubed; ppb = parts per billion; CTE = central tendency exposure (typical); RME = reasonable maximum exposure (higher); yrs = years
* The calculations in this table were generated using ATSDR's PHAST v2.5.0.0. The noncancer hazard quotients were calculated using the chronic (greater than 1 year) minimal risk level of 6.4 µg/m³ and the cancer risks were calculated using the inhalation unit risk of 7.8E-6 $(\mu g/m^3)^{-1}$.

**Carbon tetrachloride**

**Table D9. Occupational: Default exposure point concentrations for chronic exposure to carbon tetrachloride in air at 1.4 µg/m³ (0.22 ppb) along with noncancer hazard quotients and cancer risk estimates***

| Exposure Group | CTE Adjusted EPC (µg/m³) | CTE Adjusted EPC (ppb) | CTE Noncancer Hazard Quotient | CTE Cancer Risk | CTE Exposure Duration (yrs) | RME Adjusted EPC (µg/m³) | RME Adjusted EPC (ppb) | RME Noncancer Hazard Quotient | RME Cancer Risk | RME Exposure Duration (yrs) |
|---|---|---|---|---|---|---|---|---|---|---|
| Full-time worker | 0.34 | 0.054 | 0.0034 | 1.3E-7 | 5 | 0.34 | 0.054 | 0.0034 | 5.2E-7 | 20 |
| Part-time worker | 0.20 | 0.032 | 0.0020 | 4.9E-8 | 3.1 | - | - | - | - | - |

Source: [Final Vapor Intrusion Investigation Report – Fair Lawn Well Field Superfund Site – Langan Engineering and Environmental Services Inc. June 2018]
Abbreviations: adjusted EPC = the exposure point concentration (EPC) times the appropriate exposure factors; µg/m³ = micrograms per meter cubed; ppb = parts per billion; CTE = central tendency exposure (typical); RME = reasonable maximum exposure (higher); yrs = years
* The calculations in this table were generated using ATSDR's PHAST v2.5.0.0. The noncancer hazard quotients were calculated using the chronic (greater than 1 year) reference concentration of 100 µg/m³ and the cancer risks were calculated using the inhalation unit risk of 6.0E-6 $(\mu g/m^3)^{-1}$.

**Chloroform**

**Table D10. Occupational: Default exposure point concentrations for chronic exposure to chloroform in air at 1.0 µg/m³ (0.20 ppb) along with noncancer hazard quotients and cancer risk estimates***

| Exposure Group | CTE Adjusted EPC (µg/m³) | CTE Adjusted EPC (ppb) | CTE Noncancer Hazard Quotient | CTE Cancer Risk | CTE Exposure Duration (yrs) | RME Adjusted EPC (µg/m³) | RME Adjusted EPC (ppb) | RME Noncancer Hazard Quotient | RME Cancer Risk | RME Exposure Duration (yrs) |
|---|---|---|---|---|---|---|---|---|---|---|
| Full-time worker | 0.24 | 0.050 | 0.12 | 3.6E-7 | 5 | 0.24 | 0.050 | 0.12 | 1.4E-6 ‡ | 20 |
| Part-time worker | 0.15 | 0.030 | 0.073 | 1.3E-7 | 3.1 | - | - | - | - | - |

Source: [Final Vapor Intrusion Investigation Report – Fair Lawn Well Field Superfund Site – Langan Engineering and Environmental Services Inc. June 2018]
Abbreviations: adjusted EPC = the exposure point concentration (EPC) times the appropriate exposure factors; µg/m³ = micrograms per meter cubed; ppb = parts per billion; CTE = central tendency exposure (typical); RME = reasonable maximum exposure (higher); yrs = years
* The calculations in this table were generated using ATSDR's PHAST v2.5.0.0. The noncancer hazard quotients were calculated using the chronic (greater than 1 year) minimal risk level of 2 µg/m³ and the cancer risks were calculated using the inhalation unit risk of 2.3E-5 $(\mu g/m^3)^{-1}$.
‡ Indicates that the cancer risk exceeds one extra case in a million people similarly exposed, which ATSDR evaluates further.

**Tetrachloroethylene**

**Table D11. Occupational: Default exposure point concentrations for chronic exposure to tetrachloroethylene in air at 5.4 µg/m³ (0.80 ppb) along with noncancer hazard quotients and cancer risk estimates***

| Exposure Group | CTE Adjusted EPC (µg/m³) | CTE Adjusted EPC (ppb) | CTE Noncancer Hazard Quotient | CTE Cancer Risk | CTE Exposure Duration (yrs) | RME Adjusted EPC (µg/m³) | RME Adjusted EPC (ppb) | RME Noncancer Hazard Quotient | RME Cancer Risk | RME Exposure Duration (yrs) |
|---|---|---|---|---|---|---|---|---|---|---|
| Full-time worker | 1.3 | 0.19 | 0.032 | 2.2E-8 | 5 | 1.3 | 0.19 | 0.032 | 8.8E-8 | 20 |
| Part-time worker | 0.79 | 0.12 | 0.019 | 8.2E-9 | 3.1 | - | - | - | - | - |

Source: [Final Vapor Intrusion Investigation Report – Fair Lawn Well Field Superfund Site – Langan Engineering and Environmental Services Inc. June 2018]
Abbreviations: adjusted EPC = the exposure point concentration (EPC) times the appropriate exposure factors; µg/m$^3$ = micrograms per meter cubed; ppb = parts per billion; CTE = central tendency exposure (typical); RME = reasonable maximum exposure (higher); yrs = years
* The calculations in this table were generated using ATSDR's PHAST v2.5.0.0. The noncancer hazard quotients were calculated using the chronic (greater than 1 year) minimal risk level of 41 µg/m$^3$ and the cancer risks were calculated using the inhalation unit risk of 2.6E-7 (µg/m$^3$)$^{-1}$.

**Trichloroethylene**

**Table D12. Occupational: Default exposure point concentrations for chronic exposure to trichloroethylene in air at 0.40 µg/m³ (0.07 ppb) along with noncancer hazard quotients***

| Exposure Group | CTE Adjusted EPC (µg/m³) | CTE Adjusted EPC (ppb) | CTE Noncancer Hazard Quotient | CTE Cancer Risk | CTE Exposure Duration (yrs) | RME Adjusted EPC (µg/m³) | RME Adjusted EPC (ppb) | RME Noncancer Hazard Quotient | RME Cancer Risk | RME Exposure Duration (yrs) |
|---|---|---|---|---|---|---|---|---|---|---|
| Full-time worker | 0.096 | 0.018 | 0.046 | 2.5E-8 | 5 | 0.096 | 0.018 | 0.046 | 1.0E-7 | 20 |
| Part-time worker | 0.058 | 0.011 | 0.027 | 9.4E-9 | 3.1 | - | - | - | - | - |

Source: [Final Vapor Intrusion Investigation Report – Fair Lawn Well Field Superfund Site – Langan Engineering and Environmental Services Inc. June 2018]
Abbreviations: adjusted EPC = the exposure point concentration (EPC) times the appropriate exposure factors; µg/m$^3$ = micrograms per meter cubed; ppb = parts per billion; CTE = central tendency exposure (typical); RME = reasonable maximum exposure (higher); yrs = years
* The calculations in this table were generated using ATSDR's PHAST v2.5.0.0. The noncancer hazard quotients were calculated using the chronic (greater than 1 year) minimal risk level of 2.1 µg/m$^3$ and the cancer risks were calculated using the inhalation unit risks of 2.1E-6 [NHL], 1.0E-6 [liver], 1.0E-6 [kidney] (µg/m$^3$)$^{-1}$ and age-dependent adjustment factors.

**Table D13. PHAST Default Input Parameters and Equations – 1,4 Dioxane and PFAS in Drinking Water – Residential Exposures**

PUBLIC HEALTH ASSESSMENT SITE TOOL

## Default Input Parameters and Equations – 1,4 Dioxane and PFAS in Drinking Water – Residential Exposures

### Equations

**Water Ingestion Exposure Dose Equation**

$$D_{noncancer} = (C \times IR \times EF_{noncancer}) \div BW \qquad \textbf{Equation 1}$$

$D_{noncancer}$ = dose (mg/kg/day), C = contaminant concentration (mg/L), IR = intake rate (L/day), $EF_{noncancer}$ = exposure factor (unitless), BW = body weight (kg)

**Hazard Quotient**

$$HQ = D_{noncancer} \div HG \qquad \textbf{Equation 2}$$

HQ = hazard quotient, $D_{noncancer}$ = dose (mg/kg/day), HG = health guideline (e.g., oral MRL, RfD)

**Cancer Risk Equations**

$$CR = (D_{noncancer} \times CSF) \times (ED \div LY) \text{ for each exposure group} \qquad \textbf{Equation 3}$$

$$\text{ADAF-adjusted } CR = (D_{noncancer} \times CSF) \times (ED \div LY) \times ADAF \text{ for each exposure group} \qquad \textbf{Equation 4}$$

$$\text{Total } CR = \text{Sum of the CR for all exposure groups} \qquad \textbf{Equation 5}$$

CR = cancer risk (unitless), $D_{noncancer}$ = dose, CSF = oral cancer slope factor [$(mg/kg/day)^{-1}$], EF (cancer) = exposure factor (cancer) calculated as follows: EF (noncancer; unitless) x exposure group specific exposure duration (years) ÷ lifetime of 78 years, ADAF = age-dependent adjustment factor (unitless), ED = exposure duration (years), LY = lifetime years (78 years)

## Default Exposure Factors

| Duration Category | Days per Week | Weeks per Year | Years | Exposure Group Specific $EF_{noncancer}$ | Exposure Group Specific* $EF_{cancer}$ |
|---|---|---|---|---|---|
| **Acute** | - | - | - | 1 | - |
| **Intermediate** | 7 | - | - | 1 | - |
| **Chronic** | 7 | 52.14 | See exposure group specific exposure durations | 1 | = $EF_{noncancer}$ x ($ED_{age\text{-}specific\ (yrs)}$ ÷ 78 years) |

Abbreviations: EF = exposure factor; NC = not calculated
* Cancer risk is averaged over a lifetime of exposure (78 years).

## Default Exposure Parameters

| Exposure Group | Body Weight (kg) | CTE Exposure Duration (yrs) | CTE Intake Rate (liters/day) | RME Exposure Duration (yrs) | RME Intake Rate (liters/day) |
|---|---|---|---|---|---|
| Birth to < 1 year | 7.8 | 1 | 0.595 | 1 | 1.106 |
| 1 to < 2 years | 11.4 | 1 | 0.245 | 1 | 0.658 |
| 2 to < 6 years | 17.4 | 4 | 0.337 | 4 | 0.852 |
| 6 to < 11 years | 31.8 | 5 | 0.455 | 5 | 1.258 |
| 11 to < 16 years | 56.8 | 1 | 0.562 | 5 | 1.761 |
| 16 to < 21 years | 71.6 | 0 | 0.722 | 5 | 2.214 |
| Total Child (all age groups) | - | 12 | - | 21 | - |
| Adult | 80 | 12 | 1.313 | 33 | 3.229 |
| Pregnant Women | 73 | - | 1.158 | - | 2.935 |
| Breastfeeding Women | 73 | - | 1.495 | - | 3.061 |

Abbreviations: CTE = central tendency exposure (typical); kg = kilograms; RME = reasonable maximum exposure (higher)

**Contaminant Information**

| Contaminant Name | Entered Concentration | EPC Type | Converted Concentration* |
|---|---|---|---|
| 1,4-dioxane | 0.74 µg/L | 95% UCL of the mean | 0.00074 mg/L |
| Perfluorooctanoic acid (PFOA) | 9.89 ng/L or ppt | 95% UCL of the mean | 9.89E-6 mg/L |
| Perfluorooctanesulfonic acid (PFOS) | 6.52 ng/L or ppt | 95% UCL of the mean | 6.52E-6 mg/L |

Abbreviations: µg/L = micrograms per liter; ng/L = nanograms per liter = parts per trillion (ppt); EPC = exposure point concentration; mg/L = milligram chemical per liter water; UCL = upper confidence limit

* Contaminant concentration converted to standard unit for calculating exposure.

## Drinking Water Ingestion Chronic (Default)

### 1,4-dioxane

**Table D14. Residential Default exposure doses for chronic exposure to 1,4-dioxane in drinking water at 0.00074 mg/L along with noncancer hazard quotients and cancer risk estimates***

| Exposure Group | CTE Dose (mg/kg/day) | CTE Noncancer Hazard Quotient | CTE Cancer Risk | CTE Exposure Duration (yrs) | RME Dose (mg/kg/day) | RME Noncancer Hazard Quotient | RME Cancer Risk | RME Exposure Duration (yrs) |
|---|---|---|---|---|---|---|---|---|
| Birth to < 1 year | 5.6E-5 | 0.00056 | - | 1 | 0.00010 | 0.0010 | - | 1 |
| 1 to < 2 years | 1.6E-5 | 0.00016 | - | 1 | 4.3E-5 | 0.00043 | - | 1 |
| 2 to < 6 years | 1.4E-5 | 0.00014 | - | 4 | 3.6E-5 | 0.00036 | - | 4 |
| 6 to < 11 years | 1.1E-5 | 0.00011 | - | 5 | 2.9E-5 | 0.00029 | - | 5 |
| 11 to < 16 years | 7.3E-6 | 7.3E-5 | - | 1 | 2.3E-5 | 0.00023 | - | 5 |
| 16 to < 21 years | 7.5E-6 | 7.5E-5 | - | 0 | 2.3E-5 | 0.00023 | - | 5 |
| Total Child | - | - | 2.4E-7 | 12 | - | - | 8.6E-7 | 21 |
| Adult | 1.2E-5 | 0.00012 | 1.9E-7 | 12 | 3.0E-5 | 0.00030 | 1.3E-6 ‡ | 33 |
| Pregnant Women | 1.2E-5 | 0.00012 | - | - | 3.0E-5 | 0.00030 | - | - |
| Breastfeeding Women | 1.5E-5 | 0.00015 | - | - | 3.1E-5 | 0.00031 | - | - |

Source: [NJDEP Drinking Water Watch, USEPA UCMR 3 and UCMR 5]

Abbreviations: CTE = central tendency exposure (typical); mg/kg/day = milligram chemical per kilogram body weight per day; mg/L = milligram chemical per liter water; RME = reasonable maximum exposure (higher); yrs = years

* The calculations in this table were generated using ATSDR's PHAST v2.5.0.0. The noncancer hazard quotients were calculated using the chronic (greater than 1 year) minimal risk level of 0.1 mg/kg/day and the cancer risks were calculated using the cancer slope factor of 0.1 $(mg/kg/day)^{-1}$.

‡ Indicates that the cancer risk exceeds one extra case in a million people similarly exposed, which ATSDR evaluates further.

## Drinking Water Ingestion Chronic (Default)

### Perfluorooctanoic acid (PFOA)

**Table D15. Residential Default exposure doses for chronic exposure to perfluorooctanoic acid (PFOA) in drinking water at 9.89E-6 mg/L along with noncancer hazard quotients***

| Exposure Group | CTE Dose (mg/kg/day) | CTE Noncancer Hazard Quotient | CTE Cancer Risk | CTE Exposure Duration (yrs) | RME Dose (mg/kg/day) | RME Noncancer Hazard Quotient | RME Cancer Risk | RME Exposure Duration (yrs) |
|---|---|---|---|---|---|---|---|---|
| Birth to < 1 year | 7.5E-7 | 25 † | - | 1 | 1.4E-6 | 47 † | - | 1 |
| 1 to < 2 years | 2.1E-7 | 7.1 † | - | 1 | 5.7E-7 | 19 † | - | 1 |
| 2 to < 6 years | 1.9E-7 | 6.4 † | - | 4 | 4.8E-7 | 16 † | - | 4 |
| 6 to < 11 years | 1.4E-7 | 4.7 † | - | 5 | 3.9E-7 | 13 † | - | 5 |
| 11 to < 16 years | 9.8E-8 | 3.3 † | - | 1 | 3.1E-7 | 10 † | - | 5 |
| 16 to < 21 years | 1.0E-7 | 3.3 † | - | 0 | 3.1E-7 | 10 † | - | 5 |
| Total Child | - | - | - | 12 | - | - | - | 21 |
| Adult | 1.6E-7 | 5.4 † | - | 12 | 4.0E-7 | 13 † | - | 33 |
| Pregnant Women | 1.6E-7 | 5.2 † | - | - | 4.0E-7 | 13 † | - | - |
| Breastfeeding Women | 2.0E-7 | 6.8 † | - | - | 4.1E-7 | 14 † | - | - |

Source: [NJDEP Drinking Water Watch, USEPA UCMR 3 and UCMR 5]

Abbreviations: CTE = central tendency exposure (typical); mg/kg/day = milligram chemical per kilogram body weight per day; mg/L = milligram chemical per liter water; RME = reasonable maximum exposure (higher); yrs = years

* The calculations in this table were generated using ATSDR's PHAST v2.5.0.0. The noncancer hazard quotients were calculated using the chronic (lifetime) reference dose of 3E-8 mg/kg/day.

† Indicates the hazard quotient is greater than 1, which ATSDR evaluates further.

**Perfluorooctanesulfonic acid (PFOS)**

**Table D16. Residential Default exposure doses for chronic exposure to perfluorooctane sulfonic acid (PFOS) in drinking water at 6.52E-6 mg/L along with noncancer hazard quotients***

| Exposure Group | CTE Dose (mg/kg/day) | CTE Noncancer Hazard Quotient | CTE Cancer Risk | CTE Exposure Duration (yrs) | RME Dose (mg/kg/day) | RME Noncancer Hazard Quotient | RME Cancer Risk | RME Exposure Duration (yrs) |
|---|---|---|---|---|---|---|---|---|
| Birth to < 1 year | 5.0E-7 | 5.0 † | - | 1 | 9.2E-7 | 9.2 † | - | 1 |
| 1 to < 2 years | 1.4E-7 | 1.4 † | - | 1 | 3.8E-7 | 3.8 † | - | 1 |
| 2 to < 6 years | 1.3E-7 | 1.3 † | - | 4 | 3.2E-7 | 3.2 † | - | 4 |
| 6 to < 11 years | 9.3E-8 | 0.93 | - | 5 | 2.6E-7 | 2.6 † | - | 5 |
| 11 to < 16 years | 6.5E-8 | 0.65 | - | 1 | 2.0E-7 | 2.0 † | - | 5 |
| 16 to < 21 years | 6.6E-8 | 0.66 | - | 0 | 2.0E-7 | 2.0 † | - | 5 |
| Total Child | - | - | - | 12 | - | - | - | 21 |
| Adult | 1.1E-7 | 1.1 † | - | 12 | 2.6E-7 | 2.6 † | - | 33 |
| Pregnant Women | 1.0E-7 | 1.0 † | - | - | 2.6E-7 | 2.6 † | - | - |
| Breastfeeding Women | 1.3E-7 | 1.3 † | - | - | 2.7E-7 | 2.7 † | - | - |

Source: [NJDEP Drinking Water Watch, USEPA UCMR 3 and UCMR 5]

Abbreviations: CTE = central tendency exposure (typical); mg/kg/day = milligram chemical per kilogram body weight per day; mg/L = milligram chemical per liter water; RME = reasonable maximum exposure (higher); yrs = years

* The calculations in this table were generated using ATSDR's PHAST v2.5.0.0. The noncancer hazard quotients were calculated using the chronic (lifetime) reference dose of 1E-7 mg/kg/day.

† Indicates the hazard quotient is greater than 1, which ATSDR evaluates further.

**Table D17. PHAST Wading in Little Diamond Brook - Site-specific Input Parameters and Equations**

PUBLIC HEALTH ASSESSMENT SITE TOOL

## Wading in Little Diamond Brook - Site-specific Input Parameters and Equations

### Equations

**Administered Dermal Dose Equation**

$$ADD_{noncancer} = (DA_{event} \times SA \times EV \times EF_{noncancer}) \div (BW \times ABS_{GI}) \quad \textbf{Equation 1}$$

$ADD_{noncancer}$ = administered dermal dose (mg/kg/day), $DA_{event}$ = absorbed dose per event (mg/cm$^2$/event), SA = skin surface area available for contact (cm$^2$), EV = event frequency (events/day), $EF_{noncancer}$ = exposure factor (unitless), BW = body weight (kg), $ABS_{GI}$ = gastrointestinal absorption factor (unitless)

**Hazard Quotient**

$$HQ = D_{noncancer} \div HG \quad \textbf{Equation 2}$$

HQ = hazard quotient, $D_{noncancer}$ = dose (mg/kg/day), HG = health guideline (e.g., oral MRL, RfD)

**Cancer Risk Equations**

$$CR = (D_{noncancer} \times CSF) \times (ED \div LY) \text{ for each exposure group} \quad \textbf{Equation 3}$$

$$\text{ADAF-adjusted } CR = (D_{noncancer} \times CSF) \times (ED \div LY) \times ADAF \text{ for each exposure group} \quad \textbf{Equation 4}$$

$$\text{Total } CR = \text{Sum of the CR for all exposure groups} \quad \textbf{Equation 5}$$

CR = cancer risk (unitless), $D_{noncancer}$ = dose, CSF = oral cancer slope factor [(mg/kg/day)$^{-1}$], EF (cancer) = exposure factor (cancer) calculated as follows: EF (noncancer; unitless) x exposure group specific exposure duration (years) ÷ lifetime of 78 years, ADAF = age-dependent adjustment factor (unitless), ED = exposure duration (years), LY = lifetime years (78 years)

## Site-specific Exposure Factors

| Duration Category | Event Duration (hours/event) | Event Frequency (events/day) | Days per Week | Weeks per Year | Years | Exposure Group Specific $EF_{noncancer}$ | Exposure Group Specific* $EF_{cancer}$ |
|---|---|---|---|---|---|---|---|
| **Acute** | 1 | 1 | - | - | - | 1 | - |
| **Intermediate** | 1 | 1 | 2 | 12 | - | 0.29 | - |
| **Chronic** | 1 | 1 | 2 | 12 | 33 | 0.066 | = $EF_{noncancer}$ x ($ED_{age\text{-}specific\ (yrs)}$ ÷ 78 years) |

Abbreviations: EF = exposure factor; NC = not calculated
* Cancer risk is averaged over a lifetime of exposure (78 years).

## Site-specific Exposure Parameters

| Exposure Group | Body Weight (kg) | Exposure Duration (years) | Combined Skin Surface Area ($cm^2$) | Notes |
|---|---|---|---|---|
| 6 to < 11 years | 31.8 | 5 | 3,824 | - |
| 11 to < 16 years | 56.8 | 5 | 5,454 | - |
| 16 to < 21 years | 71.6 | 5 | 6,083 | - |
| Total Child (all age groups) | - | 15 | - | - |
| Adult | 80 | 33 | 7,325 | - |

Abbreviations: $cm^2$ = centimeters square skin; kg = kilograms

## Contaminant Information

| Contaminant Name | Entered Concentration | EPC Type | Converted Concentration* | $ABS_{GI}$ | $DA_{event}$ |
|---|---|---|---|---|---|
| 1,4-dioxane | 1.02 ppb | 95% UCL of the mean | 0.00102 mg/L | 1 | 5.6E-10 mg/cm²/event |
| Carbon tetrachloride | 1.44 ppb | 95% UCL of the mean | 0.00144 mg/L | 1 | 5.67E-8 mg/cm²/event |
| Tetrachloroethylene | 2.11 ppb | 95% UCL of the mean | 0.00211 mg/L | 1 | 1.84E-7 mg/cm²/event |

Abbreviations: $ABS_{GI}$ = gastrointestinal absorption factor; $DA_{event}$ = absorbed dose per event; EPC = exposure point concentration; mg/cm²/event = milligrams per centimeter squared per event; mg/L = milligram chemical per liter water; ppb = parts per billion; UCL = upper confidence limit

* Contaminant concentration converted to standard unit for calculating exposure.

**Table D18. PHAST Site Specific Surface Water Wading Results – Little Diamond Brook**

## Site Specific Surface Water Wading Results – Little Diamond Brook

**Surface Water Dermal (skin): Chronic Exposures**

**1,4-dioxane**

**Table D19. Wading: Site-specific dermal exposure doses for chronic exposure to 1,4-dioxane in surface water at 0.00102 mg/L along with noncancer hazard quotients and cancer risk estimates***

| Exposure Group | Dose (mg/kg/day) | Noncancer Hazard Quotient | Cancer Risk | Exposure Duration (yrs) |
|---|---|---|---|---|
| 6 to < 11 years | 4.4E-9 | 4.4E-8 | - | 5 |
| 11 to < 16 years | 3.5E-9 | 3.5E-8 | - | 5 |
| 16 to < 21 years | 3.1E-9 | 3.1E-8 | - | 5 |
| Total Child | - | - | 7.1E-11 | 15 |
| Adult | 3.4E-9 | 3.4E-8 | 1.4E-10 | 33 |

Source: [Final Remedial Investigation Report – Fair Lawn Well Field Superfund Site, Langan Engineering and Environmental Services Inc. June 2018]
Abbreviations: mg/kg/day = milligram chemical per kilogram body weight per day; mg/L = milligram chemical per liter water; yrs = years
* The calculations in this table were generated using ATSDR's PHAST v2.5.1.0. The noncancer hazard quotients were calculated using the chronic (greater than 1 year) minimal risk level of 0.1 mg/kg/day and the cancer risks were calculated using the cancer slope factor of 0.1 $(mg/kg/day)^{-1}$.

**Carbon tetrachloride**

**Table D20. Wading: Site-specific dermal exposure doses for chronic exposure to carbon tetrachloride in surface water at 0.00144 mg/L along with noncancer hazard quotients and cancer risk estimates***

| Exposure Group | Dose (mg/kg/day) | Noncancer Hazard Quotient | Cancer Risk | Exposure Duration (yrs) |
|---|---|---|---|---|
| 6 to < 11 years | 4.5E-7 | 0.00011 | - | 5 |
| 11 to < 16 years | 3.6E-7 | 8.9E-5 | - | 5 |
| 16 to < 21 years | 3.2E-7 | 7.9E-5 | - | 5 |
| Total Child | - | - | 5.0E-9 | 15 |
| Adult | 3.4E-7 | 8.5E-5 | 1.0E-8 | 33 |

Source: [Final Remedial Investigation Report – Fair Lawn Well Field Superfund Site, Langan Engineering and Environmental Services Inc. June 2018]
Abbreviations: mg/kg/day = milligram chemical per kilogram body weight per day; mg/L = milligram chemical per liter water; yrs = years
* The calculations in this table were generated using ATSDR's PHAST v2.5.1.0. The noncancer hazard quotients were calculated using the chronic (lifetime) reference dose of 0.004 mg/kg/day and the cancer risks were calculated using the cancer slope factor of 0.07 $(mg/kg/day)^{-1}$.

**Tetrachloroethylene**

**Table D21. Wading: Site-specific dermal exposure doses for chronic exposure to tetrachloroethylene in surface water at 0.00211 mg/L along with noncancer hazard quotients and cancer risk estimates***

| Exposure Group | Dose (mg/kg/day) | Noncancer Hazard Quotient | Cancer Risk | Exposure Duration (yrs) |
|---|---|---|---|---|
| 6 to < 11 years | 1.5E-6 | 0.00018 | - | 5 |
| 11 to < 16 years | 1.2E-6 | 0.00015 | - | 5 |
| 16 to < 21 years | 1.0E-6 | 0.00013 | - | 5 |
| Total Child | - | - | 4.9E-10 | 15 |
| Adult | 1.1E-6 | 0.00014 | 9.8E-10 | 33 |

Source: [Final Remedial Investigation Report – Fair Lawn Well Field Superfund Site, Langan Engineering and Environmental Services Inc. June 2018]
Abbreviations: mg/kg/day = milligram chemical per kilogram body weight per day; mg/L = milligram chemical per liter water; yrs = years
* The calculations in this table were generated using ATSDR's PHAST v2.5.1.0. The noncancer hazard quotients were calculated using the chronic (greater than 1 year) minimal risk level of 0.008 mg/kg/day and the cancer risks were calculated using the cancer slope factor of 0.0021 $(mg/kg/day)^{-1}$.