# Letter Health Consultation

VOLATILE ORGANIC COMPOUNDS IN INDOOR AIR AT A CHILDCARE FACILITY

PHILADELPHIA, PHILADELPHIA COUNTY, PENNSYLVANIA

Evaluation of 2022 Indoor Air and Sub-Slab Soil Gas Data at a Childcare Facility

January 17, 2024

Bureau of Epidemiology, Division of Environmental Health Epidemiology
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Pennsylvania Department of Health
Division of Environmental Health Epidemiology
Harrisburg, PA

January 17, 2024

Madalyn Kulas
Senior Project Manager
The VERTEX Companies, LLC
3322 Route 22 West, Suite 907
Branchburg, NJ 08876

Re: Review of VERTEX 2022 sub-slab soil gas and indoor air data at a Philadelphia childcare facility

Dear Madalyn Kulas,

The Pennsylvania Department of Health (PADOH) evaluated the sub-slab soil gas and indoor air data that you shared with us on March 6, 2023, and prepared this Letter Health Consultation (LHC). Although acrolein was the contaminant of concern that you requested us to evaluate, as a health protective measure we also evaluated the other contaminants that were reported/included in the dataset. Based on our evaluation of the data for potential health effects, we provided conclusions and recommendations to protect human health. PADOH worked on this evaluation under a cooperative agreement with the Agency for Toxic Substances and Disease Registry (ATSDR), although the conclusions of this report are solely those of PADOH.

The focus of this LHC is on indoor air volatile organic compounds (VOCs) at the childcare center, which were sampled by VERTEX on one day each during January, February, March, and November of 2022. PADOH screened all VOCs and evaluated any VOC that exceeded a health-based screening value (which PADOH designated as a "potential contaminant of concern"). PADOH assessed the potential for cancer and non-cancer health effects for each potential contaminant of concern (COC). As a public health protective approach, PADOH used the maximum and/or most representative detected value of each COC to estimate exposures and assumed exposures occurred for 12 hours per day, 5 days per week, and 52 weeks per year among children and staff.

**Conclusions**

Based on the concentrations of VOCs detected in 2022, and assuming that they are representative of daily exposures at the center, current and future adverse non-cancer health effects are unlikely to occur. Concentrations of nearly all COCs, including acrolein, were lower than or similar to VOC levels typically found in indoor air. The estimated lifetime excess cancer

risks for COCs detected in indoor air were similar to or lower than what would be expected from typical indoor settings without known sources of contamination.

PADOH did find one detection of trichloroethylene (TCE) on January 25, 2022, which could have harmed the health of pregnant staff (specifically, a developing fetus). TCE was not detected in subsequent sampling of the center's indoor air for the rest of 2022, including on the next sampling date (February 16, 2022). TCE was detected in sub-slab soil gas in January 2022, but was lower than the indoor air detected concentration. Typically, to confirm a vapor intrusion pathway, the concentration of indoor air of a contaminant would be lower than the sub-slab soil gas concentration; therefore, PADOH cannot confirm at this time if the TCE detected in the center is from the vapor intrusion pathway or another indoor air source. Additionally, PADOH cannot draw conclusions for TCE exposure for periods where data were missing or incomplete (i.e., before January 25, 2022 or between January 25 and February 16, 2022).

Specific to your request and concern about acrolein, indoor air levels were 1-2 orders of magnitude (80-115 times) below adverse effect levels and lower than concentrations reported in other indoor settings.

**Limitations**

- Acrolein is a compound that is difficult to measure in air. This is due to potential issues with the air sampling canisters, calibration standards, and the time it takes to analyze a sample. In this dataset, air canisters were batch-certified prior to sample collection. However, without individually certifying each canister, it is possible there was acrolein contamination that could potentially result in an acrolein concentration that is overestimated. Other indoor air sources of acrolein, such as cooking oil at high temperatures, could also contribute to detections of acrolein.
- Indoor air contaminants can fluctuate over the course of a day, week, or season. While there are data for one day per month for a few months, there was no indoor air data representative of summer and fall months; therefore, the seasonality of indoor air could not be fully evaluated. VERTEX consulted a contractor in July 2022 to begin Heating, Ventilation, and Air Conditioning (HVAC) system and other building adjustments, which may have affected summer and fall sampling.
- Sub-slab soil gas was taken on a single date in January 2022, whereas indoor air sampling took place for 4 dates in total (January, February, March, and November 2022). The single round of sub-slab soil gas sampling occurred one day later (January 26) than the initial indoor air sampling event (January 25). Ideally, soil gas samples should be collected at the same time as indoor air sampling because soil gas measurements can vary [ATSDR 2016].
- Samples taken pre-building mitigation were of longer duration (8 hours for each of the 3 dates of sampling) and likely a better representation of indoor air quality than post building-mitigation samples (two 30-minute grab samples taken on a single sampling date).

- Indoor air sampling was limited to 4 dates and PADOH currently does not have data to assess whether TCE or other contaminants were present at the center on dates outside the sampling period and were at levels that could harm health.

**Recommendations**

As a precautionary measure, PADOH recommends additional periodic sampling that is representative of a full day's exposure during all seasons to ensure TCE levels remain non-detect or below the Pennsylvania Department of Environmental Protection (PADEP) Land Recycling Program's Act 2 vapor intrusion statewide screening standard for TCE in residential indoor air, of 2.1 µg/$m^3$. Should levels exceed 2.1 µg/$m^3$, PADOH recommends that VERTEX immediately notify PADOH so that measures can be taken to address the exposures and inform building occupants, particularly people who might be pregnant.

Any additional sub-slab soil gas sampling should ideally occur on the same date as indoor air sampling.

The exposure to acrolein at levels detected in indoor air at the center are unlikely to result in harmful health effects. However, acrolein is a difficult chemical to measure in air, and any future acrolein sampling should ensure canisters are clean and individually certified and instruments have been calibrated according to EPA guidelines.

PADOH acknowledges the due diligence performed by VERTEX to sample and improve indoor air quality at the center.

Attached to this letter ("Attachment A") is the assessment supporting our findings. If you have any questions about this LHC, please contact me at 717-787-3350 or by email at nmccray@pa.gov.

Sincerely,

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Division of Environmental Health Epidemiology
Pennsylvania Department of Health

cc: Julie V. Miller, PhD, DABT, Public Health Toxicologist, PADOH
Anil Nair, PhD, Director, Division of Environmental Health Epidemiology, PADOH

# ATTACHEMENT A: DATA REVIEW AND HEALTH EVALUATION

## Contents

## 1. Site Background and Environmental Sampling

As mentioned in VERTEX's Project No. 79883 report, the childcare center is located in Philadelphia, Pennsylvania and shares building space with a larger storage unit business. The two businesses do not share rooms or Heat, Ventilation and Air Conditioning (HVAC) systems [The VERTEX Companies, LLC 2023]. For the purpose of this LHC, references to "the site" refer to the entire building space while references to the "center" refer solely to the childcare center.

Near the site are several commercial businesses including a gym, clothing store, warehouse, and hospice center. Also nearby are a delivery station, truck center, distribution centers, and residential buildings. To the site's northeast is Northeast Philadelphia Airport. It is Pennsylvania's fourth busiest airport, with most aircraft consisting of single-engine, twins, jets, turboprops and helicopters ["Northeast Philadelphia Airport," n.d.].

The storage unit business purchased the site in March 2022. As part of due diligence involving the purchasing, a Phase I Environmental Site Assessment (ESA) was performed prior to acquisition of the property [The VERTEX Companies, LLC 2023]. The ESA included a Phase II Limited Site Investigation (LSI), conducted from January – November 2022, to assess historic uses and operations on the subject property and its proximity to Northeast Philadelphia Airport. Historic site uses included box and die manufacturing, auto repair, and industrial use. The site property also had underground storage tanks containing heating oil that were lawfully decommissioned. The 2022 LSI found no recognized environmental conditions associated with these historic operations [The VERTEX Companies, LLC 2023].

The LSI included sampling of site indoor air, sub-slab soil gas, and outdoor ambient air for volatile organic compounds (VOCs). The primary focus of this LHC are the indoor air results from the LSI at the childcare center, due to the nature of its operations and occupancy.

## 2. Subsurface Slab, Indoor Air, and Outdoor Air Sampling

The LSI was conducted from January through November 2022. It was performed in general conformance with the Vapor Intrusion section of the Pennsylvania Department of Environmental Protection (PADEP) Land Recycling Program Technical Guidance Manual [The VERTEX Companies, LLC 2023].

The first round of indoor air sampling occurred on January 25, 2022. Eight-hour samples were collected using 2.7 liter Summa canisters to mimic a typical work period at the center and potential exposure duration for children and employees. At the childcare center, indoor air samples were collected at a front/registration desk entrance area (VIA-5) and in a classroom/common area open and accessible to an adjacent, small kitchen (VIA-6). This kitchen at VIA-6 contains an electric range, microwave, and warming oven.

The next day (January 26, 2022) a single round of sub-slab soil gas was collected. Sub-slab samples were collected at 7 site locations including 3 beneath the childcare center. Sub-slab

samples VSG-5 and VSG-6 were collocated with the indoor air samples taken the previous day (VIA-5 and VIA-6). A third sub-slab soil gas sample was also collected, beneath a hallway of the center with a small kitchenette (VSG-7). The samples were collected over a 15-minute duration using 2.7 liter Summa canisters. No additional sub-slab soil gas sampling occurred after January 26, 2022.

Based on the sub-slab soil gas results on January 26, 2022, and presumably because there were some detected compounds that exceeded sub-slab soil gas screening levels, laboratory analysis was activated for the indoor air samples collected the previous day. Samples were analyzed using the United States Environmental Protection Agency (EPA) Method TO-15 for VOCs.

Subsequent/confirmatory 8-hour indoor air sampling occurred on two additional dates: February 16 and March 31, 2022, per the methods described above. Prior to the February 16 sampling, 2 of the 4 center HVAC units were modified to maximize fresh air intake, and the remaining 2 units were modified to add a fresh air intake. The February 16 and March 31 samples were also taken at the center's front/registration desk (VIA-5) and classroom/small kitchen area (VIA-6), as well as the hallway area with small kitchenette (VIA-7). The kitchenette of sample VIA-7 is separate from the small kitchen of VIA-6 and contains a sink but no microwave or oven.

Nearby outdoor air sampling also occurred on the same dates as indoor air sampling but only at the storage facility.

During the initial sampling period (January – March 2022), VERTEX noted that the VOC acrolein was consistently detected in indoor air above Pennsylvania Department of Environmental Protection (PADEP) residential and non-residential screening values. Therefore, efforts were made to mitigate acrolein indoor air levels at the childcare center.

In July 2022, VERTEX consulted with a contractor familiar with site's HVAC systems. During the following months, several adjustments were made to the childcare center, including:

1. Installing activated carbon/potassium permanganate blend air filters with a particulate filter into the 4 HVAC units serving the daycare area (installed on November 3, 2022).
2. Further modifying the HVAC units to ensure maximum amounts of fresh air intake.
3. Checking and configuring HVAC system and return lines. Any issues with the configuration or connections were noted, and disconnected supply and return ducts were repaired on November 3, 2022.
4. Checking building pressure.
5. Using a photoionization detector to examine penetrations in floor slab for the presence of VOCs (which were inconclusive and did not identify a source of acrolein).
6. Evaluating indoor unvented/open flame combustion sources, of which none were noted.

On November 22, 2022, a round of post-modification indoor air sampling occurred (sample IDs VIA-25, VIA-26 and VIA-27) at the 3 center locations described previously (VIA-5, VIA-6, and VIA-7). Two grab samples were taken at each location – a daytime sample from 12:40-1:10 p.m. to represent heavier traffic conditions of the nearby Northeast Philadelphia Airport, and an evening sample from 10:40-11:10 p.m. to represent lighter traffic conditions. Two grab samples of outdoor air were also collected at these time points, at a doorway near the ground to mimic air entering through the doorway, and near the rooftop, approximately 16 feet above ground, to mimic ambient air possibly entering the building through the HVAC units [The VERTEX Companies, LLC 2023].

Post-mitigation acrolein was detected above PADEP residential and nonresidential standards at slightly higher concentrations than pre-mitigation levels. Post-mitigation indoor acrolein was slightly higher than outdoor acrolein, with outdoor concentrations at the rooftop higher than outdoor concentrations at the doorway.

PADOH reviewed concentrations of acrolein and other compounds sampled at the childcare center to determine whether they could harm health. PADOH referred to guidance from ATSDR's public health assessment guidance manual [ATSDR 2023a].

## 3. Exposure Pathway Analysis (Vapor Intrusion)

Detectable vapor-forming chemicals in indoor air can come from indoor and outdoor sources. Vapor intrusion is the migration of vapor-forming chemicals and gases from any subsurface source, such as contaminated groundwater or soil, into indoor air [PADOH 2017]. If vapor intrusion is occurring, the measured concentration of a contaminant will lessen as it migrates from a subsurface source to sub-slab soil gas and then to indoor air, provided there are no other indoor sources of that contaminant [PADOH 2017]. The presence of indoor air contaminants does not necessarily indicate that vapor intrusion is occurring; common indoor sources of contaminants can include household cleaning products, stored fuels, furniture, flooring, and dry-cleaned clothing [ATSDR 2016].

PADOH conducted an exposure pathway analysis to determine whether human exposure from vapor intrusion could have occurred (past exposure), may be occurring (present exposure) or could occur (future exposure) at the center. Exposure pathway analyses consist of five components: a **source, environmental fate and transport,** an **exposure point,** an **exposure route,** and a **receptor population.** Exposure pathways are designated as "completed" if all five components are present, "potential" if one component is missing but cannot be eliminated, or "eliminated" if one or more component is missing and will never be present. Compounds in completed or potential pathways are evaluated further [ATSDR 2023a].

For this site, PADOH determined that a **potential exposure pathway** existed for past, present, and future vapor intrusion exposures, with the one component missing being the specific source. VOCs were detected in sub-slab soil gas; however, they were also present in nearby

ambient air. While most detected VOCs were lower in indoor air than in sub-slab soil gas, concentrations for some indoor VOCs exceeded sub-slab soil gas, and some VOCs detected in sub-slab soil gas were not detected in indoor air. Thus, the true source of the compounds is unknown. The remaining components of the exposure pathway were present:

1. **Source:** Although specific source is unknown, compounds were found in the sub-slab soil gas and in ambient air at the site.
2. **Environmental Fate and Transport:** Contaminants in sub-slab soil gas and ambient air can be transferred into indoor spaces.
3. **Exposure Point:** People could be exposed at the childcare center if vapors enter the facility.
4. **Exposure Route:** People could inhale the indoor air contaminants that may be present.
5. **Receptor Population:** Children and staff at the childcare center are the receptor populations.

PADOH then screened VOCs within this potential exposure pathway to determine which VOCs required further evaluation.

## 4. PADOH Screening and Health Effect Evaluation Process

To determine which VOCs required further investigation, PADOH compared their maximum concentrations to Agency for Toxic Substances and Disease Registry (ATSDR) comparison values (CVs). CVs are chemical levels in air, water and soil that are used for screening purposes. They are non-site specific. Contaminant levels below CVs are not expected to harm health. Contaminant levels above CVs are further assessed to see whether harmful health effects could occur. For evaluation of the childcare center, PADOH assessed ATSDR CVs for air and sub-slab soil gas. PADOH used ATSDR's Public Health Assessment Site Tool (PHAST) to screen compounds.

There are ATSDR CVs for cancer and non-cancer effects. Non-cancer CVs are called ATSDR Environmental Media Evaluation Guides (EMEGs) and represent acute (≤14 day), intermediate (15-364 days) or chronic (a year or longer) exposures. Air EMEGs are derived from ATSDR Minimal Risk Levels (MRLs) for inhalation, using default exposure assumptions. Reference Dose Media Evaluation Guides (RMEG) are derived from EPA Reference Concentrations (RfCs) representing lifetime exposures. Contaminant concentrations below EMEGs and RMEGs are also below respective MRLs and RfCs and are not expected to cause adverse non-cancer health effects.

Cancer CVs are called cancer Risk Evaluation Guides (CREGs). Concentrations of a cancer-causing substance below a CREG are unlikely to result in an increased cancer risk at 1 person per 1 million people exposed for a lifetime (a default estimate of 78 years).

PADOH also examined ATSDR sub-slab soil gas CVs, which are derived from compounds that have an air CV and a Henry's Law Constant. Further information on their derivation is provided in ATSDR 2016.

For indoor air VOCs that 1) exceeded an ATSDR CV, 2) exceeded a Pennsylvania Department of Environmental Protection (PADEP) screening value, or 3) lacked a screening value, PADOH deemed them **potential contaminants of concern (COCs)** and evaluated them further. This included VOCs that were undetected but for which the analytical method detection limit (MDL) exceeded a CV. For each COC PADOH calculated an adjusted exposure point concentration (EPC) to determine whether the detected contaminant could harm health among center occupants: children from birth through 5 years of age, and full-time adult workers.

## 4.1. Exposure Assumptions

As a public health protective approach, PADOH calculated adjusted exposure point concentrations (EPCs) based on the maximum or most representative concentration for each COC. If pre- and post-mitigation concentrations for a COC were similar, PADOH used pre-mitigation concentrations as basis for its calculations because the 8-hour sampling duration on these dates was more representative of daily exposure than the 30-minute post-mitigation grab samples that were taken once during the day and once in the evening. For acrolein and trichloroethylene, PADOH calculated an adjusted EPC based on their maximum concentrations, as a "worst case scenario" approach.

PADOH adjusted maximum/most representative COC concentrations by childcare center hours. Appendix 1 explains this calculation.

PADOH assumed a maximum, "worst-case" childcare exposure scenario of 12 hours per day, 5 days a week, for 52 weeks per year. PADOH acknowledges that this scenario is unlikely; a 12 hour per day exposure scenario exceeds the center's hours of operation (M-F, 6:30 a.m.-6 p.m.). It also slightly exceeds a default Reasonable Maximum Exposure (RME) assumption for childcare centers of 11.8 hours a day for young children (birth to <1 years of age) and for full-time adult workers. Although this scenario is unlikely, these exposures could potentially occur if children, or more likely an adult worker, are at the center longer than its operating hours (11.5 hours per day), and year-round (52 weeks). **As the scenarios presented in this report represent this "worst-case" assumption, children and staff attending the childcare center for *less than* 12 hours per day and *less than* 52 weeks per year would have lower exposure estimates than calculations presented in this report.** Appendix 1 provides an example of how EPCs are lowered if assuming less than 12 hours per day exposure.

## 4.2. Assessing the Potential for Adverse Health Effects

To determine the potential for non-cancer effects, PADOH compared adjusted EPCs to chronic, intermediate and acute ATSDR inhalation MRLs and EPA chronic reference concentrations (RfCs). A contaminant's adjusted EPC divided by its health guideline produces a hazard quotient. PADOH further assessed COCs where adjusted EPCs produced hazard quotients > 1 by conducting a toxicological evaluation.

To determine the potential for cancer effects, PADOH calculated estimated lifetime excess cancer risk. For VOCs that are known or probable carcinogens (e.g., compounds that exceeded a

CREG CV), PADOH multiplied the adjusted EPC by EPA's Inhalation Unit Risk (IUR) for each COC and divided it by the estimated time (in years) spent during a lifetime at the daycare. PADOH used PHAST for this calculation and its default childcare center assumption of 6 years' exposure for a child (birth to 6 years of age) and 20 years' exposure for an adult full-time worker:

**Lifetime Excess Cancer Risk Calculation for Each Carcinogen COC =**

$$\frac{\text{Adjusted EPC} * \text{EPA IUR} * [(6 \text{ exposure years, child}) \text{ OR } (20 \text{ exposure years, adult})]}{78 \text{ years (default assumption for a lifetime)}}$$

*Sources: ATSDR 2023a; ATSDR 2021*

The results of this report assume that exposures remain constant to the detected (maximum or most representative) concentration. However, indoor air contaminant concentrations can fluctuate widely based on a variety of factors such as seasonality, air flow from HVAC systems, etc., so actual exposures may vary from this assumed scenario.

## 5. Screening Results

Table 1 shows the COCs for sub-slab soil gas and indoor air at the childcare center, based on their maximum concentrations and other considerations. Screening results for all VOCs can be found in Tables 1a and 1b (Appendix 2).

**Table 1. Potential Contaminant of Concern (COC) results based on their maximum detected concentrations in center sub-slab soil gas and indoor air**

| No. | COC | Why designated as a COC |
|---|---|---|
| 1 | 1,2-Dichloroethane | - Exceeded CREG CV for indoor air |
| 2 | 1,3-Butadiene | - Exceeded CREG CVs for sub-slab soil gas and indoor air |
| 3 | 1,4-Dioxane | - Exceeded CREG CVs for sub-slab soil gas and indoor air |
| 4 | Benzene | - Exceeded CREG CVs for sub-slab soil gas and indoor air |
| 5 | Carbon Tetrachloride | - Exceeded CREG CV for indoor air |
| 6 | Chloroform | - Exceeded CREG CV for indoor air |
| 7 | Acrylonitrile | - Exceeded CREG CV for sub-slab soil gas and indoor air |
| 8 | Naphthalene | - Exceeded CREG CV for sub-slab soil gas and indoor air |
| 9 | Vinyl Chloride* | - Was not detected, but the MDL exceeded CREG CV for indoor air |
| 10 | 1,2-Dibromoethane* | - Was not detected, but the MDL exceeded CREG CVs for sub-slab soil gas and indoor air |
| 11 | 1,3-Dichloropropane* | - Was not detected, but a current CV or screening value could not be located |
| 12 | Dibromochloromethane* | - Was not detected, but a current CV or screening value could not be located |
| 13 | Isopropanol | - Exceeded PADEP screening value for indoor air |
| 14 | Trichloroethylene (TCE) | - Exceeded CREG, cEMEG, iEMEG and RMEG CVs, and PADEP screening value for indoor air |

| | | |
|---|---|---|
| 15 | Acrolein | - Exceeded RMEG, iEMEG and PADEP screening value for sub-slab soil gas and indoor air<br>- Was petitioner concern due to consistent PADEP screening value exceedances |

*Not detected but deemed a COC for reasons described in the table. COC = Potential Contaminant of Concern; CV = Comparison Value; CREG = Cancer Risk Evaluation Guide CV (cancer effects); EMEG = Environmental Media Evaluation Guide (non-cancer effects); iEMEG = EMEG representing intermediate duration exposures (15-364 days); cEMEG = EMEG representing chronic duration exposures (1 year or more); MDL = method detection limit; RMEG = Reference Media Evaluation Guide for chronic exposures (non-cancer effects)

There were **15** CoCs (Table 1). Based on their maximum detected concentrations, there were **6** COCs for sub-slab soil gas **(1,3-butadiene, 1,4-dioxane, acrolein, benzene, naphthalene, and acrylonitrile)** and **11** COCs for indoor air **(1,2-dichloroethane, 1,3-butadiene, 1,4-dioxane, benzene, carbon tetrachloride, chloroform, acrylonitrile, naphthalene, isopropanol, trichloroethylene, and acrolein).**

Most commonly, the above compounds were designated COCs because of low CREG values that were also below the analytical MDL (shown further in Appendix 2 Tables 1a, 1b and 2a-2l). In addition, several COC concentrations were lab-estimated J-values. A J-value is a laboratory notation for a reported sample concentration that is estimated because a contaminant/analyte was detected above the MDL but below the quantification or reporting limit (Tables 2a-2l). Three indoor air COCs (1,2-dichloroethane, 1,3-butadiene, and acrylonitrile) were only detected post-mitigation.

With three exceptions—a single detection of **trichloroethylene** at 11.2 µg/$m^3$, **isopropanol** and **acrolein** – all detected COCs in indoor air were below non-cancer CVs or screening values.

There were **4** COCs that were **not** detected – **vinyl chloride, 1,2-dibromoethane, 1,3-dichloropropane** and **dibromochloromethane (Table 1)** – but were deemed COCs because the MDLs exceeded CVs (vinyl chloride and 1,2-dibromoethane), or because current CVs or screening values could not be located (1,3-dichloropropane and dibromochloromethane).

We assessed the potential for health effects for all of the above compounds, discussed in section 6 below. **The tables referenced in section 6 (Tables 1a-1b, 2a-2l, and 3a-3c) can be found in Appendix 2.** We also compared COC indoor concentrations to background levels typically found indoors and to outdoor levels measured at the site.

## 6. Public Health Implications – COCs Further Evaluated for Estimated Lifetime Excess Cancer Risk

The COCs discussed below exceeded CREGs but did not exceed non-cancer CVs, MRLs or RfCs. Therefore, **for these compounds below, non-cancer health effects are not expected to occur. The cancer risk estimates reported below assume exposures for 12 hours per day, 5 days per week, 52 weeks per year for 6 years (child) or for 20 years (full-time worker).** Estimates would be lower for exposures of shorter duration.

## 6.1 1,2-Dichloroethane

The maximum indoor air concentration of 1,2-dichloroethane at the childcare center was 0.441 (J) µg/m$^3$. It was a value detected in an evening, post-mitigation grab sample taken in the childcare classroom area with adjacent kitchen (sample ID: VIA-26). 1,2-Dichloroethane was not detected in pre-mitigation indoor air, in sub-slab soil gas, or in outdoor air. The lab estimated value of 0.441 (J) µg/m$^3$ exceeded ATSDR's CREG, of 0.038 µg/m$^3$ (Table 1b).

> 1,2-Dichloroethane is a man-made chemical primarily used to make plastic and vinyl products such as polyvinyl chloride (PVC) pipes [ATSDR 2022a]. It is also added to leaded gasoline that is used in aircrafts, racing vehicles and farm equipment. 1,2-Dichloroethane was previously used in consumer household products such as cleaning agents and adhesives but is generally no longer available for commercial purposes. In a survey of New Jersey and Pennsylvania homes by Heavner et al. 1996, 1,2-dichloroethane was detected at mean concentrations of 0.32 µg/m$^3$ (max: 9.72 µg/m$^3$) in the homes of smokers and 0.03 µg/m$^3$ (max 0.54 µg/m$^3$) in the homes of nonsmokers [ATSDR 2022b].
>
> The U.S. Department of Health and Human Services (DHHS) and U.S. Environmental Protection Agency (EPA) have classified 1,2-dichloroethane as a probable/reasonably anticipated to be carcinogenic to humans based on laboratory animal studies [ATSDR 2022b].

**The estimated lifetime excess cancer risk** from a 1,2-dichloroethane concentration of 0.441 (J) µg/m$^3$, adjusted for a childcare center exposure scenario (adjusted EPC: 0.16 µg/m$^3$) is **1.0E-6 (1 in 1 million) for a full-time adult worker and 3.2E-7 (3 in 10 million) for a child (Table 2a).**

The 1,2-dichloroethane detection at the center's indoor air was higher than concentrations found in non-smoking homes according to Heavner et al.'s 1996 study [ATSDR 2022b]. However, the center's concentration is lab-estimated, and the MDL (0.244 µg/m$^3$) also exceeds the background concentrations reported by Heavner et al. in indoor air.

## 6.2. 1,3-Butadiene

The maximum indoor air concentration of 1,3-butadiene was 0.168 (J) µg/m$^3$, which was a value detected in an evening sample and represents a post-mitigation grab sample taken in the hallway area with kitchenette (VIA-27). 1,3-Butadiene was not detected in pre-mitigation indoor air. It was detected in outdoor air at a slightly higher maximum concentration of 0.312 (J) µg/m$^3$. 1,3-Butadiene was detected in 2 of 3 sub-slab soil gas samples, the highest of which (3.12 µg/m$^3$) exceeded an ATSDR sub-slab CREG CV (1.1 µg/m$^3$, Table 1a). The highest indoor lab-estimated concentration of 0.168 (J) µg/m$^3$ exceeded ATSDR's air CREG of 0.033 µg/m$^3$ (Table 1b).

> 1,3-Butadiene is a chemical made from the processing of petroleum [ATSDR 2012a]. It has a mild gasoline-like odor. It is used to make man-made rubber that is then used for

car and truck tires, as well as certain plastics [ATSDR 2012b]. Common sources are automobile exhaust and cigarette and wood smoke. Large amounts are released by industrial sources; although in the atmosphere, 1,3-butadiene evaporates quickly with a short half-life of 6 hours. A 2004 study by Sax et al. found mean indoor air concentrations of 1.0 µg/m$^3$ and 1.2 µg/m$^3$, respectively, in homes in New York City in the winter and summer [ATSDR 2012b]. 1,3-Butadiene has been found at higher indoor concentrations in smoking settings.

DHHS, EPA and the International Agency for Research on Cancer (IARC) have classified 1,3-butadiene as a known human carcinogen based on sufficient evidence in humans (certain worker populations) and laboratory animals.

**The estimated lifetime excess cancer risk** from an indoor air 1,3-butadiene concentration of 0.168 (J) µg/m$^3$, adjusted for a childcare center exposure scenario (adjusted EPC: 0.06 µg/m$^3$) **is 4.6E-7 (5 in 10 million) for a full-time adult worker and 1.4E-7 (1 in 10 million) for a child (Table 2b).** The estimated concentrations for 1,3-butadiene at the center were lower than concentrations typically found in indoor air.

## 6.3. 1,4-Dioxane

The maximum 1,4-dioxane indoor air concentration was a single, pre-mitigation detection of 0.386 (J) µg/m$^3$ in the classroom area with adjacent kitchen (VIA-6) on March 31, 2022. It was not detected post-mitigation or in outdoor air. It was detected in sub-slab soil gas at a maximum of 8.58 µg/m$^3$ (VSG-6), which exceeded ATSDR's sub-slab CREG (6.7 µg/m$^3$, Table 1a). The indoor lab-estimated detection of 0.386 (J) µg/m$^3$ exceeded ATSDR's CREG air CV of 0.20 µg/m$^3$ (Table 1b).

1,4-Dioxane is used primarily as a solvent in the manufacture of other chemicals and as a laboratory reagent [ATSDR 2012c]. It has a pleasant odor and is a trace contaminant in some chemicals used in detergents, cosmetics and shampoos. Manufacturers reduce 1,4-dioxane in these chemicals to low levels before they are made into household products. In air, 1,4-dioxane rapidly breaks down into different compounds. Current background concentrations of 1,4-dioxane in air are unknown [ATSDR 2012d]. In the mid-1980s, average levels of the compound in air in U.S. air samples were 4 µg/m$^3$ for indoor air and 0.4 µg/m$^3$ for outdoor air [ATSDR 2012d].

The limited number of studies available do not show whether 1,4-dioxane causes cancer in humans [ATSDR 2012c]. DHHS has classified 1,4-dioxane as reasonably anticipated to be a human carcinogen based on some evidence in laboratory mice and rats. EPA has deemed it as likely to be carcinogenic to humans. Scientists have debated the degree to which 1,4-dioxane cancer findings in rats and mice apply to exposure situations commonly encountered by people.

**The estimated lifetime excess cancer risk** from a 1,4 dioxane concentration of 0.386 (J) µg/m$^3$, adjusted for a childcare center exposure scenario (adjusted EPC: 0.14 µg/m$^3$) is **1.8E-7 (2 in 10 million) for a full-time adult worker and 5.3E-8 (5 in 100 million) for a child (Table 2c).**

## 6.4. Benzene

The maximum indoor benzene concentration was 0.997 µg/m$^3$, which was a value detected in an evening, post-mitigation grab sample taken in the front-desk area (VIA-25). We assessed the pre-mitigation maximum of 0.888 µg/m$^3$ because it was an 8-hour sample taken in the classroom area with adjacent kitchen (sample VIA-6) and more representative of exposure. Outdoor benzene concentrations (maximum: 1.61 µg/m$^3$) were higher than indoor levels. In sub-slab soil gas, benzene was detected in 3 of 3 samples at a maximum of 16.5 µg/m$^3$ (VSG-7).

All benzene concentrations as well as the MDL exceeded ATSDR's CREG, of 0.13 µg/m$^3$ (Table 1b). Sub-slab gas samples VSG-6 (6.26 µg/m$^3$) and VSG-7 (16.5 µg/m$^3$) exceeded ATSDR's CREG for benzene sub-slab soil gas (4.3 µg/m$^3$, Table 1a).

> Benzene is a colorless liquid with a sweet odor that evaporates very quickly in air [ATSDR 2007a]. It is widely used in the U.S. and ranks in the top 20 chemicals for production volume. It is used to make other chemicals and some types of rubbers, lubricants, dyes, detergents, pesticides and drugs. Everyone is exposed to low amounts of benzene, which is found in tobacco smoke, automobile service stations, gasoline, exhaust from motor vehicles, and industrial emissions. It can also be released from certain household products and is found in cigarettes. The average smoker inhales about 10 times more benzene than non-smokers daily. Once in air, benzene reacts with other chemicals to break down within a few days [ATSDR 2007b]. EPA air monitoring in Philadelphia found median outdoor benzene levels at 1 ppb (3.19 µg/m$^3$) [ATSDR 2007b]. Indoor air means and medians have ranged around 0.64 ppb – 2.3 ppb (2.0 µg/m$^3$ to 7.3 µg/m$^3$). Higher levels have been found in homes of smokers or in smoke-filled restaurants.
>
> DHHS, EPA, the International Agency for Research on Cancer (IARC) and other agencies have classified benzene as a known human carcinogen. Long-term exposure to high levels in air can cause leukemia [ATSDR 2007b].

**The estimated lifetime excess cancer risk** from a benzene concentration of 0.888 µg/m$^3$, adjusted for a childcare exposure scenario (adjusted EPC: 0.32 µg/m$^3$) **is 6.3E-7 (6 in 10 million) for a full-time adult worker and 1.9E-7 (2 in 10 million) for a child (Table 2d).** Indoor benzene at the childcare center was lower than background levels reported in non-smoking homes.

## 6.5. Carbon tetrachloride

The maximum carbon tetrachloride indoor air concentration was 0.604 µg/m$^3$, which was taken pre-mitigation in the classroom area with adjacent kitchen (VIA-6). In sub-slab soil gas, carbon tetrachloride was detected in 3 of 3 locations at concentrations below ATSDR's soil gas vapor

intrusion CV (Table 1a). Outdoor air concentrations were similar to indoor air. All indoor air carbon tetrachloride concentrations as well as the MDL exceeded ATSDR's CREG CV for carbon tetrachloride, of 0.17 µg/m$^3$ (Table 1b).

> Carbon tetrachloride is a manufactured chemical with a sweet smell [ATSDR 2005a]. In air it is most often found as a colorless gas. In the past, it had a number of uses, such as a pesticide, in the production of refrigeration fluid, as a cleaning fluid and degreasing agent, in fire extinguishers, and in spot removers. These uses are now banned and it is currently only used in some industrial applications. It is a very stable compound in air (30-100 years) with very low background levels in the environment. Several studies have reported indoor air concentrations of carbon tetrachloride in U.S. homes to be 1 – 9 µg/m$^3$ (0.16 – 1.4 ppb) and are usually higher than outdoor air, indicating sources that may include building materials or products such as pesticides or cleaning agents [ATSDR 2005b].
>
> DHHS and EPA have classified carbon tetrachloride as reasonably/likely to be carcinogenic to humans, based on sufficient laboratory animal evidence. Studies in humans have not been able to determine whether carbon tetrachloride causes cancer because usually there has been exposure to other compounds [ATSDR 2005a].

**The estimated lifetime excess cancer risk** from a carbon tetrachloride concentration of 0.604 µg/m$^3$, adjusted for a childcare exposure scenario (adjusted EPC: 0.22 µg/m$^3$) **is 3.3E-7 (3 in 10 million) for a full-time adult worker and 1E-7 (1 in 10 million) for a child (Table 2e).** Carbon tetrachloride concentrations found in indoor air at the childcare center were lower than background levels reported in other scientific studies.

## 6.6. Chloroform

The maximum chloroform concentration was 3.37 µg/m$^3$, which was a value detected in a daytime, post-mitigation grab sample taken in the hallway area with kitchenette (VIA-27). This value was higher than the pre-mitigation maximum of 1.25 µg/m$^3$. We assessed the pre-mitigation maximum because it was an 8-hour sample detected in the classroom area with adjacent kitchen (sample VIA-6) and more representative of exposure. In sub-slab soil gas, chloroform was detected in 2 of 3 samples (VSG-5 and VSG-6) at levels below CVs. Chloroform was not detected in outdoor air.

All chloroform detections as well as the MDL exceeded ATSDR's CREG CV of 0.043 µg/m$^3$, and several detections exceeded PADEP's Act 2 Statewide Vapor Intrusion Screening Standard for Residential Indoor Air of 1.1 µg/m$^3$ (Table 1b). For non-cancer effects, PADOH prioritizes ATSDR CVs for chloroform, which range from 98-490 µg/m$^3$ (Table 1b) and were not exceeded.

> Chloroform is a colorless liquid that evaporates easily in air and has a pleasant, non-irritating odor [ATSDR 2014]. Its historical uses include as an anesthetic during surgery, but it is not used today for this purpose. Today it is used to make other chemicals and it

can also be formed in small amounts when chlorine is added to water. Chloroform can enter air directly from factories that make or use it and by evaporating from water and soil that contain it [ATSDR 1997]. One of the most significant indoor air sources of chloroform is chlorinated tap water or breathing air where it has been released from shower water. Concentrations are usually <1 – 25 times higher in indoor than in outdoor settings [ATSDR 1997]. Typical median indoor air concentrations range from approximately 0.2-4 ppb (0.98-19.6 µg/m$^3$) [ATSDR 1997].

DHHS and EPA have classified chloroform as "reasonably" and "likely" to be carcinogenic to humans, respectively, based on sufficient laboratory animal data.

**The estimated lifetime excess cancer risk** from a chloroform concentration of 1.25 µg/m$^3$ **is 2.6E-6 (3 in 1 million) for a full-time adult worker and 7.9E-7 (8 in 10 million) for a child (Table 2f).**

If using the maximum detected chloroform concentration overall for the cancer calculation (3.37 µg/m$^3$), estimated lifetime cancer risk is 7.1E-6 (7 in 1 million) for an adult worker and 2.1E-6 (2 in 1 million) for a child. Chloroform detected at the childcare center was similar to and lower than concentrations typically found in indoor air.

## 6.7. Acrylonitrile

The maximum acrylonitrile concentration was 0.078 (J) µg/m$^3$, which was a value detected in an evening, post-mitigation grab sample taken in the center's front desk area (VIA-25). Acrylonitrile was not detected pre-mitigation or in outdoor air. In sub-slab soil gas, acrylonitrile was detected in 2 of 3 samples, the highest of which (0.621 µg/m$^3$) exceeded a sub-slab soil gas CREG CV (0.50 µg/m$^3$). The lab-estimated indoor concentration of 0.078 (J) µg/m$^3$ exceeded a CREG air CV of 0.015 µg/m$^3$ (Table 1b).

Acrylonitrile is a volatile substance used to manufacture acrylic fibers, plastics, and other chemicals [ATSDR 2023b]. People can be exposed to very low levels by contacting consumer products such as acrylic carpeting, ingesting low levels from food stored in acrylic plastic containers, or inhaling smoke from tobacco, marijuana, or other acrylonitrile-containing burning biomass. Workers involved in the production of acrylic fibers and other products may be more highly exposed.

Based on a review of 148 vapor intrusion public health assessments and health consultations, ATSDR noted 3 sites with air concentrations above 2.0 µg/m$^3$. In 3 residential buildings, indoor air concentrations ranged from 2.7 µg/m$^3$ to 4.8 µg/m$^3$ [ATSDR 2023c].

EPA and DHHS have designated acrylonitrile as a "probable" and "reasonably anticipated" to be a human carcinogen, respectively.

**The estimated lifetime excess cancer risk** from an acrylonitrile concentration of 0.078 (J) µg/m$^3$, adjusted for a childcare center scenario (0.028 µg/m$^3$) **is 4.9E-7 (5 in 10 million) for a full-time worker and 1.5E-7 (2 in 10 million) for a child (Table 2g).**

## 6.8. Naphthalene

The maximum naphthalene concentration was 0.629 µg/m$^3$, which was a value detected in an evening, post-mitigation grab sample taken in the center's front desk area (VIA-25). This post-mitigation concentration was slightly higher than the pre-mitigation maximum, of 0.592 µg/m$^3$ (Table 1b). We assessed the pre-mitigation maximum (sample VIA-6) because it was an 8-hour sample taken in a classroom area with adjacent kitchen and more representative of exposure. In sub-slab soil gas, naphthalene was detected in 3 of 3 samples (max: 3.69 µg/m$^3$) all of which exceeded ATSDR's soil gas vapor intrusion CREG (0.97 µg/m$^3$; Table 1a). Naphthalene was detected once in outdoor air at a concentration of 0.393 µg/m$^3$.

All indoor air naphthalene concentrations as well as the MDL exceeded ATSDR's indoor air CREG, of 0.029 µg/m$^3$ (Table 1b).

> Naphthalene is a white solid that evaporates easily and is found in petroleum and coal. It has a strong but not unpleasant smell. Most naphthalene that enters the environment is from burning of woods and fossil fuels in the home [ATSDR 2005c]. Its major commercial use is in the manufacture of polyvinyl chloride (PVC) plastics. Its major consumer use is in moth repellants and toilet deodorant blocks. Naphthalene evaporates easily in air and breaks down to other chemicals, often within one day. Indoor averages typically range from 0.86-32 µg/m$^3$ according to some studies [ATSDR 2005d] and are usually higher than outdoor concentrations. In homes with and without smokers, indoor concentrations were measured around 2.2 and 1.0 µg/m$^3$, respectively [ATSDR 2005d].
>
> DHHS has classified naphthalene as a reasonably anticipated to be a human carcinogen based on findings in laboratory rodent studies. There is no direct evidence in humans that naphthalene causes cancer [ATSDR 2005c].

**The estimated lifetime excess cancer risk** from a naphthalene concentration of 0.592 µg/m$^3$, adjusted for a childcare center exposure scenario (adjusted EPC: 0.21 µg/m$^3$) **is 1.8E-6 (2 in 1 million) for a full-time adult worker and 5.5E-7 (6 in 10 million) for a child (Table 2h).** Naphthalene detected at the center was similar to and lower than concentrations typically found in indoor air.

## 6.9. Vinyl chloride and 1,2-dibromoethane

Vinyl chloride and 1,2-dibromoethane were **not detected** but had MDLs that exceeded ATSDR CREG CVs. We briefly evaluated the lifetime excess cancer risk from these VOCs by estimating exposure as ½ of each compound's MDL. Results are shown in Table 2i.

**Vinyl chloride** is a colorless gas with a mild, sweet odor [ATSDR 2023d]. It is a manufactured substance used to make polyvinyl chloride (PVC), which is used in a variety of plastic products such as pipes and packaging materials. It can also be formed when other substances such as trichloroethylene break down. Air in rural/remote and urban/suburban areas of the U.S. typically contains very low or no detectable amounts of vinyl chloride [ATSDR 2023e]. DHHS has classified vinyl chloride as a known human carcinogen. EPA has classified it as known human carcinogen by inhalation.

**1,2-Dibromoethane** is a colorless liquid with a mild, sweet odor. Most of it is man-made but small amounts occur in the ocean (thought to be made by algae) [ATSDR 2018a]. Most of its past uses, such as an additive in leaded gasoline and as a pesticide on soil and citrus, vegetable and grain crops, have been stopped or banned. Today it is used to treat logs for termites and beetles, in beehives to control moths, on ornamental plants to control beetles, and in the production of dyes, resins, gums and waxes. It breaks down slowly in air (over 4-5 months). Outdoors, 1-2 dibromoethane has been found in the parts per trillion ranges [ATSDR 2018b]. Inhaling volatized 1-2 dibromoethane from contaminated groundwater (e.g., during showering) may be an important source in indoor air but there are limited indoor air data for the compound. DHHS and EPA have classified 1,2-dibromoethane as "reasonably" and "likely" to be a human carcinogen, respectively [ATSDR 2018b].

Under the assumptions that the above non-detected compounds were potentially half their respective MDL, **the highest estimated lifetime excess cancer risk was** found for 1,2-dibromoethane, at **1.7E-6 (2 in 1 million) for full-time adult workers and 5.1E-7 (5 in 10 million) for children (Table 2i).** Lifetime excess cancer risk for vinyl chloride was lower, at 3.2E-8 (3 in 100 million) for full-time adult workers and 1.9E-8 (2 in 100 million) for a child.

## 6.10. 1,3-Dichloropropane and Dibromochloromethane

1,3-Dichloropropane and dibromochloromethane were **not detected** but lack a current CV or screening value from ATSDR or PADEP (Tables 1a, 1b). EPA's Provisional Peer Reviewed Toxicity Value reports for 1,3-dichloropropane and dibromochloromethane could not determine a subchronic or chronic reference concentration (RfC) for inhalation exposures to either compound due to the lack of human or animal toxicity data [EPA 2006; EPA 2009a]. Similarly, there are currently no inhalation-based health guidelines on these compounds from ATSDR. EPA has classified dibromochloromethane as a possible human carcinogen due to laboratory animal studies involving oral exposures to the compound in drinking water [EPA 2009a]. EPA determined that data were inadequate to estimate 1,3-dichloropropane's carcinogenetic potential [EPA 2006].

The Texas Commission on Environmental Quality provided short- and long-term Effect Screening Levels (ESLs) of 460 µg/m$^3$ and 46 µg/m$^3$ for 1,3-dichloropropane, and 20 µg/m$^3$ and 2 µg/m$^3$ for dibromochloromethane, respectively [TCEQ 2016]. These screening levels are

above the MDL for 1,3-dichloropropane (0.49 µg/m$^3$) and dibromochloromethane (0.073 µg/m$^3$) that were used to measure these compounds in indoor air at the childcare center. **Based on these limited data, it is unlikely that 1,3-dichlorpopane or dibromochloromethane would impact health, if present in indoor air at ½ their respective MDL.**

# 7. Public Health Implications – COCs Further Evaluated for Potential Non-cancer Health Effects

We assessed the potential for **non-cancer effects** for the following three VOCs – **isopropanol, trichloroethylene, and acrolein** – because their indoor air concentrations exceeded non-cancer CVs and/or screening values. For these COCs, PADOH assessed the potential for non-cancer health effects from childcare center exposures of acute (≤14 days), intermediate (15-364 days), and chronic (1 year or more) duration. Also included below is the estimated lifetime excess cancer risk for trichloroethylene (TCE). The remaining two VOCs are not classified as human carcinogens.

## 7.1. Isopropanol

The maximum isopropanol concentration was 602 µg/m$^3$, detected in the classroom area with adjacent kitchen (VIA-6) on March 31, 2022. Post-mitigation isopropanol was lower, at 401 µg/m$^3$ during the day and 65.9 µg/m$^3$ at night (Table 2j). The maximum outdoor isopropanol of 4.23 µg/m$^3$ was below indoor levels. Isopropanol was detected in 2 of 3 sub-slab locations (max: 34.9 µg/m$^3$) at concentrations below PADEP Act 2 Statewide Residential Vapor Intrusion Screening Standards for sub-slab soil gas (8,000 µg/m$^3$, Table 1a).

There are no ATSDR CVs for isopropanol; however, several detected indoor air values, including the maximum of 602 µg/m$^3$, exceeded the PADEP Act 2 Residential Vapor Intrusion Screening Standard for indoor air of 210 µg/m$^3$ (Table 1b).

> Isopropanol is also known as isopropyl alcohol and is a volatile liquid with a sharp, musty alcohol smell [EPA 2014a]. It is used in making a variety of products such as cosmetics, skin and hair products, dyes, cleaners, antifreezes, and other chemicals [NJ DOH 2016]. It can be found in wipes and solutions to clean and disinfect workplaces [WA State Department of Labor & Industries 2021].
>
> Short-term overexposure to high amounts of isopropanol can cause headache, dizziness, or unconsciousness. Inhalation exposures can also irritate mucous membranes of the upper respiratory tract [OEHHA 2008]. There is a lack of human data on long-term exposures to isopropanol but the available laboratory animal studies suggest the neurological and reproductive systems are targets for toxicity, as are developmental effects from exposure to high levels [EPA 2014a].
>
> EPA and ATSDR lack general population health guidelines for isopropanol. Isopropanol has not been classified as to its carcinogenicity.

***7.1.1. Acute exposures (<1-14 days) to isopropanol.*** There are no ATSDR or EPA health guidelines for general population acute exposures to isopropanol. The State of California has an acute recommended exposure limit (REL) for 1 hour inhaled isopropanol of 3,200 µg/m$^3$ to protect against eye and respiratory effects found in humans, which has a safety factor applied for human variability [OEHHA 2008, 2020]. Although EPA lacks inhalation health guidelines for isopropanol, it identified 3 human studies from 2002-2003 that found odor, irritation, annoyance, discomfort in throat, fatigue, and suggestive sex-related differences in lung metabolism in humans exposed for 2-to-4-hour periods at isopropanol levels up to 350,000 µg/m$^3$ [EPA 2014a]. The lowest exposure level identified for these effects was 31,000 µg/m$^3$, for discomfort in the throat and airways as well as fatigue in a study of 56 human adults [EPA 2014a].

The acute EPC for isopropanol exposures at the childcare center was 300 µg/m$^3$ (Table 3a). Although there are limited human data available, this estimate is well below the state of California's 1-hour REL for isopropanol exposures, of 3,200 µg/m$^3$, and effect levels found in humans (31,000 µg/m$^3$). **Based on the limited human data available, we would not expect adverse health effects to occur from acute exposures to isopropanol levels detected at the center.**

***7.1.2. Intermediate (15-364 days) and chronic exposures (1 year or more) to isopropanol.*** There are no agency health guidelines for general population intermediate and chronic exposures to isopropanol, and EPA has not identified long-term human inhalation studies on isopropanol. However, EPA established an isopropanol Provisional Peer-Reviewed Toxicity Value (PPRTV) based on studies evaluating exposures in laboratory animals [EPA 2014a].

For subchronic exposures (defined by EPA as 30-90 days), EPA's PPRTV provisional Reference Concentration (p-RfC) is 7,000 µg/m$^3$. It's derived from a Burleigh-Flayer 1994 study on rats exposed to isopropanol for 6 hours a day, 5 days a week, for 13 weeks. At a human-equivalent concentration (HEC) of 2,198,000 µg/m$^3$ (the highest exposure tested), female rats experienced increased mean cumulative motor activity, with a no observed adverse effect level (NOAEL) of 661,800 µg/m$^3$. EPA divided the study NOAEL by a composite uncertainty factor of 100 to result in a subchronic, p-RfC of 7,000 µg/m$^3$.

The adjusted EPC for intermediate-duration isopropanol exposures at the childcare center was 220 µg/m$^3$ (Table 3a). This concentration is well below EPA's p-RfC for isopropanol subchronic exposures (7,000 µg/m$^3$). Thus we would not expect adverse non-cancer health effects to occur from intermediate isopropanol exposures at the center.

The adjusted center EPC for *chronic* isopropanol exposures was also 220 µg/m$^3$ (Table 3a). This value exceeded EPA's chronic p-RfC of 200 µg/m$^3$ .

EPA's chronic p-RfC is derived from Burleigh-Flayer et al. 1997 that found decreased absolute and relative testes weights in male mice exposed to isopropanol by inhalation for 78 weeks. EPA identified a human equivalent concentration for these effects at a Lowest Observed

Adverse Effect Level (LOAEL) of 221,000 µg/m$^3$. After applying a composite uncertainty factor of 1,000, a chronic provisional RfC was set at 200 µg/m$^3$ [EPA 2014a]. The PADEP Act 2 Residential Vapor Intrusion Screening Standard for indoor air of 210 µg/m$^3$ is based on the EPA PPRTV chronic p-RfC.

The adjusted EPC for chronic isopropanol exposure at the childcare center (220 µg/m$^3$) is well below the human equivalent LOAEL of 221,000 µg/m$^3$ from which EPA's chronic p-RfC is derived. **Therefore, based on the limited data available, we would not expect adverse health effects to occur from intermediate or chronic exposures to the highest detected isopropanol concentrations in indoor air.**

## 7.2. Trichloroethylene (TCE)

Trichloroethylene (TCE) was detected on a single occasion at an indoor air concentration of 11.2 µg/m$^3$ on January 25, 2022. This detection occurred in the front desk area (sample VIA-5). TCE was not detected in subsequent samples of indoor air, or in outdoor air. It was detected in 2 of 3 sub-slab soil gas samples, at 3.78 µg/m$^3$ (VSG-5) and 0.328 µg/m$^3$ (VSG-6).

Although it was the sole detection of TCE in air, the TCE concentration of 11.2 µg/m$^3$ exceeded ATSDR cancer (0.21 µg/m$^3$) and non-cancer CVs (2.1 µg/m$^3$), and PADEP's Act 2 Vapor Intrusion Screening Standard for residential (2.1 µg/m$^3$) and nonresidential (8.8 µg/m$^3$) indoor air (Table 1b). Thus, we further evaluated the exposure to a 11.2 µg/m$^3$ TCE concentration in air.

Sub-slab soil gas TCE concentrations, including the collocated sample with the sole detection of TCE among indoor air locations (VSG-5), were lower and did not exceed sub-slab soil gas CVs (Table 1a).

> TCE is a colorless, volatile liquid with a sweet odor that evaporates quickly into the air [ATSDR 2019a]. Its two major uses are as a solvent to remove grease from metal parts and as a chemical used to make other chemicals, especially the refrigerant HFC-134a. It has several other uses including in dry-cleaning operations and as a component of adhesives, lubricants, paints, pesticides, cold metal cleaners and other substances [ATSDR 2019b]. The most common exposure sources for TCE are drinking contaminated water or breathing air released that has volatized from contaminated water. TCE and other volatile chemicals can also diffuse from contaminated groundwater or soil and migrate into buildings via vapor intrusion. Atmospheric TCE has a short half-life of about 3-7 days.
>
> TCE is widely detected in ambient air with data suggesting a general decline in the past two decades, at concentrations generally less than 1 ppb (5.4 µg/m$^3$) [ATSDR 2019b]. Studies on indoor air TCE have reported varied detection frequencies and concentrations. A 2011 review of scientific literature published by EPA of 2,503 samples taken between 1990 and 2005 in North American residences found it detected in 43% of homes, with 50$^{th}$ percentiles ranging from below device reporting limits to 1.1 µg/m$^3$ [EPA 2011a]. Indoor TCE levels of studies cited in ATSDR's 2019 TCE profile generally

report levels <1 µg/m$^3$, although medians have been reported as high as 27 µg/m$^3$ and maximums as high as 115-118 µg/m$^3$ [ATSDR 2019b].

Available human and animal data identify the central nervous system, kidney, liver, immune system, male reproductive system, and developing fetus as potential targets for TCE toxicity, with animal studies suggesting the immune system and developing fetus as particularly sensitive targets [ATSDR 2019b]. The systemic effects of TCE are not route specific; similar effects can be found from oral- or inhalation-based exposures [ATSDR 2019b].

DHHS, EPA and IARC have classified TCE as a known human carcinogen due to sufficient evidence from epidemiological studies of kidney cancer in humans [ATSDR 2019a]. There is also some evidence of an association between TCE and Non-Hodgkin's Lymphoma in humans.

The maximum TCE level of 11.2 µg/m$^3$ is higher than averages typically found from indoor air studies, although it represents a sole detection, on January 25, 2022. Typically, to confirm a vapor intrusion pathway, the indoor air concentration is lower than the sub-slab gas concentration. In this instance, the indoor air concentration is higher than the measured sub-slab soil gas concentration at the same location, which suggests a possible indoor air source for the TCE other than or in addition to the vapor intrusion pathway. PADOH notes that sub-slab soil gas sampling took place one day later than indoor air sampling, on January 26, 2022.

***7.2.1. Acute exposures (<1-14 days) to TCE.*** The sole TCE detection of 11.2 µg/m$^3$ was adjusted to 5.6 µg/m$^3$ to account for an acute childcare center exposure scenario (Table 3b; Appendix 1). ATSDR lacks an acute inhalation Minimal Risk Level (MRL) for TCE due to lack of adequate human or animal data for acute exposures. However, ATSDR noted that sensitive developmental effects (cardiac malformations, developmental immunotoxicity) found from intermediate-duration oral gestational or early postnatal exposure studies could potentially be elicited from acute, inhalation exposures of ≤14 days, if occurring during critical periods of development [ATSDR 2019b]. These sensitive developmental endpoints, which were found in laboratory animals, serve as the basis for ATSDR's intermediate and chronic inhalation MRLs for TCE. They are therefore considered relevant for acute human exposures. The acute EPC at the center (5.6 µg/m$^3$) exceeded ATSDR's intermediate and chronic inhalation TCE MRLs (2.1 µg/m$^3$). These MRLs are discussed further below.

***7.2.2. Guidelines for intermediate and chronic exposures and their relevance to acute TCE exposures.*** ATSDR's chronic and intermediate inhalation MRLs for TCE (2.1 µg/m$^3$) are derived from two oral exposure studies that reported fetal heart malformation in rats and decreased thymus weight in female mice [Johnson et al. 2003; Keil et al. 2009; ATSDR 2019b]. In the Johnson et al. 2003 study, pregnant rats were dosed at several TCE drinking water concentrations during gestation. In the Keil et al. 2009 study, mice were administered TCE in drinking water at several concentrations for 30 weeks.

In its Integrated Risk Information System (IRIS) assessment for TCE, EPA developed a physiologically based pharmacokinetic (PBPK) model to extract these oral studies to human equivalent concentrations (HECs) for inhalation exposures [ATDSR 2019b]. The resulting $HEC_{99}$ concentrations were 0.00037 ppm (20 μg/$m^3$) for fetal heart malformations and 0.0033 ppm (180 μg/$m^3$) for decreased thymus weight. After applying uncertainty factors to each effect level, EPA set its final TCE chronic RfC at 2.0 μg/$m^3$. ATSDR agreed with EPA's approach and set its chronic and intermediate MRLs at 2.1 μg/$m^3$ based on these studies [Johnson et al. 2003 and Keil et al. 2009].

In deriving its RfC, EPA provided a supporting study by the National Toxicology Program in 1988 that found kidney toxicity in female rats exposed for 104 weeks at a HEC of 30 μg/$m^3$ [EPA 2011b].

Human epidemiological data support animal findings of developmental, immunological, and renal toxicity. Indoor air and workplace exposures to TCE are of particular concern to women who are pregnant, particularly early in pregnancy before the fetal heart is fully formed. An epidemiological study of Endicott, New York women found associations between proximity to a soil gas plume containing TCE and cardiac defects and other adverse birth outcomes [Forand et al. 2012]. The plume occurred because of a large spill at a manufacturing facility; soil gas sampling revealed high TCE levels (100 to 10,000 μg/$m^3$). TCE indoor air levels were 0.18-140 μg/$m^3$; 67% of measured samples exceeded 3.3 μg/$m^3$ [ATSDR 2019b]. This epidemiological study [Forand et al. 2012] did not capture exposure at the individual or household level, and exposure to other contaminants (such as perchloroethylene) also occurred; however, the study provides some epidemiological support for adverse developmental effects.

Due to concerns for increased risk of elevated TCE exposure to the developing fetus, some states and agencies have action levels for indoor air TCE from vapor intrusion, in which efforts should be made to quickly minimize exposures to residents or workers. A few states have set these levels between 5 and 24 μg/$m^3$ for pregnant women, depending on residential or worker exposures [MA DPH 2017; CT DPH 2015]. An EPA Region 9 memo in 2014 stated, "Accelerated Response Actions" be taken at 2 μg/$m^3$ for residential TCE levels, at 8 μg/$m^3$ for 8-hour workplace levels, and at 7 μg/$m^3$ for 10-hour workplace levels. In the same memo, EPA set an "Urgent Response Action Level" of 6 μg/$m^3$ for residential exposures, 24 μg/$m^3$ for 8-hour commercial/industrial exposures, and 21 μg/$m^3$ for 10-hour commercial/industrial exposures [EPA 2014b]. In a 2017 public health assessment, the New Jersey Department of Health noted that indoor air concentrations of TCE about 3 times greater than the ATSDR's inhalation MRL of 2.1 μg/$m^3$ might become a concern for health effects [NJ DOH 2017]. Each of the above guidelines and action levels approach or exceed the most sensitive TCE effect level of 20 μg/$m^3$ for fetal heart effects.

***7.2.3. Assessing the potential for health effects from acute exposures (≤14 days) to TCE.***
PADOH currently does not have data to conclude if TCE was present at the center prior to January 25, 2022, at levels that could harm people's health from acute exposures.

**However, if pregnant staff were exposed to 11.2 µg/m$^3$ TCE on January 25, 2022, and up to approximately 3 weeks thereafter (adjusted EPC: 5.6 µg/m$^3$), this exposure may have harmed the health of the developing fetus. This scenario assumes a "worst case scenario" of 12 hours per day exposure during this period.** The adjusted EPC from this acute center exposure scenario is 3.6 times below a 20 µg/m$^3$ effect level for fetal heart malformations.

As mentioned, TCE was not detected in any subsequent/confirmatory sampling, including on the next testing date (February 16, 2022). PADOH currently lacks information on whether TCE might have been present and at which levels between January 25, 2022, and February 16, 2022.

***7.2.4. Assessing the potential for health effects from intermediate (15-364 days) or chronic exposures (1 year or more) to TCE.*** The adjusted EPCs for intermediate and chronic TCE exposures at the center (4.0 µg/m$^3$) also exceeded ATSDR chronic and intermediate MRLs (2.1 µg/m$^3$).

The human-equivalent effect levels for TCE inhalation exposure include low levels for kidney toxicity (30 µg/m$^3$) and immunotoxicity (180 µg/m$^3$). Along with fetal malformation effects, the kidney and immunotoxicity effects mentioned above were used to derive and support ATSDR and EPA intermediate and chronic health guidelines. These effects occurred from studies of longer exposure periods – 30 weeks for immunotoxicity [Keil et al. 2009] and 2 years for kidney toxicity [NTP 1988]. Human epidemiological evidence suggests that kidney and immune system effects are most likely to occur from occupational-based exposures (such as among TCE factory workers) over long periods. ATSDR noted that TCE may lead to non-cancer kidney effects but studies on humans are hampered by lack of exposure data and concomitant exposure to other chemicals [ATSDR 2019b].

TCE can also affect the human liver, neurological, and reproductive systems but these effects are about 1-3 orders of magnitude less sensitive than developmental, immune system, and kidney effects [EPA 2011c].

**Because TCE was not detected during sampling events after January 25, 2022, (i.e., from the next testing date of February 16, 2022, and onward), it does not appear that other non-cancer health effects from TCE (such as immune system and kidney effects) are likely to occur from intermediate or chronic exposures at the center after this date, assuming TCE remains non-detect or detected at much lower levels than on January 25, 2022.** However, as stated previously, PADOH does not have data to conclude if intermediate or chronic exposures from breathing TCE at levels that may have existed prior to January 25, 2022, or up to three weeks after, could cause adverse health effects.

***7.2.5. Lifetime excess cancer risk from TCE exposure.*** **The estimated lifetime excess cancer risk** from the single TCE concentration of 11.2 µg/m$^3$, adjusted for a chronic childcare center exposure scenario (adjusted EPC: 4.0 µg/m$^3$) is **2.6E-6 (3 in 1 million) for a child and 4.2E-6 (4 in 1 million) for a full-time adult worker (Table 2k).** We note that this lifetime excess cancer risk estimate is unlikely for center occupants because TCE was not detected after the sole detection

on January 25, 2022, and we do not have any information on indoor air concentrations prior to January 25, 2022.

## 7.3. Acrolein

Acrolein was detected with a maximum concentration of 0.674 µg/m$^3$, which was a daytime value of a post-mitigation grab sample in the classroom area with adjacent kitchen (VIA-26). This maximum was slightly higher than the pre-mitigation maximum (0.642 µg/m$^3$), which was also taken at this location.

All indoor and outdoor acrolein detections (0.101-0.674 µg/m$^3$) exceeded EPA's RfC (0.020 µg/m$^3$), ATSDR's intermediate MRL (0.092 µg/m$^3$) and PADEP's Act 2 vapor intrusion screening values for residential (0.02 µg/m$^3$) and non-residential indoor air (0.088 µg/m$^3$). The MDL for acrolein (0.089 µg/m$^3$) also exceeded EPA's RfC and PADEP Act 2 screening levels. No detections exceeded ATSDR's acute MRL (6.9 µg/m$^3$).

In sub-slab soil gas, acrolein was detected in 3 of 3 samples. One sample (VSG-6, at 2.14 µg/m$^3$) exceeded a chronic non-cancer sub-slab CV, and one sample (VSG-5, at 3.12 µg/m$^3$) exceeded both chronic (0.67 µg/m$^3$) and intermediate (3.1 µg/m$^3$) non-cancer soil gas CVs (Table 1a).

Post-mitigation acrolein at the childcare center (n=6 samples; average: 0.578 µg/m$^3$) was slightly higher than pre-mitigation levels (n=8 samples; average: 0.443 µg/m$^3$). Indoor air levels were higher than outdoor levels (n=7 samples; average: 0.274 µg/m$^3$).

Childcare center acrolein was also slightly higher than acrolein at the storage facility (n=6 samples; average: 0.344 µg/m$^3$). Sub-slab soil gas at the childcare center (n=3 detections, range: 0.468-3.12 µg/m$^3$) was also higher than at the storage facility (n=3 detections; range: 0.504-1.32 µg/m$^3$).

PADOH assessed the potential for non-cancer health effects from acrolein exposure based on the concentrations detected inside the childcare center. Acrolein has not been classified as a carcinogen by DHHS or EPA.

> Acrolein is a colorless or yellow liquid with a burnt and pungent odor [ATSDR 2007c]. It ignites and burns easily in air. It is primarily used to make other chemicals. Small amounts are released when organic matter such as trees and plants are burned, and when fuels such as gasoline and oil are burned. Acrolein is also found in tobacco smoke. Low amounts can be found in automobile exhaust and in fried foods, cooking oils, and roasted coffee. Major acrolein emissions from nonroad sources include nonroad diesel vehicles and airports [ATSDR 2007d]. In air, acrolein changes into other chemicals within days. Acrolein that enters soil can change into vapor and enter air.
>
> There is very little information about how exposure to acrolein affects people's health [ATSDR 2007c]. Breathing large amounts can damage the lungs or lead to more serious health effects. Lower amounts may cause eye watering and burning of the nose and

throat and a decreased breathing rate, which usually disappears after exposure stops. Laboratory animal studies indicate that breathing acrolein causes irritation to the nasal cavity, lowered breathing rate, and damage to the lining of the lungs.

EPA's National Air Quality and Emissions Trend Report for 1998 listed ambient acrolein in urban locations at 0.20 μg/m$^3$ based on 1996 data [ATSDR 2007d]. U.S. ambient air data from 2006-2009 found average measurements ranging from non-detect to 2.1 μg/m$^3$ [EPA 2009b]. In indoor air, acrolein has been measured at concentrations of <0.05-29 μg/m$^3$ [ATSDR 2007d]. When collocated samples are taken, indoor air concentrations are typically higher than outdoor air. Based on the International Agency for Research on Cancer (IARC's) review of studies on indoor acrolein levels in Japan, China and U.S. homes (including in California, New Jersey and Texas), acrolein concentrations ranged from <0.01-39 μg/m$^3$, with medians of 1 to 8 μg/m$^3$ [IARC 2021]. Primary indoor sources include smoking or cooking with oils and fats at high temperatures. Concentrations detected in a California school by a 2004 study by Sawant et al. were attributed to building elements such as carpet, drywall and adhesives [IARC 2021].

Acrolein is a very difficult compound to measure in air because it is highly reactive [EPA 2012]. It can react with other compounds, and potentially other compounds can react to form acrolein inside sampling canisters. Monitoring results can be subject to uncertainty and affected by factors such as how the canisters are cleaned in preparation for sample collection, and gas standards used to calibrate analytical equipment [EPA 2010]. In 2012, EPA decided not to use outdoor ambient acrolein data it collected around schools due to significant questions about the reliability of results [EPA 2012]. EPA has since worked on providing guidance for improving acrolein measurements. Recommendations using the TO-15 method have specific guidelines for canister cleaning and calibration [EPA 2010].

There are scant human data on acrolein, though it is a strong respiratory irritant. We compared adjusted center EPCs for acute, intermediate and chronic exposures to ATSDR MRLs and EPA RfCs and the principal studies used to derive these guidelines. We also assessed a few studies from the limited body of epidemiological research to date on acrolein exposures.

***7.3.1. Acute exposures (<1-14 days) to acrolein.*** The primary toxic effects from acrolein are to the mucous membranes, with the nasal tissues appearing to be the most sensitive target of inhalation exposure. Onset of noticeable irritation in humans occurs at 0.3 ppm (690 μg/m$^3$) with higher airborne concentrations (4,590-11,500 μg/m$^3$) resulting in severe manifestations of irritation over the entire respiratory tract [ATSDR 2007d]. Acrolein can also irritate the throat, lungs, stomach and skin [ATSDR 2007d]. ATSDR's acute MRL of 6.9 μg/m$^3$ is derived from a 1977 study by Weber-Tschoop et al. on human volunteers that found decreased respiratory rates and nose and throat irritation at exposures to 690 μg/m$^3$ acrolein. In this study, no acute effects were observed at 390 μg/m$^3$.

The adjusted acute EPC at the childcare center was 0.34 μg/m$^3$ (Table 3c). This exposure estimate is below ATSDR's acute MRL for acrolein (6.9 μg/m$^3$) and well below the most sensitive acute effect levels (690 μg/m$^3$ and above). **Thus, adverse health effects are not expected to occur from acute acrolein exposures at the center.**

***7.3.2. Intermediate (15-364 days) and chronic exposures (a year or more) to acrolein.*** The adjusted EPC for intermediate and chronic acrolein exposures at the center was 0.24 μg/m$^3$ (Table 3c). This value exceeded EPA's chronic RfC (0.02 μg/m$^3$) and ATSDR's intermediate MRL (0.092 μg/m$^3$) and produced hazard quotients of 12 and 2.6, respectively, for both center employees and children.

EPA's chronic RfC of 0.02 μg/m$^3$ is derived from a study by Feron et al. 1978 of laboratory hamsters, rats, and rabbits [EPA 2003]. The animals were exposed to acrolein in a whole-body exposure chamber for 6 hours a day, 5 days per week for 13 weeks at 0, 900, 3,200 and 11,000 μg/m$^3$. Nasal cavities were slightly affected in 1 of 12 rats at the lowest level of exposure tested (900 μg/m); severity in these histopathologic changes increased at higher levels of exposure, and rats were the most affected out of the tested species. The effect level of 900 μg/m$^3$ found in rats was adjusted for continuous, human exposure, to an effect level 20 μg/m$^3$. From this lowest observed adverse effect level (LOAEL) EPA applied a total uncertainty factor (UF) of 1,000: x3 for use of a minimal LOAEL, x3 intraspecies extrapolation, x10 for human variability and x10 for use of a subchronic study. The final chronic RfC was ($20_{LOAEL}/1000_{UF}$ = 0.02 μg/m$^3$).

The study's critical effect at a human equivalent concentration of 20 μg/m$^3$ is approximately 83 times higher than the adjusted exposure concentration at the childcare center (0.24 μg/m$^3$).

ATSDR's intermediate MRL for acrolein (0.092 μg/m$^3$) is also derived from the Feron et al. 1978 study finding nasal cavities in rats exposed to 900 μg/m$^3$. ATSDR converted the effect level to a human-equivalent, intermediate-exposure LOAEL of 27.5 μg/m$^3$ and applied a total UF of 300 (x10 for use of a LOAEL, x3 for extrapolation from animals to humans using dosimetric adjustments, x10 for human variability) to arrive at an intermediate MRL of 0.092 μg/m$^3$ (27.5 μg/m$^3{}_{LOAEL}/300_{UF}$ = 0.092 μg/m$^3$).

The above human equivalent effect levels for intermediate (27.5 μg/m$^3$) and chronic (20 μg/m$^3$) acrolein exposures are about 80-115 times above childcare center exposure estimates (0.24 μg/m$^3$) based on the maximum acrolein concentration detected. **Therefore, adverse health effects are unlikely to occur from intermediate or chronic acrolein exposures at the center.**

***7.3.3. Epidemiological studies and sensitive populations.*** In general, children are not likely to be affected by acrolein more than adults; however, children who are sensitive to air irritants, such as children with asthma, may be more sensitive to lung irritation from acrolein [ATSDR 2007d]. Generally, individuals with compromised respiratory function such as asthma might be at risk for developing adverse respiratory responses when exposed to a strong respiratory irritant such as acrolein [ATSDR 2007d]. However, outside of short-term volunteer studies (e.g.,

Weber-Tschopp et al. 1977) and cases of accidental acute inhalation to unknown levels of acrolein, there are very limited data on humans and acrolein exposure [ATSDR 2007d].

A cross-sectional study by Annesi-Maesano et al. [2012] reported that it was first to examine the effect of acrolein in classrooms. The study assessed school indoor air quality in 6 French cities (n=6,590 children, mean age 10 y) and found that the medium and highest tertiles of indoor acrolein (range: above limit of detection to higher than 1.55 $\mu g/m^3$) were significantly associated asthma prevalence, allergic asthma, and exercise-induced asthma. However, the detection limit was not reported and other compounds, including $NO_2$, $PM_{2.5}$, formaldehyde and acetaldehyde, were also measured with acrolein during a 5-day period. Certain classrooms had poorer air quality with indoor $PM_{2.5}$ and $NO_2$ levels above World Health Organization threshold limits, and asthma associations were also found for $PM_{2.5}$. Though this study was the first to report on acrolein in this setting, the authors did not exclude that the acrolein finding could have been a proxy for other indoor pollutants not assessed [Annesi-Maesano et al. 2012].

A 2014 study by deCastro [2014] reported that it was the first to report an association between outdoor air acrolein and adverse effects in the general population. It used outdoor dispersion modeling based on EPA National Ambient Air Toxics (NATA) data and reported a marginal significant increase in prevalent adult asthma attack odds, reported in the previous year, for exposures to the highest outdoor acrolein quintile (0.05-0.46 $\mu g/m^3$) relative to the lowest (0.000138-0.0109 $\mu g/m^3$). The study is not a direct equivalent to the childcare center's indoor air concentrations as it is based on outdoor dispersion modeling using several EPA data inputs.

The current body of epidemiological evidence remains limited on the association of low acrolein concentrations and asthma exacerbations or other effects; more studies are needed, which is complicated by the difficulty in measuring acrolein. Concentrations of acrolein found at the center were within and below ranges (<0.05-29 $\mu g/m^3$) [ATSDR 2007d] and medians (1-8 $\mu g/m^3$) found inside homes based on the latest scientific studies (most recently compiled by IARC 2021).

***7.3.4. Possible acrolein sources at the center.*** Common indoor sources of acrolein are from cooking at high temperatures and tobacco smoke. Potential sources at the center could include any cooking in the small kitchen adjacent to the classroom (sample location VIA-6) that involves the heating of oils or fats to high temperatures; smoking tobacco products is unlikely to be occurring indoors at the childcare center. Acrolein was detected in sub-slab soil gas (maximum: 3.12 $\mu g/m^3$) suggesting the possibility of VI from acrolein in soil, potentially from sources near the center (such as the airport). Alternatively, it could be an indicator of outdoor ambient acrolein migrating indoors. Regardless, should indoor air concentrations remain at or below detected values at the center, adverse health effects are unlikely to occur. As mentioned, in addition to detected acrolein, the MDL was also above the EPA chronic health guideline and PADEP's Act 2 residential and non-residential screening values.

# 8. Conclusions

Under the assumption that concentrations of VOCs detected in 2022 represent indoor air levels at the childcare center, PADOH concludes that current and future exposures to detected VOCs are not expected to result in adverse non-cancer health effects. Nearly all VOCs that exceeded screening levels, with a notable exception of TCE on a single occasion, were within or below levels typically found in indoor air, including acrolein. With regard to acrolein, adjusted EPCs at the center were below the ATSDR acute inhalation MRL that is protective against effects from short-term exposures, and 80-115 times below toxicological effect levels from intermediate and chronic exposures.

PADOH concludes that a past, acute exposure (≤14 days' duration) to the concentration of TCE detected (11.2 µg/$m^3$) could have harmed the health of pregnant staff, specifically the developing fetus, on or around January 25, 2022. This conclusion assumes a "worst case scenario" of 12 hours per day exposure during this period, if a worker was pregnant during this time. The sole TCE detection on January 25, 2022, occurred at the front desk area and not in the remaining two locations. TCE was not detected in succeeding months at any location including the next date tested (February 16, 2022) and it therefore appears unlikely to have been present at the center following February 16, 2022. However, PADOH cannot draw conclusions for TCE exposure for periods where data were missing or incomplete (i.e., before January 25, 2022 or between January 25 and February 16, 2022).

PADOH calculated lifetime excess cancer risk for any known or probable carcinogen that was detected or had a method detection that exceeded a cancer screening value. Lifetime excess risks for most compounds were lower than 1 in 1 million after adjusting for childcare center hours. The highest estimated lifetime excess risk was for TCE (4 in 1 million for a full-time adult worker); however, it was based on the single detection as mentioned and is unlikely for center occupants because TCE was not detected after January 25, 2022.

## Limitations

- Acrolein is a compound that is difficult to measure in air. This is due to potential issues with the air sampling canisters, calibration standards, and the time it takes to analyze a sample. In this dataset, air canisters were batch-certified prior to sample collection. However, without individually certifying each canister, it is possible there was acrolein contamination that could potentially result in an acrolein concentration that is overestimated. Other indoor air sources of acrolein, such as cooking oil at high temperatures, could also contribute to detections of acrolein.
- Indoor air contaminants can fluctuate over the course of a day, week, or season. While there are data for one day per month for a few months, there was no indoor air data representative of summer and fall months; therefore, the seasonality of indoor air could not be fully evaluated. VERTEX consulted a contractor in July 2022 to begin Heating,

Ventilation, and Air Conditioning (HVAC) system and other building adjustments, which may have affected summer and fall sampling.

- Sub-slab soil gas was taken on a single date in January 2022, whereas indoor air sampling took place for 4 dates in total (January, February, March, and November 2022). The single round of sub-slab soil gas sampling occurred one day later (January 26) than the initial indoor air sampling event (January 25). Soil gas measurements can vary and should ideally be taken at the same time as indoor air sampling [ATSDR 2016].
- Samples taken pre-building mitigation were of longer duration (8 hours for each of the 3 dates of sampling) and likely a better representation of indoor air quality than post building-mitigation samples (two 30-minute grab samples taken on a single sampling date).
- Indoor air sampling was limited to 4 dates and PADOH currently does not have data to assess whether TCE or other contaminants were present at the center on dates outside of the sampling period and were at levels that could harm health.

## Recommendations

- As a precautionary measure, PADOH recommends additional periodic sampling that is representative of a full day's exposure during all seasons to ensure TCE levels remain non-detect or below the Pennsylvania Department of Environmental Protection (PADEP) Land Recycling Program's Act 2 vapor intrusion statewide screening standard for TCE in residential indoor air, of 2.1 $\mu g/m^3$. Should levels exceed 2.1 $\mu g/m^3$, PADOH recommends that VERTEX immediately notify PADOH so that measures can be taken to address the exposures and inform building occupants, particularly people who might be pregnant.
- Any additional sub-slab soil gas sampling should ideally occur on the same date as indoor air sampling.
- The detected acrolein levels are unlikely to harm health. However, acrolein is a difficult compound to measure in air and any future sampling should ensure canisters are cleaned and individually certified, and methods are performed according to EPA guidelines.

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# Appendix 1. Adjusted Exposure Point Calculations for the Childcare Center

To estimate children and staff inhalation exposures to detected chemicals at the childcare facility, PADOH calculated an **Adjusted Exposure Point Concentration (EPC).** The calculation incorporates the concentrations of a chemical detected ("exposure point concentration") and adjusts for hours spent at the childcare center ("exposure factor").

As a public health protective assumption, PADOH used the maximum or most representative concentration detected as the EPC for each potential contaminant of concern (COC). For 3 COCs, benzene, chloroform and naphthalene, PADOH used the highest pre-mitigation value, which was lower than the highest post-mitigation value, because the sampling time (8 hours) and location of the highest pre-mitigation value was more representative of exposure.

PADOH adjusted the maximum or most representative EPC to account for childcare center hours. As discussed in the main report, PADOH used a "worst-case scenario" assuming 12 hours of exposure per day, 5 days per week, for 52 weeks per year. PADOH acknowledges that such a scenario is unlikely, but possible.

The **adjusted EPC formula is below:**
**EPC** (maximum/most representative concentration detected in indoor air, µg/$m^3$) *** EF** (Exposure Factor, unitless) = **Adjusted EPC**

EFs=1 for residential-based exposures (24 hours a day, 7 days a week, 365 days per year), which serves at the denominator in the EF equation. Site-specific exposures serve as the numerator. For PADOH's childcare center scenario for chronic (year or longer) exposures, the EF = 0.36:

**EF (PADOH chronic exposure childcare center scenario):**
12 hours of exposure per day, 5 days per week, 52 weeks out of the year
= (12 hours * 5 days * 52 weeks) / (24 hours * 7 days * 52 weeks) = **0.36**

- **Adjusted chronic and intermediate EPC based on the maximum (and only) trichloroethylene detection:**
  = 11.2 µg/$m^3$ (EPC) * 0.36 (EF) = **4.0** µg/$m^3$ (adjusted EPC)
- **Adjusted chronic and intermediate EPC based on the maximum acrolein detection:**
  = 0.674 µg/$m^3$ (EPC) * 0.36 (EF) = **0.24** µg/$m^3$ (adjusted EPC)

If children and adults were present at the center year-round (52 weeks) at the center but for fewer than 12 hours a day, exposure factors (EFs) and hence adjusted EPCs decrease.

EF changes based on amount of exposure:

- 12 hour per day exposure, 5 days a week, for 52 weeks per year
  - (12 * 5 * 52) / (24 * 7 * 52) = 0.36
- 10 hour per day exposure, 5 days a week, for 52 weeks per year

    - (10 * 5 * 52) / (24 * 7 * 52) = 0.29
- 8 hour per day exposure, 5 days a week, for 52 weeks per year
    - (8 * 5 * 52) / (24 * 7 *52) = 0.24
- 4 hour per day exposure, 5 days a week, for 52 weeks per year
    - (4 * 5 *52) / (24 * 7 * 52) = 0.12
- 2 hour per day exposure, 5 days a week, for 52 weeks per year
    - (2 * 5 *52) / (24 * 7 * 52) = 0.06

Adjusting the EF as shown above changes the adjusted EPCs for detected chemicals at the site. Examples are shown below for trichloroethylene and acrolein.

| **Chemical** | **Maximum Unadjusted concentration (µg/m³)** | **Adjusted EPC for 12 hour exposure (µg/m³)** | **Adjusted EPC for 10 hour exposure (µg/m³)** | **Adjusted EPC for 8 hour exposure (µg/m³)** | **Adjusted EPC for 4 hour exposure (µg/m³)** | **Adjusted EPC for 2 hour exposure (µg/m³)** |
|---|---|---|---|---|---|---|
| - | - | *EF: 0.36* | *EF: 0.29* | *EF: 0.24* | *EF: 0.12* | *EF: 0.06* |
| Trichloroethylene (TCE) | 11.2 | 4.0 | 3.2 | 2.7 | 1.3 | 0.68 |
| Acrolein | 0.674 | 0.24 | 0.20 | 0.06 | 0.02 | 0.04 |

Adjusted Exposure Point Concentration (EPC) = EPC * Exposure Factor (EF)

Several compounds, such as chloroform, do not adjust by an EF when calculating a chronic EPC for non-cancer effects (e.g., their EF always = 1). This is because their non-cancer health guideline levels were derived from an unadjusted concentration of that compound. Further details are provided in ATSDR 2021.

For **acute** exposures (≤14 days), the denominator in the EF equation is simply set for 24 hours. The EPC is multiplied by the exposure duration (in this case, 12 hours) and divided by 24 hours.

Adjusted EPC, acute exposures = EPC * EF:

- Childcare center adjusted EPC based on a concentration of TCE of 11.2 µg/m$^3$ = 11.2 * (12 / 24 hours) = 5.6 µg/m$^3$
- Childcare center adjusted EPC based on a concentration of 0.674 µg/m$^3$ acrolein = 0.674 * (12 / 24 hours) = 0.34 µg/m$^3$

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# Appendix 2. Additional Tables Referenced in Main Report

**Table 1a. Comparison Value and Screening Results for Sub-slab Soil Gas at the Childcare Center (µg/m$^3$)**

| Contaminant | Analytical MDL | Max detected in sub-slab soil gas | ATSDR CREG | ATSDR cEMEG | ATSDR RMEG | ATSDR iEMEG | ATSDR aEMEG | PA-VI-SSG-R |
|---|---|---|---|---|---|---|---|---|
| 1,1,1-Trichlorethane | 0.273 | 0.431 | - | - | 170,000 | 130,000 | 180,000 | 200,000 |
| 1,1,2,2-Tetrachloroethane | 0.422 | ND | - | - | - | - | - | 16 |
| 1,1,2-Trichloroethane | 0.366 | ND | 2.1 | - | - | 370 | 5,300 | 8 |
| 1,1,-Dichloroethane | 0.254 | ND | - | - | - | - | - | 590 |
| 1,1-Dichloroethene | 0.255 | 0.42 | - | 130 | 6,700 | 130 | - | 8,000 |
| 1,2,4-Trichlorobenzene | 0.500 | ND | - | - | - | - | - | 80 |
| 1,2,4-Trimethylbenzene | 0.181 | 10.3 | - | - | 2,000 | - | - | 2,400 |
| **1,2-Dibromoethane[1]** | **0.431** | ND | **0.057** | - | 300 | - | - | 1.6 |
| 1,2-Dichlorobenzene | 0.378 | ND | - | - | - | - | - | 8,000 |
| 1,2-Dichloroethane | 0.244 | ND | 1.3 | - | - | - | 40,000 | 36 |
| 1,2,-Dichloropropane | 0.282 | ND | - | - | 130 | 310 | 3,100 | 0.25[2] |
| 1,3,5-Trimethylbenzene | 0.332 | 2.62 | - | - | 2,000 | - | - | 2,400 |
| **1,3-Butadiene** | 0.148 | **3.12** | **1.1** | - | 67 | - | - | 31 |
| **1,3-Dichloropropane[3]** | 0.490 | ND | - | - | - | - | - | NS |
| 1,4-Dichlorobenzene | 0.154 | 0.926 | - | 2,000 | 27,000 | 40,000 | 400,000 | 85 |
| **1,4-Dioxane** | 0.290 | **8.58** | **6.7** | 3,700 | 1,000 | 24,000 | 240,000 | 190 |
| 3-Chloropropene | 0.183 | ND | - | - | 33 | - | - | 40 |
| 4-Methyl-2-pentanone | 0.173 | 4.71 | - | - | 100,000 | - | - | 120,000 |
| Acetone | 1.64 | 278 | - | - | - | - | 630,000 | 1,200,000 |
| **Acrolein** | 0.137 | **3.12** | - | - | **0.67** | **3.1** | 230 | **0.8** |
| **Acrylonitrile** | 0.120 | **0.621** | **0.50** | - | 67 | - | 7,300 | 14 |
| **Benzene** | 0.156 | **16.5** | **4.3** | 320 | 1,000 | 630 | 970 | 120 |
| Benzyl chloride | 0.250 | ND | - | - | - | - | - | 19 |
| Bromodichloromethane | 0.338 | ND | - | - | - | - | - | 25 |
| Bromoform | 0.663 | ND | 30 | - | - | - | - | 850 |
| Bromomethane | 0.300 | ND | - | 130 | 170 | 2,600 | - | 200 |
| Carbon disulfide | 0.174 | 4.7 | - | 31,000 | 23,000 | | | 28,000 |
| Carbon tetrachloride | 0.314 | 0.459 | 5.7 | 6,300 | 3,300 | 6,300 | - | 160 |
| Chlorobenzene | 0.287 | ND | - | - | - | - | - | 2,000 |
| Chloroethane | 0.212 | ND | - | - | 330,00 | - | 1,300,000 | 400,000 |
| Chloroform | 0.309 | 1.21 | 1.4 | 3,300 | - | 8,000 | 16,000 | 41 |
| Chloromethane | 0.142 | 1.56 | - | 2,100 | 3,000 | - | 33,000 | 520 |
| Cyclohexane | 0.127 | 0.458 | - | - | 200,000 | - | - | 240,000 |
| **Dibromochloromethane[4]** | 0.523 | ND | - | - | - | - | - | 35 |
| Dichlorodifluoromethane | 0.288 | 2.29 | - | - | - | - | - | 4,000 |
| Ethylbenzene | 0.188 | 3.78 | - | 8,700 | 33,000 | 290,000 | 730,000 | 370 |
| Freon-113 | 0.503 | 0.59 | - | - | - | - | - | 200,000 |

| Contaminant | Analytical MDL | Max detected in sub-slab soil gas | ATSDR CREG | ATSDR cEMEG | ATSDR RMEG | ATSDR iEMEG | ATSDR aEMEG | PA-VI-SSG-R |
|---|---|---|---|---|---|---|---|---|
| Isopropanol | 1.17 | 34.9 | - | - | - | - | - | 8,000 |
| Isopropyl benzene | 0.241 | 0.487 | - | - | 13,000 | - | - | 16,000 |
| Methyl Methacrylate | 0.285 | 2.46 | - | - | 23,000 | - | - | 28,000 |
| Methyl tert Butyl Ether | 0.189 | ND | - | 120,000 | 100,000 | 120,000 | 240,000 | 3,600 |
| Methylene Chloride | 0.466 | 0.823 | 2,100 | 33,000 | 20,000 | 33,000 | 70,000 | 24,000 |
| **Naphthalene** | **0.464** | **3.69** | **0.97** | 120 | 100 | - | - | 28 |
| n-Hexane | 0.128 | 2.96 | - | 70,000 | 23,000 | - | - | 28,000 |
| o-xylene[5] | 0.197 | 5.43 | - | 7,300 | 3,300 | 87,000 | 290,000 | 4,000 |
| p/m-xylene[5] | 0.395 | 9.86 | - | 7,300 | 3,300 | 87,000 | 290,000 | 4,000 |
| Styrene | 0.185 | 7.83 | - | 28,000 | 33,000 | - | 700,000 | 40,000 |
| Tetrachloroethene | 0.444 | 39.3 | 130 | 1,400 | 1,300 | 1,400 | 1,400 | 1,600 |
| Tetrahydrofuran | 0.168 | 0.242 | - | - | 67,000 | - | - | 480 |
| Toluene | 0.196 | 100 | - | 130,000 | 170,000 | - | 250,000 | 200,000 |
| Trans-1,2-dichloroethene | 0.255 | ND | - | - | - | 26,000 | 26,000 | NS |
| Trichloroethylene (TCE) | 0.271 | 3.78 | 7.0 | 70 | 67 | 70 | - | 80 |
| Trichlorofluoromethane | 0.386 | 52.2 | - | - | - | - | - | 28,000 |
| Vinyl bromide | 0.313 | ND | - | - | 100 | - | - | 29 |
| Vinyl chloride | 0.160 | 0.639 | 3.7 | - | 3,300 | 1,700 | 43,000 | 31 |
| Xylenes, Total | 0.197 | 15.2 | - | 7,300 | 3,300 | 87,000 | 290,000 | 4,000 |

**Bold =** potential Contaminant of Concern for sub-slab soil gas. Bolded values indicate the detected concentration and/or analytical method detection limit and comparison value (CV) that was exceeded. Blank cells = no available CV or screening value; EMEG = Environmental Media Evaluation Guide CVs for acute (≤14 days, aEMEG), intermediate (15-364 days, iEMEG) or chronic (1 year or longer, cEMEG) exposures. MDL = Method Detection Limit; RMEG = Reference Media Evaluation Guide CV. PA-VI-SSG-R: Pennsylvania Residential Sub-Slab Soil Gas Statewide Health Standard Vapor Intrusion Screening Values listed by the Pennsylvania Department of Environmental Protection's Land Recycling Program for Vapor Intrusion into Buildings from Groundwater and Soil under Act 2 [PADEP 2022]; ND = Not detected; NS = No standard

[1] Not detected but identified as a potential Contaminant of Concern because the MDL exceeded a CV.

[2] MDL exceeded PA-VI-SSG-R screening value; however, PADOH prioritizes the Agency of Toxic Substances and Disease Registry (ATSDR) CVs.

[3] Not detected but identified as a potential Contaminant of Concern because a CV from ATSDR or screening value from PADEP could not be located.

[4] PADOH could not identify a current Act 2 sub-slab soil gas CV or PADEP screening value for this compound. The screening values provided in this table are from PADEP 2016. PADOH considered this compound as a potential contaminant of concern for indoor air.

[5] CVs provided are for total xylenes.

**Table 1b. Comparison Value and Screening Results for Indoor Air at the Childcare Center (µg/m³)**

| Contaminant | Analytical MDL | Max in Indoor Air, pre-mitigation | Max in Indoor Air, post-mitigation | ATSDR CREG | ATSDR cEMEG | ATSDR RMEG | ATSDR iEMEG | ATSDR aEMEG | PA-VI-IA-R |
|---|---|---|---|---|---|---|---|---|---|
| **Volatile Organics in Air** | - | - | - | - | - | - | - | - | - |
| 1,1,1-Trichloroethane | 0.273 | ND | ND | - | - | 5,000 | 3,800 | 5,500 | 5,200 |
| 1,1-Dichloroethane | 0.254 | ND | ND | - | - | - | - | - | 15 |
| 1,1-Dichoroethene | 0.255 | ND | ND | - | 4.0 | 200 | 4.0 | - | 210 |
| 1,2,4-Trichorobenzene | 0.500 | ND | ND | - | - | - | - | - | 2.1 |
| 1,2,4-Trimethylbenzene | 0.181 | 0.836 | 0.708 J | - | - | 60 | - | - | 63 |
| 1,2-Dichlorobenzene | 0.378 | ND | ND | - | - | - | - | - | 210 |
| **1,2-Dichloroethane** | **0.244** | ND | **0.441 J (n)** | **0.038** | - | - | - | 1,200 | 0.94 |
| 1,2-Dichloropropane | 0.282 | ND | ND | - | - | 4.0 | 9.2 | 92 | 0.0066[1] |
| 1,3,5-Trimethylbenzene | 0.332 | ND | ND | - | - | 60 | - | - | 63 |
| **1,3-Butadiene** | **0.148** | ND | **0.168 J (n)** | **0.033** | - | 2.0 | - | - | 0.81 |
| **1,3-Dichloropropane[2]** | 0.490 | ND | ND | - | - | - | - | - | NS |
| 1,4-Dichlorobenzene | 0.382 | ND | ND | - | 60 | 800 | 1,200 | 12,000 | 2.2 |
| **1,4-Dioxane** | **0.290** | **0.386 J** | ND | **0.20** | 110 | 30 | 720 | 7,200 | 4.9 |
| 3-Chloropropene | 0.183 | ND | ND | - | - | 1.0 | - | - | 1.0 |
| 4-Methyl-2-pentanone | 0.173 | 0.66 J | 0.385 J | - | - | 3,000 | - | - | 3,100 |
| Acetone | 1.64 | 38.5 | 60.3 | - | - | - | - | 19,000 | 32,000 |
| **Benzene** | **0.156** | **0.888** | **0.997 (n)** | **0.13** | 9.6 | 30 | 19 | 29 | 3.1 |
| Bromoform | 0.663 | ND | ND | 0.91 | - | - | - | - | 22 |
| Bromomethane | 0.300 | ND | ND | - | 3.9 | 5.0 | 78 | - | 5.2 |
| Carbon disulfide | 0.174 | ND | 0.405 J | - | 930 | 700 | - | - | 730 |
| **Carbon tetrachloride** | **0.314** | **0.604** | **0.591 J** | **0.17** | 190 | 100 | 190 | - | 4.1 |
| Chlorobenzene | 0.287 | ND | ND | - | - | - | - | - | 52 |
| Chloroethane | 0.212 | ND | ND | - | - | 10,000 | - | 40,000 | 10,000 |
| **Chloroform** | **0.309** | **1.25** | **3.37** | **0.043** | 98 | - | 240 | 490 | 1.1[1] |
| Chloromethane | 0.142 | 1.76 | 1.28 | - | 62 | 90 | 620 | 1,000 | 14 |
| Cyclohexane | 0.127 | 0.757 | 0.447 J | - | - | 6,000 | - | - | 6,300 |
| Dichlorodifluoromethane | 0.288 | 2.67 | 2.67 | - | - | - | - | - | 100 |
| Ethylbenzene | 0.188 | 0.586 J | 0.617 J | - | 260 | 1,000 | 8,700 | 22,000 | 9.7 |
| Freon-113 | 0.503 | 0.667 | 0.544 J | - | - | - | - | - | 5,200 |
| **Isopropanol** | 1.17 | **602** | **401** | - | - | - | - | - | **210** |
| Isopropyl benzene | 0.241 | ND | 1.11 | - | - | 400 | - | - | 420 |
| Methyl Methacrylate | 0.285 | 0.557 | ND | - | - | 700 | - | - | 730 |
| Methyl tert butyl ether | 0.189 | ND | ND | - | 3,600 | 3,000 | 3,600 | 7,200 | 94 |
| Methylene chloride | 0.466 | 4.31 | 4.79 | 63 | 1,000 | 600 | 1,000 | 2,100 | 630 |
| n-Hexane | 0.128 | 1.62 | 1.70 | - | 2,100 | 700 | - | - | 730 |
| o-Xylene[3] | 0.197 | 0.756 J | 0.817 J | - | 220 | 100 | 2,600 | 8,700 | 100 |
| p/m-Xylene[3] | 0.395 | 1.98 | 2.08 | - | 220 | 100 | 2,600 | 8,700 | 100 |

| Contaminant | Analytical MDL | Max in Indoor Air, pre-mitigation | Max in Indoor Air, post-mitigation | ATSDR CREG | ATSDR cEMEG | ATSDR RMEG | ATSDR iEMEG | ATSDR aEMEG | PA-VI-IA-R |
|---|---|---|---|---|---|---|---|---|---|
| Styrene | 0.185 | 1.94 | 1.38 | - | 850 | 1,000 | - | 21,000 | 1,000 |
| Tetrachloroethene | 0.444 | 0.773 | 0.515 J | 3.8 | 41 | 40 | 41 | 41 | 42 |
| Tetrahydrofuran | 0.168 | 1.65 | 0.584 J | - | - | 2,000 | - | - | 13 |
| Toluene | 0.196 | 3.84 | 3.03 | - | 3,800 | 5,000 | - | 7,500 | 5,200 |
| Trans-1,2-Dichloroethene | 0.255 | ND | ND | - | - | - | | 12,000 | NS |
| **Trichloroethylene (TCE)** | **0.271** | **11.2** | ND | **0.21** | **2.1** | **2.0** | **2.1** | **-** | **2.1** |
| Trichlorofluoromethane | 0.386 | 1.51 | 1.61 | - | - | - | - | - | 730 |
| Vinyl bromide | 0.313 | ND | ND | - | - | 3.0 | - | - | 0.76 |
| **Vinyl chloride[4]** | **0.160** | ND | ND | **0.11** | - | 100 | 51 | 1,300 | 0.81 |
| Xylenes, Total | 0.197 | 2.74 J | 2.89 J | - | 220 | 100 | 2,600 | 8,700 | 100 |
| **Volatile Organics in Air by Selective Ion Monitoring** | - | - | - | - | - | - | - | - | - |
| 1,1,2,2-Tetrachloroethane | 0.039 | ND | ND | - | - | - | - | - | 0.42 |
| 1,1,2-Trichloroethane | 0.032 | ND | ND | 0.063 | - | - | 11 | 160 | 0.21 |
| **1,2-Dibromoethane[4]** | **0.062** | ND | ND | **0.0017** | - | 9.0 | - | - | **0.041** |
| **Acrolein** | **0.089** | **0.642** | **0.674** | - | - | **0.020** | **0.092** | 6.9 | **0.021** |
| **Acrylonitrile** | **0.053** | ND | **0.078 J (n)** | **0.015** | - | 2.0 | 2.0 | 220 | 0.36 |
| Benzyl chloride | 0.037 | ND | ND | - | - | - | - | - | 0.5 |
| Bromodichloromethane | 0.045 | 0.241 | 0.342 | - | - | - | - | - | 0.66 |
| **Dibromochloromethane[5]** | 0.073 | ND | ND | - | - | - | - | - | 0.9 |
| **Naphthalene** | **0.184** | **0.592** | **0.629 (n)** | **0.029** | 3.7 | 3.0 | - | - | 0.72 |

**Bold =** potential Contaminant of Concern for indoor air. Bolded values indicate the detected concentration and/or analytical method detection limit and comparison value (CV) that was exceeded. Blank cells = no available CV or screening value; EMEG = Environmental Media Evaluation Guide CVs for acute (≤14 days, aEMEG), intermediate (15-364 days, iEMEG) or chronic (1 year or longer, cEMEG) exposures. MDL = Method Detection Limit; RMEG = Reference Media Evaluation Guide CV. PA-VI-SIA-R: Pennsylvania Residential Indoor Air Statewide Health Standard Vapor Intrusion Screening Values, and listed by the Pennsylvania Department of Environmental Protection's Land Recycling Program for Vapor Intrusion into Buildings from Groundwater and Soil under Act 2 [PADEP 2022]; (n) = 30 minute post-mitigation sample taken during the evening (10:40-11:10 p.m.); ND = Not detected; NS = No standard

[1] Exceeded; however, PADOH prioritizes the Agency of Toxic Substances and Disease Registry (ATSDR) CVs.

[2] Not detected but identified as a potential Contaminant of Concern because a CV from ATSDR or screening value from PADEP could not be located.

[3] CVs provided are for total xylenes.

[4] Not detected but identified as a potential Contaminant of Concern because the analytical method detection limit exceeded a CV.

[5] PADOH could not identify a current Act 2 VI indoor air CV or PADEP screening value for this compound and thus considered it a potential contaminant of concern for indoor air. The screening values provided in this table for this compound are from PADEP 2016.

## Tables 2a–2l Lifetime Excess Cancer Risk Estimates for Indoor Air Contaminants that Exceeded an ATSDR Cancer Risk Evaluation Guide (CREG) CV (µg/m$^3$). Detection summaries are also included for isopropanol and acrolein, which are not classified as carcinogens

Analytical reporting and method detection limits of these tables refer to indoor air. CREG = cancer risk evaluation guide comparison value; EPC = Exposure Point Concentration; J = lab-estimated result; NA = Not Applicable (indicating that VIA-7 was not sampled in January 2022). Indoor air CREGs are provided in each table. Sub-slab soil gas CVs, if exceeded, are also bolded; their CVs can be found in Table 1a of the report.

Samples shown in the table are collocated. VSG = sub-slab soil gas samples and VIA = indoor air samples. Samples at locations 5, 6 and 7 were taken pre-mitigation in indoor air. Samples 25/35 (day sample/night sample), 26/36, and 27/37 were taken at these locations post-mitigation (at day and at night). Contaminant concentrations are presented in µg/m$^3$.

**Table 2a. 1,2-Dichloroethane**

CREG: 0.038 µg/m$^3$. Reporting Limit: 0.809 µg/m$^3$. Method Detection Limit: 0.244 µg/m$^3$

| Sampling Location | Sub-Slab Soil Gas | Pre-mitigation indoor air | Pre-mitigation Indoor Air | Pre-mitigation Indoor Air | Post-mitigation Indoor Air | Post-mitigation Indoor Air |
|---|---|---|---|---|---|---|
| Date (2022) | Jan-26 | Jan-25 | Feb-16 | Mar-31 | Nov-22 (Day) | Nov-22 (Night) |
| VSG / VIA 5, 25, 35 | ND | ND | ND | ND | ND | **0.376 (J)** |
| VSG / VIA 6, 26, 36 | ND | ND | ND | ND | ND | **0.441 (J)*** |
| VSG / VIA 7 27, 37 | ND | NA | ND | ND | 0.34 (J) | ND |

**Bold = value that exceeds a CV (CREG). *** Value used to estimate exposure. J = value is lab-estimated. Note: for 1,2-dichloroethane, analytical reporting and method detection limits also exceeded this CV. Cancer risk estimate based on this maximum exposure concentration (0.441 J µg/m$^3$), adjusted for a childcare center exposure scenario (adjusted EPC: 0.16 µg/m$^3$): 1.0E-6 (adult full-time worker), 3.2E-7 (child). Assumes exposure for 12 h/day, 5 days/wk, 52 weeks/yr, for 6 years (child) and 20 years (adult). EPA Inhalation Unit Risk (IUR): 2.6E-5 (µg/m$^3$)$^{-1}$

**Table 2b. 1,3-Butadiene**

CREG: 0.033 µg/m$^3$. Reporting Limit: 0.442 µg/m$^3$. Method Detection Limit: 0.148 µg/m$^3$

| Sampling Location | Sub-Slab Soil Gas | Pre-mitigation indoor air | Pre-mitigation indoor air | Pre-mitigation indoor air | Post-mitigation indoor air | Post-mitigation indoor air |
|---|---|---|---|---|---|---|
| Date (2022) | Jan-26 | Jan-25 | Feb-16 | Mar-31 | Nov-22 (Day) | Nov-22 (Night) |
| VSG / VIA 5, 25, 35 | **3.12** | ND | ND | ND | ND | **0.166 (J)** |
| VSG / VIA 6, 26, 36 | 0.498 | ND | ND | ND | ND | ND |
| VSG / VIA 7 27, 37 | ND | NA | ND | ND | ND | **0.168 (J)*** |

**Bold = value that exceeds a CV (CREG). *** Value used to estimate exposure. J = value is lab-estimated. Note: for 1,3-butadiene, analytical reporting and method detection limits also exceeded this CV. Cancer risk estimate based on this maximum exposure concentration (0.168 J µg/m$^3$), adjusted for a childcare center exposure scenario (adjusted EPC: 0.06 µg/m$^3$): 4.6E-7 (adult full-time worker); 1.4E-7 (child). Assumes exposure for 12 h/day, 5 days/wk, 52 weeks/yr, for 6 years (child) and 20 years (adult). EPA Inhalation Unit Risk (IUR): 3.0E-5 (µg/m$^3$)$^{-1}$

**Table 2c. 1,4-Dioxane**

CREG: 0.20 µg/m$^3$. Reporting Limit: 0.721 µg/m$^3$ . Method Detection Limit: 0.29 µg/m$^3$

| Sampling Location | Sub-Slab Soil Gas | Pre-mitigation indoor air | Pre-mitigation indoor air | Pre-mitigation indoor air | Post-mitigation indoor air | Post-mitigation indoor air |
|---|---|---|---|---|---|---|
| Date (2022) | Jan-26 | Jan-25 | Feb-16 | Mar-31 | Nov-22 (Day) | Nov-22 (Night) |
| VSG / VIA 5, 25, 35 | 0.755 | ND | ND | ND | ND | ND |
| VSG / VIA 6, 26, 36 | **8.58** | ND | ND | **0.386 (J)*** | ND | ND |
| VSG / VIA 7 27, 37 | ND | NA | ND | ND | ND | ND |

**Bold = value that exceeds a CV (CREG). ***Value used to estimate exposure. J = value is lab-estimated. Note: for 1,4-dioxane, analytical reporting and method detection limits also exceeded this CV. Cancer risk estimate based on this maximum exposure concentration (0.386 J µg/m$^3$), adjusted for a childcare center exposure scenario (adjusted EPC, 0.14 µg/m$^3$): 1.8E-7 (adult full-time worker); 5.3E-8 (child). Assumes exposure for 12 h/day, 5 days/wk, 52 weeks/yr, for 6 years (child) and 20 years (adult). EPA Inhalation Unit Risk (IUR): 5.0E-6 (µg/m$^3$)$^{-1}$

**Table 2d. Benzene**

CREG: 0.13 µg/m$^3$. Reporting Limit: 0.639 µg/m$^3$. Method Detection Limit: 0.156 µg/m$^3$

| Sampling Location | Sub-Slab Soil Gas | Pre-mitigation indoor air | Pre-mitigation indoor air | Pre-mitigation indoor air | Post-mitigation indoor air | Post-mitigation indoor air |
|---|---|---|---|---|---|---|
| Date (2022) | Jan-26 | Jan-25 | Feb-16 | Mar-31 | Nov-22 (Day) | Nov-22 (Night) |
| VSG / VIA 5, 25, 35 | **16.5** | **0.872** | **0.626** | **0.415 (J)** | **0.684** | **0.997** |
| VSG / VIA 6, 26, 36 | **6.26** | **0.888*** | **0.604** | **0.636 (J)** | **0.808** | **0.936** |
| VSG / VIA 7 27, 37 | 0.406 | NA | **0.428** | **0.412 (J)** | **0.700** | **0.728** |

**Bold = value that exceeds a CV (CREG). ***Value used to estimate exposure. J = value is lab-estimated. Note: for benzene, analytical reporting and method detection limits also exceeded this CV. Cancer risk estimate based on the maximum concentration representative of exposure (0.888 µg/m$^3$) and adjusted for a childcare center exposure scenario (adjusted EPC: 0.32 µg/m$^3$): 6.3E-7 (adult full-time worker); 1.9E-7 (child). Assumes exposure for 12 h/day, 5 days/wk, 52 weeks/yr, for 6 years (child) and 20 years (adult). EPA Inhalation Unit Risk (IUR): 7.8E-6 (µg/m$^3$)$^{-1}$

**Table 2e. Carbon tetrachloride**

CREG: 0.17 µg/m$^3$. Reporting Limit: 1.26 µg/m$^3$. Method Detection Limit: 0.314 µg/m$^3$

| Sampling Location | Sub-Slab Soil Gas | Pre-mitigation indoor air | Pre-mitigation indoor air | Pre-mitigation indoor air | Post-mitigation indoor air | Post-mitigation indoor air |
|---|---|---|---|---|---|---|
| Date (2022) | Jan-26 | Jan-25 | Feb-16 | Mar-31 | Nov-22 (Day) | Nov-22 (Night) |
| VSG / VIA 5, 25, 35 | 0.44 | **0.566** | **0.497** | **0.478 (J)** | **0.39 (J)** | **0.579 (J)** |
| VSG / VIA 6, 26, 36 | 0.459 | **0.604*** | **0.503** | **0.516 (J)** | **0.384 (J)** | **0.591 (J)** |
| VSG / VIA 7 27, 37 | 0.403 | NA | **0.472** | **0.522 (J)** | **0.535 (J)** | **0.516 (J)** |

**Bold = value that exceeds a CV (CREG). ***Value used to estimate exposure. J = value is lab-estimated. Note: for carbon tetrachloride, analytical reporting and method detection limits also exceeded this CV. Cancer risk estimate based on this maximum exposure concentration (0.604 µg/m$^3$), adjusted for a childcare center exposure scenario (adjusted EPC: 0.22 µg/m$^3$): 3.3 E-7 (adult full-time worker); 1.0 E-7 (child). Assumes exposure for 12 h/day, 5 days/wk, 52 weeks/yr, for 6 years (child) and 20 years (adult). EPA Inhalation Unit Risk (IUR): 6.0E-6 (µg/m$^3$)$^{-1}$

**Table 2f. Chloroform**

CREG: 0.043 µg/m$^3$. Reporting Limit: 0.977 µg/m$^3$. Method Detection Limit: 0.309 µg/m$^3$

| Sampling Location | Sub-Slab Soil Gas | Pre-mitigation indoor air | Pre-mitigation indoor air | Pre-mitigation indoor air | Post-mitigation indoor air | Post-mitigation indoor air |
|---|---|---|---|---|---|---|
| Date (2022) | Jan-26 | Jan-25 | Feb-16 | Mar-31 | Nov-22 (Day) | Nov-22 (Night) |
| VSG / VIA 5, 25, 35 | 1.21 | **0.64** | **0.689** | ND | **0.508 (J)** | **0.41 (J)** |
| VSG / VIA 6, 26, 36 | 1.05 | **1.25*** | **1.05** | **1.16** | **2.02** | **1.19** |
| VSG / VIA 7 27, 37 | ND | ND | **0.801** | **0.801 (J)** | **3.37** | **1.26** |

**Bold = value that exceeds a CV (CREG). ***Value used to estimate exposure. J = value is lab-estimated. Note: for chloroform, analytical reporting and method detection limits also exceeded this CV. Cancer risk estimate based on this maximum concentration representative of exposure (1.3 µg/m$^3$; for chloroform, this value is not adjusted): 2.6E-6 (adult full-time worker) and 7.9 E-7 (child). Assumes exposure for 12 h/day, 5 days/wk, 52 weeks/yr, for 6 years (child) and 20 years (adult). If using the maximum detected chloroform concentration overall (3.37 µg/m$^3$) for the cancer calculation, estimated lifetime cancer risk is 7.1E-6 (7 in 1 million) for an adult worker and 2.1E-6 (2 in 1 million) for a child. EPA Inhalation Unit Risk (IUR): 2.35E-5 (µg/m$^3$)$^{-1}$

**Table 2g. Acrylonitrile**

CREG: 0.015 µg/m$^3$. Reporting Limit: 1.09 µg/m$^3$. Method Detection Limit: 0.053 µg/m$^3$

| Sampling Location | Sub-Slab Soil Gas | Pre-mitigation indoor air | Pre-mitigation indoor air | Pre-mitigation indoor air | Post-mitigation indoor air | Post-mitigation indoor air |
|---|---|---|---|---|---|---|
| Date (2022) | Jan-26 | Jan-25 | Feb-16 | Mar-31 | Nov-22 (Day) | Nov-22 (Night) |
| VSG / VIA 5, 25, 35 | 0.621 | ND | ND | ND | ND | **0.078 (J)*** |
| VSG / VIA 6, 26, 36 | 0.477 | ND | ND | ND | ND | **0.076 (J)** |
| VSG / VIA 7 27, 37 | ND | NA | ND | ND | **0.067 (J)** | ND |

**Bold = value that exceeds a CV (CREG). ***Value used to estimate exposure. J = value is lab-estimated. Note: for acrylonitrile, analytical reporting and method detection limits also exceeded this CV. Cancer risk estimate based on the maximum exposure concentration (0.078 J µg/m$^3$), adjusted for a childcare center exposure scenario (0.028 µg/m$^3$): 4.9E-7 (adult full-time worker); 1.5E-7 (child). Assumes exposure for 12 h/day, 5 days/wk, 52 weeks/yr, for 6 years (child) and 20 years (adult). EPA Inhalation Unit Risk (IUR): 6.8E-5 (µg/m$^3$)$^{-1}$

**Table 2h. Naphthalene**

CREG: 0.029 µg/m$^3$. Reporting Limit: 0.262 µg/m$^3$. Method Detection Limit: 0.184 µg/m$^3$

| Sampling Location | Sub-Slab Soil Gas | Pre-mitigation indoor air | Pre-mitigation indoor air | Pre-mitigation indoor air | Post-mitigation indoor air | Post-mitigation indoor air |
|---|---|---|---|---|---|---|
| Date (2022) | Jan-26 | Jan-25 | Feb-16 | Mar-31 | Nov-22 (Day) | Nov-22 (Night) |
| VSG / VIA 5, 25, 35 | **3.69** | **0.393** | **0.398** | ND | **0.357** | **0.629** |
| VSG / VIA 6, 26, 36 | **1.9** | **0.309** | **0.592*** | **0.367** | **0.388** | **0.592** |
| VSG / VIA 7 27, 37 | **1.56** | NA | **0.189** | ND | **0.367** | **0.215 (J)** |

**Bold = value that exceeds a CV (CREG). ***Value used to estimate exposure. J = value is lab-estimated. Note: for naphthalene, analytical reporting and method detection limits also exceeded this CV. Cancer risk estimate based on the maximum concentration representative of exposure (0.592 µg/m$^3$), adjusted for a childcare center exposure scenario (0.21 µg/m$^3$): 1.8E-6 (adult full-time worker); 5.5E-7 (child). Assumes exposure for 12 h/day, 5 days/wk, 52 weeks/yr, for 6 years (child) and 20 years (adult). EPA Inhalation Unit Risk (IUR): 3.4E-5 (µg/m$^3$)$^{-1}$

**Table 2i. Vinyl chloride and 1,2-dibromoethane**

(**not detected,** ½ of method detection limit used to estimate lifetime cancer risks)

| Compound | Detected in indoor air? | Analytical Method Detection Limit (MDL), µg/m³ | ATSDR CREG, µg/m³ | Adjusted EPC (Half the MDL, adjusted for a childcare center scenario), µg/m³ | Lifetime Excess Cancer Risk, using ½ MDL to estimate exposure |
|---|---|---|---|---|---|
| Vinyl Chloride | No | 0.160 | 0.11 | 0.029 | 1.9E-8 (child)<br>3.2E-8 (adult) |
| 1,2-Dibromoethane | No | 0.062 | 0.0017 | 0.011 | 5.1E-7 (child)<br>1.7E-6 (adult) |

Cancer risk estimates assume exposure for 12 h/day, 5 day/wk, 52 weeks/yr, for 6 years (child) and 20 years (adult). EPA Inhalation Unit Risk (IUR), dibromoethane: 0.0006 $(\mu g/m^3)^{-1}$. EPA Inhalation Unit Risk (IUR), vinyl chloride: 8.8E-6 $(\mu g/m^3)^{-1}$ – from birth; 4.0E-6 $(\mu g/m^3)^{-1}$ – during adulthood.

**2j. Isopropanol**

Device Reporting Limit: 1.23 µg/m³. Analytical Method Detection Limit: 1.17 µg/m³

| Sampling Location | Sub-Slab Soil Gas | Pre-mitigation indoor air | Pre-mitigation indoor air | Pre-mitigation indoor air | Post-mitigation indoor air | Post-mitigation indoor air |
|---|---|---|---|---|---|---|
| Date (2022) | Jan-26 | Jan-25 | Feb-16 | Mar-31 | Nov-22 (Day) | Nov-22 (Night) |
| VSG / VIA 5, 25, 35 | 34.9 | 147 | 9.78 | 168 | 165 | 49.7 |
| VSG / VIA 6, 26, 36 | 22.7 | 27.3 | 8.21 | **602*** | **401** | 65.9 |
| VSG / VIA 7 27, 37 | ND | NA | 5.09 | 87 | **275** | 62.7 |

**Bold = value that exceeds a PADEP Indoor Residential Screening Value, of 210 µg/m³ as listed in in PADEP 2022.**

*Value used to estimate exposure.

**Table 2k. Trichloroethylene**

CREG: 0.021 µg/m³. Device Reporting Limit: 1.07 µg/m³. Analytical Method Detection Limit: 0.271 µg/m³

| Sampling Location | Sub-Slab Soil Gas | Pre-mitigation indoor air | Pre-mitigation indoor air | Pre-mitigation indoor air | Post-mitigation indoor air | Post-mitigation indoor air |
|---|---|---|---|---|---|---|
| Date (2022) | Jan-26 | Jan-25 | Feb-16 | Mar-31 | Nov-22 (Day) | Nov-22 (Night) |
| VSG / VIA 5, 25, 35 | 3.78 | **11.2*** | ND | ND | ND | ND |
| VSG / VIA 6, 26, 36 | 0.328 | ND | ND | ND | ND | ND |
| VSG / VIA 7 27, 37 | ND | NA | ND | ND | ND | ND |

**Bold = value that exceeds a CV (CREG, cEMEG, iEMEG, and RMEG). ***Value used to estimate exposure. Cancer risk estimate based on the maximum exposure concentration (11.2 µg/m³), adjusted for a childcare center exposure scenario (adjusted EPC: 4.0 µg/m³): 4.2E-6 (adult full-time worker); 2.6E-6 (child). Assumes exposure for 12 h/day, 5 days/wk, 52 weeks/yr, for 6 years (child) and 20 years (adult). EPA Inhalation Unit Risk (IURs): 2.1E-6 (Non-Hodgkin's Lymphoma), 1.0E-6 (Liver); 1.0E-6 (Kidney) $(\mu g/m^3)^{-1}$

**2l. Acrolein**

Device Reporting Limit: 0.115 µg/$m^3$. Analytical Method Detection Limit: 0.089 µg/$m^3$

| **Sampling Location** | **Sub-Slab Soil Gas** | **Pre-mitigation indoor air** | **Pre-mitigation indoor air** | **Pre-mitigation indoor air** | **Post-mitigation indoor air** | **Post-mitigation indoor air** |
|---|---|---|---|---|---|---|
| Date (2022) | Jan-26 | Jan-25 | Feb-16 | Mar-31 | Nov-22 (Day) | Nov-22 (Night) |
| VSG / VIA 5, 25, 35 | **3.12** | **0.518** | **0.399** | **0.337** | **0.532** | **0.628** |
| VSG / VIA 6, 26, 36 | **2.14** | **0.592** | **0.445** | **0.642** | **0.674*** | **0.559** |
| VSG / VIA 7 27, 37 | 0.468 | NA | **0.222** | **0.39** | **0.537** | **0.539** |

**Bold = value that exceeds a CV (iEMEG and RMEG).** ***Value used to estimate exposure. Note – sub-slab soil gas detections at VIA-5 and VIA-6 also exceed a non-cancer CV. Lifetime excess cancer risk was not calculated for acrolein because it is not classified as a human carcinogen.

## Tables 3a-3c. Non-Cancer Health Effects Evaluation for Isopropanol, Trichloroethylene, and Acrolein

**Table 3a. Exposure Concentrations and Hazard Quotients based on the Highest Isopropanol Concentration**

| Compound | Unadjusted Concentration | Adjusted EPC (acute) | Adjusted EPC (intermediate and chronic) | Acute Hazard Quotient MRL: n/a | Intermediate Hazard Quotient MRL: n/a | Chronic Hazard Quotient MRL: n/a |
|---|---|---|---|---|---|---|
| Isopropanol | 620 µg/m$^3$ | 300 µg/m$^3$ | 220 µg/m$^3$ | - | - | - |

[-] = no health guideline to assess non-cancer hazard quotients. MRL = minimal risk level; n/a = not applicable; EPC = Exposure Point Concentration

**Table 3b. Exposure Concentrations and Hazard Quotients based on the Highest Trichloroethylene Concentration**

| Compound | Unadjusted Concentration | Adjusted EPC (acute) | Adjusted EPC (intermediate and chronic) | Acute Hazard Quotient MRL: n/a | Intermediate Hazard Quotient MRL: 2.1 µg/m$^3$ | Chronic Hazard Quotient MRL: 2.1 µg/m$^3$ |
|---|---|---|---|---|---|---|
| Trichloroethylene | 11.2 µg/m$^3$ | 5.6 µg/m$^3$ | 4.0 µg/m$^3$ | - | **1.9** | **1.9** |

**Bold** = Hazard Quotient > 1. [-] = no health guideline to assess non-cancer hazard quotients. MRL = Minimal Risk Level; n/a = not applicable; EPC = Exposure Point Concentration

**Table 3c. Exposure Concentrations and Hazard Quotients based on the Highest Acrolein Concentration**

| Compound | Unadjusted Concentration | Adjusted EPC (acute) | Adjusted EPC (intermediate and chronic) | Acute Hazard Quotient MRL: 6.9 µg/m$^3$ | Intermediate Hazard Quotient MRL: 0.092 µg/m$^3$ | Chronic Hazard Quotient RfC: 0.02 µg/m$^3$ |
|---|---|---|---|---|---|---|
| Acrolein | 0.674 µg/m$^3$ | 0.34 µg/m$^3$ | 0.24 µg/m$^3$ | <1 | **2.6** | **12** |

**Bold** = Hazard Quotient > 1. MRL = Minimal Risk Level; EPC = Exposure Point Concentration