Health Consultation

Delfasco Forge Superfund Site
Grand Prairie, Dallas County, Texas

EPA ID: TXD988034328

Prepared by the
Texas Department of State Health Services

APRIL 10, 2024

Prepared under a Cooperative Agreement with the
U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES
Agency for Toxic Substances and Disease Registry

Health Consultation: A Note of Explanation

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

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

This concludes the health consultation process for this site, unless additional information is obtained by ATSDR that, in the Agency's opinion, indicates a need to revise or append the conclusions previously issued.

The Texas Department of State Health Services (DSHS) prepared this health consultation for the Delfasco Forge Superfund site, located in Grand Prairie, Dallas County, Texas. This publication was made possible by a cooperative agreement (program # TS-23-0001) with the federal Agency for Toxic Substances and Disease Registry (ATSDR). DSHS evaluated data of verified quality using approved methods, policies, and procedures in effect at the date of publication. ATSDR reviewed this document and concurs with its findings, based on the information presented by DSHS.

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Summary

Introduction

The former Delfasco Forge facility is in Grand Prairie, Dallas County, Texas. The facility operated as a munitions manufacturing and forging facility from 1980 to 1998. Former facility operations used chlorinated solvents containing trichloroethylene (TCE) and other chemicals to degrease metal. These operations contaminated on-site soil and groundwater. The groundwater contamination has spread to the adjacent residential area where an estimated 157 occupied homes (about a 0.2-square mile area) are located above the contaminated groundwater.

In September 2018, the United States Environmental Protection Agency (EPA) added the site to the National Priorities List (NPL).

The Agency for Toxic Substances and Disease Registry (ATSDR) was established by Congress in 1980 under the Comprehensive Environmental Response, Compensation, and Liability Act, also known as the Superfund law. Since 1986, ATSDR has been required by law to conduct public health assessment activities for each site listed on or proposed to the NPL. The Texas Department of State Health Services (DSHS) has a cooperative agreement with ATSDR to perform public health assessment activities for listed or proposed NPL sites in the state of Texas.

DSHS evaluated available environmental data, including groundwater and indoor air samples, to determine if past, present, and future exposures to chemicals in groundwater and indoor air might harm people's health. This health consultation was developed based on this evaluation. DSHS will review and evaluate additional information as it becomes available.

Conclusions

From available information, DSHS reached seven conclusions in this health consultation.

Conclusion 1

For some residential properties sampled during 2008–2016, people's health might have been harmed by breathing trichloroethylene (TCE) that has evaporated into their indoor air from the underlying contaminated groundwater.

Basis for Conclusion

DSHS evaluated indoor air concentrations collected infrequently during 2008–2016 from residential properties located above the contaminated groundwater. All the indoor air samples were taken in May, except for one round of sampling in October 2014. On the basis of the maximum level of TCE detected in indoor air either before or after the installation of vapor mitigation systems, DSHS identified the following health risks:

- Exposure to TCE at 17 properties (16, 17, 18, 20, 21, 36, 38, 39, 42, 43, 57, 58, 59, 60, 81, 85, and 86) during the 3-week or longer period early in the first trimester of pregnancy could cause fetal heart malformations in children.
- Exposure to TCE at 10 properties (17, 18, 20, 21, 38, 39, 42, 43, 60, and 81) could cause harmful immune system effects (such as decreased thymus weight, which could increase the risk for autoimmune diseases) in children and adults.
- The estimated cancer risks from long-term exposure (several decades) at properties 17, 20, and 43 is a health concern for children and adults. DSHS estimated the lifetime cancer risk for children and adults to be greater than 1 in 10,000 people (1E-4).

There is uncertainty with the risk estimates because they are based on the maximum concentration detected in indoor air from limited (1 to 3) sampling events. Sampling events are when environmental samples are collected over a specific period of time.

Additionally, TCE levels detected in indoor air from some residential properties are above the 95th percentile concentrations measured in North American residences, which suggests vapor intrusion might be occurring in some homes [EPA 2011].

Conclusion 2

Worker's health might be harmed by breathing TCE that has evaporated into indoor air of the former Delfasco Forge workplace buildings from the underlying contaminated groundwater.

Basis for Conclusion

DSHS evaluated indoor air concentrations collected from the on-site commercial buildings in May and September of 2020 for occupational exposure to TCE. TCE was detected in indoor air at a level approaching the effect level for fetal heart malformations in the developing fetus of pregnant women. Therefore, workers who are pregnant while working in this building during the first 3 weeks of pregnancy might be at increased risk for fetal heart effects in their children from short-term exposure to TCE. However, there is uncertainty with the risk estimate because it is based on the maximum level detected in indoor air from one sampling event.

Conclusion 3

Future exposures to TCE and other volatile contaminants (those that evaporate easily) in indoor air at residential properties 17, 18, 43, 60, and 85 are not expected if vapor mitigation systems are operating as intended. TCE might still harm people's health at property 20, even though it has a vapor mitigation system.

DSHS cannot determine whether future exposure to TCE is harmful at the other properties with vapor mitigation systems because too few indoor air samples have been collected since installation of the systems.

Basis for Conclusion

Although vapor mitigation systems were installed at 32 properties, inhalation exposures to TCE might occur if these systems are not operating properly. DSHS compared maximum indoor air levels of TCE collected before and after the installation of vapor mitigation systems at six properties (17, 18, 20, 43, 60, and 85). In the most recent sampling results (2016), TCE levels were either not detected or were below the comparison value at five properties (17, 18, 43, 60, and 85). Adverse health effects from TCE in indoor air are not expected at these properties.

However, at property 20, TCE was above levels that could cause noncancer health effects (including fetal heart malformations in children whose mothers were exposed early in their pregnancy and immune system effects) for people living at the property. The elevated level of TCE was attributed to a hole in the property's

foundation and to DSHS's knowledge that the hole has not been repaired. EPA is communicating with the residents of the property about ongoing mitigation efforts.

At the other 26 properties with vapor mitigation systems, indoor air samples were either not collected or collected once 2 months after systems were installed in 2014. TCE levels in indoor air were above comparison values at some homes. Additional sampling is needed to fully assess the effectiveness of the vapor mitigation systems. The effectiveness of vapor mitigation systems might vary over time and with seasonality.

Conclusion 4

Exposure to tetrachloroethylene (PCE) and benzene in indoor air at some residential properties and at the former Delfasco Forge workplace buildings is not expected to harm people's health.

Basis for Conclusion

PCE and benzene were detected above comparison values (CVs) in indoor air at some residential properties and in the on-site occupational buildings. However, PCE and benzene were below levels that cause noncancer health effects. Cancer risks for PCE and benzene were estimated to be less than 1 in 1,000,000 people (1E-6). There is no concern for cancer from these exposures. However, there is uncertainty with the risk estimate because it is based on the maximum level of chemical detected in indoor air collected from one sampling event.

Conclusion 5

Due to lack of data, DSHS cannot currently conclude whether breathing indoor air at other residential and commercial buildings above the groundwater contamination might harm people's health because indoor air samples were not available.

Basis for Conclusion

DSHS estimates there are about 157 occupied residential properties and 20 occupied commercial or industrial buildings within 100 feet of the contaminated groundwater. Given the depth of groundwater (30 to 70 feet below ground surface), contaminants can move from the groundwater and soil and enter the interior of these buildings.

Most residential properties and businesses above the groundwater contamination have not been sampled. Some of these properties require more sampling to ensure that harmful exposures, if they are occurring, can be identified and stopped. Recent

groundwater sampling events show the groundwater contamination is moving to the northeast and past the current evaluated residential area. That means vapor intrusion might occur in a greater number of residential properties than previously estimated. The northeastern extent of the contamination has not been fully determined.

Conclusion 6

Water from private residential water wells that contain volatile organic compounds, such as TCE, PCE, and benzene, is not expected to harm people's health when used for irrigation, gardening, and recreational activities.

Basis for Conclusion

A 2006 drinking water survey identified 16 residential water wells within a 0.5-mile radius of the site. The wells were determined to be completed in the shallow groundwater. Although the wells are no longer used for domestic purposes, they may be used for irrigation, gardening, and recreation. A 2011 water well survey identified six unregistered wells within the groundwater contamination area. Of those six wells, one was used for irrigation and the other five were not in use. All homes are connected to city water for residential use. Therefore, exposure to contaminants in the groundwater during activities such as irrigation, gardening, and recreation using private well water could not be fully assessed because no groundwater samples from residential private water wells have been collected. However, exposure to volatile organic compounds (TCE, PCE, and benzene) in water from irrigation, gardening, and recreational activities would likely be minimal. These chemicals evaporate from water relatively quickly and readily disperse in outdoor air.

Conclusion 7

Residential exposure to drinking from the public water supply is not expected to harm people's health.

Basis for Conclusion

Residences are connected to the public water system of Grand Prairie, Texas. The 2011 well water survey found that none of the unregistered wells identified are used to supply water to homes. One state registered public drinking water well serves the public water system in the area of the groundwater contamination However, it does not connect to the contaminated groundwater and did not contain any site-related chemicals when sampled in 2011. This water well is completed in the lower Woodbine and Trinity aquifers that reach a depth of 2,163 feet below

ground surface and obtains water from 2,006 to 2,163 feet below ground surface. The Woodbine and Trinity aquifers are below the area of groundwater contamination and the Eagle Ford shale formation blocks movement between the shallower groundwater contamination and the much deeper aquifers. The Eagle Ford shale formation begins at 27 to 73 feet below ground surface and is approximately 145 feet thick and relatively impermeable.

Conclusion 8

Exposure to TCE and other site contaminants at James Fannon Middle School is not expected to harm the health of students and staff based on available information. However, there is potential for future exposure.

Basis for Conclusion

Exposure to TCE and other site contaminants is not likely to have taken place at James Fannin Middle School because the contaminated groundwater is not directly beneath the school and the direction of groundwater flow is moving away from the school. EPA also collected passive soil gas samples at the school that suggest vapor intrusion is not taking place. However, vapor intrusion might be a future potential exposure pathway because of several factors:

- Vapor dispersion of TCE from groundwater can vary over time and location
- Results from using passive soil gas samples to quantify air concentrations are not always reliable.

Recommendations

DSHS recommends that EPA do the following:

- Sample indoor air (and sub-slab soil gas and outdoor air when appropriate) in homes within 100 feet of the current groundwater plume, as delineated by 1 microgram per liter (μg/L) of TCE in groundwater. Sampling is recommended in hot and cold weather conditions when windows and doors are mostly closed to maintain climate control while heating and air conditioning operate.[1] Prioritization of indoor air sampling in residential homes should be for homes that have not yet been sampled, have not been sampled since receiving a vapor mitigation system, or have elevated indoor levels of TCE.
- Resample homes that were sampled between 2008 and 2016, due to possible changes in TCE concentrations in the groundwater since then, and consider multiple sampling events in cold and hot weather to understand potential seasonal variation.
- Continue its efforts to offer vapor mitigation systems to residents within 100 feet of the TCE groundwater contamination.
- Sample groundwater and active soil gas (near-source) within 100 feet of homes that refuse indoor air samples to assess potential risk to individual homes.
- Conduct a comprehensive groundwater sampling event to determine which additional homes might be contaminated by the TCE in groundwater. The extent of groundwater contamination moving to the northeast of the site has not been fully determined.
- Continue to monitor TCE levels in the shallow groundwater near James Fannin Middle School until remedial actions prevent further spread of contaminated groundwater.

[1] Also consider monitoring indicators, tracers, and surrogates to assess potential for active vapor intrusion conditions:

- https://iavi.rti.org/assets/docs/Temp_Measurement_Fact_Sheet_int.pdf
- https://iavi.rti.org/assets/docs/Pressure_Measurement_Fact_Sheet_Int.pdf
- https://iavi.rti.org/assets/docs/Radon_methods_fact_sheet_int.pdf

Next Steps

DSHS will

- Provide the final version of this document to community members, city officials, the Texas Commission on Environmental Quality (TCEQ), EPA, and other interested parties.
- Continue to work with EPA and TCEQ to evaluate additional data as they become available.
- Continue to engage with the community through community meetings and addressing community concerns.

For More Information

For more information about this health consultation, contact the DSHS Health Assessment and Toxicology Program at 1-888-681-0927.

Purpose and Statement of Issues

This health consultation was prepared for the Delfasco Forge Superfund site in accordance with a cooperative agreement between the Agency for Toxic Substance and Disease Registry (ATSDR) and the Texas Department of State Health Services (DSHS). The former Delfasco Forge facility operated as a munitions manufacturing and forging facility from 1980 to 1998. Facility operations contaminated soil and groundwater with chlorinated solvents, including trichloroethylene (TCE) and other volatile organic compounds (VOCs). The groundwater contamination has spread to the adjacent residential area. About 157 occupied homes (about a 0.2-square mile area) sit atop of the contaminated groundwater.

In September 2018, the U.S. Environmental Protection Agency (EPA) listed the site on the National Priorities List (NPL) of hazardous waste sites in the United States [EPA 2018a, EPA 2018b]. This health consultation evaluates the indoor air data collected from the facility buildings and surrounding residential homes from 2008 to 2020. The purpose of the health consultation is to determine whether exposure to the hazardous substances could harm public health.

Background

Site Description

The former Delfasco Forge facility is in a mixed residential, commercial, and industrial area in Dallas County, Texas, and approximately 14 miles west of downtown Dallas (Figure 1). The site is bordered by residential homes to the north and east, an empty lot and commercial business to the south, and NE 28th Street to the west. A middle school is located on the other side of NE 28th Street to the north and west of the facility. The site address is 114 NE 28th Street Grand Prairie, Texas.

While operational, the Delfasco Forge property consisted of two buildings. One building housed offices and a forge shop (Building A). The other building contained a machine shop (Building B) (Figure 2). Most of the remaining lot consisted of asphalt or concrete parking areas. Currently, the site is fenced on all sides where buildings are not present and leased to a concrete sawing business, which uses the two buildings.

Figure 1. Delfasco Forge Superfund site location [EA 2021]

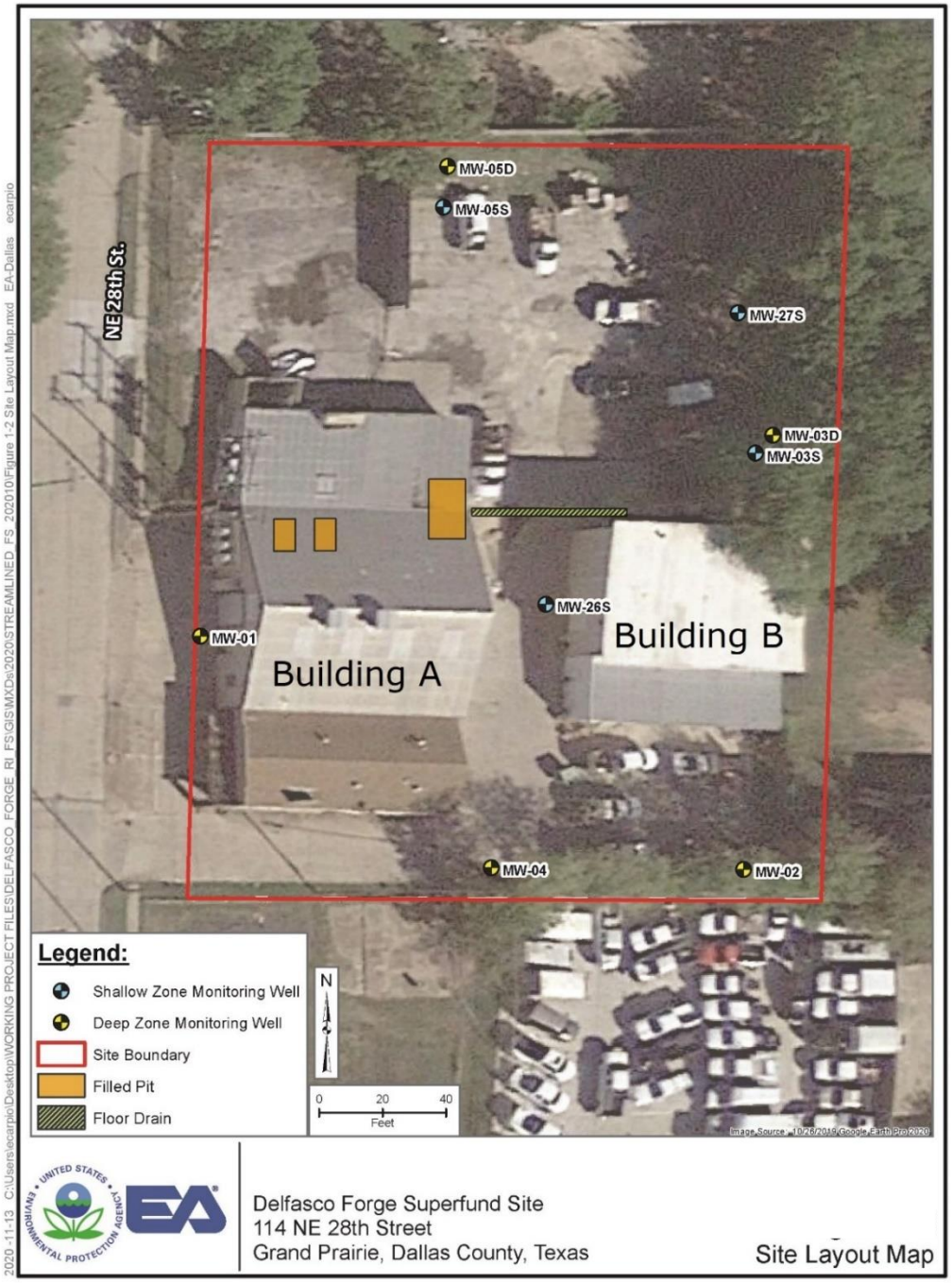

Figure 2. Current site layout - Delfasco Forge Superfund site [adpated from EA 2021]

Site History

Trichloroethylene (TCE) was reportedly used in on-site operations in small quantities by spot hand application. Used and unused TCE substances were stored in 55-gallon drums on site [EPA 2018b]. Contamination into soil and groundwater likely occurred from two sources, including an elongated storm drain and a former sump (Figure 2). In 1998, Delfasco Forge vacated the facility and ceased on-site operations.

In 2002, Delfasco Forge conducted a phase II environmental site assessment and identified TCE and TCE breakdown products in groundwater [EnSafe 2002]. Later in 2002, Delfasco Forge entered the Texas Commission on Environmental Quality (TCEQ) voluntary cleanup program. Further investigations of soil and groundwater found that the TCE-groundwater contamination covered an area of about 65 acres (approximately 1,100 feet wide and 2,650 feet long) and extended below a residential area to the northeast of the site [EnSafe 2005, 2007]. The groundwater flow direction was determined to be northeast of the site and lies beneath a residential area.

More recent sampling has shown that the groundwater contamination plume continues to move in the same northeast direction, potentially affecting a larger area than previously identified [EA 2021].

In addition to TCE, tetrachloethylene (PCE) and TCE breakdown chemicals were identified in subsequent groundwater investigations. These included 1,2-dichloroethane, cis-1,2-dichloroethene, 1,1-dichloroethene, 1,1,2-trichloroethane, and vinyl chloride. Other contaminants of concern not related to TCE were also identified. These included benzene, carbon tetrachloride, chloroform, and 1,4-dioxane [EnSafe 2002, Ensafe 2005].

Investigations at the site included the following:

- 2002 to 2005 – Investigations were conducted while the facility was in the TCEQ voluntary cleanup program. Delfasco Forge conducted a phase II environmental site assessment [Ensafe 2002] and an affected property assessment [Ensafe 2005]. Chlorinated VOCs were found in soil samples collected within the former facility boundary (depths ranging from 1 foot to 35 feet below ground surface [bgs]) [Ensafe 2002, 2005].

- 2005 – Delfasco Forge unsuccessfully applied for a municipal settings designation (MSD). An MSD allows a city to approve an ordinance or covenant to restrict the groundwater use for public consumption. EPA recommended that the city of Grand Prairie not approve the MSD because of the potential for vapor intrusion from the chemicals in the groundwater.

- 2006 – EnSafe Inc. conducted a drinking water survey and identified 16 shallow private residential water wells within a 0.5-mile radius of the site [EnSafe 2006]. EnSafe determined that the wells were generally completed in the alluvial aquifer and were not used for drinking water, although some wells were potentially used for irrigation.
- 2008 – EPA conducted a vapor intrusion investigation in the residential neighborhood northeast of the site. Sub-slab soil gas and indoor air samples were collected from a total of 16 homes and 2 commercial buildings. TCE was detected in ten of the 18 buildings [EPA 2018]. On the basis of these results, EPA ordered Delfasco Forge to delineate the extent of groundwater contamination and mitigate vapor intrusion in contaminated homes and commercial buildings.
- 2008 – EPA installed vapor mitigation systems at four homes. These homes had the highest TCE concentrations measured during the investigation [EPA 2016].
- 2008 – Delfasco Forge filed for Chapter 7 bankruptcy. TCEQ determined there was an imminent threat to human health of residents living above the plume and referred the site to EPA's Resource Conservation and Resource Act (RCRA) program for further action under RCRA authority.
- 2009 – DSHS and the Texas Environmental Health Institute, in consultation with EPA, the University of Texas-Austin, and ATSDR conducted an exposure investigation at the Delfasco Forge site and nearby residential areas. Samples of tap water, soil vapor, and indoor air from homes and blood and urine from residents of these homes were collected and analyzed for TCE. The results showed that TCE blood levels were highly correlated with the indoor air levels of TCE [DSHS 2012, Archer et al. 2015].
- 2011 – TCEQ collected a total of seven groundwater samples from two public supply wells serving the public water system and from four monitoring wells, and six soils samples from the Delfasco Forge property [EPA 2018]. TCE was detected in four monitoring wells above the EPA Hazard Ranking System groundwater pathway benchmark level but was not detected in the two wells serving the public water system.
- 2011 – TCEQ conducted a water well survey that identified six unregistered wells within the groundwater plume area. Of those six wells, one was used for irrigation and the other five were not in use. All the homes were connected to city water for residential use [EA 2011].
- 2013 – TCEQ collected samples from 21 monitoring wells located outside the facility boundary to delineate the extent of groundwater contamination [EA 2013].
- 2014 – TCEQ collected samples from 29 monitoring wells to delineate groundwater contamination and address data gaps in the 2013 groundwater sampling [EA 2014].

- 2014 – EPA collected indoor air samples from a total of 30 homes before and/or after the installation of vapor mitigation systems. EPA offered to install vapor mitigation systems at 87 homes located within an area EPA identified as the vapor mitigation focus area. The focus area was roughly defined by TCE levels of 5 microgram per liter (µg/L) in groundwater. Of these homes, 30 have had the vapor mitigation systems installed [EPA 2014, EPA 2016, Tetra Tech 2013, Tetra Tech 2017]. There were two types of vapor mitigation systems installed depending on the structure type of the home. Sub-slab depressurization systems were installed on homes with slab on grade foundations and crawlspace ventilation systems were installed on homes with pier and beam type foundations.
- 2016 – EPA collected a total of 23 indoor air samples from 6 homes as part of a post-mitigation assessment. These homes had vapor mitigation systems installed in 2014 [EPA 2016].
- 2016 – EnSafe collected groundwater samples from 31 monitoring wells on behalf of TCEQ's Superfund Site Discovery and Assessment program [EnSafe 2016].
- 2018 – EPA listed Delfasco Forge on the NPL.
- 2020 - EPA conducted remedial investigation activities and collected a total of 19 groundwater samples from 15 monitoring wells on or near the site, a total of 6 indoor air samples from on-site buildings, and a total of 12 on-site sub-slab and active soil gas samples. Much of the sampling was conducted on-site to test the soil vapor extraction and passive barrier treatment remediation techniques [EA 2021].

Site Visits

In August 2008, DSHS staff attended an EPA-hosted public meeting.

In 2009, DSHS and ATSDR participated in an EPA-hosted open house meeting in Grand Prairie. The meeting informed community members about the results of the vapor intrusion investigation. It also gave participants the opportunity to discuss their results individually with DSHS and ATSDR staff members.

In August and October 2013, DSHS staff conducted door-to-door community outreach activities to inform residents of homes about the availability of free vapor mitigation systems from EPA.

In 2018 and 2022, DSHS staff participated in EPA-hosted public meetings virtually (due to the Coronavirus Disease 2019 pandemic and travel restrictions) and addressed health concerns from community members.

In June 2022, DSHS staff participated in an in-person EPA-hosted public meeting at the Shotwell Library in Grand Prairie to discuss the on-going remedial investigation.

In August 2023, DSHS staff participated in an in-person EPA-hosted public meeting at the Tony Shotwell Life Center in Grand Prairie to discuss the proposed cleanup strategy for the contaminated groundwater at the site.

Land Use

DSHS estimates that 170 properties are within 100 feet (either vertically or horizontally) of the TCE groundwater contamination, as delineated by 5 µg/L, which is EPA's maximum contaminant level (MCL) for TCE in drinking water (Figure 6) [EnSafe 2016]. This includes 136 occupied residential properties, 12 vacant residential properties, 17 occupied commercial/industrial properties, and 5 vacant commercial/industrial properties. The hydraulic groundwater gradient (direction of flow) of the shallow groundwater has been determined to be to the northeast of the site (Figure 6).

Site Geology and Hydrogeology

The groundwater beneath the site is in the uppermost groundwater bearing unit (GWBU) of the Eagle Ford Aquifer. The GWBU consists of a shallow zone (20 to 30 feet bgs) and a deep zone (40 to 70 feet bgs) that are hydraulically connected through layers of clay and silty sands [EPA 2018b, EnSafe 2007]. TCE contamination has been detected in both the shallow and deep zones of the GWBU. Both the shallow and deep zones are within 100 feet of the ground surface, which is close enough to potentially be a vapor intrusion concern.

The shallow subsurface is highly variable and consists of inter-bedded units of clay, silt, and sand. Water moves laterally north and east from the Delfasco Forge facility through these sands (Figure 8) [EPA 2018, EnSafe 2016]. The vadose zone, the area above the water table, varies from approximately 10 to 45 feet bgs. It has high-swelling inorganic clays that might crack as they dry and shrink, resulting in a pathway for water and vapor migration [EPA 2018, EnSafe 2007, Wise and Hudson 1971].

Demographics

The 2010 United States Census Bureau reported the total population for Dallas County and the City of Grand Prairie as 2,368,139 and 175,396 persons, respectively [USCB 2010]. The Census Bureau reported 5,800 people residing in 1,692 housing units within a 1-mile radius of the site in 2010. At the time of the census, 794 children under the age of 6 years and 1,257 women of child-bearing age (15–44 years) resided in this area (Figure 3).

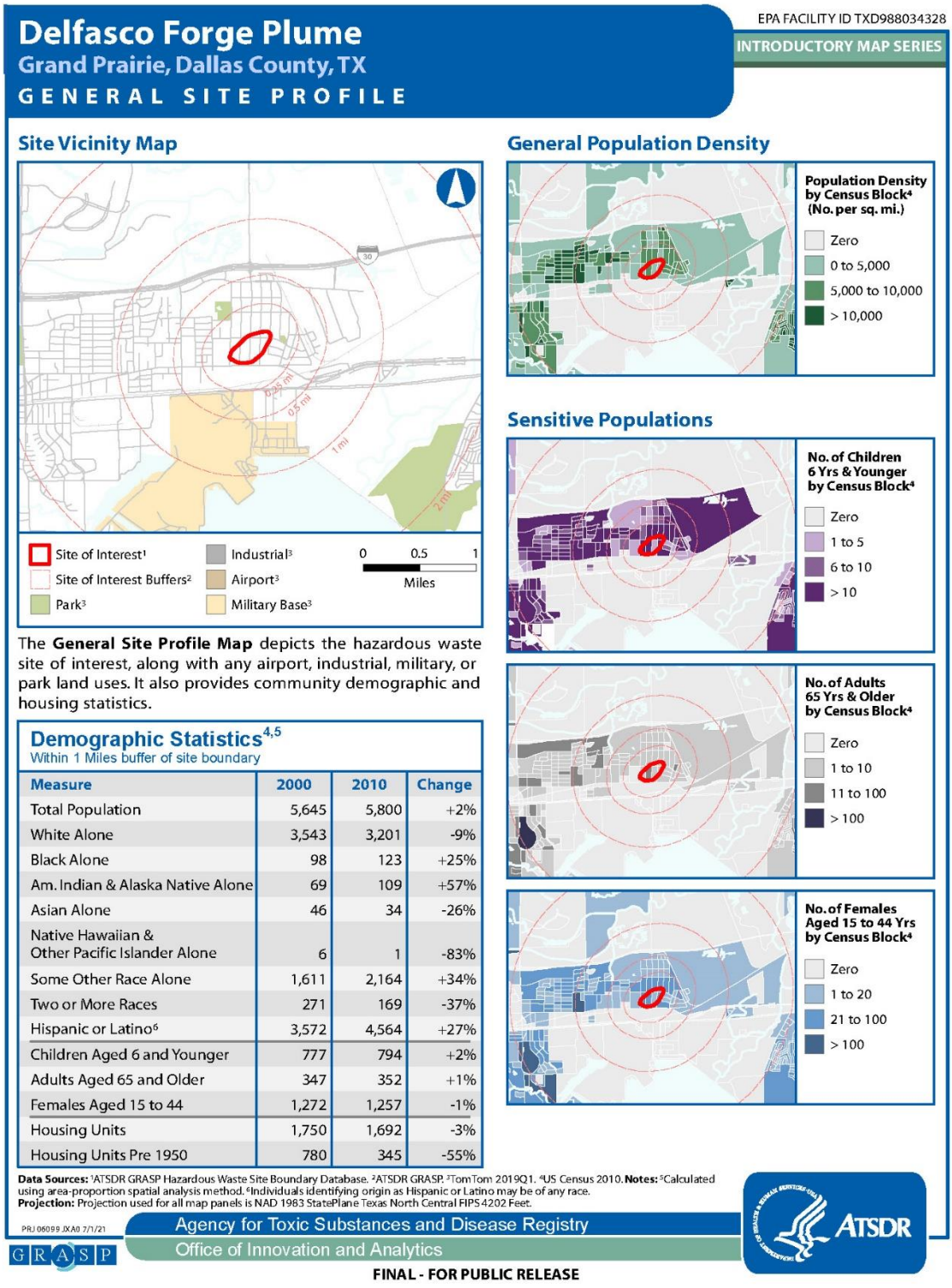

Figure 3. Demographic information for area within 1 mile of the Delfasco Forge Superfund site

Discussion

Environmental Data

DSHS evaluated results of indoor air, groundwater, and soil gas samples collected by EPA and TCEQ. The samples were collected during site investigation activities and to evaluate the vapor intrusion pathway. The samples were collected and analyzed following EPA's standard protocols and quality assurance/quality control guidelines. DSHS assumed adequate quality assurance/quality control procedures were followed regarding data collection, chain of custody, laboratory procedures, and data reporting. Residents were asked to remove other potential sources of indoor contamination before indoor air samples were taken.

Samples were analyzed for VOCs. Duplicate samples were collected for quality control purposes. DSHS used the higher concentration of the duplicate samples when determining exposure point concentration. Environmental data used in the health consultation are listed below.

Groundwater

DSHS evaluated groundwater sampling data collected from monitoring wells to help determine the potential for vapor intrusion at the residential area and on-site business. This included groundwater samples collected from monitoring wells placed in the residential area and at the on-site business. Sampling data included

- 21 groundwater samples and two duplicates from five on-site and 16 off-site monitoring wells (Figure 4) [EA 2013],
- 29 groundwater samples and four duplicates from nine on-site and 20 off-site monitoring wells (Figure 5) [EA 2014],
- 31 groundwater samples and four duplicates from nine on-site and 22 off-site monitoring wells (Figure 6) [EnSafe 2016], and
- 19 groundwater samples on and near the site from 15 monitoring wells [EA 2021].

Soil Gas and Indoor Air

DSHS evaluated indoor air samples collected from residential properties located north and east of the facility and from the on-site buildings. Sampling data included

- five indoor air samples at five residential properties in 2008 [Dynamac Corporation 2008],
- 11 indoor air samples at 11 residential properties in 2009 [DSHS 2012, Archer et al. 2015],
- 20 indoor air samples from residential properties before the installation of vapor mitigation systems and 24 indoor air samples from properties after the installation of vapor mitigation systems in a total of 30 residential properties collected in 2014 [EPA 2014], and

- six indoor air samples from a total of six residential properties after the installation of vapor mitigation systems from 2016 [EPA 2016].

DSHS evaluated sub-slab soil gas and active soil gas (ASG) samples taken in 2020 to help determine the potential for vapor intrusion at the on-site business. Sampling data included

- six indoor air samples from the on-site buildings and 12 on-site sub-slab and active soil gas samples (Figure 7) [EA 2021].

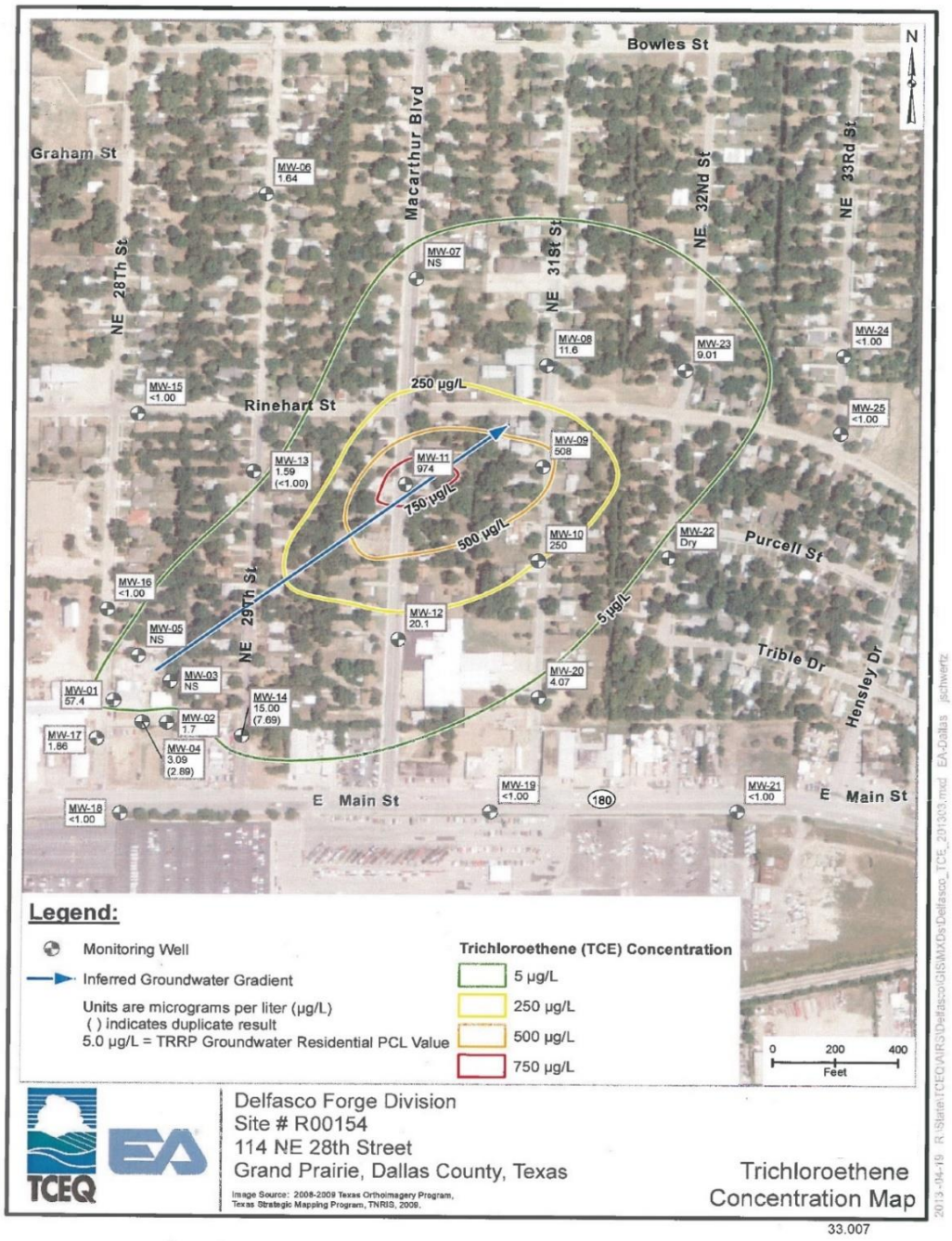

Figure 4. Groundwater sample locations that had trichloroethylene (TCE) concentrations and plume delineation in 2013. Groundwater plumes in the shallower and deeper zones of the upper aquifer were not differentiated in this plume delineation [EA 2013].

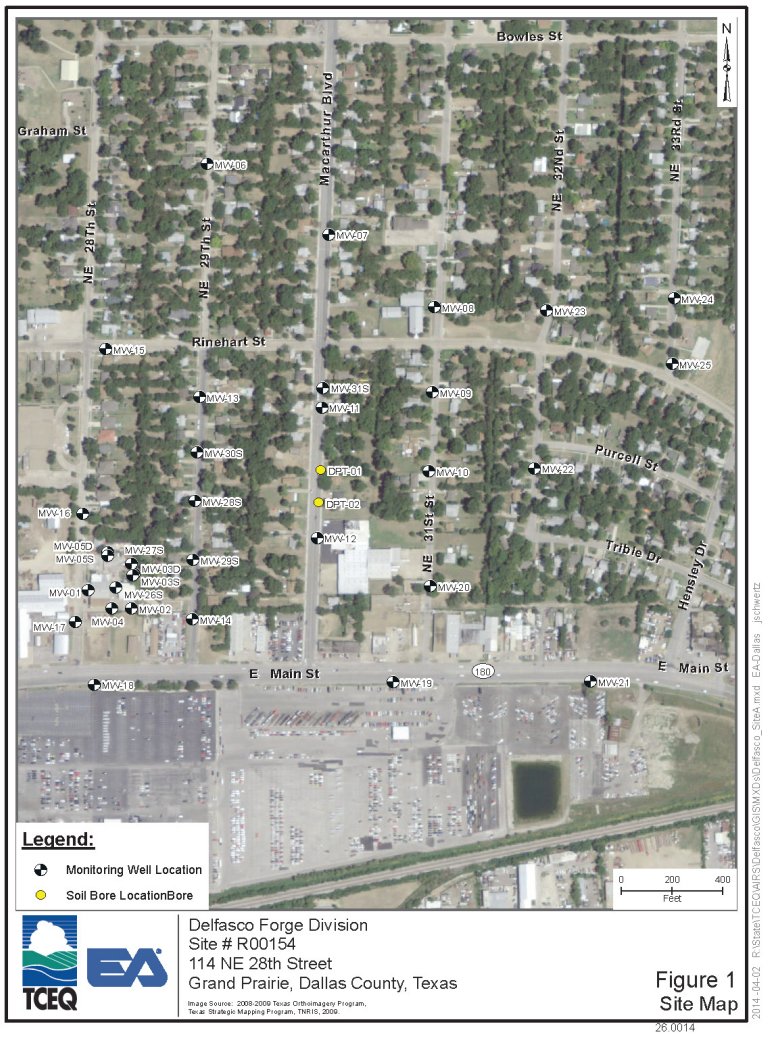

Figure 5. Groundwater sample locations in 2014 - Delfasco Forge Superfund site [EA 2014]

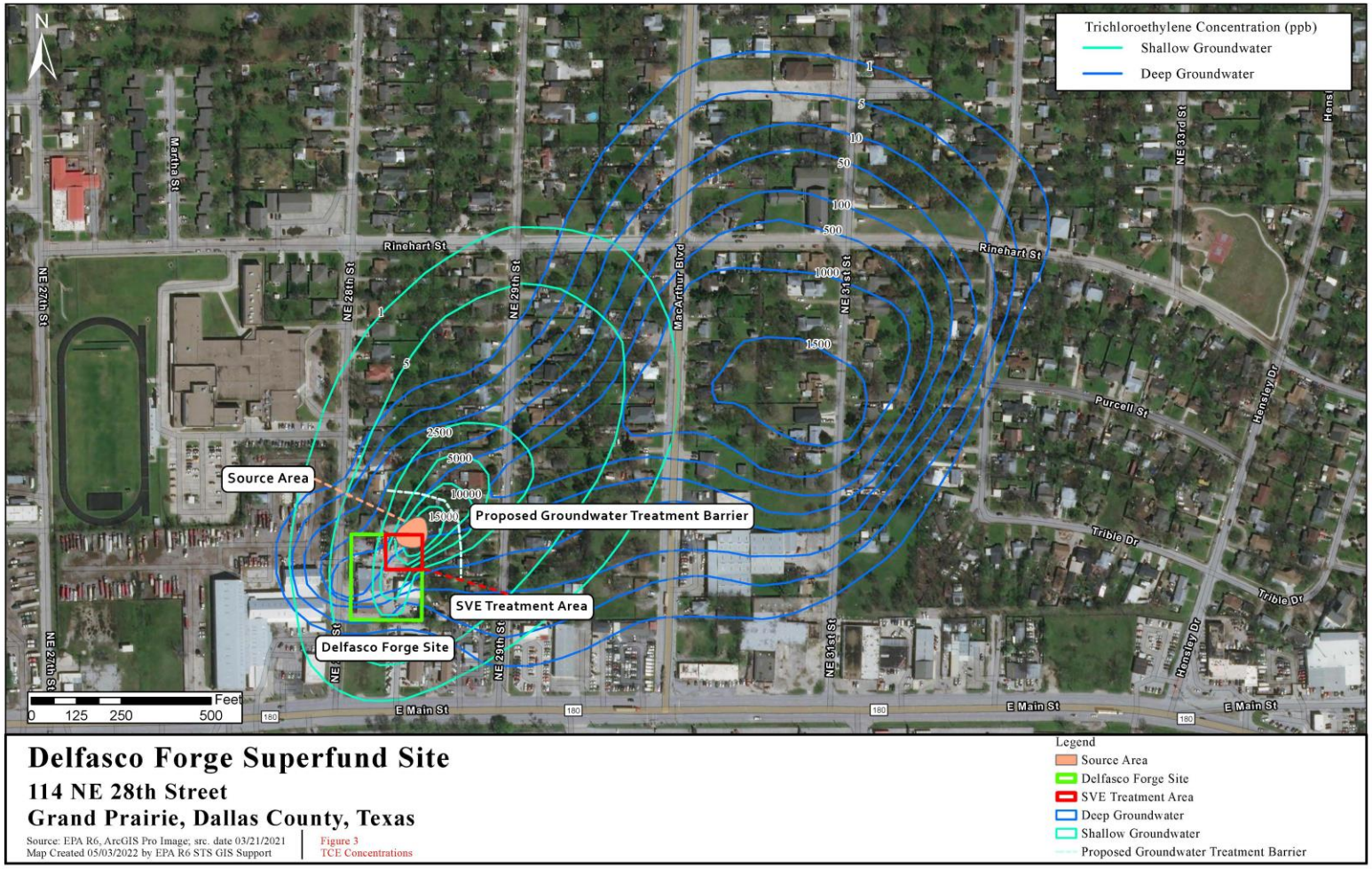

Figure 6. Trichloroethylene (TCE) shallow and deep groundwater plume delineation based on 2016 groundwater sampling. The shallow groundwater is 20–30 ft below ground surface (bgs) and the deeper groundwater is 40–70 ft bgs. Contamination identified in both levels of the aquifer can contribute to indoor air pollution through vapor intrusion [EPA 2023, EnSafe 2016].

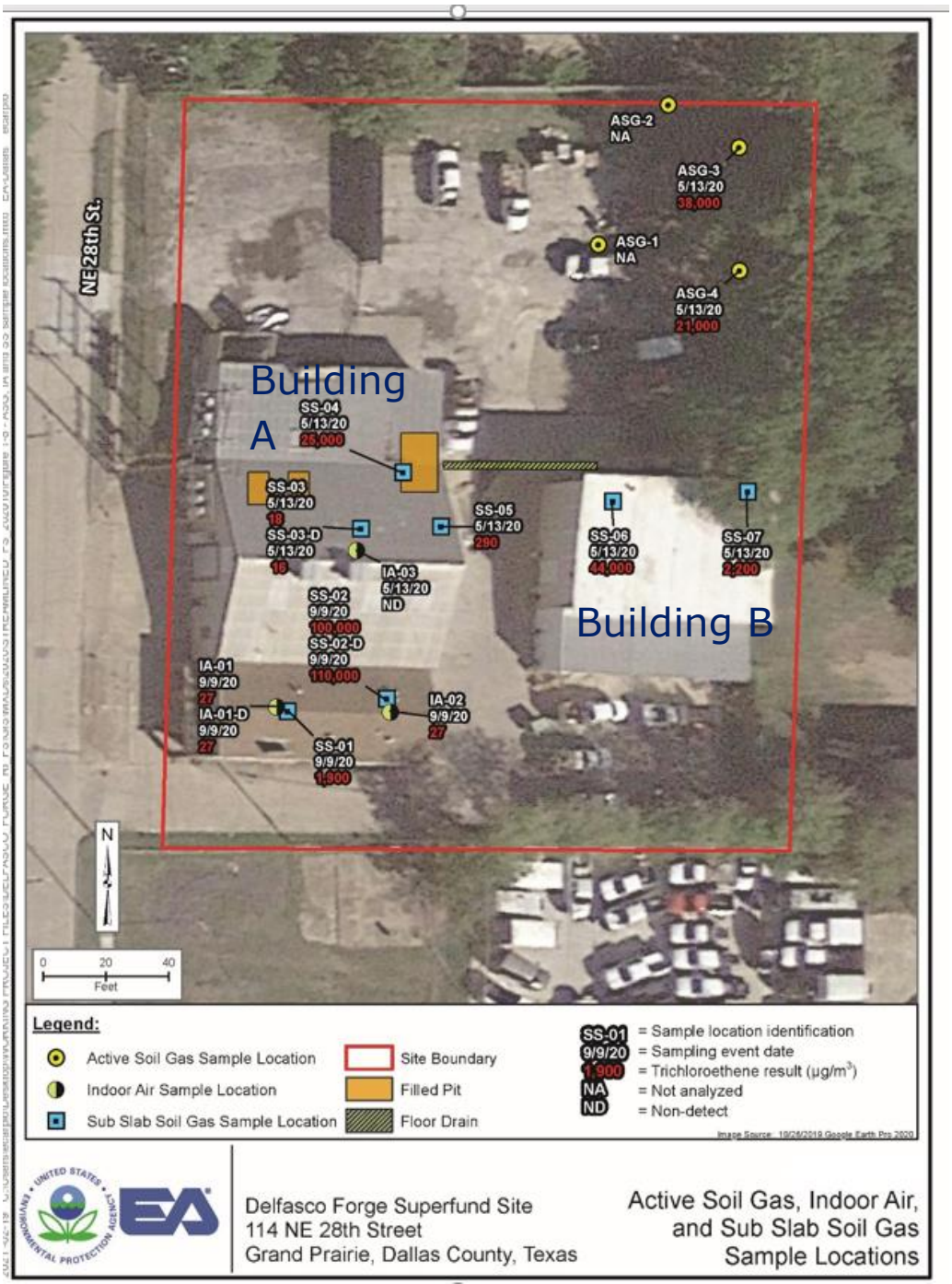

Figure 7. On-site indoor air, sub-slab soil gas, and active soil gas sample locations in 2020 – Delfasco Forge Superfund site [adapted from EA 2021]

Vapor Intrusion Pathway

Vapor Intrusion

The vapor intrusion pathway warrants consideration at this site because of the volatile nature of TCE and other VOCs detected in the groundwater and because the groundwater contamination is located within 100 feet beneath homes (24–55 feet bgs).

Vapor intrusion is the migration of VOCs from the subsurface-contaminated groundwater and soil through pore spaces of soil into the indoor air of buildings (Figure 8). However, the concentrations of contaminants entering the indoor air from the subsurface are dependent on site- and building-specific factors such as building construction, soil type and moisture content, air conditioning/heating settings in the building, ventilation in buildings, and number and spacing of cracks and holes in the foundation. Additionally, estimating indoor air concentrations that people breathe from vapor intrusion has inherent uncertainty because of the dynamic nature of the pathway in different conditions. Estimates must account for varying air exchange for a range of climatic conditions. Because of these uncertainties, indoor air samples collected in cold weather (when windows and doors are most likely to remain closed, allowing soil gas vapors to accumulate indoors) and hot weather are needed to fully characterize health risks from vapor intrusion.

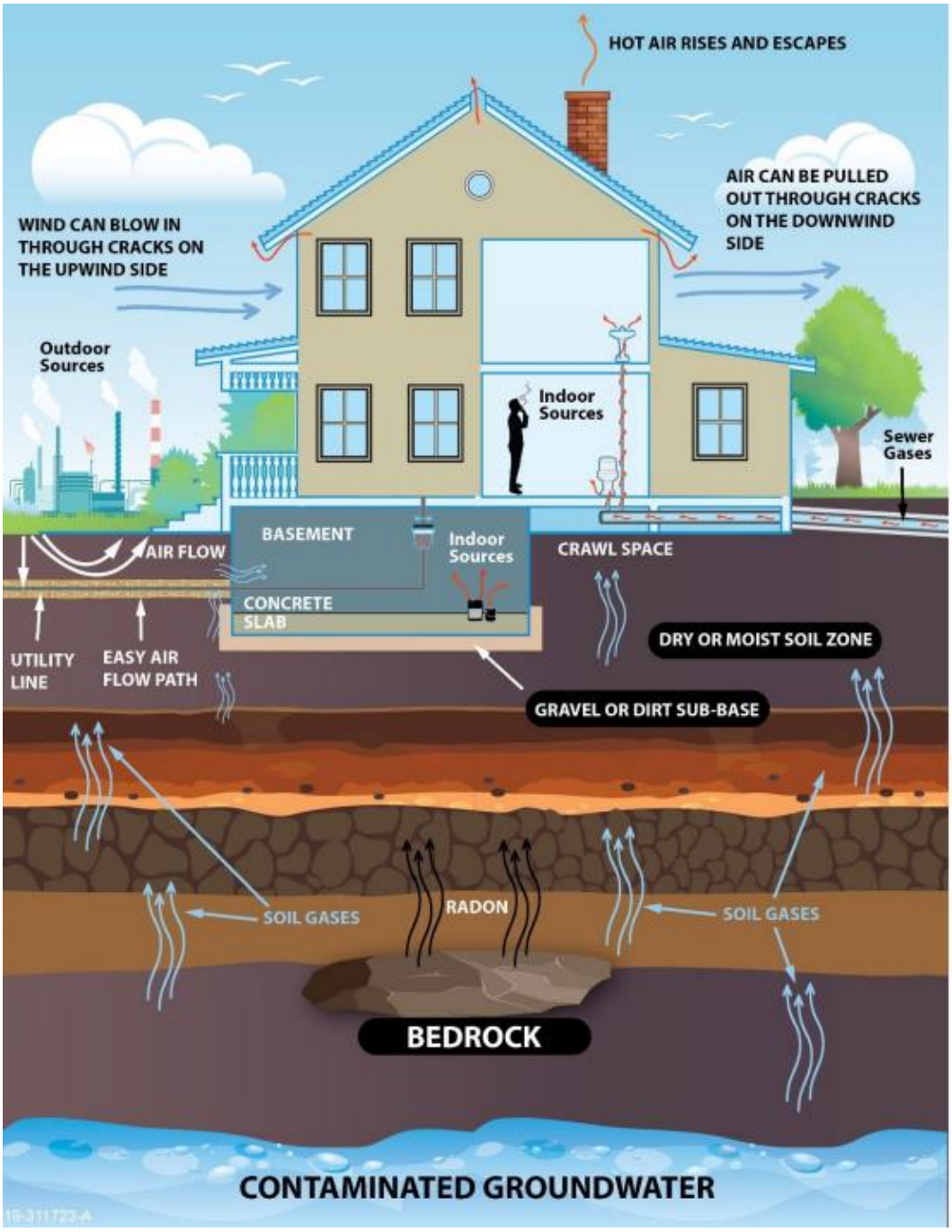

Figure 8. Simplified schematic of vapor intrusion [ATSDR 2016]

Process to Evaluate Environmental Contamination

DSHS conducted a three-step process using available environmental sampling data to evaluate the public health implications. First, DSHS conducted an exposure pathway analysis to identify how people might be exposed. Second, DSHS compared sampling data to CVs. Third, when CVs were exceeded, DSHS conducted a more detailed public health evaluation of contaminants of concern to determine whether harmful effects might be possible [ATSDR 2005a].

Exposure Pathway Analysis

An exposure pathway describes how a chemical moves from its source and comes into physical contact with people. Identifying exposure pathways is important in a health consultation because adverse health effects from contaminants can only happen if people are exposed to contaminants. The presence of a contaminant in the environment does not necessarily mean that people are coming into contact with it.

Five elements are considered in the evaluation of exposure pathways:

1. A source of contamination
2. An environmental media that could absorb or transport the contamination
3. A point of exposure where people could contact the contaminated media
4. A route of exposure, such as inhalation, ingestion, or dermal contact
5. An identifiable exposed population

DSHS divided exposure pathways into three categories: completed, potential, and eliminated.

- A completed exposure pathway occurs when all five elements are present, and exposure has occurred, is occurring, or will occur in the future.
- A potential exposure pathway occurs when one or more of the five elements cannot be identified but might have been present in the past or be present at some point in the future.
- Eliminated exposure pathways are missing one or more elements and exposure cannot occur.

The exposure pathway analysis identifies the different ways people could be or might have been exposed to the environmental contamination in the past, present, and future.

Completed Exposure Pathways

Inhalation of contaminants in residential indoor air (past, present, and future)

The pathway for inhalation of contaminants in residential indoor air is completed at 42 residential properties, based on active indoor air sampling results. This exposure pathway involves contaminant vapors moving from groundwater and soil and entering the interior of these homes (Figure 8). VOCs, such as TCE, PCE, and benzene, were detected in residential homes where indoor air samples were collected between 2008 and 2016 (Table 14, Appendix C). Environmental samples documenting the contamination were first taken in 2002; therefore, the extent of contamination and knowing when residential exposure began are uncertain. Delfasco Forge began operations in 1981. Exposure between 1981 and 2002 could not be evaluated.

To date, 32 properties have vapor mitigation systems installed. These include properties 9, 16, 17, 18, 20, 21, 25, 36, 38, 39, 42, 43, 55, 56, 57, 58, 59, 60, 61, 65, 66, 68, 69, 79, 80, 81, 85, 86, 100, 101, 104, and 105 (Table 1). Of these homes, 24 were sampled in October 2014, about 2 months after vapor mitigation systems were installed, and 6 homes that had vapor mitigation systems installed in 2014 were sampled in 2016 (Table 1).

Table 1. Timeline of indoor air sampling and vapor mitigation system installation in the residential homes north of the Delfasco Forge Superfund site

Property ID	Indoor air sampled in May 2008	Vapor mitigation system installed in 2008	Indoor air sampled in May 2009	Indoor air sampled in May 2014	Vapor mitigation system installed in July to August 2014	Indoor air sampled in October 2014	Indoor air sampled in May 2016
7	—	—	X	—	—	—	—
9	—	—	—	X	Installed	X	—
16	X	—	X	X	Installed	X	—
17	X	—	X	X	Installed	X	X
18	—	—	—	X	Installed	X	X
20*	—	Installed	X	X	Installed	X	X
21	—	Installed	X	—	—	—	—
25	—	—	—	X	Installed	X	—
30	—	—	X	—	—	—	—
36	—	—	—	X	Installed	—	—
38	X	—	—	X	Installed	X	—
39	—	—	—	X	Installed	—	—
42	X	Installed	—	—	—	—	—

Property ID	Indoor air sampled in May 2008	Vapor mitigation system installed in 2008	Indoor air sampled in May 2009	Indoor air sampled in May 2014	Vapor mitigation system installed in July to August 2014	Indoor air sampled in October 2014	Indoor air sampled in May 2016
43*	X	Installed	X	X	Installed	X	X
44	—	—	X	—	—	—	—
55	—	—	—	X	Installed	X	—
56	—	—	X	—	Installed	X	—
57	—	—	X	X	Installed	X	—
58	—	—	—	—	Installed	X	—
59	—	—	—	—	Installed	X	—
60	—	—	—	X	Installed	X	X
61	—	—	—	—	Installed	—	—
65	—	—	—	—	Installed	—	—
66	—	—	—	—	Installed	X	—
67	—	—	—	X	—	—	—
68	—	—	—	—	Installed	X	—
69	—	—	—	X	Installed		—
79	—	—	—	—	Installed	X	—
80	—	—	—	X	Installed	X	—
81	—	—	—	—	Installed	X	—
85	—	—	X	X	Installed	X	X
86	—	—	—	X	Installed	X	—
100	—	—	—	—	Installed	—	—
101	—	—	—	—	Installed	X	—
104	—	—	—	—	Installed	X	—
105	—	—	—	X	Installed	X	—

Abbreviation: X = indoor-air sampling event.
*Properties 20 and 43 had vapor mitigation systems installed in both 2008 and 2014.

Inhalation of contaminants in on-site occupational indoor air (past, present, and future)

The pathway for on-site occupational indoor air inhalation is complete. Vapor intrusion is documented with measured contaminants in indoor air in both buildings (Buildings A and B). These indoor air samples are supported by sub-slab soil gas and shallow groundwater samples showing significant contamination of these media (Figure 7).

Potential Exposure Pathways

Inhalation of contaminants in residential indoor air of homes not sampled (past, present, and future)

There are 157 occupied residential homes that are located within 100 feet of the TCE groundwater plumes, as delineated at 1 μg/L. As shown in Figure 6, 1 μg/L is the closest value to the ATSDR vapor intrusion CV (0.52 μg/L) for TCE in groundwater. Only 32 of these homes have vapor mitigation systems. Only limited sampling has been conducted after the vapor mitigation systems were installed to confirm that the systems are functioning to reduce indoor TCE levels.

Given the concentration of VOCs in groundwater and the shallow depth of groundwater (20–70 feet bgs), vapor intrusion is a potential exposure pathway for homes not tested or without vapor mitigation systems. Figure 6 shows that as of 2016, the highest concentrations of TCE in the shallow groundwater are located outside the northeastern corner of the Delfasco site. The highest concentrations of TCE in the deeper groundwater plume are located beneath the residential neighborhood. The shallow and deep groundwater plume gradients are moving northeast toward the crossroads of NE 31st Street and Rinehart Street. These results suggest that there is potential for vapor intrusion in a greater number of residential properties than previously estimated [EnSafe 2016, EA 2021].

Ingestion, inhalation, and skin absorption of contaminants in water from private residential wells (past, present, and future)

Exposure to water from private wells is a potential exposure pathway. Although the residential area is connected to the public water system, which is free from contamination, 16 private residential water wells have been identified within a 0.5-mile radius of the site (Figure 9) [Ensafe 2006]. The wells are generally completed in the alluvial layer, between 20 to 45 feet bgs, and three of the wells were located within the contaminated groundwater plume area. Of those three wells, two were dry at the time of sampling and one had a TCE concentration (19 µg/L) above EPA's MCL of 5 µg/L [Ensafe 2006]. Additionally, there were an additional 5 wells that were identified from the surveys but could not be physically located.

According to an Ensafe report, residential private wells are not presently being used for drinking water, although some might be used for irrigation [Ensafe 2006]. Additionally, people could have been exposed by incidentally swallowing water and through skin contact with water during recreational activities. However, this exposure would likely be minimal. Inhalation of chemicals through vaporization in outdoor air is not likely because these chemicals evaporate from water relatively quickly and readily disperse in outdoor air. Given the limited information available

from the private residential water wells, DSHS could not fully evaluate this exposure pathway.

In 2011, TCEQ's water well survey identified six unregistered wells within the groundwater plume area [EA 2011]. Of those six wells, one was used for irrigation and the other five were not in use. All homes were connected to city water for residential use.

Inhalation of contaminants in indoor air at James Fannin Middle School (future)

James Fannin Middle School is located northwest from the Delfasco Forge site across NE 28th Street. A portion of the school property is within 100 ft of the TCE groundwater contamination (Figure 7). The current direction of shallow groundwater movement is to the northeast, away from the school. In response to community concerns, EPA collected 13 passive soil gas samples at the James Fannin Middle School in August 2022 [Personal communication with Delfasco Forge site remedial project manager on passive soil gas sample results for James Fannin Middle School collected during August 2022; US Environmental Protection Agency, Region 6]. TCE was not detected in any of the passive soil gas samples. The direction of groundwater flow away from the school, the low levels of TCE in groundwater samples collected near the school, and the passive soil gas sample results suggest that this exposure pathway is not a current health concern. However, because of the location of the TCE in the shallow groundwater, the variability of vapor dispersion from groundwater over time and place, and uncertainty in using passive soil gas samples to quantify air concentrations, vapor intrusion might be a potential exposure pathway at the school in the future.

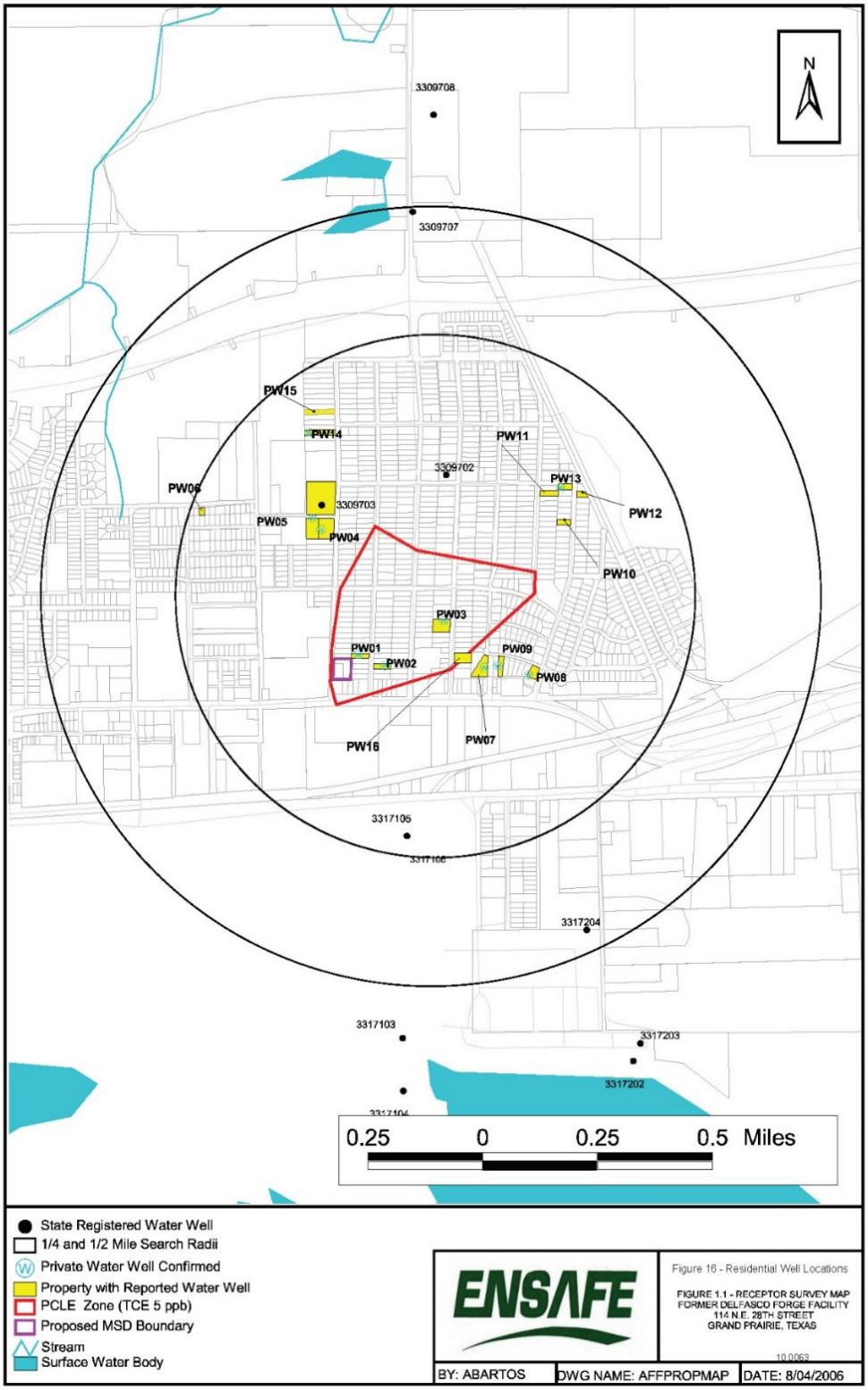

Figure 9. Locations of residential private water wells in the area of the Delfasco Forge Superfund site [Ensafe 2006]

Eliminated Exposure Pathways

Ingestion, inhalation, and skin absorption of contaminants in water from public water system (past, present and future)

Exposure to water from the City of Grand Prairie public water system was eliminated as an exposure pathway. Ensafe [2006] identified several state registered water wells (identified by 7-digit numbers in Figure 9: 3309703, 3309702, 3317105, 3317106, 3309707, and 3317204) within a 0.5-mile radius around the Delfasco Forge site that are owned by the City of Grand Prairie public water system. The public water system wells are completed in either the Woodbine and Trinity aquifers, which are located at depths of 2,006–2,163 feet bgs, and beneath the Eagle Ford Shale formation, which begins at 27–73 feet bgs.

TCE has not been detected in any of these public water system wells [TCEQ 2022]. Contamination from the shallow groundwater (located at 20–55 feet bgs) is not likely to have migrated to deeper aquifers because the Eagle Ford shale formation serves as a barrier for vertical migration between the contaminated shallow aquifer and the deeper aquifers [Ensafe 2006]. The Eagle Ford Shale is approximately 145 feet thick and relatively impermeable to water exchange between the two aquifers.

Table 2. Human exposure pathway evaluation – Delfasco Superfund site

Source	Medium	Point of exposure	Route of exposure	Potentially exposed population	Time frame & type of exposure pathway
Delfasco site groundwater contamination	Groundwater, soil vapor	Indoor air from vapor intrusion	Inhalation	Residents in homes with vapor mitigation systems	Past – potential Present – complete Future – potential
Delfasco site groundwater contamination	Groundwater, soil vapor	Indoor air from vapor intrusion	Inhalation	Residents in homes without vapor mitigation systems	Past – potential Present – complete Future – potential
Delfasco site groundwater contamination	Groundwater, soil vapor	Indoor air from vapor intrusion	Inhalation	Workers	Past – potential Present – complete Future – complete
Delfasco site groundwater contamination	Groundwater, soil vapor	Indoor air from vapor intrusion	Inhalation	Students and school staff	Past – eliminated Present – eliminated Future – potential
Delfasco site groundwater contamination	Groundwater	Private wells	Ingestion, inhalation, dermal	Residents with private water wells	Past – potential Present – potential Future – potential
Public water system	Residential drinking water	Residential tap	Ingestion, inhalation, dermal	Residents, workers	Past – eliminated Present – eliminated Future – eliminated

Screening Analysis

Following identification of completed and potential exposure pathways, DSHS conducted a screening analysis to identify potential contaminants of concern. The maximum concentration for each contaminant was compared to comparison values (CVs) published by ATSDR. CVs and other screening criteria are media-specific (e.g., air, soil, and water) levels below which no adverse health effects are expected to occur. It is important to note that if a chemical concentration exceeds a CV, it does not necessarily mean there is a health concern. It means the chemical- and site-specific exposure scenario warrants further public health evaluation based on site-specific exposure conditions. Chemicals without CVs were further analyzed.

Inhalation of contaminants in residential indoor air (past, present, and future)

Indoor air results in residential properties without vapor mitigation systems

Indoor air was sampled in 33 residential properties in the area adjacent to the facility. This includes samples collected from homes without vapor mitigation systems and from homes before vapor mitigation systems were installed. While samples were analyzed for VOCs, not all VOCs were analyzed nor detected in each residential property consistently over the sampling events. Additionally, a limited number of samples (one to five samples per home) were collected at each residential property from 2008 to 2016. During this period, at least one of three chemicals, including TCE, PCE and benzene, was detected above CVs in indoor air at 20 residential properties (Table 3). These chemicals were further evaluated.

Indoor air results in residential properties with vapor mitigation systems

A total of 32 homes had vapor mitigation systems installed either in 2008 or 2014 and 24 of these properties were sampled after vapor mitigation systems were installed (Table 1). All samples collected in indoor air after vapor mitigation system installation were evaluated. Although chemical levels (TCE, PCE and benzene) decreased in most homes after vapor mitigation systems were installed, chemicals were still detected in some homes above CVs (Table 4). These chemicals were further evaluated.

Table 3. Summary of chemicals detected above air comparison values in indoor air collected from residences without vapor mitigation systems or before vapor mitigation systems were installed from 2008 to 2016

Contaminant	# Detected out of # samples	Concentration range (median) µg/m³	Comparison value µg/m³: type	Residential properties with detections above comparison value (number of detections)
Benzene*	18 out of 18	0.32–4.19 (0.34)	0.13: ATSDR CREG	9(2), 16(1), 17(1), 18(1), 25(1), 36(1), 38(1), 39(1), 55(1), 57(1), 60(1), 67(1), 69(1), 80(1), 85(1), 86(1), 105(1)
Tetrachloroethylene (PCE)	8 out of 23	0.054–6.18 (0.77)	3.8: ATSDR CREG	16(1)
Trichloroethylene (TCE)*	22 out of 31	0.1–65.6 (2.45)	0.21: ATSDR CREG	16(3), 17(3), 18(1), 25(1), 36(1), 38(2), 39(1), 42(1), 43(1), 57(2), 60(1), 80(1), 85(1), 86(1), 105(1)

Abbreviations: µg/m³ = microgram per cubic meter of air; ATSDR = Agency for Toxic Substances Disease Registry; CREG = cancer risk evaluation guidelines.
*The detection limits for TCE and benzene are higher than the comparison values, so the number of exceedances were counted only in the samples that were detected.

Table 4. Summary of chemicals detected above air comparison values in indoor air collected from residences after vapor mitigation systems were installed from 2008 to 2016

Contaminant	# Detected out of # samples	Concentration range (median) µg/m³	Comparison value µg/m³: type	Residential properties with detections above comparison values (number of detections)
Benzene*	27 out of 27	0.29–6.15 (0.77)	0.13: ATSDR CREG	9(1), 16(1), 17(1), 18(1), 20(2), 25(1), 38(1), 43(3), 55(1), 56(1), 57(1), 58(1), 59(1), 60(1), 66(1), 68(1), 79(1), 80(1), 81(1), 85(1), 86(1), 101(1), 104(1), 105(1)
Tetrachloroethylene (PCE)	7 out of 33	0.34–6.18 (0.34)	3.8: ATSDR CREG	17(1)
Trichloroethylene (TCE)*	26 out of 36	0.27–153 (0.92)	0.21: ATSDR CREG	16(1), 17(2), 18(1), 20(4), 21(1), 38(1), 43(3), 55(1), 57(1), 58(1), 59(1), 60(2), 79(1), 80(1), 81(1), 85(1), 86(1), 104(1), 105(1)

Abbreviations: µg/m³ = microgram per cubic meter of air; ATSDR = Agency for Toxic Substances Disease Registry; CREG = cancer risk evaluation guidelines.
* The detection limits for TCE and benzene are higher than the screening criteria, so the number of exceedances were counted only in the samples that were detected.

Groundwater results collected from monitoring wells located in the residential area

Groundwater samples were collected from 31 monitoring wells in the residential area. The monitoring wells were installed to help determine the vertical and lateral extent of VOC contamination. The results also provide information on the potential for vapor intrusion in residential homes. VOCs detected in groundwater were compared to CVs derived to evaluate the potential vapor intrusion from groundwater. Several chemicals, including 1,1-dichloroethene, 1,1,2-trichloroethane, 1,2-dichloroethane, benzene, chloroform, PCE, TCE, and vinyl chloride, were detected above the CVs (Table 5). The results support the potential for vapor intrusion in the residential area.

Table 5. Summary of groundwater sampling results collected from monitoring wells detected above soil vapor intrusion groundwater comparison values, 2013 to 2016

Contaminant	# Detected out of total samples	Concentration range (median) µg/L	CV µg/L: CV type	# Detected samples exceeded CV
1,1-Dichloroethene	24 out of 80*	0.35–162 (1.0)	3.7: ATSDR chronic EMEG	18
1,1,2-Trichloroethane	8 out of 81	0.18–21.1 (0.42)	1.9: ATSDR CREG	4
1,2-Dichloroethane	12 out of 81	0.31–9.05 (0.46)	0.79: ATSDR CREG	8
Benzene	11 out of 81	0.37–12.4 (0.58)	0.57: ATSDR CREG	8
Chloroform	8 out of 31*	0.45–7.72 (1.0)	0.29: ATSDR CREG	8
Tetrachloroethylene (PCE)	23 out of 81	0.48–166 (0.95)	5.3: ATSDR CREG	12
Trichloroethylene (TCE)	59 out of 81	0.36–16,100 (5.74)	0.52: ATSDR CREG	56
Vinyl chloride	12 out of 81	0.36–518 (0.46)	0.097: ATSDR CREG	12

Abbreviations: µg/L= micrograms per liter; ATSDR = Agency for Toxic Substances Disease Registry; CREG = cancer risk evaluation guidelines; CV = comparison value; EMEG/MRL= environmental media evaluation guide/minimum risk level; EPA = Environmental Protection Agency.
*1,1-Dichloroethene was not sampled in well 5 on 2/21/2014 and chloroform was not analyzed during the 2013 or 2014 sampling events.

Inhalation of contaminants in on-site occupational indoor air (present and future)

A total of 18 groundwater samples were collected from on-site monitoring wells in May 2020 [EA 2021]. Several chemicals in groundwater were detected above CVs derived to evaluate the potential for vapor intrusion from groundwater. These included 1,1-dichloroethene, 1,2-dichlorethane, benzene, carbon tetrachloride, chloroform, PCE, TCE, and vinyl chloride (Table 6). Additionally, a total of 12 ASG and sub-slab samples were collected near on-site buildings in 2020 at a depth of 25 feet bgs. VOCs, including 1,2-dichloroethane, 1,4-dioxane, benzene, chloroform, tetrachloroethylene, TCE, and vinyl chloride, were detected above CVs derived to evaluate potential for vapor intrusion from sub-slab and near-source soil gas (Table 7). Past exposure to on-site chemical concentrations were not evaluated because no environmental samples were collected before 2020.

Due to the detections of VOCs in shallow groundwater, ASG, and sub-slab samples, in 2020, EPA also collected six indoor air samples from the on-site buildings [EA 2021]. VOCs (including 1,4-dioxane, benzene, carbon tetrachloride, and TCE) were detected above air CVs (Table 8). The results support the potential for vapor intrusion in on-site occupational buildings.

Table 6. Summary of chemicals detected above soil vapor intrusion groundwater comparison values in samples collected from on-site monitoring wells in 2020

Contaminant	# Detected out of total samples	Concentration range (median) µg/L	CV: CV type µg/L	# Detected samples exceeding the CV
1,1-Dichloroethene	8 out of 18	0.192–46.1 (0.19)	3.7: ATSDR chronic EMEG	7
1,2-Dichloroethane	6 out of 18	0.116–2.93 (0.43)	0.79: ATSDR CREG	5
Benzene	10 out of 18	0.176–5.21 (0.28)	0.57: ATSDR CREG	7
Carbon tetrachloride	1 out of 18	0.183	0.15: ATSDR CREG	18
Chloroform	12 out of 18	0.151–6.19 (0.37)	0.29: ATSDR CREG	11
Tetrachloroethylene (PCE)	13 out of 18	0.333–57.6 (10.6)	5.3: ATSDR CREG	9
Trichloroethylene (TCE)	17 out of 18	0.138–9,340 (810)	0.52: ATSDR CREG	16
Vinyl chloride	11 out of 18	0.248–104 (1.1)	0.097: ATSDR CREG	18

Abbreviations: µg/L= micrograms per liter; ATSDR = Agency for Toxic Substances Disease Registry; CV = comparison value; EMEG = environmental media evaluation guide; CREG = cancer risk evaluation guidelines.

Table 7. Summary of chemicals detected above soil vapor intrusion sub-slab and near-source soil gas comparison values in on-site active soil gas and sub-slab samples in 2020

Contaminant	# Detected out of total samples	Concentration range (median) µg/m³	CV:CV type µg/m³	# Detected samples exceeding CV
1,2-Dichloroethane	1 out of 12	1.4	1.3: ATSDR CREG	1
1,4-Dioxane	1 out of 12	16	6.7: ATSDR CREG	1
Benzene	4 out of 12	4–150 (24)	4.3: ATSDR CREG	3
Chloroform	3 out of 12	4.2–65 (9.4)	1.4: ATSDR CREG	3
Tetrachloroethylene (PCE)	12 out of 12	8.8–4,400 (375)	130: ATSDR CREG	11
Trichloroethylene (TCE)	12 out of 12	18–210,000 (21,500)	7: ATSDR CREG	12
Vinyl chloride	3 out of 12	0.59–290 (39)	3.7: ATSDR CREG	2

Abbreviations: µg/L= micrograms per liter; ATSDR = Agency for Toxic Substances Disease Registry; CREG = cancer risk evaluation guidelines; CV = comparison value.

Table 8. Summary of chemicals detected above air comparison values from on-site indoor air samples collected in 2020

Contaminant	# Detected out of total samples	Concentration range (median) µg/m³	CV:CV type µg/m³	# Detected samples exceeding CV
1,4-Dioxane	1 out of 6	0.62	0.2: ATSDR CREG	1
Benzene	4 out of 6	0.78–12 (6.3)	0.13: ATSDR CREG	4
Carbon tetrachloride	1 out of 6	0.44	0.17: ATSDR CREG	1
Trichloroethylene (TCE)	4 out of 6	27–48 (37)	0.21: ATSDR CREG	4

Abbreviations: µg/m³= micrograms per cubic meter; ATSDR = Agency for Toxic Substances Disease Registry; CREG=cancer risk evaluation guidelines; CV = comparison value.

Health Effects Evaluations

The potential contaminants of concern exceeding screening values were further evaluated by calculating exposure point concentrations (EPCs) [ATSDR 2019c]. The EPC is a representative chemical concentration that is calculated for an environmental medium, such as indoor air, soil, or water. Because the number of indoor air samples collected in each home during the vapor intrusion investigation varied from one to less than eight, DSHS could not accurately determine a 95% upper confidence limit of the arithmetic mean as the EPC. Therefore, the EPC for potential contaminants of concern detected at the residential properties and on-site commercial buildings was determined based on the maximum concentration. For residential properties this included either the maximum concentration detected before or after vapor mitigation systems were installed.

DSHS estimated noncancer and cancer risks using the EPC. No site-specific exposure information was available, so DSHS calculated the health effects evaluation results using health protective exposure assumptions for two exposure scenarios. Those were the typical or central tendency exposure (CTE) and the high or reasonable maximum exposure (RME), as recommended by ATSDR (Appendix D). The RME refers to persons who are at the upper end of the exposure distribution (about the 95%). The CTE refers to persons who have an average or typical exposure distribution.

Noncancer Health Effects

To evaluate noncancer health effects, DSHS compared EPCs to appropriate health guidelines, such as ATSDR'S chronic minimal risk level (MRL) and EPA's reference concentration (RfC). A health-based guideline is an estimate of daily exposure dose to a substance over a specified duration that is unlikely to cause harmful, noncancer health effects in humans. If an estimated exposure dose is lower than the health-based guideline, adverse noncancer health effects are not expected to occur. If an estimated dose is higher than the health-based guideline, it does not necessarily mean it will harm people's health. It does, however, mean that DSHS must conduct an in-depth evaluation to determine if adverse health effects are possible and if the exposure poses a health hazard. This is done by comparing the dose to known noncancer health effect levels reported in the scientific literature.

DSHS calculated hazard quotients (HQs) to compare estimated exposure doses to health guidelines. The HQs were calculated by dividing the estimated exposure doses by the health guideline. If the HQ is less than 1, then adverse health effects are not likely because the estimated dose in people is below the health guideline. If the HQ is greater than 1, DSHS further evaluated the margin of exposure (MOE). The MOE is a measure of how close the estimated dose is to harmful levels. The

smaller the MOE, the closer the exposure is to effect levels. When the MOE is less than 1, then exposures exceed effect levels.

Cancer Health Effects

To estimate cancer risk for cancer-causing contaminants, the EPC was multiplied by the inhalation unit risk factor (IUR). The cancer risk is an excess lifetime cancer risk, which estimates the proportion of a population that might be affected by a carcinogen during their lifetime (365 days/year for 78 years) (Appendix D and F). An excess lifetime cancer risk represents the additional risk above the existing background cancer risk. For example, an estimated cancer risk of two per million (or 2E-6) potentially represents two excess cancer cases in a population of 1 million over their lifetime. In the United States, the background cancer risk (or the probability of developing cancer at some point during a person's lifetime) is about two in five for men and women [ACS 2020]. Note, the cancer risk estimates in this document are not a measure of the actual cancer cases in the neighborhood northeast of the site; rather, they are a tool used by DSHS for making public health recommendations in this document.

Inhalation of contaminants in residential indoor air

DSHS estimated noncancer and cancer health risks for TCE, PCE, and benzene in residential indoor air using indoor air sampling results collected from multiple sampling events sporadically between 2008 and 2016. This included samples collected before and after vapor mitigation systems were installed. There were 31 residential properties with at least one chemical detected above its CV. (See Appendix B, Tables 11–13 for detailed Health Effects Evaluation Results.)

Trichloroethylene (TCE)

TCE is mainly used as a degreaser for metal parts, as a solvent, and to make other chemicals [ATSDR 2019a]. It is a colorless liquid that evaporates easily and can be found in some household products, including paint removers, adhesives, and spot removers. People are generally exposed to TCE from breathing air or drinking water containing TCE. About half of the TCE breathed in will get into the bloodstream and organs. Once in the bloodstream, the liver will convert much of the TCE into other chemicals. Most of these chemicals will leave the body in the urine within a day. Some of these chemicals can be stored in body fat for a brief period and build up if exposure continues [ATSDR 2019a].

TCE was detected above the CV in 24 residential properties, either without vapor mitigation systems or after vapor mitigation systems were installed, with

concentrations of all samples ranging from 0.1 μg/m^3 to 153 μg/m^3 (Tables 11 and 12).

Noncancer effects

DSHS used ATSDR's chronic inhalation MRL for TCE (2.1 μg/m^3). The MRL is based on the results of two critical studies. One study reported immune system effects (decreased thymus weight) in female mice drinking water containing 1.4 milligrams per liter (mg/L) TCE (EPA estimated dose in mice of 0.35 mg/kg/day TCE) over 9 weeks [Keil et al. 2009]. In the other study, Johnson et al. [2003] reported developmental effects (fetal heart malformations) in pregnant rats drinking water containing 0.25 mg/L TCE (estimated dose in rats of 0.048 mg/kg/day) during the gestation period (22 days). EPA derived TCE concentrations that might be expected to have the same effects in humans. The 99th percentile of those human equivalent concentrations are 21 μg/m^3 TCE for short-term exposures potentially associated with cardiac malformations and 190 μg/m^3 TCE for chronic exposures potentially associated with immunological effects [ATSDR 2019a].

TCE concentrations in residential indoor air exceeded the MRL (HQs greater than 1) at 17 properties (Tables 11 and 12). For nine of these properties (17, 18, 20, 21, 38, 39, 43, 60, and 81), the TCE concentration was greater than the effect level for fetal heart malformations (21 μg/m^3) (MOE less than 1). For the remaining eight properties (16, 36, 42, 57, 58, 59, 85, and 86), TCE concentrations approached the fetal heart malformations effect level (21 μg/m^3). The difference between the concentration and the effect level was low (MOEs ranged from 1 to 8) and harmful developmental effects might occur. Pregnant women who breathe TCE in indoor air at levels that approach or exceed harmful levels are at increased risk of having their fetus develop with a heart defect. This health effect could occur during the 3-week period early in the first trimester when the fetal heart forms and begins to function.

TCE concentrations were below the effect level for immune system effects (190 μg/m^3) in all properties (Tables 11 and 12, Appendix B). However, in 10 properties (17, 18, 20, 21, 38, 39, 42, 43, 60, and 81) the difference between the concentration and the effect level is low (MOEs ranged from 1 to 9); thus, the measured concentrations were approaching the effect level for harmful immune system effects. In these homes, harmful effects to the immune system (such as decreased thymus weight, which could increase the risk for autoimmune diseases) might occur in children and adults after long-term exposure to TCE.

Cancer effects

EPA classifies TCE as carcinogenic to humans by all routes of exposure [EPA 2012]. The National Toxicology Program (NTP) has determined that TCE is reasonably anticipated to be a human carcinogen based on animal studies and limited human studies [NTP 2021]. Animal studies showed increased numbers of liver, kidney, testicular, and lung tumors following exposure to TCE [EPA 2011, Charbotel et al. 2006, Raaschou-Nielsen et al. 2003].

In 2012, EPA published a revised inhalation unit risk of 4.1E-6 $(ug/m^3)^{-1}$ reflecting total incidence of kidney, non-Hodgkin's lymphoma, and liver cancers [USEAP 2012]. EPA concluded, by a weight of evidence evaluation, that TCE is carcinogenic by a mutagenic mode of action for induction of kidney tumors [EPA 2012]. As a result, increased early life susceptibility is assumed for kidney cancer, and age-dependent adjustment factors (ADAFs) are used for the kidney cancer component of the total cancer risk when estimating age-specific cancer risks. ADAFs are factors by which cancer risk is multiplied to account for increased susceptibility to mutagenic compounds when exposure occurs early in life. The standard ADAFs are 10 (for children younger than 2 years), 3 (for children ages 2–16 years), and 1 (for children older than 16 years and adults).

DSHS calculated RME cancer risk for inhalation of TCE at 24 residential properties using EPA's IUR of 4.1E-6 $(\mu g/m^3)^{-1}$ [EPA 2019] (Tables 11 and 12). DSHS found the cancer risk for children and adults to be as follows:

- Between 1 and 3 cases in 10,000 people (1E-4 and 3E-4) at properties 17, 20, and 43
- Between 1 and 8 cases in 100,000 people (1E-5 and 8E-5) for properties 18, 21, 38, 39, 42, 57, 58, 60, 81, and 85
- Between 2 and 9 cases in 1,000,000 people (1E-6 and 9E-6) for properties 16, 18, 25, 36, 38, 57, 59, 79, 80, 86, and 105

On the basis of this information, cancer risk at properties 17, 20, and 43 might be a health concern for children and adults after long-term exposure. Cancer risks at the other properties are not a health concern. It is important to remember that these cancer risks are based on using the maximum TCE air concentration from just a few indoor air samples. In estimating the cancer risk, DSHS assumed that people were exposed for decades to the maximum concentration; therefore, there is some uncertainty in these estimates.

TCE indoor air sampling results after the installation of vapor mitigation systems

Indoor air samples were collected in October 2014, in 24 residential properties where vapor mitigations systems were installed in July through August 2014 (Table 1). TCE was detected at levels that exceeded the CV at 19 properties (16, 17, 18, 20, 21, 38, 43, 55, 57, 58, 59, 60, 79, 80, 81, 85, 86, 104, and 105) in 2014 (Tables 4 and 12, Appendix B). The maximum TCE concentrations decreased from levels collected before the installation of vapor mitigation systems at eight properties (16, 25, 43, 57, 60, 80, 86, and 105) and increased at four properties (17, 18, 38, and 85) (Table 13, Appendix B). The sampling results might be a result of the uncertainty and variability inherent in indoor air sampling. However, not enough indoor air samples were taken in individual homes to quantify the variability of indoor air TCE concentrations or to determine the effectiveness of the vapor mitigation systems.

Six of the properties that had vapor mitigation systems installed in 2014 were sampled again in May 2016 (Tables 1 and 13). From the 2016 sampling results, TCE levels were either not detected or were below the MRL (HQs less than 1) at five properties (17, 18, 43, 60, and 85). Adverse noncancer and cancer health effects are not likely from inhaling TCE in indoor air at these properties.

Although TCE levels at property 20 decreased from 122 $\mu g/m^3$ to 53.1 $\mu g/m^3$ over the 2-year period (2014–2016), TCE in 2016 was detected above the MRL (HQ above 1). At this property, EPA noted a 6-inch hole in the foundation during sample collection in 2016 and suggested that this was the likely source of the elevated TCE concentration [EPA 2016]. EPA also noted that the property owner created the hole in the foundation to install a floor safe. To DSHS's knowledge, the hole has not been repaired. Therefore, TCE levels at property 20 might still be a health concern for people living at the property. EPA is communicating with the residents of property 20 about ongoing mitigation efforts.

Tetrachloroethylene (PCE)

Tetrachloroethylene (PCE) is widely used for dry cleaning of fabrics and for metal degreasing operations. It is also used as a building block for making other chemicals and is used in some consumer products. PCE enters the environment by evaporating into the air during use. It can also get into the water supply and soil during disposal of sewage sludge and factory waste and when leaking from underground storage tanks. It can stay in air for several months before it is broken down into other chemicals or is brought back down to soil and water from rain [ATSDR 2019b]. Consumer products that might contain PCE include water repellants, fabric softeners, spot removers, adhesives, and wood cleaners.

Noncancer effects

DSHS used the ATSDR chronic inhalation MRL of 41 $\mu g/m^3$. That MRL is based on an occupational epidemiology study that found that dry cleaning workers experienced decreased color vision with the lowest observed adverse effect level of 11,544 $\mu g/m^3$ [ATSDR 2019b, Gobba et al 1994].

PCE was detected above the CV in one residential property (property 16), at a concentration of 4.6 $\mu g/m^3$. The measured concentration of PCE in residential indoor air was lower than the chronic MRL (HQs less than 1). Therefore, adverse noncancer health effects are not likely from inhaling PCE in indoor air (Tables 11 and 12 Appendix B).

Cancer effects

EPA considers PCE likely to be carcinogenic to humans by all routes of exposure [EPA 2012]. The NTP classifies PCE as reasonably anticipated to be a human carcinogen based on sufficient evidence in experimental animals [NTP 2021]. Human studies suggest that exposure to PCE might increase the risks for developing bladder cancer, multiple myeloma, or non-Hodgkins lymphoma. Animal studies have shown that PCE can cause liver, kidney, and blood system cancers.

DSHS used EPA's IUR of 2.6E-7 $(\mu g/m^3)^{-1}$. It is based on an animal study that showed hepatocellular adenomas or carcinomas development in mice exposed to PCE [EPA 2012, Chiu and Ginsberg 2011].

DSHS calculated cancer risk for inhalation of PCE at property 16 (Tables 11 and 12, Appendix B). DSHS found the cancer risk for children and adults to be less than 1 case in 1,000,000 people (1E-6). These cancer risks are not a health concern.

PCE sampling results after vapor mitigation system installation

PCE was detected in indoor air samples collected after vapor mitigation systems were installed at levels that exceeded the CV at one property (property 17) (Table 4 and Table 12. Appendix B). However, the detected PCE concentration was below the MRL (HQs less than 1) and adverse noncancer health effects are not likely.

DSHS found the cancer risk for children and adults to be less than 1 case in 1,000,000 people (1E-6) for children and for adults. This cancer risk is not a health concern.

Benzene

Benzene is a colorless, sweet-smelling liquid that evaporates quickly and dissolves slightly into water. It is highly flammable and can be formed naturally and by

human industry. Industry is the main source of benzene in the environment. Benzene is produced from petroleum and is used in a variety of industries, such as chemical manufacturing, and in manufacturing rubbers, lubricants, dyes, detergents, drugs, and pesticides. Benzene usually breaks down within a few days in the air but breaks down much more slowly when in the water or soil. Benzene can enter a human body through the lungs, gastrointestinal track, or across the skin.

Noncancer effects

DSHS used the ATSDR chronic inhalation MRL of 9.6 $\mu g/m^3$. The chronic MRL for benzene is based on study of workers exposed to benzene in two shoe manufacturing facilities in China [Lan et al. 2004]. The critical effect in developing the MRL was a decrease in B lymphocytes in the blood of these workers. Using benchmark dose modeling and adjusting the worker exposure to a continuous exposure, ATSDR estimated that the lowest level for blood-related effects to be 96 $\mu g/m^3$. (Appendix G) [ATSDR 2007].

Benzene was detected at levels exceeding the CV at 24 properties (properties 9, 16, 17, 18, 20, 25, 36, 38, 39, 43, 55, 57, 58, 59, 60, 66, 79, 80, 81, 85, 86, 101, 104, and 105), with concentrations of all samples ranging from 0.32 $\mu g/m^3$ to 6.2 $\mu g/m^3$. The concentrations in residential indoor air did not exceed the chronic MRL (HQs less than 1) in any sample. Therefore, adverse noncancer health effects are not likely from inhaling benzene in indoor air (Tables 11 and 12, Appendix B).

Cancer effects

Long term exposure to benzene in air at high enough concentrations can cause acute myelogenous leukemia (a type of cancer that affects the blood-forming organs). EPA and the NTP have classified benzene as a known human carcinogen based on evidence from human and animal studies [ATSDR 2007, NTP 2021, EPA 2007].

DSHS used EPA's IUR 7.8E-6 $(\mu g/m^3)^{-1}$ to calculate cancer risk for benzene. That IUR is based on the incidence of leukemia in several human studies [EPA 2000]. DSHS calculated cancer risk for inhalation of benzene at 24 residential properties (Tables 11 and 12, Appendix B). DSHS found the cancer risk from benzene for adults and children to be between 1 and 9 cases in 1,000,000 people (1E-6 and 9E-6). This applies to properties 9, 16, 17, 20, 25, 36, 38, 39, 43, 55, 57, 58, 59, 66, 67, 68, 69, 79, 80, 81, 85, 96, 101, and 105 for either children or adults. These cancer risks are not a health concern.

DSHS found the cancer risk from benzene for adults and children to be between 1 and 2 cases in 100,000 people (1E-5 and 2E-5) for properties 58 and 60. These cancer risks are not a health concern.

Benzene sampling results after vapor mitigation system installation

Benzene was detected in samples collected after vapor mitigation systems were installed at levels that exceeded the CV at 24 properties (9, 16, 17, 18, 20, 25, 38, 43, 55, 56, 57, 58, 59, 60, 66, 68, 79, 80, 81, 85, 86, 101, 104, and 105) (Table 13, Appendix B). However, all concentrations were below the MRL (HQs less than 1) and adverse noncancer health effects are not likely from inhaling benzene in indoor air in homes with vapor mitigation systems.

DSHS found the cancer risk from benzene in homes for children and adults to be 9 cases in 1,000,000 people (9E-6) or less at properties 9, 16, 17, 18, 20, 25, 38, 43, 55, 56, 57, 59, 66, 68, 79, 80, 81, 85, 86, 101, 104, and 105. These cancer risks are not a health concern.

DSHS found the cancer risk from benzene for adults and children to be between 1 and 2 cases in 100,000 people (1E-5 and 2E-5) for properties 58 and 60. These cancer risks are not a health concern.

Inhalation of contaminants in on-site occupational indoor air

DSHS estimated noncancer and cancer health risks for TCE, benzene, 1,4-dioxane, and carbon tetrachloride in indoor air for on-site workers (Table 9). DSHS used the default RME full-time worker exposure parameters in ATSDR's Public Health Assessment Site Tool (PHAST) to calculate an adjusted EPC. The parameters used were 8.5 hours of exposure daily, 5 days a week, 50 weeks a year, for 20 years (Appendix F).

Trichloroethylene (TCE)

Noncancer

The maximum TCE concentration measured in on-site indoor air (48 $\mu g/m^3$) was time adjusted to 12 $\mu g/m^3$ (adjusted EPC) to represent a 24-hour per day continuous exposure. This adjustment allows the adjusted air concentration to be compared to ATSDR's chronic inhalation MRL, which is based on a 24-hour per day continuous exposure. The adjusted EPC (12 $\mu g/m^3$) for workers was above the MRL of 2.1 $\mu g/m^3$ (HQ greater than 1) (Table 9). The adjusted EPC is approaching the effect level for fetal heart malformations of 21 $\mu g/m^3$ (MOE of 2), suggesting harmful developmental effects to the fetus are possible for pregnant women exposed during their first trimester of pregnancy. However, the adjusted EPC is

below the effect level for immune system effects (190 μg/m^3) (MOE of 16), and harmful immune system effects are not expected to occur in adult workers. Therefore, workers who are pregnant while working in this building early in their pregnancy might be at increased risk for fetal heart effects in their children from short-term exposure to TCE.

Cancer

DSHS used the time-adjusted EPC on the combined risks for non-Hodgkin's lymphoma, liver cancer, and kidney cancer to calculate cancer risk for inhalation of TCE in on-site indoor air. DSHS found the cancer risk for workers to be 1 case per 100,000 people (1E-5). This cancer risk is not a health concern.

Benzene

Noncancer

The time-adjusted EPC for benzene in indoor air (3.0 μg/m^3) was below the chronic inhalation MRL (9.6 μg/m^3). Harmful noncancer health effects are not likely to occur from inhalation of benzene.

Cancer

DSHS used the time-adjusted EPC to calculate cancer risk for inhalation of benzene in on-site indoor air. DSHS found the cancer risk for workers to be 6 cases in 1,000,000 people (1E-6). This cancer risk is not a health concern.

1,4-Dioxane

1,4-Dioxane is a clear liquid that easily dissolves in water. It is used primarily as a solvent in the manufacture of chemicals and as a laboratory reagent. 1,4-Dioxane is a trace contaminant of some chemicals used in cosmetics, detergents, and shampoos. However, manufacturers now reduce 1,4-dioxane to low levels before using these chemicals in household products.

Noncancer

DSHS used the ATSDR chronic inhalation MRL (110 μg/m^3). That MRL is based on a 2-year inhalation study of chronic toxicity in male rats [Kasai et al. 2009]. The most sensitive endpoint was loss of cells of the olfactory epithelium of the nasal cavity. The lowest observed adverse effect level of 180,000 μg/m^3 (Appendix G) was adjusted to reflect continuous duration and extrapolated from animals to humans.

To account for these factors, a total uncertainty factor of 300 was used to calculate the MRL [ATSDR 2012].

The time-adjusted EPC for 1,4-dioxane in on-site indoor air (0.16 $\mu g/m^3$) was below the chronic MRL (110 $\mu g/m^3$). Therefore, adverse noncancer health effects are not likely.

Cancer

EPA has classified 1,4-dioxane as likely to be carcinogenic to humans, based on a finding of sufficient evidence of carcinogenicity in animals but inadequate evidence of carcinogenicity in humans [EPA 2013]. The NTP classified 1,4-dioxane as reasonably anticipated to be a human carcinogen, based on sufficient evidence of carcinogenicity from studies in experimental animals [NTP 2021].

DSHS used EPA's IUR of 5.0E-6 $(\mu g/m^3)^{-1}$ [EPA 2013]. EPA calculated IUR based on the combined tumor incidence in male rats exposed to 1,4-dioxane by inhalation for 2 years [Kasai et al. 2009]. The types of tumors observed in the study and used as the basis of the IUR calculation are nasal, liver, kidney, peritoneal, and mammary gland tumors. The rat multi-tumor benchmark concentration limit ($BMCL_{10}$) of 111,000 $\mu g/m^3$ (Appendix G) was the point of departure used to calculate the human equivalent concentration (BMC HEC = 19,500 $\mu g/m^3$) and is the basis of the IUR [EPA 2013].

DSHS used the time-adjusted EPC to calculate cancer risk for inhalation of 1,4-dioxane in on-site indoor air (0.15 $\mu g/m^3$). DSHS found the cancer risk for workers to be 2 cases in 10,000,000 people (2E-7). This cancer risk is not a health concern.

Carbon tetrachloride

Carbon tetrachloride is a clear liquid that can quickly vaporize into the air. It is a manufactured chemical that was used to make refrigeration fluid and propellants for aerosol cans. It was also used as a pesticide, cleaning fluid, degreaser, in fire extinguishing chemical, and spot remover. Carbon tetrachloride was banned in the 1970s for most applications because of its harmful effects. [ATSDR 2005a].

Noncancer

DSHS used the EPA reference concentration (RfC) of 100 $\mu g/m^3$. That RfC is based on a study by the Japan Bioassay Research Center in which rats were exposed to carbon tetrachloride vapor for 6 hours a day, 5 days a week, for 14 weeks [Nagano et al. 2007, JBRC 1998]. The study identified fatty changes in the liver, an indicator for cellular damage of the liver, as the most sensitive endpoint. EPA calculated a

benchmark concentration (using the 95% lower confidence limit at the 10% response level) of 14,300 $\mu g/m^3$ as the point of departure and applied an uncertainty factor of 100 to calculate the RfC [EPA 2010].

The time-adjusted EPC for carbon tetrachloride in on-site indoor air (0.11 $\mu g/m^3$) was less than the EPA RfC (100 $\mu g/m^3$). Therefore, adverse noncancer health effects are not likely.

Cancer

EPA has classified carbon tetrachloride as likely to be carcinogenic to human [EPA 2010]. The NTP has classified carbon tetrachloride to be reasonably anticipated to be a human carcinogen [NTP 2021].

DSHS used EPA's IUR of 6.0E-6 $(\mu g/m^3)^{-1}$. That IUR is based on a study from Nagano et al. [1998] in which male and female rats were exposed to carbon tetrachloride vapors for 104 weeks. EPA evaluated concentration response relationships between carbon tetrachloride concentrations and frequency of liver tumors and pheochromocytomas (a hormone-secreting tumor that can occur in the glands on top of the kidneys) to calculate the human equivalent concentration and calculate the IUR [EPA 2010].

DSHS used the time-adjusted EPC (0.11 $\mu g/m^3$) to calculate the cancer risk for inhalation of carbon tetrachloride in on-site indoor air. DSHS found the cancer risk for workers to be two cases in 10,000,000 people (2E-7). This cancer risk is not a health concern.

Table 9. Noncancer and cancer risk estimates for workers exposure to chemicals detected in indoor air from on-site buildings*

Contaminant	Time-adjusted EPC ($\mu g/m^3$)	Health guidelines ($\mu g/m^3$)	RME noncancer HQ	Cancer inhalation unit risk $(\mu g/m^3)^{-1}$	RME cancer risk
1,4-Dioxane	0.16	110 (ATSDR chronic inhalation MRL)	<1	5.0E-6	2E-7
Benzene	3.0	9.6 (ATSDR chronic inhalation MRL)	<1	7.8E-6	6E-6
Carbon tetrachloride	0.11	100 (EPA RfC)	<1	6.0E-6	2E-7
Trichloroethylene (TCE)	12	2.1 (ATSDR chronic inhalation MRL)	6‡	2.1E-6 (NHL), 1.0E-6 (liver), 1.0E-6 (kidney)	1E-5‡

Abbreviations: $\mu g/m^3$= micrograms per cubic meter of air; ATSDR = Agency for Toxic Substance and Disease Registry; EPA = Environmental Protection Agency; EPC = exposure point concentration; HQ = hazard quotient; RfC = reference concentration; RME = reasonable maximum exposure; TCE = trichloroethylene; MRL = minimal risk level; NHL = Non-Hodgkin lymphoma.
*The calculations in this table were generated using ATSDR's Public Health Assessment Site Tool (PHAST version 2.1.1.0).
†Worker exposure for 20 years, 5 days per week, 50 weeks per year.
‡Indicates a value where HQ is greater than 1 or cancer risk is greater than 1E-6.

Comparison of VOCs Detected in On-site and Off-site Indoor Air to Background Indoor Air Concentrations

Indoor air typically contains VOCs from a variety of sources, including consumer products, building materials, and outdoor air. Indoor air concentrations resulting from these sources are referred to as "background" when assessing the potential for intrusion of subsurface contaminant vapors into the indoor air of overlying buildings. Any indoor air sample collected for site-specific assessment of soil gas vapor intrusion is likely to detect chemicals from these other sources. Table 10 shows a comparison of indoor air concentrations detected in the workplace buildings and residential homes to background levels (50th and the 95th percentile

concentrations) measured in North American residences between 1990 and 2005 [EPA 2011]. TCE detected in indoor air from some residential properties and the workplace buildings was above the 95th percentile concentrations. Other VOCs were below the 95th percentile concentrations.

Table 10. Comparison of VOCs detected in on-site and residential indoor air to VOCs measured in North American residences during 1990–2005 [EPA 2011]

Chemical	Indoor air levels in residential properties near the Delfasco site (µg/m^3)	Indoor air levels in the former Delfasco on-site buildings (µg/m^3)	Indoor air levels in North American residences – 50th percentile (µg/m^3)	Indoor air levels in North American residences – 95th percentile (µg/m^3)
1,4-Dioxane	ND	0.62	NA	NA
Benzene	0.32–6.15	0.78–12	RL* to 4.7	9.9–29
Carbon tetrachloride	ND	0.44	<RL* to 0.68	<RL* to 1.1
Trichloroethylene (TCE)	0.1–153	27–48	RL* to 1.1	0.56–3.3
Tetrachloroethylene (PCE)	0.054– 6.18	ND	RL* to 4.1	4.1–9.5

Abbreviations: µg/m^3 = micrograms per cubic meter; NA = not available; ND = not detected; VOCs = volatile organic compounds. RL = reporting limit.
*Reporting limits represent the lowest concentration that the laboratory will report for a compound without data qualifiers. In this report, the term "reporting limits" is used synonymously with the term "detection limits" because the different studies compiled used varying conventions for these two terms.

Children's Health Considerations

In communities faced with air, water, or soil contamination, children could be at greater risk than adults from certain kinds of exposure to hazardous substances. A child's lower body weight and higher breathing rate per body mass could result in a greater exposure to hazardous substances in air compared to adults. Sufficient exposure levels during critical growth stages can result in permanent damage to the developing body systems of children. Children are dependent on adults for access to housing and medical care, and for risk identification and exposure prevention. Consequently, adults need as much information as possible to make informed decisions regarding their children's health. DSHS took this into account, and specifically evaluated exposures among young children, breastfeeding women, and pregnant women.

Community Health Concerns

Community members have expressed concern that there are unregistered private residential water wells that are unreported to the Texas Water Development Board within the contaminated area that are being used by residents. Any residents with concerns about their private water wells should contact EPA for help in evaluating the well.

Community members expressed concerns about the nearness of a school to the site. James Fannin Middle School is located northwest from the Delfasco Forge site across NE 28th street. The direction of groundwater flow and the results of the passive soil gas samples suggest that this exposure pathway is not a current health concern. However, vapor intrusion might be a future potential exposure pathway because of several factors:

- The school is near the TCE groundwater plume
- Vapor dispersion from groundwater can vary over time and location
- Results from using passive soil gas samples to quantify air concentrations are not always reliable

Community members expressed concern about health effects to persons who worked at the Delfasco Forge site during the years it operated (1981 through 1998). Any former worker at the Delfasco Forge site who has concerns about possible health effects from chemical exposure while working should consult their doctor.

Limitations

- A small number of samples were collected in indoor air at 33 residential properties (one to five samples per home) and the two on-site buildings (two to three samples per building). Sampling results might not adequately represent exposure pathways because seasonal variability or trends over time were not considered.

- The concentrations of contaminants entering the indoor air from the subsurface are dependent on site and building-specific factors. Those factors include building construction, soil type and moisture content, air conditioning and heating settings in the building, ventilation in buildings, and number and spacing of cracks and holes in the foundation. Estimating indoor air concentrations that people breathe from vapor intrusion also has inherent uncertainty because of the dynamic nature of the pathway in different conditions. Estimates must account for varying air exchange for a range of climatic conditions. Indoor air samples collected in cold weather and hot weather are needed to fully characterize health risks from vapor intrusion. In cold weather, windows and doors are most likely to remain closed, allowing soil gas vapors to accumulate indoors.

- Indoor air TCE concentrations can be affected by other sources within the home, such as cleaning products, adhesives, and paint removers. Indoor air results might be unclear if residents did not remove other potential sources before sampling.

- Approximately 124 occupied homes are located above shallow contaminated groundwater and have not been sampled for chemicals in indoor air.

- A limited number of samples (one to three samples per home) have been collected after vapor mitigation systems were installed. In some homes, no samples have been collected after vapor mitigation system were installed. Therefore, it is difficult to determine the effectiveness of vapor mitigation systems in reducing chemicals in indoor air.

Conclusions

From available information, DSHS reached seven conclusions in this health consultation.

Conclusion 1

For some residential properties sampled during 2008–2016, people's health might have been harmed by breathing trichloroethylene (TCE) that has evaporated into their indoor air from the underlying contaminated groundwater.

Basis for Conclusion

DSHS evaluated indoor air concentrations collected infrequently during 2008–2016 from residential properties located above the contaminated groundwater. All the indoor air samples were taken in May, except for one round of sampling in October 2014. On the basis of the maximum level of TCE detected in indoor air either before or after the installation of vapor mitigation systems, DSHS identified the following health risks:

- Exposure to TCE at 17 properties (16, 17, 18, 20, 21, 36, 38, 39, 42, 43, 57, 58, 59, 60, 81, 85, and 86) during the 3-week or longer period early in the first trimester of pregnancy could cause fetal heart malformations in children.
- Exposure to TCE at 10 properties (17, 18, 20, 21, 38, 39, 42, 43, 60, and 81) could cause harmful immune system effects (such as decreased thymus weight, which could increase the risk for autoimmune diseases) in children and adults.
- The estimated cancer risks from long-term exposure (several decades) at properties 17, 20, and 43 is a health concern for children and adults. DSHS estimated the lifetime cancer risk for children and adults to be greater than 1 in 10,000 people (1E-4).

There is uncertainty with the risk estimates because they are based on the maximum concentration detected in indoor air from limited (1 to 3) sampling events. Sampling events are when environmental samples are collected over a specific period of time.

Additionally, TCE levels detected in indoor air from some residential properties are above the 95th percentile concentrations measured in North American residences, which suggests vapor intrusion might be occurring in some homes [EPA 2011].

Conclusion 2

Worker's health might be harmed by breathing TCE that has evaporated into indoor air of the former Delfasco Forge workplace buildings from the underlying contaminated groundwater.

Basis for Conclusion

DSHS evaluated indoor air concentrations collected from the on-site commercial buildings in May and September of 2020 for occupational exposure to TCE. TCE was detected in indoor air at a level approaching the effect level for fetal heart malformations in the developing fetus of pregnant women. Therefore, workers who are pregnant while working in this building during the first 3 weeks of pregnancy might be at increased risk for fetal heart effects in their children from short-term exposure to TCE. However, there is uncertainty with the risk estimate because it is based on the maximum level detected in indoor air from one sampling event.

Conclusion 3

Future exposures to TCE and other volatile contaminants (those that evaporate easily) in indoor air at residential properties 17, 18, 43, 60, and 85 are not expected if vapor mitigation systems are operating as intended. TCE might still harm people's health at property 20, even though it has a vapor mitigation system.

DSHS cannot determine whether future exposure to TCE is harmful at the other properties with vapor mitigation systems because too few indoor air samples have been collected since installation of the systems.

Basis for Conclusion

Although vapor mitigation systems were installed at 32 properties, inhalation exposures to TCE might occur if these systems are not operating properly. DSHS compared maximum indoor air levels of TCE collected before and after the installation of vapor mitigation systems at six properties (17, 18, 20, 43, 60, and 85). In the most recent sampling results (2016), TCE levels were either not detected or were below the comparison value at five properties (17, 18, 43, 60, and 85). Adverse health effects from TCE in indoor air are not expected at these properties.

However, at property 20, TCE was above levels that could cause noncancer health effects (including fetal heart malformations in children whose mothers were exposed early in their pregnancy and immune system effects) for people living at the property. The elevated level of TCE was attributed to a hole in the property's

foundation and to DSHS's knowledge that the hole has not been repaired. EPA is communicating with the residents of the property about ongoing mitigation efforts.

At the other 26 properties with vapor mitigation systems, indoor air samples were either not collected or collected once 2 months after systems were installed in 2014. TCE levels in indoor air were above comparison values at some homes. Additional sampling is needed to fully assess the effectiveness of the vapor mitigation systems. The effectiveness of vapor mitigation systems might vary over time and with seasonality.

Conclusion 4

Exposure to tetrachloroethylene (PCE) and benzene in indoor air at some residential properties and at the former Delfasco Forge workplace buildings is not expected to harm people's health.

Basis for Conclusion

PCE and benzene were detected above comparison values (CVs) in indoor air at some residential properties and in the on-site occupational buildings. However, PCE and benzene were below levels that cause noncancer health effects. Cancer risks for PCE and benzene were estimated to be less than 1 in 1,000,000 people (1E-6). There is no concern for cancer from these exposures. However, there is uncertainty with the risk estimate because it is based on the maximum level of chemical detected in indoor air collected from one sampling event.

Conclusion 5

Due to lack of data, DSHS cannot currently conclude whether breathing indoor air at other residential and commercial buildings above the groundwater contamination might harm people's health because indoor air samples were not available.

Basis for Conclusion

DSHS estimates there are about 157 occupied residential properties and 20 occupied commercial or industrial buildings within 100 feet of the contaminated groundwater. Given the depth of groundwater (30 to 70 feet below ground surface), contaminants can move from the groundwater and soil and enter the interior of these buildings.

Most residential properties and businesses above the groundwater contamination have not been sampled. Some of these properties require more sampling to ensure that harmful exposures, if they are occurring, can be identified and stopped. Recent

groundwater sampling events show the groundwater contamination is moving to the northeast and past the current evaluated residential area. That means vapor intrusion might occur in a greater number of residential properties than previously estimated. The northeastern extent of the contamination has not been fully determined.

Conclusion 6

Water from private residential water wells that contain volatile organic compounds, such as TCE, PCE, and benzene, is not expected to harm people's health when used for irrigation, gardening, and recreational activities.

Basis for Conclusion

A 2006 drinking water survey identified 16 residential water wells within a 0.5-mile radius of the site. The wells were determined to be completed in the shallow groundwater. Although the wells are no longer used for domestic purposes, they may be used for irrigation, gardening, and recreation. A 2011 water well survey identified six unregistered wells within the groundwater contamination area. Of those six wells, one was used for irrigation and the other five were not in use. All homes are connected to city water for residential use. Therefore, exposure to contaminants in the groundwater during activities such as irrigation, gardening, and recreation using private well water could not be fully assessed because no groundwater samples from residential private water wells have been collected. However, exposure to volatile organic compounds (TCE, PCE, and benzene) in water from irrigation, gardening, and recreational activities would likely be minimal. These chemicals evaporate from water relatively quickly and readily disperse in outdoor air.

Conclusion 7

Residential exposure to drinking from the public water supply is not expected to harm people's health.

Basis for Conclusion

Residences are connected to the public water system of Grand Prairie, Texas. The 2011 well water survey found that none of the unregistered wells identified are used to supply water to homes. One state registered public drinking water well serves the public water system in the area of the groundwater contamination However, it does not connect to the contaminated groundwater and did not contain any site-related chemicals when sampled in 2011. This water well is completed in the lower Woodbine and Trinity aquifers that reach a depth of 2,163 feet below

ground surface and obtains water from 2,006 to 2,163 feet below ground surface. The Woodbine and Trinity aquifers are below the area of groundwater contamination and the Eagle Ford shale formation blocks movement between the shallower groundwater contamination and the much deeper aquifers. The Eagle Ford shale formation begins at 27 to 73 feet below ground surface and is approximately 145 feet thick and relatively impermeable.

Conclusion 8

Exposure to TCE and other site contaminants at James Fannon Middle School is not expected to harm the health of students and staff based on available information. However, there is potential for future exposure.

Basis for Conclusion

Exposure to TCE and other site contaminants is not likely to have taken place at James Fannin Middle School because the contaminated groundwater is not directly beneath the school and the direction of groundwater flow is moving away from the school. EPA also collected passive soil gas samples at the school that suggest vapor intrusion is not taking place. However, vapor intrusion might be a future potential exposure pathway because of several factors:

- Vapor dispersion of TCE from groundwater can vary over time and location
- Results from using passive soil gas samples to quantify air concentrations are not always reliable.

Recommendations

DSHS recommends that EPA do the following:

- Sample indoor air (and sub-slab soil gas and outdoor air when appropriate) in homes within 100 feet of the current groundwater plume, as delineated by 1 microgram per liter (μg/L) of TCE in groundwater. Sampling is recommended in hot and cold weather conditions when windows and doors are mostly closed to maintain climate control while heating and air conditioning operate.[2] Prioritization of indoor air sampling in residential homes should be for homes that have not yet been sampled, have not been sampled since receiving a vapor mitigation system, or have elevated indoor levels of TCE.
- Resample homes that were sampled between 2008 and 2016, due to possible changes in TCE concentrations in the groundwater since then, and consider multiple sampling events in cold and hot weather to understand potential seasonal variation.
- Continue its efforts to offer vapor mitigation systems to residents within 100 feet of the TCE groundwater contamination.
- Sample groundwater and active soil gas (near-source) within 100 feet of homes that refuse indoor air samples to assess potential risk to individual homes.
- Conduct a comprehensive groundwater sampling event to determine which additional homes might be contaminated by the TCE in groundwater. The extent of groundwater contamination moving to the northeast of the site has not been fully determined.
- Continue to monitor TCE levels in the shallow groundwater near James Fannin Middle School until remedial actions prevent further spread of contaminated groundwater.

[2] Also consider monitoring indicators, tracers, and surrogates to assess potential for active vapor intrusion conditions:

- https://iavi.rti.org/assets/docs/Temp_Measurement_Fact_Sheet_int.pdf
- https://iavi.rti.org/assets/docs/Pressure_Measurement_Fact_Sheet_Int.pdf
- https://iavi.rti.org/assets/docs/Radon_methods_fact_sheet_int.pdf

Public Health Action Plan

The public health action plan for the site contains a description of actions that have been or will be taken by DSHS, ATSDR, and other government agencies at the site. The purpose of the public health action plan is to ensure that this health consultation identifies public health hazards and provides a plan of action designed to mitigate and prevent harmful human health effects resulting from breathing, ingesting, or skin contact with hazardous substances found in the environment. Included is a commitment on the part of DSHS and ATSDR to follow up on this plan to ensure that it is put into effect.

Actions Planned

This document will be made available to community members, city officials, the Texas Commission on Environmental Quality (TCEQ), EPA, and other interested parties.

The DSHS Health Assessment and Toxicology Program will continue to work with EPA and TCEQ and other interested parties to ensure safety of the public.

The DSHS Health Assessment and Toxicology Program will also continue to engage with the community through community meetings and addressing community concerns.

Preparers of Report

The Texas Department of State Health Services (DSHS) prepared this health consultation for the Delfasco Forge Superfund Site, which is in Grand Prairie, Dallas County, Texas, under a cooperative agreement (#TS20-2001) with ATSDR. DSHS evaluated data of verified quality using approved methods, policies, and procedures in effect at the date of publication. ATSDR reviewed this document and concurs with its findings based on the information presented by DSHS.

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References

[ACS 2020] American Cancer Society. Cancer facts & figures 2020. Kennesaw, GA: American Cancer Society [accessed: 2020 October 15]. Available from: https://www.cancer.org/research/cancer-facts-statistics/all-cancer-facts-figures/cancer-facts-figures-2020.html.

Archer NP, Bradford CM, Villanacci JF, Crain NE, Corsi RL, Chambers DM, et al. 2015. Relationship between vapor intrusion and human exposure to trichloroethylene. J Environ Sci Health A Tox Hazard Subst Environ Eng. 50(13):1360–8. DOI: 10.1080/10934529.2015.1064275.

[ATSDR] Agency for Toxic Substances and Disease Registry. 2005a. Public Health Assessment Guidance Manual. Atlanta, GA: US Department of Health and Human Services, Public Health Service. Available from: https://www.atsdr.cdc.gov/hac/phamanual/pdfs/phagm_final1-27-05.pdf

[ATSDR] Agency for Toxic Substances and Disease Registry. 2005b. Toxicological profile for carbon tetrachloride. Atlanta, GA: US Department of Health and Human Services, Public Health Service. Available from: https://www.atsdr.cdc.gov/ToxProfiles/tp30.pdf

[ATSDR] Agency for Toxic Substances and Disease Registry. 2007. Toxicological profile for benzene. Atlanta, GA: US Department of Health and Human Services, Public Health Service. Available from: https://www.atsdr.cdc.gov/ToxProfiles/tp3.pdf

[ATSDR] Agency for Toxic Substances and Disease Registry. 2012. Toxicological profile for 1,4-dioxane. Atlanta, GA: US Department of Health and Human Services, Public Health Service. Available from: https://www.atsdr.cdc.gov/ToxProfiles/tp187.pdf

[ATSDR] Agency for Toxic Substances and Disease Registry. 2016. ATSDR's Guidance for Evaluating Vapor Intrusion Pathways. p. 54. Available from: https://www.atsdr.cdc.gov/pha-guidance/resources/atsdr-svi-guidance-508.pdf

[ATSDR] Agency for Toxic Substances and Disease Registry. 2019a. Toxicological profile for trichloroethylene. Atlanta, GA: US Department of Health and Human Services, Public Health Service. Available from: https://www.atsdr.cdc.gov/toxprofiles/tp19.pdf

[ATSDR] Agency for Toxic Substances and Disease Registry. 2019b. Toxicological profile for tetrachloroethylene. Atlanta, GA: US Department of Health and Human

Services, Public Health Service. Available from: https://www.atsdr.cdc.gov/ToxProfiles/tp18.pdf

[ATSDR] Agency for Toxic Substances and Disease Registry. 2019c. Exposure point concentration guidance for discrete sampling. Atlanta, GA: US Department of Health and Human Services, Public Health Service. Available from: https://www.atsdr.cdc.gov/pha-guidance/resources/epc-guidance-for-discrete-sampling-508.pdf

Dynamac Corporation. 2008. Site inspection report for Delfasco Forge Grand Prairie, Dallas County, Texas. Prepared for US Environmental Protection Agency Region 6. Richardson, TX: Dynamac Corporation; July 15, 2008.

Charbotel B, Fevotte J, Hours M, Martin JL, Bergeret A. 2006. Case-control study on renal cell cancer and occupational exposure to trichloroethylene. Part II: Epidemiological aspects. Ann Occup Hyg 50(8):777–87.

Chiu WA, Ginsberg GL. 2011. Development and evaluation of a harmonized physiologically based pharmacokinetic (PBPK) model for perchloroethylene toxicokinetics in mice, rats, and humans. Toxicol Appl Pharmacol 253(3):203–34.

[DSHS] Texas Department of State Health Services. 2012. Grand Prairie vapor intrusion investigation. Dallas County, Texas. p. 46.

[EA] EA Engineering, Science and Technology, Inc. 2011. Drinking water survey report. Delfasco Forge Division 114 NE 28th Street, Grand Prairie, Texas. Project No. 1486502. Hunt Valley, MD: EA Engineering, Science and Technology, Inc. p. 91.

[EA] EA Engineering, Science and Technology, Inc. 2013. Field activities and sampling results: Delfasco Forge site. Project No. 1486502. Hunt Valley, MD: EA Engineering, Science and Technology, Inc. p. 324.

[EA] EA Engineering, Science and Technology, Inc. 2014. Remedial investigation and sampling technical memorandum. Delfasco Forge Division, 114 NE 28th Street, Grand Prairie, Texas. Prepared for TCEQ Remediation Division, State Superfund. April 2014. EA Project 14865.23. Hunt Valley, MD: EA Engineering, Science and Technology, Inc.

[EA] EA Engineering, Science and Technology, Inc. 2021. Focused Feasibility Study Report Delfasco Forge Superfund Site Grand Prairie, Dallas County, Texas. ID: TXD98803428. Hunt Valley, MD: EA Engineering, Science and Technology, Inc.

EnSafe. 2002. Phase II environmental site assessment. Former Delfasco Forge facility 114 Northeast 28th St., Grand Prairie, Texas 75050. Ensafe Project 7540-005. Prepared for Delfasco Inc. Memphis, TN: EnSafe.

EnSafe. 2005. Affected property assessment report, VCP No 1571, former Delfasco Forge facility, 114 Northeast 28th St., Grand Prairie, Texas 75050. Prepared for Delfasco Inc. Memphis, TN: EnSafe.

EnSafe. 2006. Drinking water survey report, Former Delfasco Forge Facility, VCP No. 1571, 114 Northeast 28th Street, Grand Prairie, Texas 79044. Memphis, TN: EnSafe Inc. September 2006.

EnSafe. 2007. (Excerpts) affected property assessment report, VCP No. 1571, former Delfasco Forge facility, 114 Northeast 28th Street. Grand Prairie, Texas. Memphis, TN: EnSafe. p. 207.

EnSafe. 2016. Superfund site discovery and assessment program letter report Delfasco Forge Division site, 114 NE 28th St, Grand Prairie, Dallas County, Texas 75050, Site No. R00154. Memphis, TN: EnSafe.

[EPA] US Environmental Protection Agency. 2007 Integrated Risk Information System (IRIS) on benzene CASRN 71-43-2. National Center for Environmental Assessment, Office of Research and Development, Washington, DC. 2007 Available from: https://cfpub.epa.gov/ncea/iris/iris_documents/documents/subst/0276_summary.pdf#nameddest=cancerinhal

[EPA] US Environmental Protection Agency. 2010. Integrated Risk Information System (IRIS) on carbon tetrachloride CASRN 56-23-5. Washington, DC: National Center for Environmental Assessment, Office of Research and Development. Available from: https://iris.epa.gov/static/pdfs/0020_summary.pdf

[EPA] US Environmental Protection Agency. 2011. Background Indoor Air Concentrations of Volatile Organic Compounds in North American Residences (1990-2005): A Compilation of Statistics for Assessing Vapor Intrusion. Washington, DC: Office of Solid Waste and Emergency Response. EPA 530-R-10-001.

[EPA] US Environmental Protection Agency. 2012. Integrated Risk Information System (IRIS) on tetrachloroethylene CASRN 127-18-4. Washington, DC: National Center for Environmental Assessment, Office of Research and Development. Available from:

https://cfpub.epa.gov/ncea/iris/iris_documents/documents/subst/0106_summary.pdf

[EPA] US Environmental Protection Agency. 2013. Integrated Risk Information System (IRIS) on 1,4-dioxane CASRN 123-91-1. Washington, DC: National Center for Environmental Assessment, Office of Research and Development. Available from: https://cfpub.epa.gov/ncea/iris/iris_documents/documents/subst/0326_summary.pdf#nameddest=cancerinhal

[EPA] US Environmental Protection Agency. 2014. Final analytical report. Grand Prairie Property Liquidating, Inc. Sample collection dates 29 October – 20 October 2014. Project No. 15CAA018. p. 56.

[EPA] US Environmental Protection Agency. 2016. Trip report for indoor, crawl space and outdoor air sampling at the former Delfasco Forge site.

[EPA] US Environmental Protection Agency (EPA). 2018a. National Priorities List Final Rule, Vol. 83 Federal Register No. 96, Thursday, May 17, 2018. Rules and Regulations. Available from: https://www.govinfo.gov/content/pkg/FR-2018-05-17/pdf/2018-10464.pdf

[EPA] US Environmental Protection Agency. 2018b. Hazardous Ranking System. ID: TXD988034328. EPA Region 6: Grand Prairie, Dallas County, Texas.

[EPA] US Environmental Protection Agency. 2019. Integrated Risk Information System (IRIS) on trichloroethylene CASRN 79-01-6. Washington, DC: National Center for Environmental Assessment, Office of Research and Development. Available from: https://iris.epa.gov/static/pdfs/0199_summary.pdf

Gobba F, Righi E, Fantuzzi G, Predieri G, Cavazzuti L, Aggazzotti G. 1998. Two-year evolution of perchloroethylene-induced color-vision loss. Arch Environ Health 54(3):196–8.

[JBRC] Japan Bioassay Research Center. 1998. Subchronic inhalation toxicity and carcinogenicity studies of carbon tetrachloride in F344 rats and BDF1 mice (Studies Nos. 0020, 0021, 0043, and 0044). Kanagawa: Japan Industrial Safety and Health Association, Japan Bioassay Research Center.

Johnson PD, Goldberg SJ, Mays MZ, Dawson BV. 2003. Threshold of trichloroethylene contamination in maternal drinking waters affection fetal heart development in the rat. Environ Health Perspect 111(3):289–92.

Kasai T, Kano H, Umeda Y, Sasaki T, Ikawa N, Nishizawa T, et al. 2009. Two-year inhalation study of carcinogenicity and chronic toxicity of 1,4-dioxane in male rats. Inhal Toxicol (11):889–97.

Keil DE, Peden-Adams MM, Wallace S, Ruiz P, Gilkeson GS. 2009. Assessment of trichloroethylene (TCE) exposure in murine strains genetically-prone and non-prone to develop autoimmune disease. J Environ Sci Health A Tox Hazard Subst Environ Eng 44(5):443–53.

Lan Q, Zhang L, Li G, Vermeulen R, Weinberg RS, Dosemeci M, et al. 2004. Hematotoxicity in workers exposed to low levels of benzene. Science 306(5702):1774–6.

Nagano K, Nishizawa T, Yamamoto S, Matsushima T. 1998. Inhalation carcinogenesis studies of six halogenated hydrocarbons in rats and mice. In: Chiyotani K, Hosoda Y, Aizawa Y, eds. Advances in the prevention of occupational respiratory diseases: Proceedings of the 9th International Conference on Occupational Respiratory Diseases, Kyoto, Japan, 13-16 October 1997. Amsterdam: Elsevier Science BV. pp 741–6.

Nagano K, Umeda Y, Saito M, Nishizawa T, Ikawa N, Arito H, et al. 2007. Thirteen-week inhalation toxicity of carbon tetrachloride in rats and mice. J Occup Health 49:249–59.

[NTP 2021] National Toxicology Program. 2021. 15th Report on carcinogens. Research Triangle Park, NC: US Department of Health and Human Services, Public Health Service. Available from: https://ntp.niehs.nih.gov/go/roc15.

Raaschou-Nielsen O, Hansen J, McLaughlin JK, Kolstad H, Christensen JM, Tarone RE, et al. 2003. Cancer risk among workers at Danish companies using trichloroethylene: a cohort study. Am J Epidemiol 158(12):1182–92.

[TCEQ] Texas Commission on Environmental Quality. Texas Drinking Water Watch. Release: 3.7. Online database for public water supply system. [Accessed 2022 May 12]. Available from: https://dww2.tceq.texas.gov/DWW/JSP/NonTcrSampleResultsbyAnalyte.jsp?tinwsys_is_number=1256&tinwsys_st_code=TX&wsnumber=TX0570048%20%20%20&DWWState=TX&tsaanlyt_is_number=378&tsaanlyt_st_code=HQ&history=1&counter=0

[Tetra Tech] Tetra Tech. 2013. Environmental consulting services for the installation of chemical vapor mitigation systems, Grand Prairie, Texas. Pasadena, CA: Tetra Tech. p. 13.

[Tetra Tech] Tetra Tech. 2017. Installation of vapor intrusion mitigation systems. Pasadena, CA: Tetra Tech. p. 12.

[USCB] United States Census Bureau. 2010. Dallas, Texas. 2010 demographic profile. Accessed: July 21, 2020.

Wise JR, Hudson R. 1971. An Examination of expansive clay problems in Texas. The University of Texas at Austin. p. 98. Available from: https://library.ctr.utexas.edu/digitized/texasarchive/phase1/118-5-chr.pdf

Appendix A: Acronyms and Abbreviations

mg/kg/day — milligrams per kilogram per day

ADAF — age dependent adjustment factors

ATSDR — Agency for Toxic Substances and Disease Registry

BMCL — benchmark concentration lower confidence limit

BMDL — benchmark dose lower confidence limit

CV — comparison value

CREG — cancer risk evaluation guide

CSF — cancer slope factors

CTE — central tendency exposure

DSHS — Texas Department of State Health Services

EPA — Environmental Protection Agency

EPC — exposure point concentration

EMEG — environmental media evaluation guide

GWBU — groundwater bearing unit

HEC — human equivalent concentration

IUR — inhalation unit risk

MOE — margin of exposure

MRL — minimum risk level

NTP — National Toxicology Program

ND — not detected

NS — not sampled

NPL — National Priority List

NOAEL — no observed adverse effects level

PCE — tetrachloroethylene

RfD — reference dose

RME — reasonable maximum exposure

SVI — soil vapor intrusion

TCE — trichloroethylene

TCEQ — Texas Commission on Environmental Quality

VOCs — volatile organic compounds

Appendix B: Health Effects Evaluation Results

Table 11. Noncancer and cancer risks of chemicals detected above comparison values in indoor air at residential properties before mitigation system installation or in homes without vapor mitigation systems*

Property	Chemical	Exposure group	EPC ($\mu g/m^3$)	RME noncancer HQ†	RME cancer risk‡	MOE (fetal heart malformation)	MOE (immune system)
9	Benzene	Child	0.86	0.09	2E-6§	—	—
9	Benzene	Adult	0.86	0.09	3E-6§	—	—
16	Benzene	Child	0.32	0.03	7E-7	—	—
16	Benzene	Adult	0.32	0.03	1E-6	—	—
16	TCE	Child	5.2	3§	9E-6§	4	37
16	TCE	Adult	5.2	3§	9E-6§	4	37
16	PCE	Child	4.6	<1	3E-7	—	—
16	PCE	Adult	4.6	<1	5E-7	—	—
17	Benzene	Child	1.5	<1	3E-6§	—	—
17	Benzene	Adult	1.5	<1	5E-6§	—	—
17	TCE	Child	13.5	6.4§	2E-5§	2	14
17	TCE	Adult	13.5	6.4§	2E-5§	2	14
18	Benzene	Child	0.45	<1	9E-7	—	—
18	Benzene	Adult	0.45	<1	2E-6§	—	—
18	TCE	Child	3.8	1.8§	6E-6§	5	50
18	TCE	Adult	3.8	1.8§	7E-6§	5	50
25	Benzene	Child	0.45	<1	9E-7	—	—
25	Benzene	Adult	0.45	<1	2E-6§	—	—
25	TCE	Child	0.96	<1	2E-6	—	—
25	TCE	Adult	0.96	<1	2E-6	—	—
36	Benzene	Child	0.48	<1	1E-6	—	—
36	Benzene	Adult	0.48	<1	2E-6§	—	—
36	TCE	Child	4	1.9§	7E-6§	5	48

Property	Chemical	Exposure group	EPC ($\mu g/m^3$)	RME noncancer HQ†	RME cancer risk‡	MOE (fetal heart malformation)	MOE (immune system)
36	TCE	Adult	4	1.9§	7E-6§	5	48
38	Benzene	Child	0.9	<1	2E-6§	—	—
38	Benzene	Adult	0.9	<1	3E-6§	—	—
38	TCE	Child	3.2	1.5§	5E-6§	7	59
38	TCE	Adult	3.2	1.5§	5E-6§	7	59
39	Benzene	Child	0.48	<1	1E-6	—	—
39	Benzene	Adult	0.48	<1	2E-6§	—	—
39	TCE	Child	29	14§	5E-5§	<1	7
39	TCE	Adult	29	14§	5E-5§	<1	7
42	TCE	Child	22	10§	4E-5§	1	9
42	TCE	Adult	22	10§	4E-5§	1	9
43	TCE	Child	66	31§	1E-4§	<1	3
43	TCE	Adult	66	31§	1E-4§	<1	3
44	TCE	Child	0.16	<1	3E-7	—	—
44	TCE	Adult	0.16	<1	3E-7	—	—
55	Benzene	Child	0.54	<1	1E-6	—	—
55	Benzene	Adult	0.54	<1	2E-6§	—	—
57	Benzene	Child	1.3	<1	3E-6§	—	—
57	Benzene	Adult	1.3	<1	4E-6§	—	—
57	TCE	Child	7.2	3.4§	1E-5§	3	26
57	TCE	Adult	7.2	3.4§	1E-5§	3	26
60	Benzene	Child	4.2	<1	9E-6		
60	Benzene	Adult	4.2	<1	1E-5§		
60	TCE	Child	29	14§	5E-5§	<1	7
60	TCE	Adult	29	14§	5E-5§	<1	7
67	Benzene	Child	0.35	<1	7E-7	—	—
67	Benzene	Adult	0.35	<1	1E-6	—	—

Property	Chemical	Exposure group	EPC ($\mu g/m^3$)	RME noncancer HQ[†]	RME cancer risk[‡]	MOE (fetal heart malformation)	MOE (immune system)
69	Benzene	Child	0.96	<1	2E-6[§]	—	—
69	Benzene	Adult	0.96	<1	3E-6[§]	—	—
80	Benzene	Child	0.67	<1	1E-6	—	—
80	Benzene	Adult	0.67	<1	2E-6[§]	—	—
80	TCE	Child	1.7	<1	3E-6[§]	—	—
80	TCE	Adult	1.7	<1	3E-6[§]	—	—
85	Benzene	Child	1.2	<1	3E-6[§]	—	—
85	Benzene	Adult	1.2	<1	4E-6[§]	—	—
85	TCE	Child	3.7	1.8[§]	6E-6[§]	6	52
85	TCE	Adult	3.7	1.8[§]	6E-6[§]	6	52
86	Benzene	Child	0.86	<1	2E-6[§]	—	—
86	Benzene	Adult	0.86	<1	3E-6[§]	—	—
86	TCE	Child	2.8	1.3[§]	5E-6[§]	8	68
86	TCE	Adult	2.8	1.3[§]	5E-6[§]	8	68
105	Benzene	Child	0.54	<1	1E-6	—	—
105	Benzene	Adult	0.54	<1	2E-6[§]	—	—
105	TCE	Child	1.7	<1	3E-6[§]	—	—
105	TCE	Adult	1.7	<1	3E-6[§]	—	—

Abbreviations: < = less than; $\mu g/m^3$= micrograms per cubic meter of air; EPC = exposure point concentration; HQ = hazard quotient; MOE = margin of exposure; PCE = tetrachloroethylene; RME = reasonable maximum exposure; TCE = trichloroethylene.
*The calculations in this table were generated using ATSDR's Public Health Assessment Site Tool (PHAST version 2.1.1.0). Exposure parameters used in PHAST: chronic exposure = 24 hours per day, 7 days per week, 52 weeks per year, 21 years (child) or 33 years (adult). Exposure duration for children = 21 years and exposure duration for adults = 33 years. The Texas Department of State Health Services used the RME to calculate noncancer hazard quotients and cancer risk.
[†]The noncancer hazard quotients were calculated using the chronic minimal risk levels (MRLs): 2.1 $\mu g/m^3$ (trichloroethylene), 9.6 $\mu g/m^3$ (benzene), and 41 $\mu g/m^3$ (tetrachloroethylene).
[‡]The cancer risks were calculated using the inhalation unit risks of 2.1E-6 (non-Hodgkin's lymphoma), 1.0E-6 (liver), 1.0E-6 ([kidney) $(ug/m^3)^{-1}$ (trichloroethylene); 7.8E-6 $(\mu g/m^3)^{-1}$ (benzene); and 2.6E-7 $(\mu g/m^3)^{-1}$ (tetrachloroethylene).
[§]Indicates a value where HQ is greater than 1 or cancer risk is greater than 1E-6.

Table 12. Noncancer and cancer risks of chemicals detected in indoor air at residential properties with vapor mitigation systems, before and after installation (2009–2016)*

Property	Chemical	Exposure group	EPC (μg/m³) before installation	EPC (μg/m³) after installation †	RME noncancer HQ‡ after installation	RME cancer risk§ after installation	MOE (fetal heart malformation) after installation	MOE (immune system) after installation
9	Benzene	Child	0.86	0.67	<1	1E-6	—	—
9	Benzene	Adult	0.86	0.67	<1	2E-6	—	—
16	Benzene	Child	0.32	0.74	<1	2E-6¶	—	—
16	Benzene	Adult	0.32	0.74	<1	2E-6¶	—	—
16	TCE	Child	5.2	4.7	2.2¶	8E-6¶	4	41
16	TCE	Adult	5.2	4.7	2.2¶	8E-6¶	4	41
17	Benzene	Child	1.5	1.7	<1	4E-6¶	—	—
17	Benzene	Adult	1.5	1.7	<1	6E-6¶	—	—
17	TCE	Child	13.5	150	73¶	3E-6¶	<1	1
17	TCE	Adult	13.5	150	73¶	3E-6¶	<1	1
17	PCE	Child	NS	6.2	<1	4E-7	—	—
17	PCE	Adult	NS	6.2	<1	7E-7	—	—
18	Benzene	Child	0.45	0.61	<1	1E-6	—	—
18	Benzene	Adult	0.45	0.61	<1	2E-6¶	—	—
18	TCE	Child	3.82	40	19¶	7E-5¶	<1	5
18	TCE	Adult	3.82	40	19¶	7E-5¶	<1	5

Property	Chemical	Exposure group	EPC ($\mu g/m^3$) before installation	EPC ($\mu g/m^3$) after installation †	RME noncancer HQ‡ after installation	RME cancer risk§ after installation	MOE (fetal heart malformation) after installation	MOE (immune system) after installation
20	Benzene	Child	NS	0.77	<1	2E-6¶	—	—
20	Benzene	Adult	NS	0.77	<1	3E-6¶	—	—
20	TCE	Child	NS	122	58¶	2E-4¶	<1	2
20	TCE	Adult	NS	122	58¶	2E-4¶	<1	2
21	TCE	Child	NS	23	11¶	4E-5¶	<1	8
21	TCE	Adult	NS	23	11¶	4E-5¶	<1	8
25	Benzene	Child	0.96	1.09	<1	2E-6¶	—	—
25	Benzene	Adult	0.96	1.09	<1	2E-6¶	—	—
25	TCE	Child	0.38	ND	—	—	—	—
25	TCE	Adult	0.38	ND	—	—	—	—
38	Benzene	Child	0.86	0.9	<1	2E-6¶	—	—
38	Benzene	Adult	0.86	0.9	<1	3E-6¶	—	—
38	TCE	Child	3.23	23	11¶	4E-5¶	<1	8
38	TCE	Adult	3.23	23	11¶	4E-5¶	<1	8
43	Benzene	Child	NS	0.7	<1	2E-6¶	—	—
43	Benzene	Adult	NS	0.7	<1	2E-6¶	—	—
43	TCE	Child	65.6	13.8	6.6¶	2E-5¶	2	14
43	TCE	Adult	65.6	13.8	6.6¶	2E-5¶	2	14

Property	Chemical	Exposure group	EPC ($\mu g/m^3$) before installation	EPC ($\mu g/m^3$) after installation †	RME noncancer HQ‡ after installation	RME cancer risk§ after installation	MOE (fetal heart malformation) after installation	MOE (immune system) after installation
55	Benzene	Child	0.54	0.61	<1	1E-6	—	—
55	Benzene	Adult	0.54	0.61	<1	2E-6¶	—	—
55	TCE	Child	NS	0.32	<1	5E-7	—	—
55	TCE	Adult	NS	0.32	<1	6E-7	—	—
57	Benzene	Child	1.25	1.18	<1	3E-6¶	—	—
57	Benzene	Adult	1.25	1.18	<1	4E-6¶	—	—
57	TCE	Child	7.22	4.25	2¶	7E-6¶	5	45
57	TCE	Adult	7.22	4.25	2¶	7E-6¶	5	45
58	Benzene	Child	NS	3.5	<1	7E-6¶	—	—
58	Benzene	Adult	NS	3.5	<1	1E-5¶	—	—
58	TCE	Child	NS	6.3	3.0¶	1E-5¶	3	30
58	TCE	Adult	NS	6.3	3.0¶	1E-5¶	3	30
59	Benzene	Child	NS	0.67	<1	1E-6	—	—
59	Benzene	Adult	NS	0.67	<1	2E-6¶	—	—
59	TCE	Child	NS	3.0	1.4¶	5E-6¶	7	63
59	TCE	Adult	NS	3.0	1.4¶	5E-6¶	7	63
60	Benzene	Child	4.19	6.2	<1	1E-5¶	—	—
60	Benzene	Adult	4.19	6.2	<1	2E-5¶	—	—

Property	Chemical	Exposure group	EPC ($\mu g/m^3$) before installation	EPC ($\mu g/m^3$) after installation †	RME noncancer HQ‡ after installation	RME cancer risk§ after installation	MOE (fetal heart malformation) after installation	MOE (immune system) after installation
60	TCE	Child	28.8	15.6	7.4¶	3E-5¶	1	12
60	TCE	Adult	28.8	15.6	7.4¶	3E-5¶	1	12
66	Benzene	Child	NS	2.8	<1	6E-6¶	—	—
66	Benzene	Adult	NS	2.8	<1	9E-6¶	—	—
68	Benzene	Child	NS	0.74	<1	2E-6¶	—	—
68	Benzene	Adult	NS	0.74	<1	2E-6¶	—	—
79	Benzene	Child	NS	0.58	<1	1E-6	—	—
79	Benzene	Adult	NS	0.58	<1	2E-6¶	—	—
79	TCE	Child	NS	0.92	<1	2E-6¶	—	—
79	TCE	Adult	NS	0.92	<1	2E-6¶	—	—
80	Benzene	Child	0.67	0.83	<1	2E-6¶	—	—
80	Benzene	Adult	0.67	0.83	<1	3E-6¶	—	—
80	TCE	Child	1.72	0.81	<1	1E-6	—	—
80	TCE	Adult	1.72	0.81	<1	1E-6	—	—
81	Benzene	Child	NS	0.99	<1	2E-6¶	—	—
81	Benzene	Adult	NS	0.99	<1	3E-6¶	—	—
81	TCE	Child	NS	48	23¶	8E-5¶	<1	4
81	TCE	Adult	NS	48	23¶	8E-5¶	<1	4

Property	Chemical	Exposure group	EPC ($\mu g/m^3$) before installation	EPC ($\mu g/m^3$) after installation †	RME noncancer HQ‡ after installation	RME cancer risk§ after installation	MOE (fetal heart malformation) after installation	MOE (immune system) after installation
85	Benzene	Child	1.2	1.1	<1	2E-6¶	—	—
85	Benzene	Adult	1.2	1.1	<1	4E-6¶	—	—
85	TCE	Child	3.7	23	11¶	4E-5¶	—	—
85	TCE	Adult	3.7	23	11¶	4E-5¶	—	—
86	Benzene	Child	0.86	1.2	<1	2E-6¶	—	—
86	Benzene	Adult	0.86	1.2	<1	4E-6¶	—	—
86	TCE	Child	2.8	0.65	<1	1E-6	—	—
86	TCE	Adult	2.8	0.65	<1	1E-6	—	—
101	Benzene	Child	NS	1.1	<1	2E-6¶	—	—
101	Benzene	Adult	NS	1.1	<1	4E-6¶	—	—
104	Benzene	Child	NS	0.48	<1	1E-6	—	—
104	Benzene	Adult	NS	0.48	<1	2E-6¶	—	—
104	TCE	Child	NS	0.54	<1	9E-7	—	—
104	TCE	Adult	NS	0.54	<1	9E-7	—	—
105	Benzene	Child	0.54	0.7	<1	2E-6¶	—	—
105	Benzene	Adult	0.54	0.7	<1	2E-6¶	—	—
105	TCE	Child	1.67	0.27	<1	5E-7	—	—
105	TCE	Adult	1.67	0.27	<1	5E-7	—	—

Abbreviations: µg/m^3= micrograms per cubic meter of air; EPC = exposure point concentration; HQ = hazard quotient; MOE = margin of exposure; ND = not detected; NS = not sampled; PCE = tetrachloroethylene; RME = reasonable maximum exposure; TCE = trichloroethylene.
*The calculations in this table were generated using ATSDR's Public Health Assessment Site Tool (PHAST version 2.1.1.0). Exposure parameters used in PHAST: chronic exposure 24 hours per day, 7 days per week, 52 weeks per year, 24 years (child) or 33 years (adult). Exposure duration for children = 21 years and exposure duration for adults = 33 years. The Texas Department of State Health Services used the RME to calculate noncancer and cancer risk.
†The post-mitigation EPC is the first sample taken after a mitigation system was installed from 2009, 2014, or 2016.
‡The noncancer hazard quotients were calculated using the chronic minimal risk levels (MRLs): 2.1 µg/m^3 (trichloroethylene), 9.6 µg/m^3 (benzene), and 41 µg/m^3 (tetrachloroethylene).
§The cancer risks were calculated using the inhalation unit risks of 2.1E-6 (non-Hodgkin's lymphoma), 1.0E-6 (liver), 1.0E-6 (kidney) (µg/m^3)$^{-1}$ (trichloroethylene); 7.8E-6 (µg/m^3)$^{-1}$ (benzene); and 2.6E-7 (µg/m^3)$^{-1}$ (tetrachloroethylene).
¶Indicates a value where HQ is greater than 1 or cancer risk is greater than 1E-6.

Table 13. Noncancer and cancer risks of chemicals detected in indoor air in 2016 at residential properties with vapor mitigation systems, based on most recent sampling results*

Property	Chemical	Exposure group	EPC† (µg/m³) Before mitigation	EPC† (µg/m³) Most Recent	RME noncancer‡ HQ	RME cancer risk§	MOE (fetal heart malformation)	MOE (immune system)
17	Benzene	Child	1.7	NS	—	—	—	—
17	Benzene	Adult	1.7	NS	—	—	—	—
17	TCE	Child	150	0.92	<1	**2E-6**¶	—	—
17	TCE	Adult	150	0.92	<1	**2E-6**¶	—	—
17	PCE	Child	6.2	ND	—	—	—	—
17	PCE	Adult	6.2	ND	—	—	—	—
18	Benzene	Child	0.61	NS	—	—	—	—
18	Benzene	Adult	0.61	NS	—	—	—	—
18	TCE	Child	40	ND	—	—	—	—
18	TCE	Adult	40	ND	—	—	—	—
20	Benzene	Child	0.77	NS	—	—	—	—
20	Benzene	Adult	0.77	NS	—	—	—	—
20	TCE	Child	120	53.1	**25**¶	**9E-5**¶	<1	4
20	TCE	Adult	120	53.1	**25**¶	**9E-5**¶	<1	4
20	PCE	Child	1.3	0.34	<1	—	—	—
20	PCE	Adult	1.3	0.34	<1	—	—	—
43	Benzene	Child	0.7	NS	—	—	—	—

Property	Chemical	Exposure group	EPC† (µg/m³) Before mitigation	EPC† (µg/m³) Most Recent	RME noncancer‡ HQ	RME cancer risk§	MOE (fetal heart malformation)	MOE (immune system)
43	Benzene	Adult	0.7	NS	—	—	—	—
43	TCE	Child	13.8	ND	—	—	—	—
43	TCE	Adult	13.8	ND	—	—	—	—
60	Benzene	Child	6.2	NS	—	—	—	—
60	Benzene	Adult	6.2	NS	—	—	—	—
60	TCE	Child	16	0.75	<1	1E-6	—	—
60	TCE	Adult	16	0.75	<1	1E-6	—	—
85	Benzene	Child	1.1	NS	—	—	—	—
85	Benzene	Adult	1.1	NS	—	—	—	—
85	TCE	Child	23	ND	—	—	—	—
85	TCE	Adult	23	ND	—	—	—	—

Abbreviations: µg/m³= micrograms per cubic meter of air; EPC = exposure point concentration; HQ = hazard quotient; MOE = margin of exposure; ND = not detected; NS = not sampled; PCE = tetrachloroethylene; RME = reasonable maximum exposure; TCE = trichloroethylene.

Note: Bold values indicated HQ greater than 1 or cancer risk greater than 1E-6.

*The calculations in this table were generated using ATSDR's Public Health Assessment Site Tool (PHAST version 2.1.1.0). Exposure parameters used in PHAST: chronic exposure 24 hours per day, 7 days per week, 52 weeks per year, 24 years (child) or 33 years (adult). Exposure duration for children = 21 years and exposure duration for adults = 33 years. DSHS used the RME to calculate noncancer and cancer risk.

†The post-mitigation EPC is the first sample taken after a vapor mitigation system was installed from 2009, 2014, or 2016.

‡The noncancer hazard quotients were calculated using the chronic minimal risk levels (MRLs): 2.1 µg/m³ (trichloroethylene), 9.6 µg/m³ (benzene), and 41 µg/m³ (tetrachloroethylene).

§The cancer risks were calculated using the inhalation unit risks of 2.1E-6 (non-Hodgkin's lymphoma), 1.0E-6 (liver), 1.0E-6 (kidney) (µg/m3)$^{-1}$ (trichloroethylene), 7.8E-6 (µg/m3)$^{-1}$ (benzene), and 2.6E-7 (µg/m3)$^{-1}$ (tetrachloroethylene).

¶Indicates a value where HQ is greater than 1 or cancer risk is greater than 1E-6.

Appendix C: Screening Analysis

Table 14. Maximum contaminant concentrations in residential indoor air sampling from 2008 to 2016*

Property ID	Benzene (μg/m³)	TCE (μg/m³)	PCE (μg/m³)	Contaminants
16	0.3	5.2	4.6	Benzene, TCE, PCE
17	1.7	153	6.2	Benzene, TCE, PCE
18	0.6	39.7	0.5	Benzene, TCE
20	0.8	122.0	1.3	Benzene, TCE
21	ND	22.8	ND	TCE
25	0.96	0.38	ND	Benzene, TCE
36	0.5	4.0	ND	Benzene, TCE
38	0.9	23.1	0.34	Benzene, TCE
39	0.5	28.5	ND	Benzene, TCE
42	ND	21.9	0.30	TCE
43	0.7	65.6	0.96	Benzene, TCE
44	ND	0.2	ND	
55	0.6	0.3	ND	Benzene, TCE
57	1.3	7.2	1.5	Benzene, TCE
58	3.5	6.3	ND	Benzene, TCE
59	0.7	3.0	ND	Benzene, TCE
60	6.2	28.8	ND	Benzene, TCE

Property ID	Benzene (μg/m³)	TCE (μg/m³)	PCE (μg/m³)	Contaminants
79	0.6	0.9	0.41	Benzene, TCE
80	0.8	1.7	ND	Benzene, TCE
81	1.0	47.7	ND	Benzene, TCE
85	1.2	23.2	ND	Benzene, TCE
86	1.2	2.8	ND	Benzene, TCE
104	0.5	0.5	ND	Benzene, TCE
105	0.5	1.7	ND	Benzene, TCE

Abbreviations: μg/m³ = micrograms per cubic meter or air; ND = not detected; PCE = tetrachloroethylene; TCE = trichloroethylene.
*The Texas Department of State Health Services used the screening value for trans-1,2-dichloroethene to screen cis-1,2-dichloroethene because there is no ATSDR screening value available for cis-1,2-dichlorothene. Trans-1,2-dichlorethene and cis-1,2-dichlorethene did not screen above the comparison value in any samples. ATSDR cancer risk evaluation guidelines comparison values were used. The comparison value for benzene was 0.13 μg/m³ for TCE was 0.21 μg/m³ and for PCE was 3.8 μg/m³.

Appendix D: Air Inhalation Pathway Assessment Calculations

Air Inhalation Exposure Equation

Adjusted EPC = EPC x EFnoncancer

Where

EPC = exposure point concentration,
EFnoncancer = exposure factor (unitless)

Hazard Quotient

HQ = Adjusted EPC ÷ HG

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

Cancer Risk Equations

CR = Adjusted EPC x IUR x (ED ÷ LY) Equation 3

ADAF-adjusted CR = (Adjusted EPC x IUR) x (ED ÷ LY) x ADAF Equation 4

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

Where

CR = cancer risk (unitless),
EPC = exposure point concentration ($\mu g/m^3$),
IUR = inhalation unit risk ($[\mu g/m^3]^{-1}$),
ED = exposure duration (years)

LY = lifetime years (78 years)

ADAF = age-dependent adjustment factor (unitless),

EF (cancer) = exposure factor (cancer);

Where

EF (cancer) = ($EF_{noncancer}$ x Exposure Duration for $Cancer_{Exposure\ Group}$ [years])/ 78 years

The EPC for noncancer health effects in indoor air contaminants were calculated using the following formula:

EPC noncancer = C x ET x EF

where

EPC = exposure point concentration of contaminant in air ($\mu g/m^3$),
C = 95% upper confidence limit or maximum concentration of contaminant in air ($\mu g/m^3$),
ET = exposure time (hours/24 hours), and
EF = exposure frequency (days/365 days).

Appendix E: Trichloroethylene Cancer Risk Calculations

Cancer risk (CR) calculations for TCE incorporate three cancer types, including non-Hodgkin's lymphoma (NHL), kidney and liver cancer. Because TCE is only considered to be mutagenic for kidney cancer, age-dependent adjustment factors (ADAFs) are only applied to the kidney cancer portion of the cancer slope factor (CSF) and inhalation unit risk (IUR).

Therefore, TCE cancer risk = (non-Hodgkin's lymphoma risk + liver cancer risk) + (kidney cancer risk with ADAFs applied)

For inhalation:

- Total TCE CR = (NHL and liver CR) + (ADAF-adjusted kidney CR)
- NHL & liver CR = (C × (IUR for NHL + IUR for liver cancer)) × (ED/LY)
- ADAF-adjusted kidney CR = (C × IUR for kidney cancer) × ED/LY) × ADAF

Where

CR = cancer risk,
C = air concentration ($\mu g/m^3$),
IUR = inhalation unit risk ($[\mu g/m^3]^{-1}$),
ED = age-specific exposure duration (years),
LY = lifetime in years (78 years),
ADAF = age-dependent adjustment factor (unitless),
NHL = non-Hodgkin's lymphoma

The following ADAFs are applied for the evaluation of kidney cancer:

- ADAF of 10 for exposures occurring from birth to <2 years
- ADAF of 3 for exposures occurring from 2 to <16 years

Table 15. Site-specific residential exposure factors

Duration category	Hours per day	Days per week	Weeks per year	Years	Exposure group specific $EF_{noncancer}$	Exposure group specific* EF_{cancer}
Acute	24	—	—	—	1	—
Intermediate	24	7	—	—	1	—

Duration category	Hours per day	Days per week	Weeks per year	Years	Exposure group specific $EF_{noncancer}$	Exposure group specific* EF_{cancer}
Chronic	24	7	52.14	33	1	= ($EF_{noncancer}$ x Exposure duration for $cancer_{Exposure\ Group}$ (years))/78 years

Abbreviation: EF = exposure factor.
*Cancer EFs are not shown in the table because they are calculated using age-specific durations. The general formula is EF_{cancer} = ($EF_{noncancer}$ × Exposure Duration for $Cancer_{Exposure\ Group}$ (years))/78 years.

Table 16. Site-specific residential exposure factors for exposure groups

Exposure group	Age-specific exposure duration (years)
Birth to <1 year	1
1 to <2 years	1
2 to <6 years	4
6 to <11 years	5
11 to <16 years	5
16 to <21 years	5
Adult	33

Table 17. Site-specific occupational noncancer exposure factors*

Exposure group	Chronic CTE	Chronic RME	Intermediate CTE	Intermediate RME	Acute CTE	Acute RME
Full-time worker	0.24	0.24	0.25	0.25	0.35	0.35

Abbreviations: CTE = central tendency exposure; RME = reasonable maximum exposure.
*Cancer exposure factors (EF) are not shown in the table because they are calculated using age-specific durations. The general formula is $EF_{cancer} = EF_{noncancer} \times$ Exposure Duration for $Cancer_{Exposure\ Group}$ (years) ÷ 78 years.

Table 18. Site-specific occupational exposure factors per exposure duration

Exposure group	Daily (hours/day) CTE	Daily (hours/day) RME	Weekly (days/week) CTE	Weekly (days/week) RME	Annually (weeks/year) CTE	Annually (weeks/year) RME	Age-specific exposure duration (years) CTE	Age-specific exposure duration (years) RME
Full-time worker	8.5	8.5	5	5	50	50	5	20

Abbreviations: CTE = central tendency exposure; RME = reasonable maximum exposure.

Appendix F: Occupational Cancer Results

1,4-dioxane

Table 19. Occupational: Site-specific exposure point concentrations for chronic exposure to 1,4-dioxane in indoor air at 0.62 µg/m³ (0.17 ppb) along with noncancer hazard quotients and cancer risk estimates*

Exposure group	CTE Adjusted EPC† (µg/m³)	CTE Adjusted EPC† (ppb)	CTE Noncancer hazard quotient	CTE Cancer risk	CTE Exposure duration (years)	RME Adjusted EPC (µg/m³)	RME Adjusted EPC (ppb)	RME Noncancer hazard quotient	RME Cancer risk	RME Exposure duration (years)
Full-time worker	0.15	0.042	<1	5E-8	5	0.15	0.042	<1	2E-7	20

Abbreviations: µg/m^3 = micrograms per cubic meters; CTE = central tendency exposure; EPC = the exposure point concentration; mg/kg/day = milligram chemical per kilogram body weight per day; ppb = parts per billion; RME = reasonable maximum exposure.

***The values in this table were generated using ATSDR's Public Health Assessment Site Tool (PHAST v2.1.1.0). The noncancer hazard quotients were calculated using the chronic (greater than 1 year) minimal risk level of 110 µg/m^3 and the cancer risks were calculated using the inhalation unit risk of 5.0E-6 (µg/m^3)$^{-1}$.

†Adjusted EPC is the EPC times the appropriate exposure factors.

Benzene

Table 20. Occupational: Site-specific exposure point concentrations for chronic exposure to benzene in indoor air at 12 µg/m³ (3.8 ppb) along with noncancer hazard quotients and cancer risk estimates*

Exposure group	CTE Adjusted EPC† (µg/m³)	CTE Adjusted EPC† (ppb)	CTE Noncancer hazard quotient	CTE Cancer risk	CTE Exposure duration (years)	RME Adjusted EPC† (µg/m³)	RME Adjusted EPC† (ppb)	RME Noncancer hazard quotient	RME Cancer risk	RME Exposure duration (years)
Full time worker	2.9	0.91	<1	**2E-6‡**	5	2.9	0.91	<1	**6E-6‡**	20

Abbreviations: µg/m^3 = micrograms per meter cubed; CTE = central tendency exposure; EPC = exposure point concentration; mg/kg/day = milligram chemical per kilogram body weight per day; ppb = parts per billion; RME=reasonable maximum exposure.

***The calculations in this table were generated using ATSDR's Public Health Assessment Site Tool (PHAST v2.1.1.0). The noncancer hazard quotients were calculated using the chronic (greater than 1 year) minimal risk level of 9.6 µg/m^3 and the cancer risks were calculated using the inhalation unit risk of 7.8E-6 (µg/m^3)$^{-1}$.

†Adjusted EPC is the exposure point concentration (EPC) times the appropriate exposure factors.

‡A bolded value indicates that the cancer risk exceeds one extra case in a million people.

Carbon tetrachloride

Table 21. Occupational: Site-specific exposure point concentrations for chronic exposure to carbon tetrachloride in air at 0.44 µg/m³ (0.07 ppb) along with noncancer hazard quotients and cancer risk estimates*

Exposure group	CTE Adjusted EPC† (µg/m³)	CTE Adjusted EPC† (ppb)	CTE Noncancer hazard quotient	CTE Cancer risk	CTE Exposure duration (years)	RME Adjusted EPC† (µg/m³)	RME Adjusted EPC† (ppb)	RME Noncancer hazard quotient	RME cancer risk	RME Exposure duration (years)
Full time worker	0.11	0.017	<1	4E-8	5	0.11	0.017	<1	2E-7	20

Abbreviations: µg/m^3 = micrograms per meter cubed; CTE = central tendency exposure; EPC = exposure point concentration; mg/kg/day = milligram chemical per kilogram body weight per day; ppb = parts per billion; RME = reasonable maximum exposure.

***The calculations in this table were generated using ATSDR's Public Health Assessment Site Tool (PHAST v2.1.1.0). The non-cancer hazard quotients were calculated using the chronic (greater than 1 year) minimal risk level of 190 µg/m^3 and the cancer risks were calculated using the inhalation unit risk of 6.0E-6 (µg/m^3)$^{-1}$.

†Adjusted EPC is the EPC times the appropriate exposure factors.

Trichloroethylene

Table 22. Occupational: Site-specific exposure point concentrations for chronic exposure to trichloroethylene (TCE) in indoor air at 48 µg/m³ (8.9 ppb) along with noncancer hazard quotients and cancer risk estimates*

Exposure group	CTE Adjusted EPC† (µg/m³)	CTE Adjusted EPC† (ppb)	CTE Noncancer hazard quotient	CTE Cancer risk	CTE Exposure duration (years)	RME Adjusted EPC (µg/m³)	RME Adjusted EPC (ppb)	RME Noncancer hazard quotient	RME Cancer risk	RME Exposure duration (years)
Full-time worker	12	2.2	**5.5‡**	**3E-6§**	5	12	2.2	**5.5‡**	**1E-5§**	20

Abbreviations: µg/m^3 = micrograms per cubic meter; CTE = central tendency exposure; EPC = exposure point concentration; mg/kg/day = milligram chemical per kilogram body weight per day; ppb = parts per billion; RME = reasonable maximum exposure.

*****The calculations in this table were generated using ATSDR's Public Health Assessment Site Tool (PHAST v2.1.1.0). The non-cancer hazard quotients were calculated using the chronic (greater than 1 year) minimal risk level of 2.1 µg/m^3 and the cancer risks were calculated using the inhalation unit risks of 2.1E-6 (non-Hodgkin's lymphoma), 1.0E-6 (liver), 1.0E-6 (kidney) (µg/m^3)$^{-1}$ and age-dependent adjustment factors.

†Adjusted EPC is the EPC times the appropriate exposure factors.

‡Bolded value indicates hazard quotient is greater than 1.

§Bolded value indicates that the cancer risk exceeds one extra case in a million people.

Appendix G: Air Concentration Conversion Calculation

The Texas Department of State Health Services used the equation below to convert contaminant air concentration units from parts per billion (ppb) to micrograms per meter cubed ($\mu g/m^3$) and parts per million (ppm) to $\mu g/m^3$.

$\mu g/m^3 = (ppb * 12.187 * M) / (273 + °C)$

$\mu g/m^3 = ((ppm*1000) * 12.187 * M) / (273 + °C)$

Where:

- ppb or ppm = the contaminant concentration in ppb or ppm
- M = the molecular weight of the contaminant
- °C = ambient air temperature in Celsius; 20°C was used as the standard ambient temperature.
- An atmospheric pressure of 1 atmosphere was assumed.