Health Consultation

Off-Site Vapor Intrusion Investigation

SRG GLOBAL, INC.
COVINGTON, NEWTON COUNTY, GEORGIA

Prepared by
Georgia Department of Public Health

JANUARY 29, 2016

Prepared under a Cooperative Agreement with the
U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES
Agency for Toxic Substances and Disease Registry
Division of Community Health Investigations
Atlanta, Georgia 30333

Health Consultation: A Note of Explanation

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In addition, consultations may recommend additional public health actions, such as conducting health surveillance activities to evaluate exposure or trends in adverse health outcomes; conducting biological indicators of exposure studies to assess exposure; and providing health education for health care providers and community members. This concludes the health consultation process for this site, unless additional information is obtained by ATSDR or ATSDR's Cooperative Agreement Partner which, in the Agency's opinion, indicates a need to revise or append the conclusions previously issued.

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Glossary of Acronyms

ATSDR	Agency for Toxic Substances and Disease Registry
COC	Contaminants of Concern
CREG	Cancer Risk Evaluation Guide
CSF	Cancer Slope Factor
CVs	Comparison Values
EMEG	Environmental Media Evaluation Guide
EPA	United States Environmental Protection Agency
EPD	Georgia Environmental Protection Division
DPH	Georgia Department of Public Health
HEC	Human Equivalent Concentration
IARC	International Agency for Research on Cancer
IRIS	Integrated Risk Information System
LOAEL	Lowest Observed Adverse Effects Level
MRL	Minimal Risk Level
NOAEL	No Observed Adverse Effects Level
NPL	National Priorities List
NTP	National Toxicology Program
PCE	Tetrachloroethene
RfC	Reference Concentration
RSL	Regional Screening Levels
ug/m^3	micrograms per cubic meter
ppb	micrograms per liter
TCE	Trichloroethene
VOCs	Volatile Organic Compounds

Summary -

The Georgia Environmental Protection Division (EPD) requested that the Georgia Department of Public Health (DPH) provide public health input for a vapor intrusion investigation related to the SRG Global, Inc. manufacturing facility in Covington, Newton County, Georgia. A chlorinated solvent groundwater plume migrated from the SRG Global facility under the Settlers Grove residential neighborhood located south and southeast of the manufacturing plant. DPH evaluated available indoor air sample results from the Settlers Grove community to determine if people exposed to chlorinated solvents might be harmed. All residents in the community are connected to the public water supply, which is not affected by the offsite groundwater contamination. DPH reached the following three conclusions based on a review and evaluation of the sample results.

Conclusion 1

In the past, breathing trichloroethene (TCE) in House #68 via the vapor intrusion pathway, could have harmed people's health based on the available data. Babies born to pregnant women exposed to TCE in early pregnancy at these levels may have had an increased risk for heart development problems. All residents exposed to TCE at pre-mitigation levels may have had a smaller risk of immune system impacts.

Currently, post-mitigation sampling data show that these residents are no longer being exposed to concentrations of TCE in indoor air that may harm their health. However, even with a venting system in operation, indoor air samples remain above an Agency for Toxic Substances and Disease Registry (ATSDR) Cancer Risk Evaluation Guide (CREG), but even so, there is no measurable excess cancer risk from this exposure. This also applies to residents occupying House #3. Nonetheless, a low-level risk from vapor intrusion will exist until contamination in groundwater is sufficiently reduced or removed.

Basis for Conclusion

The highest indoor air concentration detected at this residence (House #68) before mitigation is 5.5 times above the ATSDR minimal risk level (MRL) for chronic and intermediate inhalation of TCE and approaches the human equivalent concentration (HEC) of 21µg/m^3 in air for a 1% extra risk in fetal cardiac malformations based on modeling and estimates from animal studies. Exposure of pregnant women to TCE levels above the Reference concentration (RfC) does not mean that fetal heart development will be impaired. However, breathing air exceeding these levels of TCE begins to introduce a small amount of risk to proper fetal heart development. Although the highest indoor air concentration detected at this residence is approximately 17 times lower than the HEC of 190 µg/m^3 for decreased thymus weight observed in mice, a small amount of risk to impacts on immune system may exist in immunosuppressed populations. Data show that the currently operating mitigation system is reducing indoor air concentrations to less than the RfC for this residence.

Next Steps

Take steps and to ensure proper maintenance and effectiveness of the venting systems until the VOC source is removed and/or degraded.

Conclusion 2

The residents in House #171 are breathing PCE (although the source is not known, PCE was not found in shallow groundwater at sampling locations nearest this residence) at levels below those that may increase the risk of adverse non-cancer health effects from this exposure. However, if the concentration of PCE found in both sampling events continues to be high, a slight increase in excess cancer risk from this exposure is expected; however, risk is still low.

Basis for Conclusion

Being that the RfC is an estimate of continuous inhalation exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime of exposure, the highest PCE concentration measured at this residence is approximately 43 times below the midpoint of the lowest observed adverse health effects (LOAEL) ranges observed in humans referenced in the studies used to derive the RfC. Thus, DPH concludes that residents exposed to PCE at levels found in indoor air at this residence are exposed to levels below those that may increase the risk of adverse non-cancer health effects. However, if the concentration of PCE continues to be present in indoor air at the levels measured in two sampling events, residents are exposed to an excess cancer risk level that is approximately 70 times higher than the excess cancer risk from exposure to PCE at the ATSDR CREG of 3.8 $\mu g/m^3$ for air. The estimated cancer risk is between 2 to 7 $x10^{-5}$.

Next Steps

Because the source of PCE in this residence has not been unequivocally determined and is not likely due to vapor intrusion, DPH recommends that SRG Global sample the indoor air of this residence in the near future during a known period when dry-cleaned clothes have not been brought into the residence, and the carpets have not been cleaned to rule out these avenues as the source of PCE. In addition, one potential source of PCE that has not been considered is sewer gas from a cracked sewer line that may extend into the surficial aquifer. However, PCE has been found in shallow groundwater at sampling locations located north of this residence. PCE has also been found at depths greater than 25 feet below ground surface at two locations just south of this residence.

Conclusion 3

Long-term exposures and potential harm to health are uncertain in homes without mitigation systems, because temporal variability has not been evaluated and some homeowners did not authorize a vapor intrusion investigation.

Basis for Conclusion

Follow-up sampling was only performed for some residences, and about half the residents did not authorize any sampling. Vapor intrusion often varies substantially over time and requires multiple sampling events over multiple seasons to evaluate the variability.

Next Steps

In order to protect community health and well-being, DPH has recommended that SRG Global under EPD oversight continue to take steps to identify homes with vapor intrusion and interrupt the vapor intrusion pathway for affected residences in the Settlers Grove subdivision. These steps include sampling and the installation of venting systems to mitigate vapor intrusion and to ensure proper maintenance and effectiveness of the venting systems until the VOC source is removed and/or degraded.

For More Information

If you have questions or comments, you can call ATSDR toll-free at 1 800-CDC-INFO and ask for information on the SRG Global Vapor Intrusion Investigation site.

Statement of Issues

In April 2014, the Georgia Environmental Protection Division (EPD) requested that the Georgia Department of Public Health (DPH) provide public health input for a vapor intrusion investigation related to the SRG Global, Inc. manufacturing facility in Covington, Newton County, Georgia (Figure 1). A chlorinated solvent groundwater plume migrated from the SRG Global facility under the Settlers Grove residential neighborhood located south and southeast of the manufacturing plant. All residents in the community are connected to the public water supply, which is not affected by the offsite groundwater contamination.

Vapor intrusion occurs when vapors from groundwater or subsurface soil contamination move through the air spaces in the soil, enter a building through cracks or other openings in the building's foundation, and build up in the indoor air [U.S. Environmental Protection Agency (EPA) 2012 a]. Many factors, including fluctuations over time in outdoor barometric pressure, soil moisture (from precipitation) or building pressure (from heating, ventilation or air conditioning operation) can affect whether or not vapor intrusion occurs. Several sampling events over a period of varying conditions may be needed to fully evaluate the potential for vapors to enter a building.

The purpose of this health consultation is to determine whether some members of the Settlers Grove community may have been harmed by exposure to site-related contaminants from vapor intrusion, and what public health actions need to be taken to reduce harmful exposures.

Background

Site Description

SRG Global, Inc. (SRG) is located at 10116 Industrial Boulevard, Covington, Newton County, Georgia. The Site property consists of approximately 13 acres, and includes approximately 200,000 square-feet of manufacturing and office space. The site also contains a covered drum storage area and a pole barn that is used for exterior storage of packing and shipping materials. Surrounding the building and storage areas are asphalt and concrete covered parking and access areas, landscaped by grass and low-lying shrub areas. The site is partially enclosed by 3,720 linear feet of chain-link fencing equipped with a locked gate that is left unlocked during business hours. Access to the facility is from the north, via Industrial Boulevard and through three driveways located on Hazelbrand Road [BVNA 2014a].

A sedimentation pond (now overgrown with vegetation) is located in the center of the eastern boundary of the Site, adjacent to Hazelbrand Road (Figure 1). The pond is a permitted storm water management facility that receives storm water runoff from the SRG facility. Also, a small unnamed creek that originates northwest and upgradient of the Site is piped into the pond. The creek resumes southeast of the Site and Hazelbrand Road and flows southeast, parallel with the eastern boundary of the Settlers Grove subdivision, and ultimately to the Alcovy River.

Figure 1: Aerial photo of the SRG Global manufacturing plant (top of photo) and the Settlers Grove subdivision southeast of the manufacturing plant. The red arrow points to the sedimentation pond, which can be seen on the eastern boundary of the manufacturing building adjacent to Hazelbrand Road. The sedimentation pond is overgrown with vegetation. The entrance into the Settlers Grove Subdivision on Waterford Road can also be seen directly across the southern-most section of the sedimentation pond.

Adjacent properties include commercial/industrial businesses to the north and southwest; commercial properties to the east; private residences to the south and southeast; and light industrial buildings to the southwest. The southeastern side of SRG Global is bounded by Hazelbrand Road, which separates the site from the adjacent community of Settlers Grove. An elementary school and a child daycare center are located approximately 650 feet northeast of the site. The school, daycare center, and surrounding residences and commercial/industrial properties, including the Site are connected to the municipal and sanitary sewage services. The closest potable water well on record is located approximately 2.1 miles southeast of the site [BVNA 2014a].

Surface elevations on-site range from about 790-794 feet (ft) above mean sea level (msl) in the northern and western portions of the SRG property to about 781-782 ft above msl on the eastern and southern portions of the SRG property. The land surface drops off to about 700 ft above msl at the Alcovy River located approximately two miles southeast of SRG [BVNA 2014a]. Surficial

aquifer groundwater flow from the Site trends eastward toward the unnamed creek located just northeast of Waterford Road [BVNA 2014b].

History

The subject, undeveloped property was purchased by Automotive Moulding Company, Inc. of Warren, Michigan from the Covington Businessman's Association in 1968. Automotive Moulding Company formally changed its name to Guardian Automotive Corporation (d/b/a SRG Global Covington) in 2002 [BVNA 2014a]. The Covington facility began operations in 1969 and has been engaged in the manufacture of automotive trim parts ever since. Specific manufacturing operations include stamping and rolling of aluminum and stainless steel trim, co-extrusion of plastic and aluminum trim, and injection molding of polyvinyl chloride and thermoplastic olefin. Aluminum anodizing operations were performed at the facility until approximately 2006.

Trichloroethene (TCE) is understood to have been used at the facility as a degreasing solvent from 1969 until the mid to late 1970's, and as an ingredient in products that were used to clean and maintain molds as part of the injection molding operation from approximately the 1990's until 2011 [BVNA 2014a].

Site Investigative Chronology

In 1996, Guardian Automotive Corporation conducted a limited soil investigation outside the former anodizing operation area. Two soil samples from each of four borings were collected and analyzed for volatile organic compounds (VOCs), semi-volatile organic compounds (SVOCs, and Priority Pollutant Metals. All metal samples were reported as below the "Type I Georgia background and the Georgia reporting levels. Three VOCs were detected (tetrachloroethene (also known as perchloroethene, or PCE)), 1,1,1-trichloroethane (TCA), and 1,1-dichloroethene (1,1-DCE), and each were "below the Georgia cleanup criteria and the Georgia Reporting level" [BVNA 2014a]. No detections of SVOCs were reported [BVNA 2014a].

At the request of a potential equity investor in November 2012, soil and groundwater sampling was performed as part of due diligence activities. Groundwater sample results from five temporary monitoring wells that were installed showed the presence of dissolved TCE ranging from non-detect to 3,290 micrograms per liter (µg/L) [BVNA 2014a].

In January 2013, five permanent groundwater monitoring wells were installed on SRG property. Chlorinated VOCs were detected in all five groundwater monitoring wells at concentrations ranging from 6.7 to 2,650 µg/L. From April to September 2013, another 34 groundwater monitoring wells were installed on SRG property. In addition, soil sampling was also conducted during this period [BVNA 2014a]. The purpose of these efforts is to determine the potential source areas where chlorinated solvents were released at the Site that led to chlorinated VOC impacts in groundwater at the Site and to delineate vertical and horizontal extents of contamination.

Sample results from these investigations indicated a need to assess the potential for migration of dissolved chlorinated VOCs downgradient of the Site toward the Settlers Grove Subdivision. Based on knowledge of the surficial aquifer groundwater table elevations, local topography, and

geomorphology, SRG re-directed onsite sourcing and delineation efforts and initiated an assessment designed to identify locations in the shallow aquifer that could serve as a potential source of VOCs that could potentially migrate via vapor intrusion into residential structures.

Settlers Grove is a small community made up of approximately 199 single-family homes. Approximately 50 percent of the homes are rented out to tenants. The population is approximately 506 and is composed of 76 percent African Americans, 17 percent white, 6 percent Hispanic/Latinos, and 1 percent mixed races. Approximately 30 percent of the residents are under 18 years of age [U.S. Census 2010]. In addition, using 2010 U.S. Census data, ATSDR estimated that more than 20 women of child-bearing age (aged 15 to 44) live in the Settlers Grove community (Appendix A).

Vapor Intrusion Investigation

To expedite the investigation of offsite groundwater contamination, sampling was conducted by SRG Global in the Settlers Grove subdivision using direct-push (Geoprobe™) borings installed in Newton County Rights of Way (ROW), rather than through permanent groundwater monitoring wells that would require private property access and longer drilling times. The initial Geoprobe™ offsite investigation was performed October 14-17, 2013, involving the installation of nine borings, followed by three additional borings that were installed in the ROW on November 21, 2013 [BVNA 2014b]. The purpose of this expedited investigation was to determine a qualitative footprint of off-site contamination so that indoor air sampling could be targeted to the residences located inside the determined footprint.

Geoprobe™ boring locations were chosen based on the topography of the Settlers Grove subdivision, surficial aquifer flow direction, and depth to bedrock as shown in Figure 2.

Soil gas samples were collected from eight boreholes (MIP01-MIP08 [shown in Figure 2]) to determine if dissolved VOCs were volatilizing vertically through the soil overburden and providing a potential for vapor intrusion. Soil gas sampling depths ranged from five to 15 feet below ground surface. Concurrent with the soil gas sampling, four ambient air samples were also collected at various locations at the intersections of ROW roadways (at the intersections of Waterford Road and Hazelbrand Road, Waterford Road and Settlers Grove Road, Waterford Road and Hidden Branches Way, and approximately 300 feet southwest of Waterford Road on Settlers Grove Road) [BVNA 2014b]. No residential sub-slab soil gas samples were collected.

Also concurrent with the offsite soil gas investigation, soil samples were collected from 11 soil borings within the Settlers Grove ROW. Soil sampling depths were biased toward depths where elevated VOC readings were observed when direct-push borings were installed and/or just above the top of the uppermost saturated zone. Two soil samples were generally collected from each soil boring location, with the first soil sample being taken within the uppermost 0-5 feet interval. In the absence of an elevated VOC reading, the second soil sample was collected from the five feet interval just above the uppermost saturated zone [BVNA 2014b].

Groundwater samples were also collected in the Newton County ROW within the Settlers Grove subdivision. The purpose of the groundwater investigation was to provide the information necessary to identify areas with the potential for vapor intrusion into residences based on the

residences being situated above the dissolved VOC plume and to assist in the delineation of the extent of regulated chemicals found in offsite groundwater. Twenty eight groundwater grab samples were collected from up to three depths from 11 direct-push borings to help evaluate horizontal and vertical distribution of dissolved VOCs in the surficial aquifer [BVNA 2014b].

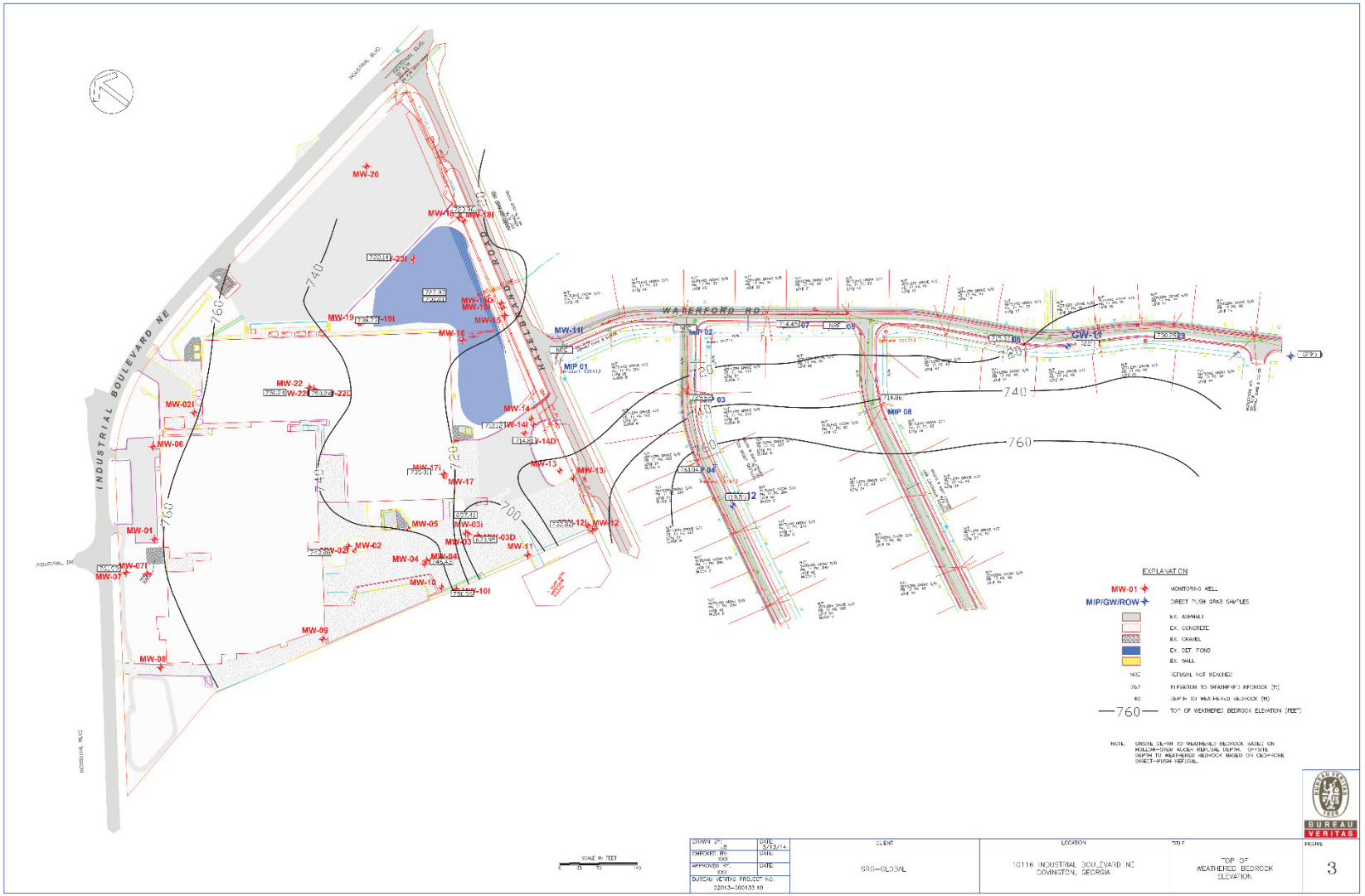

Figure 2: Contoured lines show the elevation at the top of bedrock encountered. The contoured lines also give a general overview of the topography in the investigation area where the lowest surface elevation of the Settlers Grove subdivision runs along Waterford Road and the subdivision slopes upwards south and southwest of Waterford Road.

Chemicals found in groundwater and included in the offsite indoor air evaluation were the seven VOCs detected in shallow groundwater beneath the Settlers Grove community: 1,1-dichloroethene (1,1-DCE), *cis*-1,2-DCE, *trans*-1,2-DCE, 1,1,1-trichloroethane (1,1,1,-TCA), tetrachloroethene (PCE), Trichloroethene (TCE), and vinyl chloride (VC) [BVNA 2014b].

Concerted efforts were implemented to contact all residents within 100 feet of the footprint of the groundwater plume non-detect line (Figure 2). This 100-foot buffer was selected based on the current U.S. Environmental Protection Agency (EPA) draft vapor intrusion guidance [EPA 2013a]. These residents were offered indoor air sampling and the installation of vapor mitigation systems at no cost to the residents [BVNA 2014b]. Vapor mitigation system installation was offered to every residence within the identified area regardless of authorization for sampling or actual testing results.

A total of 48 occupied homes were identified within approximately 100 feet of the detection limit of dissolved VOCs detected in groundwater. All 48 residences and/or owners of the subject homes were contacted in person, by phone, and/or by mail and provided with information describing the groundwater conditions and the proposed indoor air sampling program. Over a period of 8 weeks (from December 2013 to February 2014) SRG representatives contacted residents/owners to initiate, conduct, and communicate the results of indoor air sampling. As of March 2014 (winter sampling round), air samples were collected at 16 residences, and three home owners had venting systems installed [BVNA 2014b] as shown in Figure 3. At the end of August 2014 (summer sampling round), air samples were collected from a total of 23 residences (48 percent of homes within the determined footprint), and a total of 10 residences (21 percent of homes within the determined footprint) had venting systems installed. Three more venting systems were installed in the fall of 2014.

Discussion

Exposure Pathway

When a hazardous substance is released to the environment, people are not always exposed to it. Exposure happens when people breathe, eat, drink, or make skin contact with a contaminant. Several factors determine the type and severity of health effects associated with exposure to contaminants. Such factors include exposure concentration, frequency and duration of exposure, route of exposure, and cumulative exposures (i.e., the combination of contaminants and routes). Once exposure takes place, individual characteristics–such as age, sex, nutritional status, genetics, lifestyle, and health status–influence how that person absorbs, distributes, metabolizes, and excretes the contaminant. These characteristics, together with the exposure factors discussed above and the toxicological effects of the substance, determine health effects that may result.

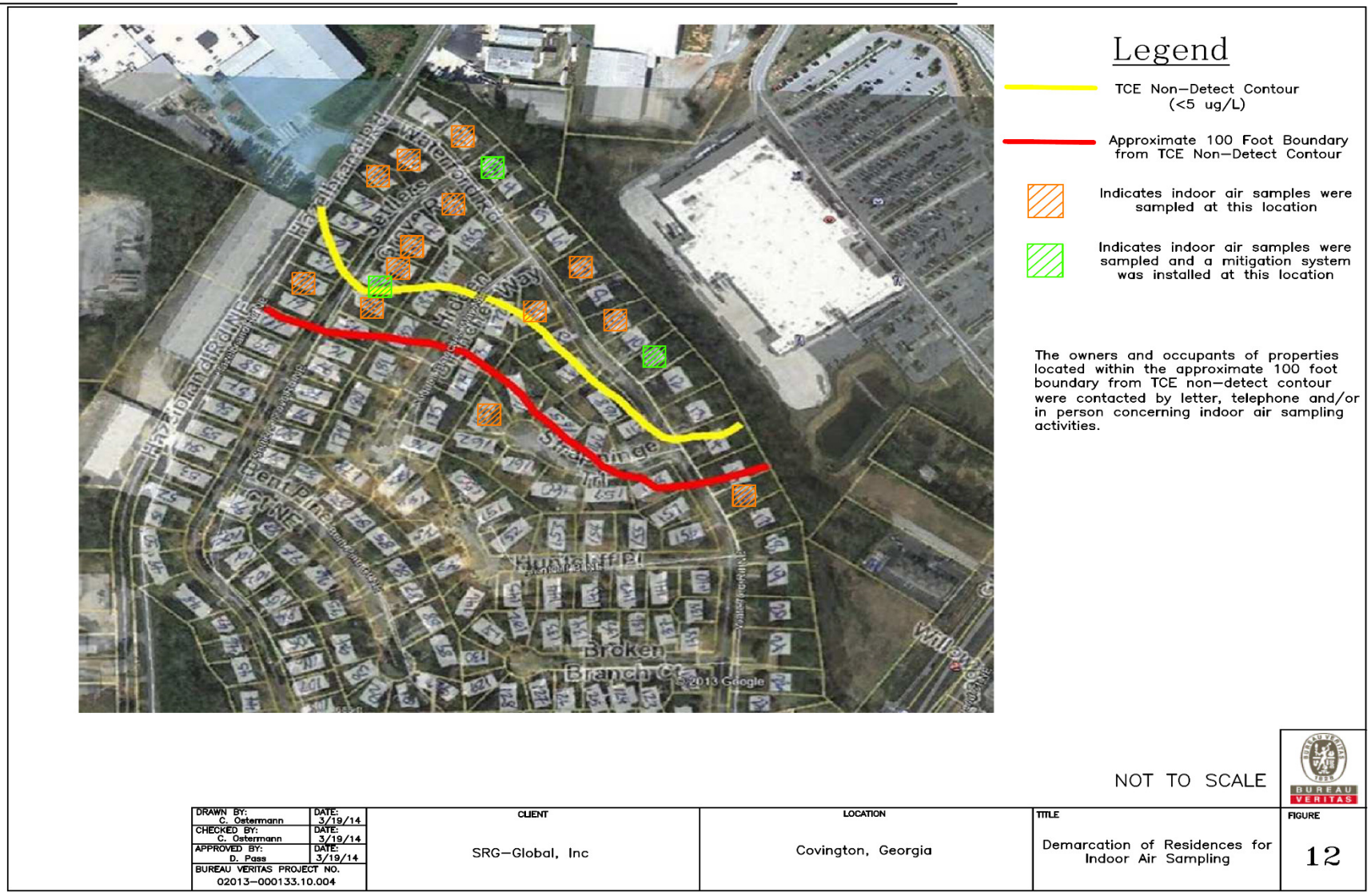

Figure 3: (As of March 2014) Settlers Grove residences sampled for indoor air include residences numbered 2, 3, 7, 9, 11, 16, 62, 67, 68, 69, 71, 72, 73, 74, 163, and 171. Vapor mitigation systems were also installed in residences numbered 3, 11, and 73.

In order for any contaminant to be a health concern, the contaminant must be present at a high enough concentration to cause potential harm, and there must be a completed route of exposure to people. A completed exposure pathway consists of five principle elements: a source of contamination, transport through an environmental medium, a point of exposure, a route of human exposure, and a receptor population. Completed exposure pathways indicate that exposure to a contaminant has occurred in the past, is presently occurring, or will occur in the future. It should be noted that the identification of an exposure pathway does not imply that health effects will occur. Exposures may, or may not be substantive. Thus, even if exposure has occurred, human health effects may not necessarily result.

In general, people can be exposed to contaminants through ingesting soil and food, drinking water, inhaling vapors and dust, and by skin contact. For this health consultation, resident-specific indoor air VOC concentrations were considered in evaluating the completed inhalation exposure pathway to these compounds by breathing indoor air. We do not expect other exposure to contaminated groundwater from drinking or bathing because the residents are on public water.

Environmental Data

The purpose of the initial soil gas, ambient air, soil, and groundwater sampling was to establish a qualitative footprint of off-site contamination so that indoor air sampling could be targeted to the residences located inside the determined footprint. All environmental sampling methods and sampling results from the contaminant footprint determination for soil gas, ambient air, soil, and groundwater, along with relevant geological descriptions and figures are shown in Appendix B.

Because the focus of this health consultation is to determine whether some members of the Settlers Grove community may be harmed by exposure to site-related contaminants from vapor intrusion, the remainder of this document will focus on indoor air.

Indoor Air Sampling Protocol

The residences located within the indoor investigation area are one- or two-story, or split-level single-family homes. Some residences have a slab-on-grade foundation, and others have a crawl space beneath the foundation. The split-level homes were constructed with portions of the floor below ground surface.

Prior to conducting indoor air sampling, occupants were interviewed and a questionnaire was completed that documented conditions that could affect measurements of VOCs in the residences. Questions were asked on the type of home construction, type of heating and cooling systems, presence of chemicals, personal practices, and household products used or stored that may contain VOCs. In addition, a portable photoionization detector (PID) was used to measure total VOCs at indoor sampling locations [ToxStrategies 2014, BVNA 2014b].

For each residence that was sampled, indoor, crawl, and outdoor air samples were collected over an approximate 24-hour period. At least one outdoor air sample was collected on each day that indoor air samples were collected. Outdoor air sample collection was started prior to collecting indoor air samples. A minimum of two primary indoor air samples were collected at each residence; one sample was collected from a main living area, and the other was collected from an

area with potential preferential pathways (e.g. the kitchen or a bathroom). Samples were collected on the lowest occupied level of any multi-story residence. Outdoor and indoor air samples were collected in a manner representative of the breathing zone (at a level 3 to 5 feet above the floor). If the house had a crawl space, at least one crawl space sample was collected. One field duplicate sample was collected during each day that indoor air sampling was conducted [BVNA 2014b].

All indoor, crawl space and outdoor air samples were collected in 6-liter Summa™ canisters, fitted with laboratory-supplied flow controllers (24-hour flow controllers). All 6-liter Summa™ canisters were individually certified by the analytical laboratory to be clean and free of EPA Method TO-15 analytes prior to sampling [BVNA 2014b]. Collected samples were analyzed by gas chromatography/mass spectrometry using EPA Method TO-15 [EPA 1999].

Indoor Air Sampling Results

Laboratory analytical data were submitted to Environmental Standards, Inc. (ESI) for independent data validation [ESI 2014]. Validated analytical results were compared to health-protective screening levels that were based on EPA's vapor intrusion screening level calculator [EPA 2014] results for the seven chemicals. For this health consultation, sample results were screened with ATSDR health-based comparison values (CVs) when an ATSDR CV was available for a particular chemical.

Sample results in the form of a letter and table were provided to each resident in person. Absentee owners of residences that elected to have sampling performed were provided a copy of the letter and table by mail.

Table 1 summarizes all indoor air sample results collected as of November 2014.

Table 1: Indoor air sampling results from Settlers Grove residential properties overlying the SRG Global VOC plume. Sampling occurred in December 2013 through August 2014.

Chemical	Number of Residences Sampled	Number of Residences with Detections	Concentration Range µg/m^3	Health-Protective Comparison Value* µg/m^3	Number of Residences Exceeding a Comparison Value
1,1-Dichloroethane	23	0	ND	7.7[a]	0
1,1-Dichloroethene	23	1	ND-0.38	79[b]	0
cis-1,2-Dichloroethene	23	5	ND-0.23	790[b]	0
trans-1,2-Dichloroethene	23	1	ND-0.047	--	0
Tetrachloroethene	23	23	ND-920	270[c], 3.8[d]	2
Trichloroethene	23	20	ND-11	2.0[c], 0.24[d]	13

µg/m^3: micrograms per cubic meter of air
ND: not detected at laboratory method detection limit
[a] EPA Soil Gas Vapor Intrusion Screening Level (VISL) divided by a slab attenuation factor of 10.

[b] EMEG/MRL: ATSDR intermediate environmental media evaluation guide/minimal risk level for air (March 2013).
[c] EMEG/MRL: ATSDR chronic environmental media evaluation guide/minimal risk level for air (March 2013).
[d] CREG: ATSDR Cancer Risk Evaluation Guideline (March 2013).

Approximately 60 percent (or 14) of the residential properties sampled had VOC concentrations detected in indoor air that were above health-protective comparison values (screening levels) and were often consistent with levels detected in outdoor air. The majority of these residences exceeded a CV based on an excess cancer risk of one in a million (CREG), and people living in these residences will be evaluated for excess cancer risk later in this document. Two of the residences sampled exceeded a CV for non-cancer health effects and will also be evaluated further in this document.

TCE was detected above a non-cancer comparison value in indoor air at one property (House #68) at levels ranging from 8.3 to 11 $\mu g/m^3$; above both the outdoor air ambient concentration (0.094 $\mu g/m^3$) and the health-protective screening level of 2.0 $\mu g/m^3$ (Appendix B, Table B.5). For reference, House #68 is located in close proximity to the groundwater migration pathway along the axis of the highest-observed dissolved-phase chlorinated solvents [BVNA 2014b]. The highest TCE sampling result was detected in the basement TV room of this residence. TCE was also detected above the indoor air screening level in the living room and kitchen of this residence.

After a venting system was installed at House #68 in May 2014, post-mitigation indoor air samples were collected on June 30, 2014. Two of the three indoor air samples were collected from locations similar to the initial indoor air samples. The initial indoor air sample location in the kitchen was moved to the basement laundry room to be closer to the preferential pathways and the sub-slab venting system. Sample results showed that TCE was detected below non-cancer indoor air screening levels in all the rooms sampled[1]. The TV room sample result was 0.72 $\mu g/m^3$, the laundry room sample result was 0.56 $\mu g/m^3$, and the living room sample result was 0. 66 $\mu g/m^3$. Two outdoor ambient air sample results averaged 0.19 $\mu g/m^3$ (Appendix B, Table B.6). However, all the indoor air sample results were above a health-protective screening level for cancer risk and will be further evaluated for cancer risk.

An uncharacteristic result was detected at one residence (House #171) during the initial winter sampling round that did not appear to be related to VOCs observed in the groundwater. At that residence, PCE was detected in the bathroom sample, at a concentration (530 $\mu g/m^3$) that exceeded both the health-based screening level (270 $\mu g/m^3$) and the outdoor air measurement (0.41 $\mu g/m^3$). PCE was detected at a low concentration (0.18 $\mu g/m^3$) in the indoor living room sample that was collected on the same day at the residence. The concentration of PCE measured in the living room was consistent with the level measured in the air outside the residence and lower than the health-based screening level. Additionally, PCE was not detected in either the crawl space air sample or the nearby grab groundwater sample. Collectively, these sample results indicate that the PCE detected in the bathroom sample was not likely related to vapor intrusion from groundwater.

[1] Letter transmittal from SRG Global to House #68 homeowner, dated August 18, 2014.

Similar to the results observed during the winter sampling round, PCE was also detected in House #171 during the summer sampling round. [2] At this residence, PCE was detected in the bathroom at an average concentration of 890 µg/m^3, and was also detected in the living room at a concentration of 820 µg/m^3. However, unlike the winter sampling results, the PCE concentration in the crawl space was elevated (33 µg/m^3) above the concentration of 0.24 µg/m^3 measured in ambient air (Table B.6). Based on other data points, including the lower concentration of PCE in the crawl space, SRG Global believes that the PCE measured at this residence is not related to vapor intrusion. Moreover, a potentially relevant activity was completed in this residence prior to the collection of the second round of indoor air samples. Potential sources of PCE in the residence may include the recent dry cleaning of clothes and carpet cleaning performed the day before the sampling[3]. However, one potential source of PCE that has not been considered is sewer gas from a cracked sewer line that may extend into the surficial aquifer [Pennell 2013].

Evaluation Process

A two-stage evaluation process was used in the assessment of indoor air data. The first step was to review available sampling data and to select contaminants that warrant further evaluation, based on the potential for exposure to these contaminants to result in adverse health effects. DPH examines the types and concentrations of contaminants of concern, which are then screened with comparison values generally established by ATSDR and EPA. Comparison Values (CVs) are concentrations of a contaminant that can reasonably (and conservatively) be regarded as harmless to human health, assuming default conditions of exposure. CVs include ample uncertainty factors to ensure protection of sensitive populations. Because CVs do not represent thresholds of toxicity, exposure to contaminant concentrations above CVs will not necessarily lead to adverse health effects [ATSDR 2005]. DPH then considers how people may come into contact with the contaminants. Because the level of exposure depends on the route, frequency, and duration of exposure and the concentration of the contaminants, this exposure information is essential to determine if a public health hazard exists.

The next step in the evaluation process involves an in-depth health-effects evaluation of the contaminants detected in the site media (in this case, indoor air) above their respective CVs. The primary focus of this effort is to evaluate the potential for the contaminant(s) to produce cancer and non-cancer health effects as a result of human exposure.

Contaminants of concern were determined by employing a screening process. Health-based screening values used by DPH include ATSDR environmental media evaluation guides for air (EMEGs), EPA Regional Screening Levels (RSLs) for residential air and ATSDR cancer risk evaluation guides (CREGs) for air. CVs such as the EMEG, RSL and CREG offer a high degree of protection and assurance that people are unlikely to be harmed by contaminants in the environment. For chemicals that cause cancer, the CREGs represent levels that are calculated to increase the estimated risk of cancer by about one additional cancer in a million people exposed.

[2] Letter transmittal from SRG Global to House #171 homeowner, dated October 9, 2014.
[3] SRG Global contacted the carpet cleaning company used by the homeowner (A Clean and Tidy Cleaner in Conyers, GA), but they would not identify the "cleaning agents" they use.

From the 23 residences in the Settlers Grove subdivision that were sampled for indoor air, two residences warranted further evaluation for non-cancer health effects. TCE levels measured in House #68 were above the ATSDR chronic EMEG of 2.0 µg/m^3 for TCE in residential air. PCE levels measured in House #171 were above the ATSDR chronic EMEG of 270 µg/m^3 for PCE in residential air.

Chemicals of Concern

Trichloroethene (TCE)
Trichloroethene (also known as trichloroethylene) is used mainly as a degreaser for metal parts. It is also used as a solvent in other ways and is used to make other chemicals. TCE can also be found in some household products, including typewriter correction fluid, paint removers, adhesives, and spot removers. The biggest source of TCE in the environment is evaporation from factories that use it. Once TCE is in the air, about half will be broken down within a week. If released to the soil, TCE generally does not break down in the soil but migrates into groundwater where it does break down, but at a very slow rate [ATSDR 1997].

People are usually exposed to TCE from breathing air or drinking water containing TCE. If you breathe the chemical, about half the amount you breathe will get into your bloodstream and organs. You will exhale the rest. If TCE comes into contact with your skin, some of it can enter your body, although not as easily as when you breathe or swallow it [ATSDR 1997].

Once in your blood, your liver changes much of the TCE into other chemicals. The majority of these breakdown products leave your body in the urine within a day. You will also quickly breathe out much of the TCE in your bloodstream. Some of the TCE or its breakdown products can be stored in body fat for a brief period, and thus may build up in your body if exposure continues [ATSDR 1997a].

Tetrachloroethene (PCE)
Tetrachloroethene (also known as tetrachloroethylene or perchloroethylene) is a synthetic chemical that 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 mostly by evaporating into the air during use. It can also get into water supplies and soil during disposal of sewage sludge and factory waste and when leaking from underground storage tanks. It can stay in the air for several months before it is broken down into other chemicals or is brought back down to soil and water from rain [ATSDR 1997b].

People can be exposed to PCE from environmental and occupational sources and from consumer products. The chemical is found most frequently in air, and less often, in water. Consumer products that may contain PCE includes water repellants, silicone lubricants, fabric softeners, spot removers, adhesives, and wood cleaners. When you bring clothes home from the dry cleaners, the clothes may release small amounts of PCE into the air. PCE can also be found in breast milk of mothers who have been exposed to the chemical [ATSDR 1997b].

PCE can enter your body when you breathe air, drink water, or eat food containing it. Most PCE leaves your body when you exhale. A small amount of PCE is changed by your body (especially your liver) into other chemicals that are removed from your body in urine. Most of the changed

PCE leaves your body in a few days. Some of the PCE you are exposed to is found in your blood and other tissues, especially body fat. PCE stored in body fat can stay in your body for several days or weeks before it is eliminated [ATSDR 1997b].

Adverse Health Effects and Cancer Risk Assessment

Trichloroethene Noncancer Health Effects

The primary health concerns for impacted residents living in the Settlers Grove subdivision are those associated with chronic inhalation of TCE that has migrated into their homes via vapor intrusion. ATSDR and EPA have concluded that TCE poses a potential human health hazard to the central nervous system, the immune system, the kidney, the liver, the male reproductive system, and the developing fetus [ATSDR 1997a; EPA 2011]. The immune system and the developing fetus are most sensitive to the toxic effects of TCE, as reductions in thymus weight [Kiel 2009] and the development of fetal malformations during a three week window of fetal heart development [Johnson 2003] are the earliest observed health effects following low level exposures. Although these studies were conducted in rats and mice exposed to TCE in drinking water, physiological-based pharmacokinetic (PBPK) modeling was used to extrapolate oral dose in animals to human equivalent concentrations (HECs) in air. The EPA RSL for TCE in residential air (2.1 µg/m^3) is based on the EPA Reference Concentration (RfC) derived from the Keil and Johnson studies. This inhalation RfC was conservatively chosen because it is below the candidate RfCs derived from the lowest concentrations associated with adverse health effects from TCE inhalation studies. The RfC was adopted by ATSDR as the both the intermediate and chronic inhalation chronic minimal risk level (MRL) for TCE exposure [ATSDR 2013].

One home in the Settlers Grove subdivision had pre-mitigation indoor air concentrations above the EPA RSL for TCE. The HEC for 1 percent extra risk of fetal cardiac malformations is 21 µg/m^3, while the HEC for decreased thymus weight is 190 µg/m^3. For fetal heart malformations, EPA used an uncertainty factor of 10x to account for human variation and species differences. For decreased thymus weight, EPA used an uncertainty factor of 100x to account for the use of the lowest observed adverse effects level (LOAEL), as well as human variation and species differences. The midpoint between the candidate RfC for the two critical effects (rounding to one significant digit) was used to derive the TCE RfC of 2.0 µg/m^3. The highest indoor air concentration detected in House #68 is 5.5 times above the RfC for chronic inhalation of TCE and approaches the HEC for fetal cardiac malformation. Exposure of pregnant women to TCE levels above the EPA RSL does not mean that fetal heart development will be impaired. However, breathing air exceeding these levels of TCE begins to introduce a small amount of risk to proper fetal development and should be avoided. Although the highest indoor air concentration detected at this residence is approximately 17 times lower than the HEC of 190 µg/m^3 for decreased thymus weight observed in mice, a small amount of risk to impacts on immune system may exist in immunosuppressed populations.

Tetrachloroethene Noncancer Health Effects

The primary health concerns for residents impacted by PCE are those associated with chronic inhalation of PCE. ATSDR and EPA have concluded that PCE poses a potential human health hazard to the nervous system [ATSDR 1997b, EPA 2012b]. In closed, poorly ventilated areas,

when PCE concentrations are high (mean PCE concentration of approximately 135,650 µg/m^3), acute and chronic exposures can cause dizziness, sleepiness, confusion, nausea, headache, difficulty in speaking and walking, and unconsciousness [Cai 1991]. The nervous system is most sensitive to the toxic effects of PCE, and occupationally-exposed adults (PCE concentration ranging from approximately 76,000 to 277,000 µg/m^3) performed below expectation on tasks assessing memory, motor skills (reaction times), visual and executive function deficits [Echeverria 1995] following low-level exposure for one year or more. Decrements in color vision were also reported among occupationally-exposed workers following long-term exposure (approximately nine years) to a time-weighted average PCE concentration of approximately 40,700 µg/m^3 [Cavalleri 1994]. The EPA RSL for PCE in residential air (42 µg/m^3) is based on the Reference Concentration (RfC) derived from the Echeverria and Cavalleri studies.

Although the source of the PCE has not been definitely determined, one home in the Settlers Grove subdivision had indoor air concentrations above the EPA RSL for PCE. Candidate RfCs were derived by dividing the LOAEL of 15,000 µg/m^3 for changes in color vision in the Cavalleri study, and the LOAEL of 56,000 µg/m^3 for cognitive and reaction time changes in the Echeverria study by an uncertainty factor of 1000x (comprised of 10x for human variability, 10x for extrapolation from an LOAEL, and 10x for database uncertainty). The candidates RfCs from these two studies span a range from 15 µg/m^3 to 56 µg/m^3. EPA used the midpoint of this range to derive the RfC for PCE of 40 µg/m^3 (rounded to one significant figure). The highest indoor air concentration detected in House #171 is 23 times above the RfC for chronic inhalation of PCE. The RfC is an estimate of continuous inhalation exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime of exposure. The highest PCE concentration measured in House #171 is approximately 43 times below the midpoint of the LOAEL ranges observed in the referenced studies and well below the most sensitive effect level . Thus, DPH concludes that residents exposed to PCE at levels found in indoor air at this residence are exposed to levels below those that may increase the risk of adverse non-cancer health effects.

Interactions of Chemicals Found in Indoor Air

Trichloroethene (TCE), and tetrachloroethene (PCE) frequently occur together in water samples collected from hazardous waste sites. These chemicals occur more frequently, along with 1,1,1-trichloroethane and 1,1-dichloroethane, than other VOCs in contaminated groundwater [ATSDR 2004]. A potential limitation of this health consultation is that each chemical found above an EPA RSL, RfC or MRL is treated individually and conclusions are derived from individual components. It is not entirely accurate to assess risk without considering joint toxicity of chemicals that have similar toxicity endpoints.

To carry out exposure-based assessments of possible health hazards associated with inhalation exposure scenarios involving indoor air exposures to mixtures of trichloroethene and tetrachloroethene, component-based approaches that assume additive joint action of the components are recommended for exposure based screening assessments [ATSDR 2004]. Applying the additivity assumption appears to be in the interest of public health since the components have several shared toxicity targets. This approach is recommended because of the lack of studies that examine relevant endpoints and describe dose-response relationships for inhalation exposures that contain mixtures of the components of concern. For noncancer

endpoints (nervous system effects, liver or kidney effects), a target-organ toxicity dose modification of the hazard index (HI) approach (Appendix C) is recommended by ATSDR given that a wide range of overlapping toxicity targets can be affected by the components.

Because PCE and TCE have similar metabolic pathways, evidence suggests that they may interfere with each other's metabolism in the body [Seiji 1989]. Occupational studies indicated that workers exposed to both PCE and TCE had lower levels of TCE metabolites in the urine than workers exposed only to TCE at about the same concentrations that occurred in the mixture. The metabolites of PCE and TCE are considered to be responsible for the chemical's toxicity to the liver and kidneys; however, it is unclear whether the parent compounds or their metabolites (particularly TCE's metabolites) have the greater impact on neurological effects. Overall, the available weight-of-evidence suggests that co-exposure of humans to tetrachloroethylene and trichloroethylene may inhibit the metabolism of trichloroethylene and thereby may inhibit carcinogenic and non-carcinogenic responses in the liver and kidney to trichloroethylene metabolites. ATSDR scientists concluded that PCE had a less-than-additive effect on TCE whereas TCE had an additive effect on PCE [ATSDR 2004].

However, DPH does not believe that the joint toxicity of these two components plays a major role in contributing to potential health hazards to residents occupying the two homes assessed in this health consultation. The reasons for this are based on using ATSDR's recommended approach to assess the joint toxicity of PCE and TCE in House #68 and #171; the TCE component contributes approximately 100 percent to the HI in House # 68, while the PCE component contributes approximately 99 percent to the HI in House #171.

Cancer Risks

In addition to noncancer toxicities associated with TCE exposure, long-term inhalation of TCE can also increase one's risk of developing certain cancers. The EPA recently released an extensive toxicological review of TCE, in which they reclassified it as "carcinogenic to humans by all routes of exposure" [EPA 2011]. The National Toxicology Program has also determined that TCE is "reasonably anticipated to be a human carcinogen". The most consistent and convincing evidence of an association between TCE exposure in humans and cancer is that for cancer of the kidney. However, there are also compelling links between TCE exposure and cancers of the lymphoid tissues (lymphoma) and liver [EPA 2011]. Please refer to Appendix D, Tables D.1 through D.5 for the estimated excess cancer risk calculations associated with exposure to the EPA RSL for residential indoor air and the average concentration of TCE detected in House #68.

The estimated lifetime excess cancer risk from TCE exposure to indoor air at the EPA RSL (2.1 $\mu g/m^3$) approaches the 1-in-100,000 excess lifetime cancer risk level (9.8×10^{-6}), which is considered low risk. DPH used two exposure durations to estimate cancer risk: 33 years and 12 years. A reasonable maximum exposure (RME) period of 33 years was used because it is the 95th percentile occupancy period, and a central tendency exposure (CTE) period of 12 years was used because that is the average period of homeownership in the United States. The estimated excess cancer risk from 33 years of exposure to the average level of TCE found in the indoor air of House #68 ranges from 2-in-100,000 (2.2×10^{-5} from birth to age 33) to 1.6-in-100,000 (1.6×10^{-5} (for adults being exposed for 33 years). The estimated excess cancer risk from 12 years of

exposure to the average level of TCE found in the indoor air of House #68 ranges from 1-in-100,000 (1.0×10^{-5} from birth to age 12) to 6-in-1,000,000 (5.9×10^{-6} for adults being exposed for 12 years). The cancer risk in both estimates is considered low. Before a venting system was installed at this residence, excess cancer risk approximately double the lifetime excess cancer risk associated with TCE exposure to the EPA RSL. Currently, post-mitigation the indoor air concentrations of TCE found in House #68 are greater than 10-fold less than pre-mitigation concentrations; thus, the estimated excess cancer risk is still low and has declined to approximately 1-in-1,000,000 (See Appendix D, Tables D6 through D.9).

Another residence (House #3) had levels of TCE detected in indoor air above the CREG in both pre-mitigation and post-mitigation sampling events. The estimated adult excess cancer risk from 33 years of exposure to the average level of TCE found in the indoor air of House #3 ranges from 1.3-in-1,000,000 (1.3×10^{-6}) to approximately 5-in-10,000000 (4.9×10^{-7}) for adults being exposed for 12 years. This range considers a cancer risk that is very low to non-existent. Excess cancer risk is also considered very low to non-existent from exposure to TCE after a venting system was installed in this residence and the adult excess cancer risk is similar to the excess cancer risk based on the EPA RSL. Post-mitigation adult excess cancer risk from exposure to TCE ranges from 1.1-in-1,000,000 (for 33 years of continuous exposure) to 4.1-in-10,000,000 (for 12 years of continuous exposure)

Long-term inhalation of PCE can also increase one's risk of developing certain cancers. The EPA considers PCE "likely to be carcinogenic to humans" by all routes of exposure. The National Toxicology Program has also determined that PCE is "reasonably anticipated to be a human carcinogen", and the International Agency for Research on Cancer (IARC) considers PCE to be "probably carcinogenic to humans". The most suggestive evidence of carcinogenicity was found in mice and rats for PCE –induced liver tumors [EPA 2012b]. PCE has also been shown to cause kidney tumors in male rats. Please refer to Appendix D, Table D.10 for the estimated excess cancer risk calculations associated with the RME (33 years and CTE (12 years) of exposure to the average concentration of PCE detected in House #171.

The lifetime excess cancer risk from PCE exposure to indoor air at the EPA RSL 42 $\mu g/m^3$) is approximately 1-in-100,000. We have no evidence that the levels of PCE in indoor air are from vapor intrusion from site-related contaminated groundwater. As stated previously, the high levels of PCE found during the two sampling events may be from other undetermined sources and may not represent conditions over a long-period of time. However, without more temporal sampling data, we cannot unequivocally determine the source. Thus, DPH chose to conservatively assume that under a worst case scenario, exposure has occurred for 33 years to estimate excess cancer risk. Under this assumption, the adult excess cancer risk for residents breathing indoor air in House #171 is approximately 7-in-100,000 (6.84×10^{-5}).

Estimated adult cancer risks using the RME and CTE for all other residences with concentrations of PCE and/or TCE, above a CREG are also shown in Appendix D, Table D.10. These estimated cancer risks range between approximately 7-in-100,000,000 to approximately 1-in-1,000,000.

It is important to note though, that approximately 50 percent of the residences located in the Settlers Grove subdivision are leased by tenants, who are more transitory than people in owner

occupied-homes. Thus, our estimated excess cancer risks represent a worst-case scenario and are likely to be much lower to some residents, especially those leasing their homes. To put cancer rates in though, according to the American Cancer Society, the lifetime risk in the U.S. that an individual will develop cancer from all causes is slightly less than 1-in-2 for men (50,000/100,000) and a little more than 1-in-3 for women (33,000/100,000) [American Cancer Society 2012].

Child Health Considerations

In communities faced with contamination of the water, soil, air, or food, ATSDR and DPH recognize that the unique vulnerabilities of infants and children demand special emphasis. Due to their immature and developing organs, infants and children are usually more susceptible to toxic substances than are adults. Children are more likely to be exposed because they play outdoors, and they often bring food into contaminated areas. They are also more likely to encounter dust, soil, and contaminated vapors close to the ground. Children are generally smaller than adults, which results in higher doses of chemical exposure because of their lower body weights relative to adults. In addition, the developing body systems of children can sustain permanent damage if toxic exposures occur during critical growth stages.

Although there are no direct, definitive links between PCE or TCE inhalation and an increased incidence of adverse health effects in children or fetuses, evidence from animal studies, together with limited information from human studies, strongly suggests that developmental and reproductive effects are of concern [ATSDR 2001, EPA 2011]. Studies show that PCE and TCE rapidly cross the placental barrier in both humans and animals, and can accumulate in the fetus. Because PCE and TCE are lipophilic, they have an affinity for fat and can be found in breast milk [ATSDR 1997b, ATSDR 2001], providing another potential source of exposure in breastfeeding infants. While a number of studies have examined acute PCE and TCE exposures in adults, similar studies in children and the effects of low-level chronic exposures typically seen in vapor intrusion cases are lacking. Also, age-dependent differences in in the absorption, distribution, metabolism, and excretion of xenobiotics[4] may also alter the susceptibility of children to PCE or TCE, compared to adults. In addition, to assess cancer risk in children for chemicals that have been determined to have a mutagenic mode-of-action for carcinogenesis, if appropriate chemical-specific data are not available, then the default age-dependent adjustment factors (ADAFs) should be applied when assessing excess cancer risk in children. These default ADAFs address the potential for differential potency associated with exposure during early life (less than 16 years of age). Data on the toxicokinetics of PCE and TCE in children are virtually nonexistent, making it difficult to predict potential differences in response between adults and children. Nonetheless, source mitigation, removal and public education directed at parents should be used to help prevent or minimize exposure to children and women of childbearing age.

[4] Xenobiotics are substances recognized as foreign in the body.

Conclusions

DPH evaluated past and current exposure to PCE and TCE from breathing indoor air at certain residences located in the Settlers Grove subdivision where indoor air sampling data was available. This evaluation included an assessment of exposure doses and estimated cancer risk from inhalation of contaminants present in indoor air. DPH reached the following three conclusions:

1. In the past, breathing trichloroethene (TCE) in House #68 via the vapor intrusion pathway, could have harmed people's health based on the available data Babies born to pregnant women intermediately exposed to TCE in early pregnancy at these levels may have been at risk for heart development problems. All residents exposed to TCE at pre-mitigation levels may have had a smaller risk of immune system impacts. Currently, post-mitigation sampling data show that these residents are no longer being exposed to concentrations of TCE in indoor air that may harm their health. However, even with a venting system in operation, indoor air samples remain above an Agency for Toxic Substances and Disease Registry (ATSDR) Cancer Risk Evaluation Guide (CREG), but even so, there is no measurable excess cancer risk from this exposure. This also applies to residents occupying House #3. Nonetheless, a low-level risk from vapor intrusion will exist until contamination in groundwater is sufficiently reduced or removed.

2. The residents in House #171 are breathing PCE (although the source is not known, PCE was not found in shallow groundwater at sampling locations nearest this residence) at levels below those that may increase the risk of adverse non-cancer health effects from this exposure. However, if the concentration of PCE found in both sampling events continues to be high, a slight increase in excess cancer risk from this exposure is expected; however, this risk is still low.

3. Long-term exposures and potential harm to health are uncertain in homes without mitigation systems, because temporal variability has not been evaluated and some homes did not authorize vapor intrusion investigation

Recommendations

DPH recommends SRG Global:

1. Under EPD oversight, continue to take steps to interrupt the vapor intrusion pathway for affected residences in the Settlers Grove subdivision. These steps include the installation of venting systems to mitigate vapor intrusion and to ensure proper maintenance and effectiveness of the venting systems until the VOC source is removed and/or degraded.

2. Sample the indoor air of House #171 in the near future during a known period when dry-cleaned clothes have not been brought into the residence, and the carpets have not been cleaned to further assess whether vapor intrusion may be contributing PCE to the indoor

air. In addition, one potential source of PCE that has not been considered is sewer gas from a cracked sewer line that may extend into the surficial aquifer.

3. In order to protect community health and well-being, DPH has recommended that SRG Global under EPD oversight continue to take steps to identify homes with vapor intrusion and interrupt the vapor intrusion pathway for affected residences in the Settlers Grove subdivision. These steps include sampling and the installation of venting systems to mitigate vapor intrusion and to ensure proper maintenance and effectiveness of the venting systems until the VOC source is removed and/or degraded.

Public Health Action Plan

DPH will:

1. Distribute this health consultation and/or a fact sheet summarizing our findings to residents living within the footprint of the VOC plume and ensure that health education reaches those residents affected by vapor intrusion in the Settlers Grove subdivision.

2. Review the information and take appropriate actions as additional data become available.

3. If PCE concentrations in House #171 remain high, yet the source remains unidentified, DPH will provide the residents with chemical specific information regarding the use of PCE in consumer products and suggest ways to minimize services that utilize PCE in their processes.

4. Continue to respond to all requests for information and health concerns regarding the safety of breathing contaminated indoor air.

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Report Preparation

This Health Consultation was prepared by the Georgia Department of Public Health under a cooperative agreement with the federal Agency for Toxic Substances and Disease Registry (ATSDR). It is in accordance with the approved agency methods, policies, procedures existing at the date of publication. ATSDR has reviewed this document and concurs with its findings based on the information presented. ATSDR's approval of this document has been captured in an electronic database, and the approving agency reviewers are listed below.

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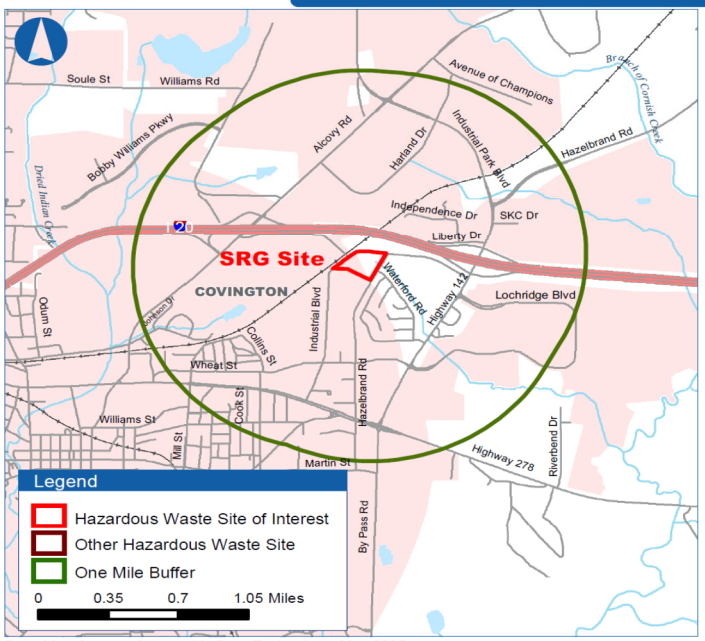
APPENDICES

Appendix A: Site Location and Demographic Map -

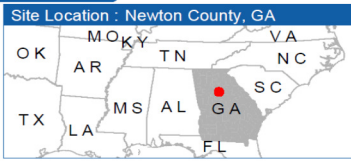
EPA Facility ID :
UNAVAILABLE

SRG SITE
NEWTON , GA



Base Map Source: Geographic Data Technology, May 2005.
Site Boundary Data Source: ATSDR Geospatial Research, Analysis, and Services Program, Current as of Generate Date (bottom left-hand corner).
Coordinate System (All Panels) : NAD_1983_StatePlane_Georgia_West_FIPS_1002_Feet

Site Location : Newton County, GA

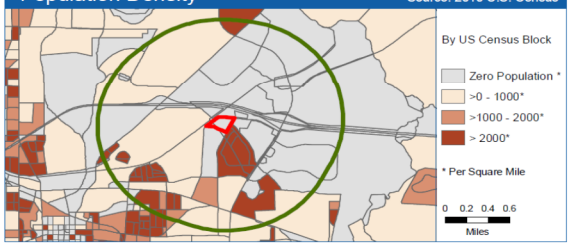


Demographic Statistics
Within One Mile of Site*

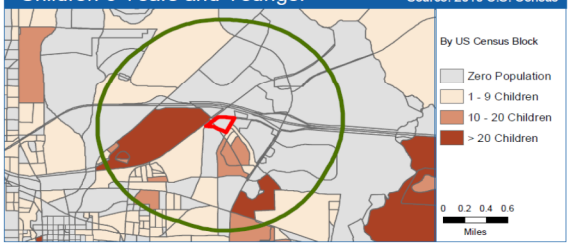
Total Population	2,180
White Alone	724
Black Alone	1,331
Am. Indian & Alaska Native Alone	4
Asian Alone	10
Native Hawaiian & Other Pacific Islander Alone	0
Some Other Race Alone	66
Two or More Races	45
Hispanic or Latino**	105
Children Aged 6 and Younger	345
Adults Aged 65 and Older	185
Females Aged 15 to 44	516
Total Housing Units	909

Demographics Statistics Source: 2010 U.S. Census
* Calculated using an area-proportion spatial analysis technique
** People who identify their origin as Hispanic or Latino may be of any race.

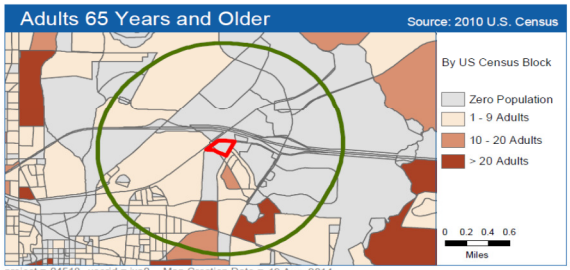
Population Density Source: 2010 U.S. Census



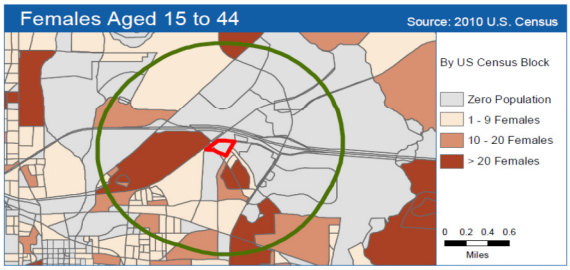
Children 6 Years and Younger Source: 2010 U.S. Census



Adults 65 Years and Older Source: 2010 U.S. Census



Females Aged 15 to 44 Source: 2010 U.S. Census



project = 04518 , userid = jxa0 , Map Creation Date = 19 Aug, 2014

GRASP

CDC ATSDR
Centers for Disease Control and Prevention
Agency for Toxic Substances and Disease Registry
Geospatial Research, Analysis & Services Program

Appendix B: Environmental Sampling Results

Soil Gas and Ambient Air Sampling Results

Soil gas and ambient air samples were collected in Summa™ canisters and analyzed for VOCs by EPA Method TO-15 [EPA 1999] at the locations shown in Figure B.1.

Analytical results for soil gas were compared to the EPA Target Soil Gas Concentration criteria for a target carcinogenic risk (TCR) of 10^{-5} (one in a hundred thousand) [EPA 2014]. None of the analytes sampled for exceeded the target soil gas concentrations. Soil gas sampling results are shown in Appendix B, Table B.1. It should be noted that the soil gas analytical results do not likely represent actual soil gas conditions of the subsurface and are suspect because incomplete ambient air purge volumes from the sampling equipment was not achieved resulting in the recovered soil gas sample being diluted with atmospheric air for every soil gas sample attempted [BVNA 2014b].

None of the analytes sampled for in ambient air exceeded the EPA Target Air Concentration criteria for a target carcinogenic risk (TCR) of 10^{-5}. Ambient air sampling results are shown in Table B.2.

Soil Sampling Results

Soil samples collected at locations shown in Figure B.1 were analyzed for VOCs by EPA Method 8260B [EPA 2007]. TCE was detected above the laboratory detection limit at only one location (MIP-02 near the intersection of Waterford Road and Settlers Grove Road) from the 0-2 feet interval at a concentration of 0.051 milligrams per kilogram (mg/kg) of soil, and at the 8-10 feet interval at a concentration of 0.05 mg/kg. No other VOC analytes were detected above the laboratory detection limits in the other soil samples collected. All soil sample results can be seen in Table B.3.

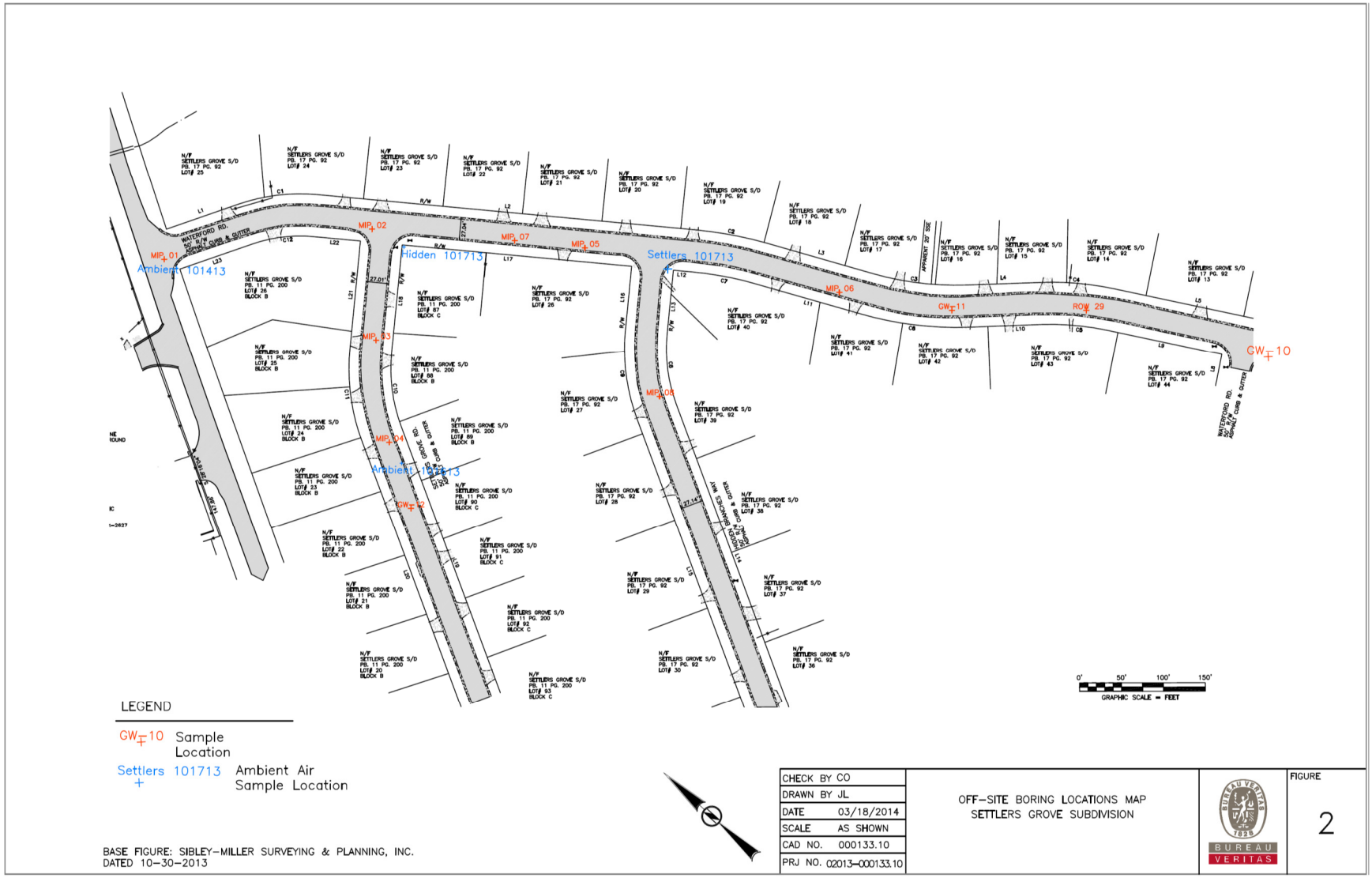


Figure B.1: Direct-push boring locations for soil gas, soil, and groundwater sample collection shown in red font. Ambient air sample locations shown in blue font.

Table B.1: Offsite Soil-Gas Sampling Results*

Contaminant	Target Indoor Air Concentration Using EPA's Vapor Intrusion Screening Level Calculator (Target Risk = 1 x 10^{-5} and Hazard Quotient = 1)	Units	Location (see Figure B1.1)							
			MIP01	MIP02	MIP03	MIP04	MIP05	MIP06	MIP07	MIP08
			Concentration							
Acetone	320,000	µg/m^3	45.4	54.6	39.2	44.7	83.1	31.6	52.5	39.7
Carbon disulfide	7,300	µg/m^3	ND(0.21)	ND(0.21)	ND(0.21)	ND(0.21)	4	ND(0.21)	ND(0.21)	ND(0.21)
Chloroform	11	µg/m^3	ND(0.36)	ND(0.36)	ND(0.36)	ND(0.36)	ND(0.36)	ND(0.36)	ND(0.36)	ND(0.36)
Chloromethane	940	µg/m^3	ND(0.27)	ND(0.27)	ND(0.27)	1.4 J	1.5J	1.4 J	1.6 J	ND(0.27)
1,1-Dichloroethene	2,100	µg/m^3	ND(0.33)	ND(0.33)	ND(0.33)	ND(0.33)	ND(0.33)	ND(0.33)	2.8 J	ND(0.33)
Dichlorodifluoromethane	1,000	µg/m^3	2.1 J	2.0 J	ND(0.31)	2.5 J	2.5 J	2.5J	2.5 J	2.1 J
m-Dichlorobenzene	--	µg/m^3	40	9	6.6	ND(0.6)	ND(0.6)	ND(0.6)	ND(0.6)	14
Ethanol	--	µg/m^3	91.2	57.3	40.3	25.4	36	23.4	35.2	40.3
Ethylbenzene	97	µg/m^3	5.2	ND(0.35)	ND(0.35)	ND(0.35)	ND(0.35)	ND(0.35)	ND(0.35)	ND(0.35)
Ethyl Acetate	730	µg/m^3	13	4	4.3	14	10	11	7.9	7.2
4-Ethyltoluene	--	µg/m^3	3.0J	ND(0.29)	ND(0.29)	ND(0.29)	ND(0.29)	ND(0.29)	ND(0.29)	ND(0.29)
Heptane	--	µg/m^3	ND(0.32)	ND(0.32)	ND(0.32)	ND(0.32)	ND(0.32)	ND(0.32)	ND(0.32)	ND(0.32)
Hexane	7,300	µg/m^3	11	8.5	8.1	2.8	3	2.9	2.7 J	16
2-Hexanone	310	µg/m^3	ND(0.4)	ND(0.4)	ND(0.4)	ND(0.4)	ND(0.4)	ND(0.4)	ND(0.4)	ND(0.4)
Isopropyl Alcohol	--	µg/m^3	59.2	12	12	8.4	7.4	5.9	21	13
Methylene chloride	6,300	µg/m^3	114	45.2	50.7	20	16	20	17	90
Methyl ethyl ketone	52,000	µg/m^3	5	8.8	10	5.6	13	4.4	6.8	6.8
Methyl Isobutyl Ketone	31,000	µg/m^3	ND(0.49)	ND(0.49)	ND(0.49)	ND(0.49)	ND(0.49)	ND(0.49)	ND(0.49)	ND(0.49)
Methyl Tert Butyl Ether	940	µg/m^3	ND(0.25)	ND(0.25)	ND(0.25)	ND(0.25)	ND(0.25)	ND(0.25)	ND(0.25)	0.72 J
Propylene	31,000	µg/m^3	1.4 J	ND(0.22)	ND(0.22)	7.6	7	1.4 J	2.9 J	ND(0.22)
Styrene	10,000	µg/m^3	ND(0.34)	ND(0.34)	ND(0.34)	ND(0.34)	ND(0.34)	ND(0.34)	0.51 J	ND(0.34)
1,1,1-Trichloroethane	52,000	µg/m^3	ND(0.36)	ND(0.36)	ND(0.36)	ND(0.36)	ND(0.36)	ND(0.36)	ND(0.36)	ND(0.36)
1,2,4-Trimethylbenzene	73	µg/m^3	12	5.4	4.4	ND(0.32)	2.2 J	ND(0.32)	ND(0.32)	6.9
1,3,5-Trimethylbenzene	--	µg/m^3	2.4 J	ND(0.29)	ND(0.29)	ND(0.29)	ND(0.29)	ND(0.29)	ND(0.29)	ND(0.29)
Tertiary Butyl Alcohol	--	µg/m^3	ND(0.55)	5.5	1.3 J	ND(0.55)	ND(0.55)	ND(0.55)	ND(0.55)	ND(0.55)
Tetrachloroethene	420	µg/m^3	ND(0.81)	ND	ND	2.4	1. 4	ND(0.81)	6.8	ND(0.81)
Tetrahydrofuran	21,000	µg/m^3	ND(0.53)	6.5	9.4	ND(0.53)	ND(0.53)	ND(0.53)	ND(0.53)	4.1
Toluene	5,200	µg/m^3	15	6.4	3.1	24	14	17	8.3	2.0 J
Trichloroethene	21	µg/m^3	ND(0.42)	3	ND(0.42)	ND(0.42)	1.9	0.86	16	ND(0.42)
Trichlorofluoromethane	7,300	µg/m^3	2.2 J	ND(0.31)	ND(0.31)	ND(0.31)	ND(0.31)	ND(0.31)	ND(0.31)	3.1 J
m,p-Xylene	1,000	µg/m^3	2.2 J	2.8 J	2.6 J	ND	2.4 J	2.0 J	2.0 J	4.8
o-Xylene	1,000	µg/m^3	2.2 J	ND(0.33)	ND(0.33)	ND(0.33)	ND(0.33)	ND(0.33)	ND(0.33)	2.3 J
Vinyl chloride	16	µg/m^3	ND(0.17)	ND(0.17)	ND(0.17)	ND(0.17)	ND(0.17)	ND(0.17)	ND(0.17)	ND(0.17)
Xylenes	1,000	µg/m^3	2.2 J	2.8 J	2.6 J	ND	2.4 J	2.0 J	2.0 J	6.9

*Source: Test America Laboratories 2013; BVNA 2014b

µg/m^3: micrograms per cubic meter of air

ND: not detected at the method detection limit in parenthesis

J: Indicates an estimated value

Table B.2: Offsite Ambient Air Sampling Results*

Contaminant	Target Indoor Air Concentration Using EPA's Vapor Intrusion Screening Level Calculator (Target Risk = 1 x 10^{-5} and Hazard Quotient = 1)	Units	Location (see Figure B.1)			
			Ambient 101413	Ambient 101613	Hidden 101713	Settlers 101713
			Concentration			
Acetone	32,000	µg/m^3	30.4	23	69.4	26.1
Carbon disulfide	730	µg/m^3	ND(0.21)	ND(0.11)	ND(0.11)	ND(0.11)
Chloroform	1.1	µg/m^3	ND(0.36)	ND(0.18)	ND(0.18)	1.5J
Chloromethane	94	µg/m^3	1.0 J	0.89	1	1
1,1-Dichloroethene	210	µg/m^3	ND(0.33)	ND(0.17)	1.5 J	ND(0.17)
Dichlorodifluoromethane	100	µg/m^3	1.9 J	1.9 J	2.2	2.1
m-Dichlorobenzene	--	µg/m^3	ND(0.6)	ND(0.3)	ND(0.3)	ND(0.3)
Ethanol	--	µg/m^3	15	10	30	11
Ethylbenzene	9.7	µg/m^3	ND(0.35)	ND(0.17)	1.2 J	ND(0.17)
Ethyl Acetate	73	µg/m^3	ND(0.83)	ND(0.4)	82.4	ND(0.4)
4-Ethyltoluene	--	µg/m^3	ND(0.29)	ND(0.15)	2.3	ND(0.15)
Heptane	--	µg/m^3	ND(0.32)	ND(0.16)	1.1J	ND(0.16)
Hexane	730	µg/m^3	2.4 J	10	12	12
2-Hexanone	31	µg/m^3	ND(0.4)	ND(0.2)	3.6	ND(0.2)
Isopropyl Alcohol	--	µg/m^3	2.9	1.7	8.4	2.1
Methylene chloride	630	µg/m^3	18	51.4	60.8	71.6
Methyl ethyl ketone	5,200	µg/m^3	5	2.8	11	3.2
Methyl Isobutyl Ketone	3,100	µg/m^3	ND(0.49)	ND(0.24)	1.6 J	ND(0.24)
Methyl Tert Butyl Ether	94	µg/m^3	ND(0.25)	ND(0.12)	ND(0.12)	ND(0.12)
Propylene	3,100	µg/m^3	0.86 J	0.72 J	1.2 J	1.1 J
Styrene	1,000	µg/m^3	ND(0.34)	ND(0.17)	ND(0.17)	ND(0.17)
1,1,1-Trichloroethane	5,200	µg/m^3	ND(0.36)	ND(0.18)	1.3 J	ND(0.18)
1,2,4-Trimethylbenzene	7.3	µg/m^3	ND(0.32)	ND(0.16)	7.9	ND(0.16)
1,3,5-Trimethylbenzene	--	µg/m^3	ND(0.29)	ND(0.15)	1.8 J	ND(0.15)
Tertiary Butyl Alcohol	--	µg/m^3	3.6	0.82 J	0.94 J	ND(0.27)
Tetrachloroethene	42	µg/m^3	ND(0.32)	2.4	16	8.8
Telrahydrofuran	2,100	µg/m^3	ND(0.25)	2.1	2.9	3.5
Toluene	5,200	µg/m^3	1.5 J	1.4 J	3.2	1.7
Trichloroethene	2.1	µg/m^3	ND(0.42)	0.46	ND(0.21)	ND(0.21)
Trichlorofluoromethane	730	µg/m^3	ND(0.31)	1.8 J	2.2	2.5
m,p-Xylene	100	µg/m^3	ND(0.56)	ND(0.28)	8.3	1.2 J
o-Xylene	100	µg/m^3	ND(0.33)	ND(0.17)	2	ND(0.17)
Vinyl chloride	1.6	µg/m^3	ND(0.17)	ND(0.087)	ND(0.087)	ND(0.087)
Xylenes	100	µg/m^3	ND(0.33)	ND(0.17)	10	1.2 J

*Source: Test America Laboratories 2013; BVNA 2014b
µg/m^3: micrograms per cubic meter of air
ND: not detected at the method detection limit in parenthesis
J: Indicates an estimated value

Table B.3: Volatile Organic Compounds in Soil Results*

Contaminant	Sample Location (See Figure B.1)									
	MIP 01	MIP02		MIP 03		MIP 04			MIP 05	
	Concentration (mg/kg)									
1,1-Dichloroethane	<0.003	<0.003	<0.006	<0.006	<0.004	<0.005	<0.005	<0.005	<0.004	<0.004
1,1-Dichloroethylene	<0.003	<0.003	<0.006	<0.006	<0.004	<0.005	<0.005	<0.005	<0.004	<0.004
cis-1,2 Dichloroethene	<0.003	<0.003	<0.006	<0.006	<0.004	<0.005	<0.005	<0.005	<0.004	<0.004
trans-1,2 Dichloroethene	<0.003	<0.003	<0.006	<0.006	<0.004	<0.005	<0.005	<0.005	<0.004	<0.004
1,1,1-Trichloroethane	<0.003	<0.003	<0.006	<0.006	<0.004	<0.005	<0.005	<0.005	<0.004	<0.004
Tetrachloroethene	<0.003	<0.003	<0.006	<0.006	<0.004	<0.005	<0.005	<0.005	<0.004	<0.004
Trichloroethylene	<0.003	0.051	0.05	<0.006	<0.004	<0.005	<0.005	<0.005	<0.004	<0.004
Vinyl Chloride	<0.006	<0.006	<0.011	<0.012	<0.008	<0.01	<0.009	<0.010	<0.008	<0.007
Depth (feet)	6'-10'	0'-2'	8'-10'	0'-2'	6'-8'	0'-2'	8'-10'	18'-20'	0'-2'	5'-7'

Contaminant	Sample Location (See Figure B.1)								
	MIP 06		MIP 07		MIP 08		GW 10	GW 11	GW 12
	Concentration (mg/kg)								
1,1-Dichloroethane	<0.003	<0.004	<0.003	<0.003	<0.005	<0.005	<0.003	<0.004	<0.004
1,1-Dichloroethylene	<0.003	<0.004	<0.003	<0.003	<0.005	<0.005	<0.003	<0.004	<0.004
cis-1,2 Dichloroethene	<0.003	<0.004	<0.003	<0.003	<0.005	<0.005	<0.003	<0.004	<0.004
trans-1,2 Dichloroethene	<0.003	<0.004	<0.003	<0.003	<0.005	<0.005	<0.003	<0.004	<0.004
1,1,1-Trichloroethane	<0.003	<0.004	<0.003	<0.003	<0.005	<0.005	<0.003	<0.004	<0.004
Tetrachloroethene	<0.003	<0.004	<0.003	<0.003	<0.005	<0.005	<0.003	<0.004	<0.004
Trichloroethylene	<0.003	<0.004	<0.003	<0.003	<0.005	<0.005	<0.003	<0.004	<0.004
Vinyl Chloride	<0.006	<0.007	<0.006	<0.006	<0.009	<0.01	<0.006	<0.008	<0.009
Depth (feet)	0'-2'	5'-7'	0'-5'	5'-10'	0'-2'	13'-15'	7'-8'	6'-7'	17'-18'

*Source: Test America Laboratories 2013; BVNA 2014b
mg/kg: milligrams per kilogram
<: less than method detection limit

Groundwater Sampling Results

Groundwater samples collected were analyzed for VOCs by EPA Method 8260B [EPA 2007]. The first groundwater sample collected at each boring was recovered from the top two-feet of the groundwater table with the top of the Screen Point sampler placed within the first few inches of the groundwater table. These observed saturated zone elevations were used to create a map of the elevation of the groundwater with a resultant accuracy of approximately +/- one foot [BVNA 2014b]. The groundwater elevation map reflects a general eastward downgradient trend toward the unnamed creek just north of Waterford Road as shown in Figure B.2.

As generally observed in the Piedmont Physiographic Province, higher concentrations of chlorinated solvent impacted groundwater will more likely be found in topographically low elevations that are coincident with thicker overburden [BVNA 2013a]. Hence, the highest concentrations of dissolved-phase chlorinated solvents would be expected to be found downgradient from the SRG site in the topographically low-lying area associated with the unnamed creek located to the north of Waterford Road.

Analytical results show that the highest VOC concentrations observed in groundwater were observed in groundwater collected from borehole MIP02. TCE was detected at 14,000 micrograms per liter (µg/L) in the groundwater sample collected at MIP02 from 26 to 28 feet below ground surface (bgs). Similar concentrations were observed in the 52 to 54 feet bgs depth interval. TCE detected at the top of the groundwater table at MIP02 (11 to 13 feet bgs) was reported at a concentration of 2,600 µg/L.

The elevated dissolved TCE concentration observed at sample location MIP02 is coincident with the interpolated thickest overburden zone (deepest depth to weathered bedrock) [BVNA 2014b]. Furthermore, dissolved chlorinated solvents such as TCE that have a higher specific gravity than water tend to increase in concentration with depth over time due to density stratification (downward flow). Therefore, the areas of thicker overburden (soft soils) beneath the saturated zone are likely to have higher dissolved-phase chlorinated solvent concentrations than thin areas of overburden that occur above bedrock [BVNA 2014b].

Figure B.3 shows the analytical results for the two primary chlorinated solvents detected, PCE and TCE, plotted in a tabular format for each depth at each sample location. The results of the groundwater analyses in the Settlers Grove subdivision are summarized in Table B.4. Table B.4 also shows that additional constituents detected in groundwater include low concentrations of 1,1-DCE, *cis* 1,2-DCE, *trans* 1,2-DCE, 1,1,1-DCA, PCE, VC, 1,1,2-TCA, 2-butanone, and chloroform. All but 2-butanone and chloroform are known to be associated with the manufacture of TCE, and/or are natural biodegradation products of PCE or TCE. Chloroform is known to be a byproduct of public water chlorination which is its likely source where it was detected [BVNA 2014b].

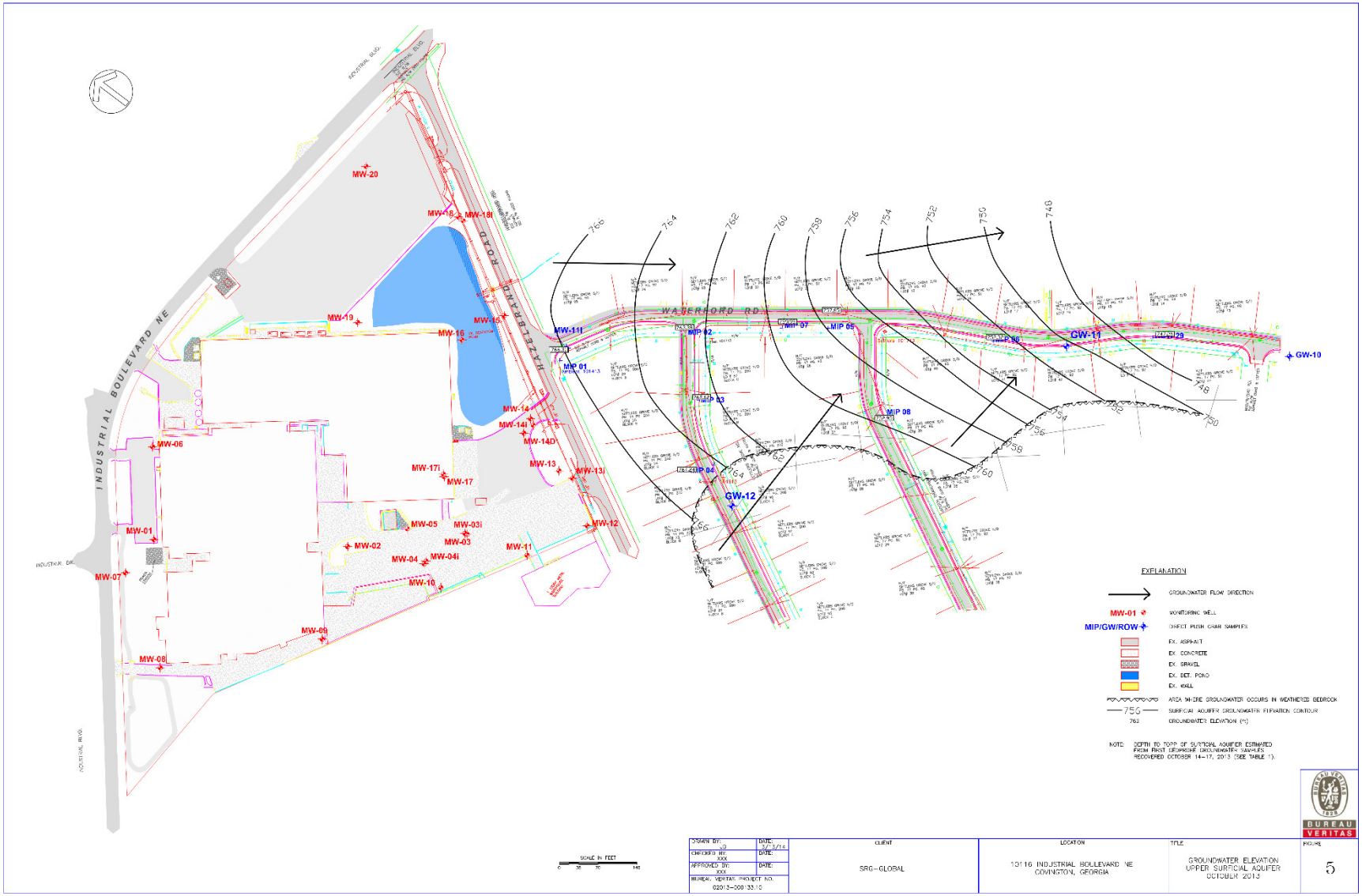


Figure B.2: Groundwater elevations in the upper surficial aquifer as well as upper aquifer groundwater flow direction.

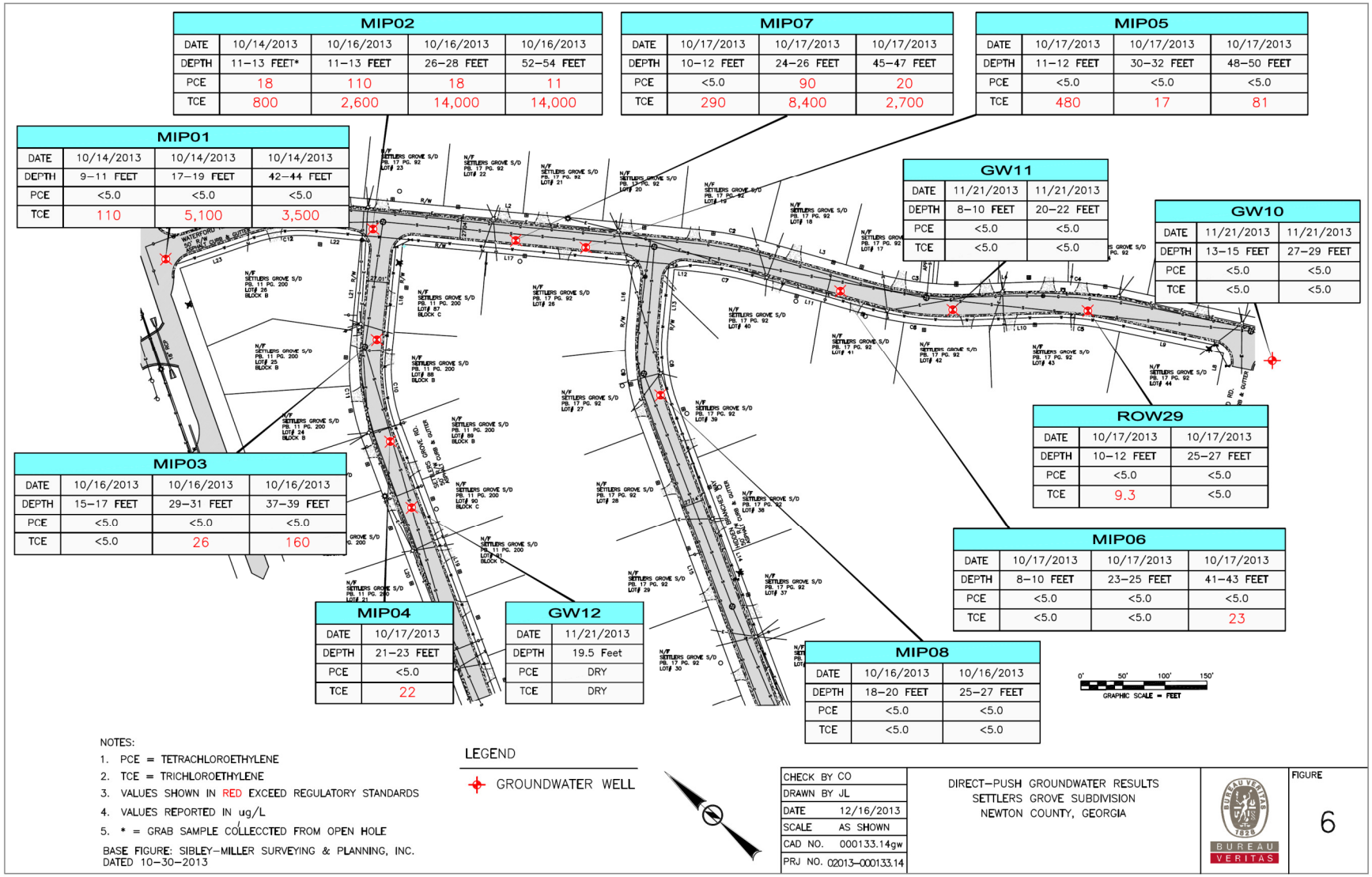

MIP02
DATE 10/14/2013 10/16/2013 10/16/2013 10/16/2013
DEPTH 11–13 FEET* 11–13 FEET 26–28 FEET 52–54 FEET
PCE 18 110 18 11
TCE 800 2,600 14,000 14,000
MIP07
DATE 10/17/2013 10/17/2013 10/17/2013
DEPTH 10–12 FEET 24–26 FEET 45–47 FEET
PCE <5.0 90 20
TCE 290 8,400 2,700
MIP05
DATE 10/17/2013 10/17/2013 10/17/2013
DEPTH 11–12 FEET 30–32 FEET 48–50 FEET
PCE <5.0 <5.0 <5.0
TCE 480 17 81
MIP01
DATE 10/14/2013 10/14/2013 10/14/2013
DEPTH 9–11 FEET 17–19 FEET 42–44 FEET
PCE <5.0 <5.0 <5.0
TCE 110 5,100 3,500
GW11
DATE 11/21/2013 11/21/2013
DEPTH 8–10 FEET 20–22 FEET
PCE <5.0 <5.0
TCE <5.0 <5.0
GW10
DATE 11/21/2013 11/21/2013
DEPTH 13–15 FEET 27–29 FEET
PCE <5.0 <5.0
TCE <5.0 <5.0
ROW29
DATE 10/17/2013 10/17/2013
DEPTH 10–12 FEET 25–27 FEET
PCE <5.0 <5.0
TCE 9.3 <5.0
MIP03
DATE 10/16/2013 10/16/2013 10/16/2013
DEPTH 15–17 FEET 29–31 FEET 37–39 FEET
PCE <5.0 <5.0 <5.0
TCE <5.0 26 160
MIP06
DATE 10/17/2013 10/17/2013 10/17/2013
DEPTH 8–10 FEET 23–25 FEET 41–43 FEET
PCE <5.0 <5.0 <5.0
TCE <5.0 <5.0 23
MIP04
DATE 10/17/2013
DEPTH 21–23 FEET
PCE <5.0
TCE 22
GW12
DATE 11/21/2013
DEPTH 19.5 Feet
PCE DRY
TCE DRY
MIP08
DATE 10/16/2013 10/16/2013
DEPTH 18–20 FEET 25–27 FEET
PCE <5.0 <5.0
TCE <5.0 <5.0
0' 50' 100' 150'
GRAPHIC SCALE = FEET
NOTES:
1. PCE = TETRACHLOROETHYLENE
2. TCE = TRICHLOROETHYLENE
3. VALUES SHOWN IN RED EXCEED REGULATORY STANDARDS
4. VALUES REPORTED IN ug/L
5. * = GRAB SAMPLE COLLECCTED FROM OPEN HOLE
BASE FIGURE: SIBLEY–MILLER SURVEYING & PLANNING, INC. DATED 10–30–2013
LEGEND
GROUNDWATER WELL
CHECK BY CO
DRAWN BY JL
DATE 12/16/2013
SCALE AS SHOWN
CAD NO. 000133.14gw
PRJ NO. 02013–000133.14
DIRECT–PUSH GROUNDWATER RESULTS
SETTLERS GROVE SUBDIVISION
NEWTON COUNTY, GEORGIA
BUREAU VERITAS
FIGURE
6

Figure B.3

Table B.4: Volatile Organic Compounds in Groundwater Results*

Contaminant	Sample Location (see Figure B.3)										
	MIP 01			MIP02				MIP 03			MIP 04
	Concentration (µg/L)										
2-Butanone	<50	<50	<50	<50	<50	<50	72	<50	<50	<50	<50
Chloroform	<5.0	<5.0	<5.0	<5.0	<5.0	<5.0	<5.0	<5.0	<5.0	<5.0	6
1,1-Dichloroethane	<5.0	7	8.4	<5.0	6.2	<5.0	<5.0	<5.0	<5.0	<5.0	<5.0
1,1-Dichloroethene	<5.0	42	48	5.1	31	21	57	<5.0	<5.0	<5.0	<5.0
cis-1,2 Dichloroethene	26	180	140	110	320	180	440	<5.0	<5.0	<5.0	<5.0
trans-1,2-Dichloroethene	<5.0	<5.0	<5.0	<5.0	<5.0	13	24	<5.0	<5.0	<5.0	<5.0
1,1,2-Trichloroethane	<5.0	<5.0	<5.0	<5.0	<5.0	5.2	6.7	<5.0	<5.0	<5.0	<5.0
Tetrachloroethene	<5.0	<5.0	<5.0	18	110	18	11	<5.0	<5.0	<5.0	<5.0
Trichloroethene	110	5,100	3,500	800	2,600	14,000	14,000	<5.0	26	160	22
Vinyl Chloride	<2.0	<2.0	<2.0	5.0	<2.0	<2.0	<2.0	<2.0	<2.0	<2.0	<2.0
Depth (feet)	9'-11	17'-19'	42'-44'	11'-13'	11 '-13'	26'-28'	52'-54'	15'-17'	29'-31'	37'-39'	21'-23'
Contaminant	**Sample Location**										
	MIP 05			MIP 06			MIP 07			MIP 08	
	Concentration (µg/L)										
2-Butanone	<50	<50	<50	<50	<50	<50	<50	<50	<50	<50	<50
Chloroform	<5.0	64	35	<5.0	<5.0	11	<5.0	<5.0	<5.0	<5.0	<5.0
1,1-Dichloroethane	<5.0	<5.0	<5.0	<5.0	<5.0	<5.0	<5.0	<5.0	<5.0	<5.0	<5.0
1,1-Dichloroethene	<5.0	<5.0	<5.0	<5.0	<5.0	<5.0	<5.0	17	6.4	<5.0	<5.0
cis-1,2 Dichloroethene	8.3	<5.0	<5.0	<5.0	<5.0	<5.0	11	130	40	<5.0	<5.0
trans-1,2-Dichloroethene	<5.0	<5.0	<5.0	<5.0	<5.0	<5.0	<5.0	<5.0	<5.0	<5.0	<5.0
1,1,2-Trichloroethane	<5.0	<5.0	<5.0	<5.0	<5.0	<5.0	<5.0	<5.0	<5.0	<5.0	<5.0
Tetrachloroethene	<5.0	<5.0	<5.0	<5.0	<5.0	<5.0	<5.0	90	20	<5.0	<5.0
Trichloroethene	480	17	81	<5.0	<5.0	23	290	8,400	2,700	<5.0	<5.0
Vinyl Chloride	<2.0	<2.0	<2.0	<2.0	<2.0	<2.0	<2.0	<2.0	<2.0	<2.0	<2.0
Depth (feet)	10'-12'	30'-32'	48'-50'	8'-10'	23'-25'	41'-43'	10'-12'	24'-26'	45'-47'	18'-20'	25'-27'
Contaminant	**Sample Location**										
	ROW29		GW 10		GW 11						
	Concentration (µg/L)										
2-Butanone	<50	<50	<50	<50	<50	<50					
Chloroform	43	63	43	63	43	63					
1,1-Dichloroethane	<5.0	<5.0	<5.0	<5.0	<5.0	<5.0					
1,1-Dichloroethene	<5.0	<5.0	<5.0	<5.0	<5.0	<5.0					
cis-1,2 Dichloroethene	<5.0	<5.0	<5.0	<5.0	<5.0	<5.0					
trans-1,2-Dichloroethene	<5.0	<5.0	<5.0	<5.0	<5.0	<5.0					
1,1,2-Trichloroethane[1]	<5.0	<5.0	<5.0	<5.0	<5.0	<5.0					
Tetrachloroethene	<5.0	<5.0	<5.0	<5.0	<5.0	<5.0					
Trichloroethene[1]	9.3	<5.0	<5.0	<5.0	<5.0	<5.0					
Vinyl Chloride[1]	<2.0	<2.0	<2.0	<2.0	<2.0	<2.0					
Depth (feet)	10'-12'	25'-27'	13'-15'	27'-29'	8'-10'	20'-22'					

*Source: Test America Laboratories 2013; BVNA 2014b

µg/L: micrograms per liter; <: less than method detection limit; Red Font: Detected above Federal MCLs for Drinking Water.

Table B.5: Indoor Air Sampling Results*. Indoor air concentrations are in micrograms per cubic meter (µg/m^3).

Map ID	Sample Type	Sample Location	1,1 Dichloroethane	1,1-Dichloroethene	*cis* 1,2-Dichloroethene	*trans* 1,2-Dichloroethene	Tetrachloroethene	Trichloroethene	Vinyl Chloride
Health-Based Comparison Values			**18[a]**	**210[a]**	**--**	**--**	**270[b], 3.8[c]**	**2.0[b], 0.24[c]**	**77[b], 0.11[c]**
2	Outdoor	Outdoor	<0.040	<0.040	<0.040	<0.040	**0.25**	**0.36**	<0.051
2	Outdoor	Outdoor (duplicate)	<0.040	<0.040	<0.040	<0.040	**0.36**	**0.31**	**0.072**
2	Indoor	Kitchen	<0.040	<0.040	<0.040	<0.040	**0.29**	**0.14**	<0.051
2	Indoor	Bedroom	<0.040	<0.040	<0.040	<0.040	**0.14**	**0.11**	<0.051
2	Crawl space	Crawl space	<0.040	<0.040	<0.040	<0.040	**0.2**	<0.054	<0.051
2 **S**	Outdoor	Outdoor	<0.040	<0.040	<0.040	<0.040	**0.12**	**0.11**	<0.051
2 **S**	Indoor	Kitchen	<0.040	<0.040	<0.040	<0.040	**0.12**	**0.19**	<0.051
2 **S**	Indoor	Back left bedroom	<0.040	<0.040	<0.040	<0.040	**0.11**	**0.22**	<0.051
2 **S**	Crawl space	Crawl space	<0.059	<0.058	<0.058	<0.058	**0.14**	**0.12**	<0.075
3	Outdoor	Outdoor	<0.040	<0.040	**0.15**	<0.040	**0.081**	**0.7**	<0.051
3	Indoor	Living room	<0.040	<0.040	<0.040	<0.040	**0.28**	**0.98**	<0.051
3	Indoor	Bathroom	<0.040	<0.040	**0.099**	<0.040	**0.39**	**0.56**	<0.051
3	Crawl space	Crawl space	<0.040	<0.040	**0.086**	<0.040	**0.28**	**0.57**	**0.06**
3	Crawl space	Crawl space	<0.040	<0.040	**0.076**	<0.040	**0.23**	**0.53**	<0.051
4	Outdoor	Outdoor	<0.040	<0.040	<0.040	<0.040	<0.068	**0.13**	<0.051
4	Indoor	Living room	<0.040	<0.040	<0.040	<0.040	**0.4**	<0.054	<0.051
4	Indoor	Kitchen	<0.040	<0.040	<0.040	<0.040	**0.15**	<0.054	<0.051
4	Crawl space	Crawl space	<0.040	<0.040	<0.040	<0.040	**0.1**	**0.21**	<0.051
4	Crawl space	Crawl space duplicate	<0.040	<0.040	<0.040	<0.040	**0.17**	**0.38**	<0.051
7	Outdoor	Outdoor	<0.040	<0.040	**0.13**	<0.040	**0.13**	**0.41**	<0.051
7	Indoor	Bathroom	<0.040	<0.040	**0.087**	<0.040	**0.19**	**0.3**	<0.051
7	Indoor	Living room	<0.040	<0.040	**0.1**	<0.040	**0.31**	**0.36**	**0.069**
7	Indoor	Living room (duplicate)	<0.040	<0.040	**0.12**	<0.040	**0.36**	**0.39**	**0.099**
7	Crawl space	Crawl space	<0.040	<0.040	<0.040	<0.040	**0.092**	**0.072**	<0.051
7 **S**	Outdoor	Outdoor	<0.040	<0.040	<0.040	<0.040	**0.35**	**0.16**	<0.051
7 **S**	Outdoor	Outdoor	<0.040	<0.040	<0.040	<0.040	**0.28**	**0.11**	<0.051
7 **S**	Indoor	Bathroom	<0.040	<0.040	<0.040	<0.040	**21**	<0.054	<0.051
7 **S**	Indoor	Living room	<0.040	<0.040	<0.040	<0.040	**22**	<0.054	<0.051
7 **S**	Crawl space	Crawl space	<0.040	<0.040	<0.040	<0.040	**15**	<0.054	<0.051
9	Outdoor	Outdoor	<0.040	<0.040	**0.14**	<0.040	**0.24[a]**	**0.49**	<0.051
9	Outdoor	Outdoor (duplicate)	<0.040	<0.040	**0.15**	<0.040	**0.14[a]**	**0.47**	<0.051
9	Indoor	Living room	<0.040	<0.040	<0.040	<0.040	**0.071**	<0.040	<0.051
9	Indoor	Bathroom	<0.040	<0.040	**0.17**	<0.040	**0.088**	**0.54**	<0.051
9	Crawl space	Crawl space	<0.040	<0.040	**0.18**	<0.040	<0.068	**0.55**	<0.051
11[b]	Indoor	Living room	<0.040	<0.040	**0.083**	<0.040	**0.18**	**0.29**	<0.051
11[b]	Indoor	Kitchen	<0.054	<0.053	**0.096**	<0.053	**0.13**	**0.32**	<0.069

12	Indoor	Living room	<0.040	<0.040	<0.040	<0.040	**0.51**	**0.11**	<0.051
12	Indoor	Bathroom	<0.040	<0.040	<0.040	<0.040	**0.53**	**0.12**	<0.051
12	Indoor	Crawl space	<0.040	<0.040	<0.040	<0.040	**0.17**	**0.60**	<0.051
16	Outdoor	Outdoor	<0.040	<0.040	0.083	<0.040	**0.14**	**0.26**	<0.051
16	Indoor	Living room/kitchen	<0.040	<0.040	0.075	<0.040	**0.53**	**0.57**	<0.051
16	Indoor	Bathroom	<0.040	<0.040	<0.040	<0.040	**0.6**	**0.59**	<0.051
16	Crawl space	Crawl space	<0.040	<0.040	0.061	<0.040	**0.35**	**0.36**	<0.051
61	Outdoor	Outdoor	<0.076	<0.074	<0.074	<0.074	<0.13	<0.10	<0.096
61	Indoor	Kitchen	<0.040	<0.040	0.075	<0.040	<0.068	**0.16**	<0.051
61	Indoor	Bedroom	<0.040	<0.040	<0.040	<0.040	**0.079**	**0.18**	<0.051
62	Outdoor	Outdoor	<0.040	<0.040	<0.040	<0.040	**0.068**	**0.055**	<0.051
62	Crawl space	Crawl space	<0.040	<0.040	<0.040	<0.040	**0.079**	**0.054**	<0.051
62	Indoor	Living room/dining room	<0.040	<0.040	<0.040	<0.040	**0.11**	**0.36**	<0.051
65	Outdoor	Outdoor duplicate	<0.040	<0.040	<0.040	<0.040	**0.39**	**0.059**	**0.062**
65	Indoor	Kitchen	<0.040	<0.040	0.083	<0.040	**0.099**	**0.077**	<0.051
65	Indoor	Lower floor bedroom	<0.040	<0.040	0.075	<0.040	**0.11**	**0.074**	<0.051
66	Outdoor	Outdoor	<0.040	<0.040	<0.040	<0.040	<0.068	<0.054	<0.051
66	Indoor	Garage storage area	<0.040	<0.040	<0.040	<0.040	**0.078**	<0.054	<0.051
66	Indoor	Garage storage area duplicate	<0.040	<0.040	<0.040	<0.040	**0.076**	<0.054	<0.051
66	Indoor	Ground floor bedroom	<0.040	<0.040	<0.040	<0.040	**0.45**	<0.054	<0.051
66 **S**	Outdoor	Outdoor	<0.040	<0.040	<0.040	<0.040	**0.11**	<0.054	<0.051
66 **S**	Indoor	Garage storage area	<0.040	<0.040	<0.040	<0.040	**0.13**	<0.054	<0.051
66 **S**	Indoor	Garage storage area duplicate	<0.040	<0.040	<0.040	<0.040	**0.11**	<0.054	<0.051
66 **S**	Indoor	Ground floor bedroom	<0.040	<0.040	<0.040	<0.040	**0.11**	<0.054	<0.051
67	Outdoor	Outdoor	<0.040	<0.040	<0.040	<0.040	**0.59**	**0.09**	<0.051
67	Indoor	Laundry room	<0.040	<0.040	<0.040	<0.040	**0.18**	**0.12**	<0.051
67	Indoor	Kitchen	<0.040	<0.040	<0.040	<0.040	**0.19**	**0.095**	<0.051
68[b]	Indoor	Basement TV room	<0.04	**0.34**	**0.21**	<0.04	**0.32**	**--[c]**	<0.051
68[b]	Indoor	Basement TV room	<0.16	**0.38**	**0.23**	<0.16	**0.34**	**11[d]**	<0.20
68[b]	Indoor	Kitchen	<0.040	**0.25**	**0.16**	<0.040	**0.35**	**--[c]**	<0.051
68[b]	Indoor	Kitchen	<0.16	**0.26**	**0.17**	<0.16	**0.36**	**8.5[d]**	<0.20
68[b]	Indoor	Living Room	<0.040	**0.29**	**0.19**	<0.040	**0.42**	**--[c]**	<0.051
68[b]	Indoor	Living Room	<0.16	**0.28**	**0.18**	<0.16	**0.33**	**8.3[d]**	<0.20
69	Indoor	Kitchen	<0.040	<0.040	<0.040	<0.040	0.14	**0.11**	<0.051
69	Indoor	Living	<0.040	<0.040	<0.040	<0.040	<0.068	**0.11**	<0.051
69 **S**	Outdoor	Outdoor	<0.040	<0.040	<0.040	<0.040	**0.11**	<0.054	<0.051
69 **S**	Indoor	Living room	<0.040	<0.040	<0.040	<0.040	**0.095**	**0.19**	<0.051
69 **S**	Indoor	Kitchen	<0.040	<0.040	<0.040	<0.040	**0.14**	**0.21**	<0.051
69 **S**	Indoor	Partial basement	<0.040	<0.040	<0.040	<0.040	**0.15**	**0.36**	<0.051
71	Outdoor	Outdoor	<0.040	<0.040	<0.040	<0.040	**0.23**	**0.094**	**0.11**
71	Indoor	Kitchen	<0.040	<0.040	<0.040	<0.040	**0.21**	**0.13**	<0.051
71	Indoor	TV room	<0.040	<0.040	<0.040	<0.040	**0.23**	**0.13**	<0.051
71 **S**	Outdoor	Outdoor	<0.040	<0.040	<0.040	<0.040	**0.12**	**0.21**	<0.051
71 **S**	Outdoor	Outdoor duplicate	<0.040	<0.040	<0.040	<0.040	**0.083**	**0.20**	<0.051
71 **S**	Indoor	Kitchen	<0.040	<0.040	<0.040	<0.040	**0.21**	**0.15**	<0.051
71 **S**	Indoor	TV Room	<0.040	<0.040	<0.040	<0.040	**0.17**	**0.15**	<0.051
72	Outdoor	Outdoor	<0.040	<0.040	<0.040	<0.040	<0.068	**0.057**	<0.051
72	Outdoor	Outdoor duplicate	<0.040	<0.040	<0.040	<0.040	**0.17**	<0.054	<0.051
72	Indoor	Kitchen	<0.040	<0.040	<0.040	<0.040	**1.1**	**0.5**	<0.051
72	Indoor	TV room	<0.040	<0.040	<0.040	<0.040	**0.95**	**0.43**	<0.051

72 **S**	Outdoor	Outdoor	<0.040	<0.040	<0.040	<0.040	**0.62**	**0.14**	**0.17**
72 **S**	Outdoor	Outdoor duplicate	<0.040	<0.040	<0.040	<0.040	**0.54**	**0.091**	**0.11**
72 **S**	Indoor	Kitchen	<0.040	<0.040	<0.040	<0.040	**0.43**	**2.0**	<0.051
72 **S**	Indoor	TV Room	<0.040	<0.040	<0.040	<0.040	**0.40**	**1.9**	<0.051
73	Outdoor	Outdoor	<0.040	<0.040	<0.040	<0.040	0.085	<0.054	<0.051
73	Indoor	Kitchen	<0.040	<0.040	<0.040	**0.047**	**1.9**	**0.41**	<0.051
73	Indoor	Laundry room	<0.040	<0.040	<0.040	<0.040	**0.54**	**0.15**	<0.051
73	Indoor	Laundry room	<0.040	<0.040	<0.040	<0.040	**0.49**	**0.13**	<0.051
74	Outdoor	Outdoor	<0.040	<0.040	<0.040	<0.040	**3.4**	**0.065**	**0.081**
74	Outdoor	Outdoor duplicate	<0.040	<0.040	<0.040	<0.040	**2.7**	**0.082**	**0.054**
74	Indoor	Basement TV room	<0.040	<0.040	<0.040	<0.040	**0.33**	<0.054	<0.051
74	Indoor	Basement unfinished	<0.040	<0.040	<0.040	<0.040	**0.23**	**0.11**	<0.051
163	Outdoor	Outdoor	<0.040	<0.040	<0.040	<0.040	**0.59**	**0.09**	<0.051
163	Indoor	Kitchen	<0.040	<0.040	<0.040	<0.040	**0.11**	<0.054	<0.051
163	Indoor	Living room	<0.040	<0.040	<0.040	<0.040	**0.11**	<0.054	<0.051
163	Crawl space	Crawl space	<0.040	<0.040	<0.040	<0.040	**0.08**	**0.064**	<0.051
163 **S**	Outdoor	Outdoor	<0.040	<0.040	<0.040	<0.040	<0.068	**0.070**	<0.051
163 **S**	Indoor	Kitchen	<0.040	<0.040	<0.040	<0.040	**0.079**	<0.054	<0.051
163 **S**	Indoor	Living room	<0.040	<0.040	<0.040	<0.040	**0.12**	<0.054	<0.051
163 **S**	Crawl space	Crawl space	<0.040	<0.040	<0.040	<0.040	<0.068	<0.054	<0.051
171	Outdoor	Outdoor	<0.040	<0.040	<0.040	<0.040	**0.41**	<0.054	<0.051
171	Indoor	Bathroom	<0.040[e]	<0.040[e]	<0.040[e]	<0.040	--[c]	**1.0[e]**	<0.051[e]
171	Indoor	Bathroom	<4.0[e]	<4.0[e]	<4.0[e]	<4.0[e]	**530**	<5.4[e]	<5.1[d,e]
171	Indoor	Living room	<0.040	<0.040	<0.040	<0.040	**0.18**	**0.081**	<0.051
171	Crawl space	Crawl space	<0.040	<0.040	<0.040	<0.040	< 0.068	<0.054	<0.051
171 **S**	Outdoor	Outdoor	<0.040	<0.040	<0.040	<0.040	**0.24**	**0.063**	<0.051
171 **S**	Indoor	Bathroom	<0.040	<0.040	<0.040	<0.040	**860**	**0.21**	<0.051
171 **S**	Indoor	Bathroom	<0.040	<0.040	<0.040	<0.040	**920**	<0.054	<0.051
171 **S**	Indoor	Living room	<1.0	<0.99	<0.99	<0.99	**820**	<1.3	<0.051
171 **S**	Crawl space	Crawl space	<0.35	<0.34	<0.34	<0.34	**33**	<0.47	<0.44
174	Outdoor	Outdoor	<0.040	<0.040	<0.040	<0.040	<0.068	**0.059**	<0.051
174	Outdoor	Outdoor duplicate	<0.040	<0.040	<0.040	<0.040	**0.58**	**0.24**	<0.051
174	Indoor	Living room	<0.040	<0.040	<0.040	<0.040	**0.17**	**0.68**	<0.051
174	Indoor	Kitchen	<0.040	<0.040	<0.040	<0.040	**0.68**	**0.53**	<0.051
174	Indoor	Crawl space	<0.040	<0.040	<0.040	<0.040	**0.53**	**0.27**	<0.051

[*]Source: Test America Laboratories 2013; BVNA 2014b, and SRG Global Letters to Settlers Grove Residents dated August 18, 2014, October 9, 2014, and November 6, 2014.

Health-based comparison values: [a]Screening level concentrations for indoor air based on EPA's VISL calculator based on EPA's Regional Screening Levels (RSLs) updated in May 2014. [b]ATSDR chronic EMEG/MRL for tetrachloroethene and trichloroethene, and the intermediate EMG/MRL for vinyl chloride. ATSDR Cancer Risk Evaluation Guide (CREG).

Indoor air sample results highlighted in yellow indicate concentrations above an RSL. Indoor air sample results highlighted in pink indicate concentrations above a CREG.

[a] – Result is considered estimated because of slight difference between duplicate sampling results.

[b] – Outdoor samples applicable to this sampling event were collected at a neighboring residence on the same day as follows:

Map #11 – outdoor air samples collected at Map #9.
Map #68 – outdoor air samples collected at Map #71.
Map #69 – outdoor air samples collected at Map #72.

[c] – Result is considered estimated because it exceeded the calibration range. The reanalyzed result supersedes the estimated value.

[d] – Sample reanalyzed to verify the chemical concentration. The analyzed result is the final result and supersedes the estimated value.

[e] – Result is considered estimated because of a decrease in canister air pressure from the field to the lab.

S: resampled in summer 2014

Bold values indicated concentrations reported above the laboratory reporting limit.

Table B.6: Post-Mitigation Indoor Air Sampling Results*. Indoor air concentrations are in micrograms per cubic meter (µg/m^3).

Map ID	Sample Type	Sample Location	1,1 Dichloroethane	1,1-Dichloroethene	*cis* 1,2-Dichloroethene	*trans* 1,2-Dichloroethene	Tetrachloroethene	Trichloroethene	Vinyl Chloride
Health-Based Comparison Values			**18[a]**	**210[a]**	**--**	**--**	**270[b], 3.8[c]**	**2.0[b], 0.24[c]**	**77[b], 0.11[c]**
3	Outdoor	Outdoor	<0.040	<0.040	<0.040	<0.040	<0.068	**0.063**	<0.051
3	Indoor	Living room	<0.040	<0.040	<0.040	<0.040	**0.30**	**0.086**	<0.051
3	Indoor	Bathroom	<0.040	<0.040	<0.040	<0.040	**0.30**	**0.079**	<0.051
3	Crawl space	Crawl space	<0.040	<0.040	<0.040	<0.040	**0.13**	**0.074**	<0.051
3	Crawl space	Crawl space	<0.040	<0.040	<0.040	<0.040	**0.14**	**0.072**	<0.051
11	Outdoor	Outdoor	<0.040	<0.040	<0.040	<0.040	**0.16**	**0.18**	<0.051
11	Indoor	Kitchen	<0.040	<0.040	<0.040	<0.040	**0.38**	**0.54**	<0.051
11	Indoor	Living room	<0.040	<0.040	<0.040	<0.040	**0.48**	**0.49**	<0.051
11	Crawl space	Crawl space	<0.040	<0.040	<0.040	<0.040	**0.21**	**0.23**	<0.051
11	Crawl space	Crawl space	<0.040	<0.040	<0.040	<0.040	**0.18**	**0.21**	<0.051
67	Outdoor	Outdoor	<0.040	<0.040	<0.040	<0.040	**0.12**	<0.054	<0.051
67	Indoor	Laundry room	<0.040	<0.040	<0.040	<0.040	**0.32**	**0.12**	<0.051
67	Indoor	Laundry room duplicate	<0.040	<0.040	<0.040	<0.040	**0.57**	**0.16**	<0.051
67	Indoor	Kitchen	<0.040	<0.040	<0.040	<0.040	<0.068	<0.054	<0.051
68	Outdoor	Outdoor	<0.059	<0.057	<0.057	<0.057	**0.62**	**0.18**	<0.057
68	Outdoor	Outdoor duplicate	<0.040	<0.040	<0.040	<0.040	**0.77**	**0.20**	<0.051
68	Indoor	Basement TV room	<0.040	<0.040	<0.040	<0.040	**0.63**	**0.72**	<0.051
68	Indoor	Living room	<0.040	<0.040	<0.040	<0.040	**0.73**	**0.66**	<0.051
68	Indoor	Basement Laundry room	<0.040	<0.040	<0.040	<0.040	**0.46**	**0.56**	<0.051
73	Outdoor	Outdoor	<0.040	<0.040	<0.040	<0.040	**0.14**	**0.13**	<0.051
73	Indoor	Kitchen	<0.040	<0.040	<0.040	<0.040	**0.11**	**0.087**	<0.051

*Source: SRG Global Letters to Settlers Grove Residents dated August 18, 2014, October 9, 2014, and November 6, 2014.
Health-based comparison values: [a]Screening level concentrations for indoor air based on EPA's VISL calculator based on EPA's Regional Screening Levels (RSLs) updated in May 2014. [b]ATSDR chronic EMEG/MRL for tetrachloroethene and trichloroethene, and the intermediate EMG/MRL for vinyl chloride. ATSDR Cancer Risk Evaluation Guide (CREG).
Bold values indicated concentrations reported above the laboratory reporting limit.

Appendix C: Chemical Interaction Analysis -

Because tetrachloroethene (PCE) and trichloroethene (TCE) share similar toxicity endpoints; namely nervous system effects (NSE), cancer and noncancer liver or kidney effects, ATSDR recommends the use of Hazard Index (HI) to evaluate the whole mixture. For example,

$$HI_{NSE} = \frac{E_{PCE}}{RfC_{PCE}} + \frac{E_{TCE}}{RfC_{TCE}}$$

where, HI_{NSE} is the hazard index for nervous system effects (the most sensitive biological endpoint), E_{PCE} is the mean indoor air exposure dose to PCE (expressed in the same units as the corresponding RfC), RfC_{PCE} is the EPA RfC for residential indoor air (40 µg/m^3), and so forth.

Preliminary evidence that an exposure to the mixture may constitute a hazard is provided when the HI for a particular exposure scenario and health endpoint exceeds 1. In practice, concern for the possibility of a health hazard increases with increasing value of the HI above 1.

Table C.1: Hazard indexes for nervous system effect components (PCE and TCE) individually and combined from past exposures.

Residence	Exposure $Dose_{PCE}/RfC_{PCE}$	Exposure $Dose_{TCE}/RfC_{TCE}$	Hazard Index
House #68	0.009	4.65	4.66
House # 171	15.65	0.23	15.88

Appendix D: Cancer Risk Evaluation

Cancer Risks

Exposure to a cancer-causing chemical, even at low concentrations, is assumed to be associated with some increased risk for evaluation purposes. The estimated risk for developing cancer from exposure to contaminants associated with breathing indoor air in the Settlers Grove subdivision was calculated by multiplying the site-specific doses by EPA's chemical-specific inhalation unit risks (IURs) available at *www.epa.gov/iris*. This calculation estimates an excess cancer risk expressed as a proportion of the population that may be affected by a carcinogen during a lifetime of exposure. For example, an estimated risk of 1×10^{-6} predicts the probability of one additional cancer over background in a population of 1 million. An increased lifetime cancer risk is not a specified estimate of expected cancers. Rather, it is an estimate of the increase in the probability that a person may develop cancer sometime in his or her lifetime following exposure to a particular contaminant under specific exposure scenarios. For children, the estimated excess cancer risk is not calculated for a lifetime of exposure, but from a fraction of lifetime; based on known or suspected length of exposure, or years of childhood.

When there is sufficient weight of evidence to conclude that a carcinogen operates through a mutagenic mode of action, and in the absence of chemical-specific data on age-specific susceptibility, EPA's *Supplemental Guidance for Assessing Susceptibility from Early-Life Exposure to Carcinogens* [EPA, 2005] advises that increased early-life susceptibility be assumed and recommends that default age-dependent adjustment factors (ADAFs) be applied to adjust for this potential increased susceptibility from early-life exposure. The current ADAFs and their age groupings are 10 for <2 years, 3 for 2–<16 years, and 1 for ≥16 years [EPA, 2005]. For risk assessments based on specific exposure assessments, the 10- and 3-fold adjustments to the slope factor or unit risk estimates are to be combined with age-specific exposure estimates when estimating cancer risks from early-life (<16-years-of-age) exposure. Currently, due to lack of appropriate data, no ADAFs are used for other life stages, such as the elderly.

In the case of TCE, the inhalation unit risk and oral slope factor estimates reflect lifetime risk for cancer at multiple sites, and a mutagenic mode of action has been established for one of these sites, the kidney. As provided in Table D.1, it is assumed that an individual is exposed to the EPA RSL of 2.1 $\mu g/m^3$ of TCE in air from birth through age 78 years. The steps in the calculation are as follows:

1. Separate the kidney cancer contribution from the non-Hodgkin's lymphoma (NHL) + liver cancer contribution to the inhalation unit risk estimate. From Section 5.2.2.1.4 [EPA 2011], the kidney lifetime unit risk is 1.0×10^{-6} per $\mu g/m^3$ in air. Subtracting this from the total lifetime unit risk of 4.1×10^{-6} per $\mu g/m^3$ from Section 5.2.2.2 [EPA 2011] results in the estimated contribution of NHL + liver cancer being 3.1×10^{-6} per $\mu g/m^3$.

2. Assign a lifetime unit risk estimate for each age group, along with the age group duration (Column D), and the fraction of lifetime each age group represents (Column E; used as a duration adjustment). For each age group, the (unadjusted) lifetime unit risk estimates for kidney cancer, total cancer, and NHL + liver cancer are shown in Column F, I, and J, respectively.

3. For each age group, the kidney cancer inhalation unit risk estimate (Column F) is multiplied by the risk per $\mu g/m^3$ equivalence (Column B), the exposure concentration (Column C), the duration adjustment (Column E), and the ADAF (Column G), to obtain the partial risk from exposure during those ages (Column H). For inhalation exposures, a risk per $\mu g/m^3$ equivalence of 1 is assumed across age groups (i.e., equivalent risk from equivalent exposure levels in air, independent of body size), as shown in Column B. In this calculation, a unit lifetime exposure of 1 $\mu g/m^3$ is assumed, as shown in Column C.

4. For each age group, the NHL + liver cancer unit risk estimate (Column J) is multiplied by the risk per $\mu g/m^3$ equivalence (Column B), the exposure concentration (Column C), and the duration adjustment (Column E), to obtain the partial risk from exposure during those ages (Column K).

5. For each age group, the ADAF-adjusted partial risk for kidney cancer (Column H) is added to the partial risk for NHL + liver cancer (Column K), resulting in the total partial risk (Column L).

Table D.1: Calculation for total lifetime cancer risk based on the kidney unit risk estimate, potential risk for NHL and liver cancer, and potential increased early-life susceptibility, assuming a constant lifetime exposure to the EPA RSL of 2.1 µg/m^3 of TCE in air.

Inhalation (concentration-equivalence across age groups)

Col A	Col B	Col C	Col D	Col E	Col F	Col G	Col H	Col I	Col J	Col K	Col L
	Exposure scenario parameters				Dose-response assessment calculations						
Units:		(µg/m^3 air)	yr	-	(µg/m^3 air)$^{-1}$	-	-	(µg/m^3 air)$^{-1}$	(µg/m^3 air)$^{-1}$	-	-
Age group	risk per µg/m^3 air equivalence	Exposure concentration	Age group duration	Duration adjustment (Col D / 78 yr)	Kidney unadjusted lifetime unit risk (p 5-137 [5.2.2.1.4])	Kidney cancer default ADAF	**Kidney ADAF-adjusted partial risk (Col B x Col C x Col E x Col F x Col G)**	Kidney+NHL+ liver unadjusted lifetime unit risk (p 5-139 [5.2.2.2])	NHL+ liver lifetime unit risk (Col I – Col F)	**NHL and liver partial risk (Col B x Col C x Col E x Col J)**	**Total partial risk (Col H + Col K)**
Birth to <1 year	1	2.100	1.000	0.0128	1.0E-06	10	**2.7E-07**	4.1E-06	3.1E-06	**8.3E-08**	**3.5E-07**
1 to <2 years	1	2.100	1.000	0.0128	1.0E-06	10	**2.7E-07**	4.1E-06	3.1E-06	**8.3E-08**	**3.5E-07**
2 to <6 years	1	2.100	4.000	0.0513	1.0E-06	3	**3.2E-07**	4.1E-06	3.1E-06	**3.3E-07**	**6.6E-07**
6 to <11 years	1	2.100	5.000	0.0641	1.0E-06	3	**4.0E-07**	4.1E-06	3.1E-06	**4.2E-07**	**8.2E-07**
11 to <16 years	1	2.100	5.000	0.0641	1.0E-06	3	**4.0E-07**	4.1E-06	3.1E-06	**4.2E-07**	**8.2E-07**
16 to <21 years	1	2.100	5.000	0.0641	1.0E-06	1	**1.3E-07**	4.1E-06	3.1E-06	**4.2E-07**	**5.5E-07**
21 to <78 years	1	2.100	57.000	0.7308	1.0E-06	1	**1.5E-06**	4.1E-06	3.1E-06	**4.8E-06**	**6.3E-06**
										Total unit risk:	**9.8E-06**

ADAF: Age Dependent Adjustment Factor
NHL: Non-Hodgkin's Lymphoma

As provided in Table D.2, it is assumed that an individual is exposed to the average concentration of TCE found in indoor air (9.3 µg/m^3) at House #68 from birth through age 33 years.

Table D.2: Calculation for total cancer risk from birth to age 33 based on the kidney unit risk estimate, potential risk for NHL and liver cancer, and potential increased early-life susceptibility, assuming constant exposure to 9.3 µg/m^3 of TCE in air.

Inhalation (concentration-equivalence across age groups)

Col A	Col B	Col C	Col D	Col E	Col F	Col G	Col H	Col I	Col J	Col K	Col L
	Exposure scenario parameters				Dose-response assessment calculations						
Units:		(µg/m^3 air)	yr	-	(µg/m^3 air)$^{-1}$	-	-	(µg/m^3 air)$^{-1}$	(µg/m^3 air)$^{-1}$	-	-
Age group	risk per µg/m^3 air equivalence	Exposure concentration	Age group duration	Duration adjustment (Col D / 78 yr)	Kidney unadjusted lifetime unit risk (p 5-137 [5.2.2.1.4])	Kidney cancer default ADAF	**Kidney ADAF-adjusted partial risk (Col B x Col C x Col E x Col F x Col G)**	Kidney+NHL+ liver unadjusted lifetime unit risk (p 5-139 [5.2.2.2])	NHL+ liver lifetime unit risk (Col I – Col F)	**NHL and liver partial risk (Col B x Col C x Col E x Col J)**	**Total partial risk (Col H + Col K)**
Birth to <1 year	1	9.300	1.000	0.0128	1.0E-06	10	**1.2E-06**	4.1E-06	3.1E-06	**3.7E-07**	**1.6E-06**
1 to <2 years	1	9.300	1.000	0.0128	1.0E-06	10	**1.2E-06**	4.1E-06	3.1E-06	**3.7E-07**	**1.6E-06**
2 to <6 years	1	9.300	4.000	0.0513	1.0E-06	3	**1.4E-06**	4.1E-06	3.1E-06	**1.5E-06**	**2.9E-06**
6 to <11 years	1	9.300	5.000	0.0641	1.0E-06	3	**1.8E-06**	4.1E-06	3.1E-06	**1.8E-06**	**3.6E-06**
11 to <16 years	1	9.300	5.000	0.0641	1.0E-06	3	**1.8E-06**	4.1E-06	3.1E-06	**1.8E-06**	**3.6E-06**
16 to <21 years	1	9.300	5.000	0.0641	1.0E-06	1	**6.0E-07**	4.1E-06	3.1E-06	**1.8E-06**	**2.4E-06**
21 to <33 years	1	9.300	12.000	0.1538	1.0E-06	1	**1.4E-06**	4.1E-06	3.1E-06	**4.4E-06**	**5.9E-06**
										Total unit risk:	**2.2E-05**

ADAF: Age Dependent Adjustment Factor
NHL: Non-Hodgkin's Lymphoma

As provided in Table D.3, it is assumed that an individual is exposed to the average concentration of TCE found in indoor air (9.3 µg/m^3) at House #68 for 33 years as an adult.

Table D.3: Calculation for total cancer risk based on the kidney unit risk estimate, potential risk for NHL and liver cancer, and potential increased early-life susceptibility, for adults occupying House #68 for 33 years assuming constant exposure to 9.3 µg/m^3 of TCE in air.

Inhalation (concentration-equivalence across age groups)

Col A	Col B	Col C	Col D	Col E	Col F	Col G	Col H	Col I	Col J	Col K	Col L
	Exposure scenario parameters				Dose-response assessment calculations						
Units:		(µg/m^3 air)	yr	-	(µg/m^3 air)$^{-1}$	-	-	(µg/m^3 air)$^{-1}$	(µg/m^3 air)$^{-1}$	-	-

Age group	risk per µg/m^3 air equivalence	Exposure concentration	Age group duration	Duration adjustment (Col D / 78 yr)	Kidney unadjusted lifetime unit risk (p 5-137 [5.2.2.1.4])	Kidney cancer default ADAF	**Kidney ADAF-adjusted partial risk (Col B x Col C x Col E x Col F x Col G)**	Kidney+NHL+ liver unadjusted lifetime unit risk (p 5-139 [5.2.2.2])	NHL+ liver lifetime unit risk (Col I – Col F)	**NHL and liver partial risk (Col B x Col C x Col E x Col J)**	**Total partial risk (Col H + Col K)**
21 to <54 years	1	9.300	33.000	0.4231	1.0E-06	1	**3.9E-06**	4.1E-06	3.1E-06	**1.2E-05**	**1.6E-05**
										Total unit risk:	**1.6E-05**

As provided in Table D.4, it is assumed that an individual is exposed to the average concentration of TCE found in indoor air (9.3 µg/m^3) at House #68 from birth to age 12.

Table D.4: Calculation for total cancer from birth to age 12 risk based on the kidney unit risk estimate, potential risk for NHL and liver cancer, and potential increased early-life susceptibility, assuming constant exposure to 9.3 µg/m^3 of TCE in air.

Inhalation (concentration-equivalence across age groups)

Col A	Col B	Col C	Col D	Col E	Col F	Col G	Col H	Col I	Col J	Col K	Col L
	Exposure scenario parameters				Dose-response assessment calculations						
Units:		(µg/m^3 air)	yr	-	(µg/m^3 air)$^{-1}$	-	-	(µg/m^3 air)$^{-1}$	(µg/m^3 air)$^{-1}$	-	-
Age group	risk per µg/m^3 air equivalence	Exposure concentration	Age group duration	Duration adjustment (Col D / 78 yr)	Kidney unadjusted lifetime unit risk (p 5-137 [5.2.2.1.4])	Kidney cancer default ADAF	**Kidney ADAF-adjusted partial risk (Col B x Col C x Col E x Col F x Col G)**	Kidney+NHL+ liver unadjusted lifetime unit risk (p 5-139 [5.2.2.2])	NHL+ liver lifetime unit risk (Col I – Col F)	**NHL and liver partial risk (Col B x Col C x Col E x Col J)**	**Total partial risk (Col H + Col K)**
Birth to <1 year	1	9.300	1.000	0.0128	1.0E-06	10	**1.2E-06**	4.1E-06	3.1E-06	**3.7E-07**	**1.6E-06**
1 to <2 years	1	9.300	1.000	0.0128	1.0E-06	10	**1.2E-06**	4.1E-06	3.1E-06	**3.7E-07**	**1.6E-06**
2 to <6 years	1	9.300	4.000	0.0513	1.0E-06	3	**1.4E-06**	4.1E-06	3.1E-06	**1.5E-06**	**2.9E-06**
6 to <11 years	1	9.300	5.000	0.0641	1.0E-06	3	**1.8E-06**	4.1E-06	3.1E-06	**1.8E-06**	**3.6E-06**
11 to <12 years	1	9.300	1.000	0.0128	1.0E-06	3	**3.6E-07**	4.1E-06	3.1E-06	**3.7E-07**	**7.3E-07**
										Total unit risk:	**1.0E-05**

ADAF: Age Dependent Adjustment Factor
NHL: Non-Hodgkin's Lymphoma

As provided in Table D.5, it is assumed that an individual is exposed to the average concentration of TCE found in indoor air (9.3 µg/m^3) at House #68 from birth to age 12.

Table D.5: Calculation for total cancer risk based on the kidney unit risk estimate, potential risk for NHL and liver cancer, and potential increased early-life susceptibility, for adults occupying the residence for 12 years assuming constant exposure to 9.3 µg/m^3 of TCE in air.

Inhalation (concentration-equivalence across age groups)

Col A	Col B	Col C	Col D	Col E	Col F	Col G	Col H	Col I	Col J	Col K	Col L
	Exposure scenario parameters				Dose-response assessment calculations						
Units:		(µg/m^3 air)	yr	-	(µg/m^3 air)$^{-1}$	-	-	(µg/m^3 air)$^{-1}$	(µg/m^3 air)$^{-1}$	-	-
Age group	risk per µg/m^3 air equivalence	Exposure concentration	Age group duration	Duration adjustment (Col D / 78 yr)	Kidney unadjusted lifetime unit risk (p 5-137 [5.2.2.1.4])	Kidney cancer default ADAF	**Kidney ADAF-adjusted partial risk (Col B x Col C x Col E x Col F x Col G)**	Kidney+NHL+ liver unadjusted lifetime unit risk (p 5-139 [5.2.2.2])	NHL+ liver lifetime unit risk (Col I – Col F)	**NHL and liver partial risk (Col B x Col C x Col E x Col J)**	**Total partial risk (Col H + Col K)**
21 to <33 years	1	9.300	12.000	0.1538	1.0E-06	1	**1.4E-06**	4.1E-06	3.1E-06	**4.4E-06**	**5.9E-06**
										Total unit risk:	**5.9E-06**

Tables D.6 through D.9 provide post-mitigation cancer risk estimates for residents living in House #68 and assumes that an individual is exposed to the average concentration of 0.65 µg/m^3. Both the RME (age 0 to 33 years, and 33 years as an adult) and CTE (age 0 to 12 years, and 12 years as an adult) are used for these cancer risk estimates.

Table D.6: Post-mitigation calculation for total cancer risk from birth to age 33 based on the kidney unit risk estimate, potential risk for NHL and liver cancer, and potential increased early-life susceptibility, assuming constant exposure to 0.65 µg/m³ of TCE in air.

Inhalation (concentration-equivalence across age groups)											
Col A	Col B	Col C	Col D	Col E	Col F	Col G	Col H	Col I	Col J	Col K	Col L
	Exposure scenario parameters				Dose-response assessment calculations						
Units:		(µg/m³ air)	yr	-	(µg/m³ air)⁻¹	-	-	(µg/m³ air)⁻¹	(µg/m³ air)⁻¹	-	-
Age group	risk per µg/m³ air equivalence	Exposure concentration	Age group duration	Duration adjustment (Col D / 78 yr)	Kidney unadjusted lifetime unit risk (p 5-137 [5.2.2.1.4])	Kidney cancer default ADAF	**Kidney ADAF-adjusted partial risk (Col B x Col C x Col E x Col F x Col G)**	Kidney+NHL+ liver unadjusted lifetime unit risk (p 5-139 [5.2.2.2])	NHL+ liver lifetime unit risk (Col I – Col F)	**NHL and liver partial risk (Col B x Col C x Col E x Col J)**	**Total partial risk (Col H + Col K)**
Birth to <1 year	1	0.650	1.000	0.0128	1.0E-06	10	**8.3E-08**	4.1E-06	3.1E-06	**2.6E-08**	**1.1E-07**
1 to <2 years	1	0.650	1.000	0.0128	1.0E-06	10	**8.3E-08**	4.1E-06	3.1E-06	**2.6E-08**	**1.1E-07**
2 to <6 years	1	0.650	4.000	0.0513	1.0E-06	3	**1.0E-07**	4.1E-06	3.1E-06	**1.0E-07**	**2.0E-07**
6 to <11 years	1	0.650	5.000	0.0641	1.0E-06	3	**1.3E-07**	4.1E-06	3.1E-06	**1.3E-07**	**2.5E-07**
11 to <16 years	1	0.650	5.000	0.0641	1.0E-06	3	**1.3E-07**	4.1E-06	3.1E-06	**1.3E-07**	**2.5E-07**
16 to <21 years	1	0.650	5.000	0.0641	1.0E-06	1	**4.2E-08**	4.1E-06	3.1E-06	**1.3E-07**	**1.7E-07**
21 to <33 years	1	0.650	12.000	0.1538	1.0E-06	1	**1.0E-07**	4.1E-06	3.1E-06	**3.1E-07**	**4.1E-07**
										Total unit risk:	**1.5E-06**

Table D.7: Post-mitigation calculation for total cancer risk based on the kidney unit risk estimate, potential risk for NHL and liver cancer, and potential increased early-life susceptibility, for adults occupying House #68 for 33 years assuming constant exposure to 0.65 µg/m³ of TCE in air.

Inhalation (concentration-equivalence across age groups)											
Col A	Col B	Col C	Col D	Col E	Col F	Col G	Col H	Col I	Col J	Col K	Col L
	Exposure scenario parameters				Dose-response assessment calculations						
Units:		(µg/m³ air)	yr	-	(µg/m³ air)⁻¹	-	-	(µg/m³ air)⁻¹	(µg/m³ air)⁻¹	-	-
Age group	risk per µg/m³ air equivalence	Exposure concentration	Age group duration	Duration adjustment (Col D / 78 yr)	Kidney unadjusted lifetime unit risk (p 5-137 [5.2.2.1.4])	Kidney cancer default ADAF	**Kidney ADAF-adjusted partial risk (Col B x Col C x Col E x Col F x Col G)**	Kidney+NHL+ liver unadjusted lifetime unit risk (p 5-139 [5.2.2.2])	NHL+ liver lifetime unit risk (Col I – Col F)	**NHL and liver partial risk (Col B x Col C x Col E x Col J)**	**Total partial risk (Col H + Col K)**
21 to <54 years	1	0.650	33.000	0.4231	1.0E-06	1	**2.8E-07**	4.1E-06	3.1E-06	**8.5E-07**	**1.1E-06**
										Total unit risk:	**1.1E-06**

Table D.8: Post-mitigation calculation for total cancer from birth to age 12 risk based on the kidney unit risk estimate, potential risk for NHL and liver cancer, and potential increased early-life susceptibility, assuming constant exposure to 0.65 µg/m³ of TCE in air.

Inhalation (concentration-equivalence across age groups)											
Col A	Col B	Col C	Col D	Col E	Col F	Col G	Col H	Col I	Col J	Col K	Col L
	Exposure scenario parameters				Dose-response assessment calculations						
Units:		(µg/m³ air)	yr	-	(µg/m³ air)$^{-1}$	-	-	(µg/m³ air)$^{-1}$	(µg/m³ air)$^{-1}$	-	-
Age group	risk per µg/m³ air equivalence	Exposure concentration	Age group duration	Duration adjustment (Col D / 78 yr)	Kidney unadjusted lifetime unit risk (p 5-137 [5.2.2.1.4])	Kidney cancer default ADAF	**Kidney ADAF-adjusted partial risk (Col B x Col C x Col E x Col F x Col G)**	Kidney+NHL+ liver unadjusted lifetime unit risk (p 5-139 [5.2.2.2])	NHL+ liver lifetime unit risk (Col I – Col F)	**NHL and liver partial risk (Col B x Col C x Col E x Col J)**	**Total partial risk (Col H + Col K)**
21 to <54 years	1	0.650	33.000	0.4231	1.0E-06	1	**2.8E-07**	4.1E-06	3.1E-06	**8.5E-07**	**1.1E-06**
										Total unit risk:	**1.1E-06**

Table D.9: Post-mitigation calculation for total cancer risk based on the kidney unit risk estimate, potential risk for NHL and liver cancer, and potential increased early-life susceptibility, for adults occupying the residence for 12 years assuming constant exposure to 0.65 µg/m³ of TCE in air.

Inhalation (concentration-equivalence across age groups)											
Col A	Col B	Col C	Col D	Col E	Col F	Col G	Col H	Col I	Col J	Col K	Col L
	Exposure scenario parameters				Dose-response assessment calculations						
Units:		(µg/m³ air)	yr	-	(µg/m³ air)$^{-1}$	-	-	(µg/m³ air)$^{-1}$	(µg/m³ air)$^{-1}$	-	-
Age group	risk per µg/m³ air equivalence	Exposure concentration	Age group duration	Duration adjustment (Col D / 78 yr)	Kidney unadjusted lifetime unit risk (p 5-137 [5.2.2.1.4])	Kidney cancer default ADAF	**Kidney ADAF-adjusted partial risk (Col B x Col C x Col E x Col F x Col G)**	Kidney+NHL+ liver unadjusted lifetime unit risk (p 5-139 [5.2.2.2])	NHL+ liver lifetime unit risk (Col I – Col F)	**NHL and liver partial risk (Col B x Col C x Col E x Col J)**	**Total partial risk (Col H + Col K)**
21 to <33 years	1	0.650	12.000	0.1538	1.0E-06	1	**1.0E-07**	4.1E-06	3.1E-06	**3.1E-07**	**4.1E-07**
										Total unit risk:	**4.1E-07**

Table D.10: Cumulative Cancer Risks for Adults Occupying All Other Residences with Indoor Air Concentrations of Contaminants detected above a CREG

Residence	Cumulative Cancer Risk			
	TCE	PCE	Using RME	Using CTE
House #3	1.3×10^{-6}	CREG not exceeded	1.3×10^{-6}	4.9×10^{-7}
House #3*	1.1×10^{-6}	CREG not exceeded	1.1×10^{-6}	4.1×10^{-7}
House #7	9.0×10^{-7}	2.35×10^{-6}	3.35×10^{-6}	1.17×10^{-6}
House #9	9.40×10^{-7}	CREG not exceeded	9.40×10^{-7}	3.4×10^{-7}
House #11	5.7×10^{-7}	CREG not exceeded	5.7×10^{-7}	1.9×10^{-7}
House #12	2.0×10^{-7}	CREG not exceeded	2.0×10^{-7}	7.3×10^{-8}
House #16	1.0×10^{-6}	CREG not exceeded	1.0×10^{-6}	3.7×10^{-7}
House #62	6.20×10^{-7}	CREG not exceeded	6.20×10^{-7}	2.3×10^{-7}
House #69	6.20×10^{-7}	CREG not exceeded	6.20×10^{-7}	2.3×10^{-7}
House #72	2.5×10^{-6}	CREG not exceeded	2.5×10^{-6}	9.0×10^{-7}
House #171	CREG not exceeded	6.84×10^{-5}	6.84×10^{-5}	2.44×10^{-5}
House #174	8.3×10^{-7}	CREG not exceeded	8.3×10^{-7}	3.0×10^{-7}

***Post-mitigation**

Note: The respective ATSDR CREGs for TCE and PCE are: 0.24 $\mu g/m^3$ and 3.8 $\mu g/m^3$. For TCE, the estimated cancer risk is based on the mean TCE concentration present in indoor air for available sample results and includes estimated cancer risk based on the kidney unit risk estimate, potential risk for NHL and liver cancer, and potential increased early-life susceptibility. For PCE, the estimated cancer risk is based on the mean PCE concentration present in indoor air for available sample results. The EPA inhalation unit risks (IURs) of 4.1×10^{-6} $(\mu g/m^3)^{-1}$ and 2.6×10^{-7} $(\mu g/m^3)^{-1}$ for PCE were used in the cancer risk calculations. Cancer risk estimations are provided for both the RME (33 years) and CTE (12 years).

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