ATSDR
AGENCY FOR TOXIC SUBSTANCES AND DISEASE REGISTRY

Public Health Assessment for

MCGAFFEY AND MAIN GROUNDWATER PLUME SITE

ROSWELL, NEW MEXICO

EPA FACILITY ID: NM0000605386

JUNE 15, 2015

U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES
PUBLIC HEALTH SERVICE
Agency for Toxic Substances and Disease Registry

THE ATSDR PUBLIC HEALTH ASSESSMENT: A NOTE OF EXPLANATION

This Public Health Assessment was prepared by ATSDR pursuant to the Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA or Superfund) section 104 (i)(6) (42 U.S.C. 9604 (i)(6)), and in accordance with our implementing regulations (42 C.F.R. Part 90). In preparing this document, ATSDR has collected relevant health data, environmental data, and community health concerns from the Environmental Protection Agency (EPA), state and local health and environmental agencies, the community, and potentially responsible parties, where appropriate.

In addition, this document has previously been provided to EPA and the affected states in an initial release, as required by CERCLA section 104 (i)(6)(H) for their information and review. The revised document was released for a 30-day public comment period. Subsequent to the public comment period, ATSDR addressed all public comments and revised or appended the document as appropriate. The public health assessment has now been reissued. This concludes the public health assessment process for this site, unless additional information is obtained by ATSDR which, in the agency's opinion, indicates a need to revise or append the conclusions previously issued.

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PUBLIC HEALTH ASSESSMENT

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Prepared by:

Central Branch
Division of Community Health Investigations
Agency for Toxic Substances and Disease Registry

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Summary

The Public Health Issues

The U.S. Environmental Protection Agency (EPA) maintains a list of hazardous waste sites across the United States. This list of hazardous waste sites is called the National Priorities List (NPL) or Superfund and helps guide environmental and public health activities to stop or reduce exposure to hazardous waste and to introduce activities to protect public health. The McGaffey and Main Groundwater Plume Site is one such site. This NPL site is located within the city limits of Roswell, Chaves County, New Mexico and consists of soil, air, and groundwater contamination at the intersection of South Main Street and McGaffey Street along with groundwater contamination that extends from this intersection to the southeast for approximately 7,500 feet to the intersection of South Atkinson Street and East Poe Street. Because the site is predominantly groundwater contamination, no visible signs of a site boundary exist. The areas above the estimated groundwater contamination are a mix of commercial, industrial and residential land uses.

This public health assessment will address the following public health questions:

- Are people being exposed to chemicals in soil, air, or groundwater from the McGaffey and Main Site?
- If so, are people being exposed at levels that might harm their health?
- Are there any public health actions that need to be taken by local, state, or federal agencies because of the McGaffey and Main Site?

The McGaffey and Main Site contaminant source area is located within the city limits of Roswell, Chaves County, New Mexico and the resulting plume of groundwater contaminants extends approximately 3.5 miles south and southeastward into unincorporated portions of Chaves County. Analyses of groundwater samples from residential wells and indoor air at buildings near the site have detected low concentrations of several volatile organic chemicals (VOCs). Tetrachloroethylene (PCE) and trichloroethylene (TCE) were the only compounds detected at concentrations above their respective health screening values.

Within the source area, some portion of the PCE/TCE contamination migrates upward via vapor intrusion into buildings. PCE air concentrations in the affected buildings were due to both vapor intrusion and fugitive emissions from a nearby operating dry cleaner. Until recent EPA building remediation, people working in those buildings were exposed by breathing the PCE/TCE from vapor intrusion and PCE from fugitive emissions; exposure to PCE from fugitive air emissions may be ongoing.

The PCE and TCE also migrated via groundwater to private wells where residents of fourteen houses may have been exposed by ingestion, inhalation, and skin contact when they used contaminated water for drinking water and other household uses.

Conclusions and Recommendations

Based on the available data and above findings ATSDR concludes that:

1. Past, present, and future inhalation exposures to PCE and TCE in indoor air in buildings adjacent to the McGaffey and Main source area are not a public health hazard.

   Basis for Conclusion: Inhalation of PCE and TCE-contaminated indoor air in buildings adjacent to the McGaffey and Main Site source area is a completed pathway of exposure. Sources of PCE include both vapor intrusion from a historic subsurface plume and fugitive emissions from a currently operating dry cleaner. TCE is only sourced from the subsurface contaminant plume. Based on the available measured PCE and TCE concentrations and estimated doses, occupational exposure via vapor intrusion and fugitive emissions to indoor air in commercial buildings adjacent to the McGaffey and Main site is unlikely to produce any harmful health effects, including cancer.

   Next Steps: Current and future PCE/TCE exposures from vapor intrusion have been mitigated by installation of a soil gas extraction and treatment system in the affected buildings. PCE exposures via fugitive emissions from the dry cleaning facility are ongoing. No additional public health actions are necessary.

2. Until 1995, at least one family used PCE and TCE-contaminated water from their private well for bathing and drinking. Based on available data (post-1995), the cumulative PCE and TCE exposures (via the private well) are unlikely to cause any harmful health effects, including cancer. However, because PCE and TCE concentrations before 1995 could have been higher than those measured post-1995, ATSDR cannot determine if these past exposures were at harmful levels.

   Basis for Conclusion: These past exposures for adults and children from this contaminated water (which stopped in 1995) occurred from drinking the water, breathing the contaminants released into indoor air (from the water), and absorbing the contaminants through their skin while bathing or showering. Past exposure to a child via the contaminated residential water well near the McGaffey and Main site for 16 years or more would have no apparent increased risk of cancer. An adult at this location for 16 years or more would have no increased risk of cancer. Non-cancer health effects from PCE exposure are unlikely for children or adults at this location.

Next Steps: No additional actions are necessary with regard to the past drinking water exposures (which stopped in 1995). Three well surveys have determined that no current residents are exposed to contaminated groundwater. ATSDR recommends that appropriate restrictions are enacted to prevent new drinking water wells in the area of groundwater contamination.

3. PCE and TCE contamination in public (or community) water systems do not present a public health hazard.

   Basis for Conclusion: Monitoring data from the public (community) water systems indicate that contaminant concentrations are below health screening values.

   Next Steps: Continue monitoring as required by the Safe Drinking Water Act.

Public Health Action Plan

As part of ATSDR's investigation at hazardous waste sites, ATSDR works with state, local, and other federal agencies to develop a public health action plan for a site. The intent of the public health action plan is to encourage actions be taken to protect public health. For this site, the groundwater exposures of primary public health concern stopped in 1995 and vapor intrusion remedial actions were completed or are ongoing. Consequently, ATSDR does not anticipate any future public health actions at this site.

Introduction and Public Health Issues

Introduction

As described in the preceding **Foreword**, ATSDR is required by law to conduct a public health assessment at sites on (or proposed for) the U.S. EPA NPL. This public health assessment (PHA) describes ATSDR's assessment of the McGaffey and Main Groundwater Contamination Site (henceforth referred to as the McGaffey and Main Site or the site) in Roswell, New Mexico. The purpose of this PHA is to present ATSDR's evaluation and conclusions on the potential public health hazards from contamination associated with this site.

This introductory section of the PHA presents the specific public health issues and community health concerns related to this site. These issues and concerns are used to frame the PHA and ensure that this document addresses and answers the public health questions of people living in the vicinity of the McGaffey and Main Groundwater Plume site.

Following this introduction is a **Background Section** summarizing a description and history of the site, the demographic character of the surrounding community, and aspects of site geology and hydrogeology. This background information is important because it establishes the timing of contaminant releases, describes the people that may be exposed, and the processes that determine contaminant migration and exposure.

The next section, **Pathways of Exposure and Environmental Sampling Results**, describes the processes of contaminant migration from the source area to locations where people may be exposed and the contaminant levels that have been measured in those locations. This section also identifies the specific contaminants that people may be exposed to and whether those contaminants are present at levels of public health concern.

Assuming that people are exposed (or potentially exposed) to contaminants at levels of health concern, the following **Public Health Implications** section discusses those exposures in relation to the known health effects for each contaminant of concern. Finally, the **Conclusions, Recommendations, and Public Health Action Plan** section presents ATSDR's determination of the public health hazard posed by exposure to site-related contaminants and recommendations for preventing or reducing such hazards.

Public Health Issues and Community Health Concerns

ATSDR has identified the following public health issues that will be investigated as part of the public health assessment process:

- Are people being exposed to chemicals from the McGaffey and Main Site?
- If so, are people being exposed at levels that might harm their health?
- Are there any public health actions that need to be taken by local, state, or federal agencies because of the McGaffey and Main Site?

On October 22, 2002, ATSDR held a public availability session at the Roswell Adult and Recreation Center on Missouri Street to learn the health concerns of local residents who might have been affected by the site. During the meeting, ATSDR gave a short presentation explaining the public health assessment process, what a public health assessment is, and a brief history of the site. Several residents attending this public meeting voiced the following concerns:

1. **Concern**: One resident reported having multiple chemical sensitivities and wanted to know if the McGaffey and Main Site could have contributed to her condition.

   ATSDR response: Since the resident did not live in the affected area, she could not have been exposed to PCE and TCE from the site. Therefore, whatever illnesses she might have could not be attributed to the McGaffey and Main Site.

2. **Concern**: One resident was concerned that his family might be at risk of cancer from the McGaffey and Main Site.

   ATSDR response: Since the resident uses city water, he and his family could not have been exposed to PCE and TCE from the McGaffey and Main Site. Therefore, he and his family are not at increased risk of cancer because of the McGaffey and Main Site. ATSDR has made recommendations to EPA about investigating the public health significance of possible soil gas migration in businesses and residences near the McGaffey and Main Street intersection.

3. **Concern**: One resident had questions about a site in Arkansas, which he visited during the summer with his parents.

   ATSDR response: ATSDR talked with the resident and gave him contact information at ATSDR where he could get his health concerns answered.

A glossary of environmental health terms can be found in Appendix A of the public health assessment.

A public comment version of this PHA was released on August 19, 2014. No comments were received from the community or State and local Agency representatives.

Background

Site Description

The McGaffey and Main Site contaminant source area is located within the city limits of Roswell, Chaves County, New Mexico and the resulting plume of groundwater contaminants extends south and southeastward into unincorporated portions of Chaves County. The source area is characterized by air and groundwater contamination at the intersection of South Main Street and McGaffey Street. Groundwater contamination also extends from to the southeast for approximately 3.5 miles. Figure 1 shows the city limits of Roswell and a rough estimate of the boundaries of the contaminated groundwater plume. Because the site is predominantly groundwater contamination, no visible signs of a site boundary exist. The area above the estimated groundwater contamination is mix of commercial, industrial, agricultural, and residential land uses (CH2M Hill, 2003a).

Demographics

Figure 1 shows the location of the source area, the resulting down-gradient groundwater plume (from URS, 2012), and the population characteristics within the plume area and for a one mile buffer around the plume. There are about 2,000 people living within the plume area (approximately 600 housing units) and the population is predominantly Hispanic (about 78%) with small percentages of African-Americans, American Indians, and Asians. There are about 200 children younger than 6 years, about 250 people over 65 years, and about 350 women between the ages of 15 and 44.

Site History

Several dry cleaning facilities used to operate on South Main Street and McGaffey Street and discharges from these facilities are believed to be the source of groundwater contamination with PCE. Other names for PCE include perchloroethylene, PERC, perchloroethene, and tetrachloroethene. This report will use the abbreviation PCE. One dry cleaning business (operating from 1955 to 1964) located at 1107 South Main Street was reported to have dumped large quantities of PCE in back of the cleaners as well as down the sewer lines, which may have leaked into surrounding soils (NMED 2001). The first indication of groundwater contamination with PCE occurred when monitoring wells near the intersection of McGaffey Street and South Main Street were sampled in 1994 by the New Mexico Environment Department (NMED). At the time, NMED was investigating a nearby leaking underground storage tank at a former Pepsi Cola bottling facility.

After finding PCE in monitoring wells, NMED sampled nearby private wells in 1994 and found several to be contaminated with PCE. Private wells at residences or businesses may be used as a source of drinking water, crop or livestock irrigation, or other purposes. These wells typically do

not include any type of water treatment or water quality testing prior to use. Municipal or public water supply wells, such as those operated by the City of Roswell or the Berrendo Water Cooperative, have specific water quality treatment and testing requirements as specified by the Safe Drinking Water Act.

If these private wells were used for cooking and drinking, the affected properties were connected to the municipal water (NMED 1996). By March 1996, NMED located and sampled numerous private wells down gradient from the intersection of McGaffey Street and South Main Street. A timeline of EPA and NMED activities at this site is presented in Appendix B.

As a result of these investigations, EPA proposed the McGaffey and Main Site to the NPL in September 2001 and listed the site on the NPL in October 2002. The site was listed because PCE was detected in groundwater underlying portions of downtown Roswell. In February 2003, the EPA released preliminary results of its remedial investigation of the site, which was designed to determine the extent of contamination at the McGaffey and Main Site.

EPA also collected soil samples from 35 borings in the parking area for 1107 South Main Street to determine the extent of PCE-contaminated soil. In addition, 8 new monitoring wells were installed and sampled along with the 20 existing monitoring wells and 10 private wells in order to determine the nature and extent of the groundwater contamination (CH2M Hill, 2003a).

As a result of the expanded groundwater monitoring, the known down-gradient extent of the contaminant plume has been extended to the south and southeast (Figure 2; CH2M Hill, 2008; URS, 2012).

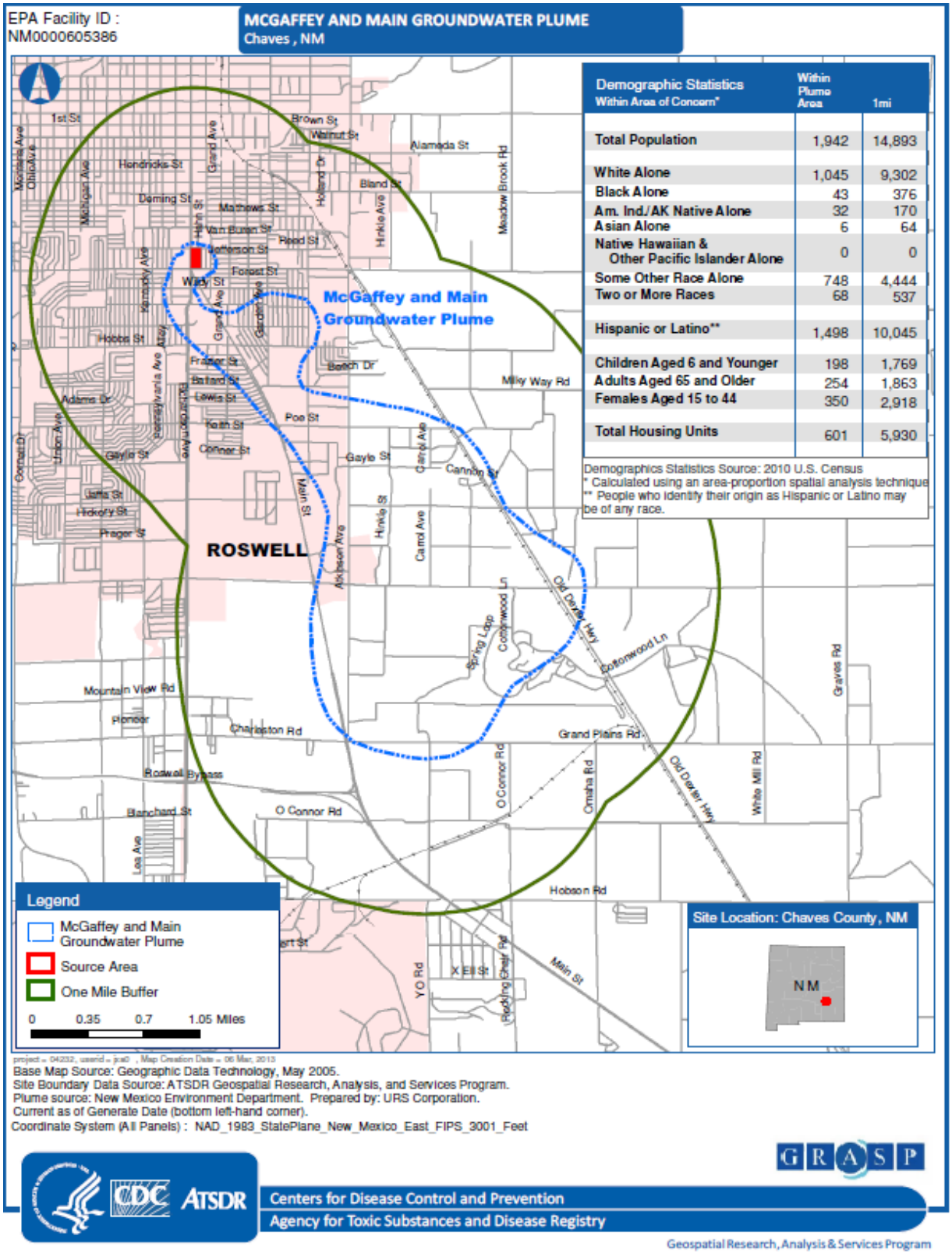

Figure 1. McGaffey and Main Groundwater Plume location and population characteristics.

Groundwater Well Surveys

Three well surveys have been conducted at the site to locate private (groundwater) wells on or near the site, to determine how wells are used, and to decide if these wells could be used to determine the extent of contaminated groundwater associated with the site. NMED conducted the first well survey in 1994 focusing on areas southeast of the site and identified 18 homes with private wells. In May 2002, EPA and NMED conducted another survey to identify homes near the site that had private wells. The 2002 survey covered an area with about 300 homes, and EPA and NMED talked to residents in about 150 of these 300 homes. EPA and NMED left a questionnaire at the remaining homes when people were not available (CH2M Hill, 2003a; 2003b).

The following procedures were followed when conducting the 2002 survey:

- If residents had a well, they were asked to fill out a questionnaire about well usage,
- If permission was granted, private wells were inspected,
- The water from some private wells was collected and sent for analysis.

It appears that only a few people who were not interviewed returned the questionnaire. However, at two properties where questionnaires were left, two people did call EPA with questions (CH2M Hill 2003b).

From information gathered during the 2002 well survey, EPA determined that about 75 residences are connected to city water while the remaining 75 households obtain drinking water through a cooperative service. Twenty-five private wells were identified during the 2002 survey, and 7 of these 25 wells were either not operable or had an unknown operating status because residents were not home to answer questions. From the 2002 survey results, the following statements can be made about how people use their private wells:

- One private well was used for drinking water,
- Fourteen private wells were used to water gardens, livestock, and pets,
- Four private wells were not used for any purpose (CH2M Hill 2003b).

The questionnaire did not ask whether or not residents with private wells used their wells for bathing. However, at least one resident reported during the 2002 survey that water from the well was used for showering. The water from some wells also may have been used to fill swimming pools. An interesting point from the 2002 survey is that several wells were rebuilt or installed in the last few years (CH2M Hill 2003b). In response to recent groundwater monitoring data showing ongoing southeastward contaminant migration, NMED commissioned an expanded well survey (AEA, 2012). The expanded well survey used the NM Office of the State Engineer

resources and County Tax Assessor information to identify potentially affected wells and well ownership and uses. The electronic record searches were followed by field surveys to verify well locations, ownership, and other information (AEA, 2012). The 2012 well survey identified 76 potentially affected wells in the expanded area of interest, including one municipal use well, 41 domestic use (private) wells, 28 irrigation wells, and 6 wells with unknown use (AEA, 2012).

Hydrogeology

EPA investigated the hydrogeology beneath the site by using monitoring wells installed by NMED and by installing their own monitoring wells (CH2M Hill 2003a). Groundwater beneath the site consists of two water-bearing soil layers separated by a non-continuous clay layer that allows groundwater to flow between the two aquifers. At 1107 South Main, the shallow aquifer begins about 45 feet below the surface and is about 150 feet thick. The shallow aquifer is also referred to as the alluvial aquifer and is made up of clay, silt, sand, and gravel. The shallow aquifer can be further divided into the 3 water-bearing zones (i.e., P1, P3, and P5). These permeable water-bearing zones are separated by clay layers (i.e., I2 and I4) that probably contain little to no water. The clay layers, however, are not continuous and so water is able to flow from one zone to the other in some areas. The bottom of the shallow aquifer is about 200 feet below the ground surface.

Between the shallow and deep aquifers is a layer of sandstone, siltstone, claystone, and carbonate rock known as the Red Beds or Grayburg aquitard. Beneath the site, the Red Beds is about 50 feet thick and occurs at a depth of about 150 to 200 feet. Since the Red Beds may not be continuous, this discontinuity may allow groundwater to flow between the shallow and deep aquifers in some areas. The deep aquifer begins about 200 feet below the surface in areas beneath the site and groundwater in the deep aquifer flows south and east toward the Pecos River. The deep aquifer is the primary source of water for the City of Roswell and is used for irrigation, municipal, and industrial purposes. Additional information about hydrogeology associated with the site can be found in EPA's Round 1 report (CH2M Hill 2003a).

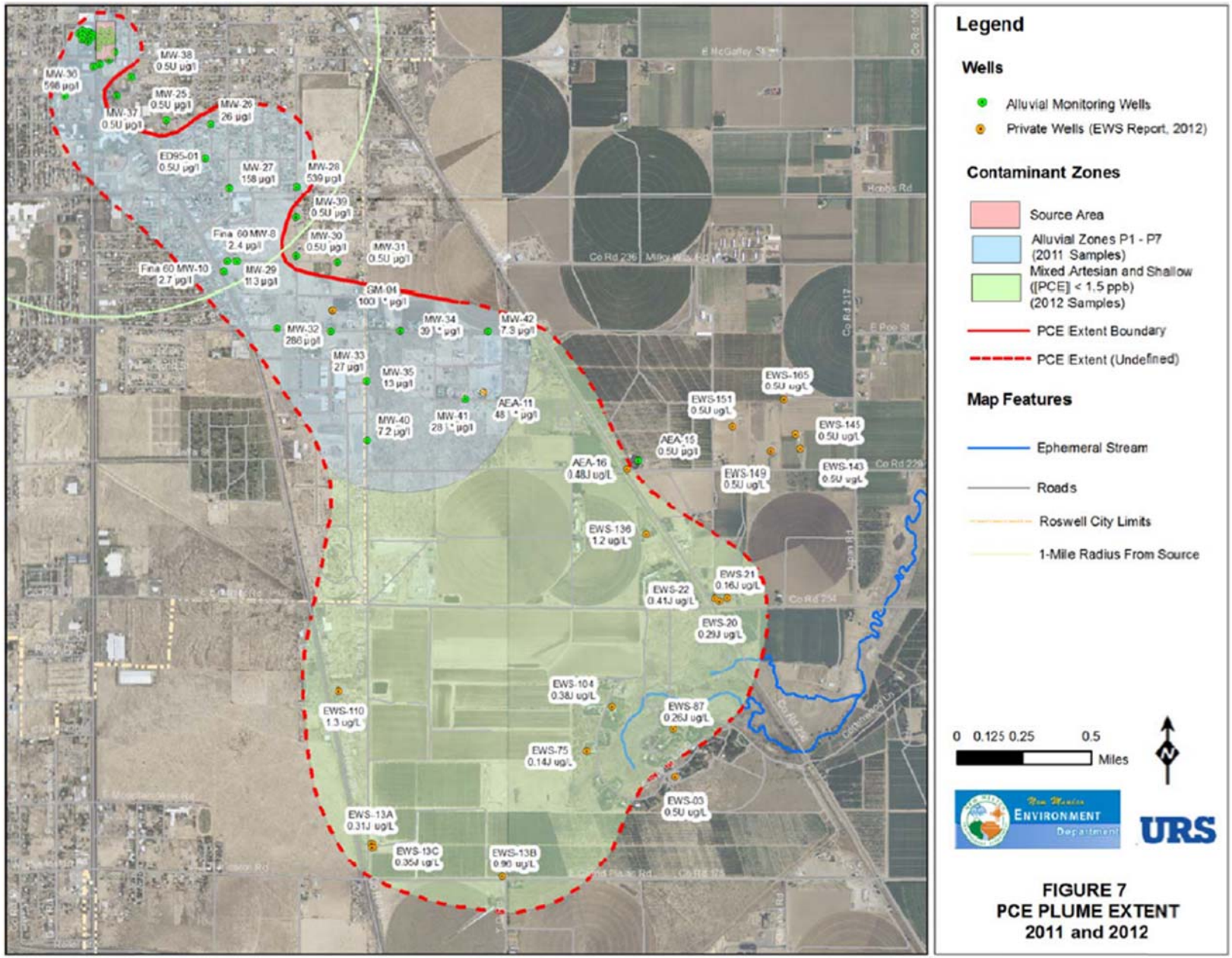

Figure 2. PCE plume (Figure 7 from URS, 2012).

Pathways of Exposure and Environmental Sampling Results

Pathways of Exposure

Historical dry cleaning operations released PCE into the subsurface. The source area is comprised of a contaminated area of approximately 5 acres and extends down to approximately 80 feet below ground surface (bgs; CH2M Hill, 2008). The subsurface source is located in the vicinity of a one block area bounded by Reed Street to the north, Hahn Street to the east, McGaffey Street to the south and the grocery store parking lot to the west. The highest concentrations were found under the alleyway beneath the source area in the vicinity of sewer piping. The most contaminated portion of groundwater in the source area is located just south of the former dry cleaners at 1107 South Main Street in the shallowest portion of the aquifer at approximately 65 feet bgs. The historic dry cleaning facility operated at this location from about1955 to 1963.

PCE, and associated contaminants such as TCE, cis 1,2-dichloroethene (1,2-DCE), and vinyl chloride are members of a class of chemicals called volatile organic compounds (VOCs). They are called "volatile" because, at standard atmospheric temperatures and pressures, a portion of these compounds will evaporate (or volatize) from a liquid into a gaseous, or airborne vapor. Another physical property of these VOCs is that in liquid form, their density is greater than water such that another portion of the VOCs will migrate downward through groundwater and may accumulate as a "dense non-aqueous phase layer" (DNAPL). Another portion of the VOCs will dissolve into groundwater and migrate down-gradient via groundwater flow. This portion of site contamination is represented by the PCE plume shown in Figures 1 and 2.

The physical properties of VOCs have specific implications for how these chemicals migrate in groundwater and air. Consequently, these physical properties determine how people may be exposed to these chemicals as they migrate away from the source area where they were released. Specifically, there are two pathways of exposure to PCE (and associated VOCs) at this site:

1) In the immediate vicinity of the source area (described above), the VOC concentrations in shallow groundwater may be high enough that volatized vapors may migrate upward through the soil and accumulate in overlying buildings. As a result, people living or working in those buildings will be exposed to the VOCs by breathing the air in those buildings. This pathway of exposure is called the *vapor intrusion pathway*.
2) People may also be exposed to the portion of the VOCs that dissolves into and migrates with groundwater via water wells located within the groundwater plume area (Figure 2). These exposures may occur from drinking the VOC-contaminated water, direct skin contact with the contaminated water, or breathing vapors that volatize from the contaminated water while showering or other household usage. This pathway of exposure is called the *groundwater pathway*.

It is important that near the source area, where vapor intrusion is occurring, people obtain their

drinking water from public water systems, such that exposure to contaminated groundwater does not occur. Conversely, in down-gradient areas, where groundwater exposures may be occurring, groundwater depths are too great (and VOC concentrations too low) for exposure via vapor intrusion.

The following section summarizing contaminant concentrations and distributions presents these results for each pathway of exposure. The units for reporting chemical levels in air and water can be confusing because the units of concentration are different. For water (private, public, and monitoring wells), chemical levels will be reported in micrograms per liter (µg/L) and for air the units are micrograms per cubic meter (µg/m^3). This system will allow the reader to easily distinguish between contaminant concentrations in water or air.[1]

Environmental Sampling Results

Vapor Intrusion Pathway

PCE and TCE vapors in affected buildings are from two sources. Historic dry cleaning operations released solvent liquids that seeped into subsurface soil and groundwater. Vapors from the subsurface liquids migrate upwards into outdoor and indoor air. In addition to the vapor intrusion source, airborne PCE emissions from a currently operating dry cleaner also contribute to vapor concentrations in the affected area and buildings. This health assessment evaluates potential health effects from breathing these vapors regardless of source.

Emissions from the currently operating dry cleaning facility at 1139 S. Main Street were evaluated using a tracer study in December 2004 (CH2M Hill, 2006). The results (Table 2) show that PCE in some buildings was wholly from the nearby operating dry cleaner, whereas other buildings had a combination of vapor intrusion and infiltration from the operating dry cleaner. Five commercial and two residential buildings were found to have the tracer and PCE in indoor air. PCE concentrations in the dry cleaner's exhaust ranged between 1,528 and 91,898 µg/m^3 and averaged 27,370 µg/m^3 over a 2-day, 8 AM to 5 PM monitoring period. The time-weighted 24-hour average PCE concentration emitted from the exhaust fan was calculated to be 14,180 µg/m^3 (CH2M Hill, 2006).

It should be noted that TCE and cis-1,2-DCE have only been detected in locations with a significant vapor intrusion source. These compounds may be produced as breakdown products of

[1] In water, the units of micrograms per liter (µg/L) are the same as parts per billion (ppb). A concentration of 10 µg/L PCE (in water) is the same as 10 ppb. In air, the mass to volume conversion must account for molecular weight and atmospheric pressure so the conversion between µg/m^3 and ppb is more complicated.

the natural degradation of PCE in the subsurface. Alternatively, they may have been present as minor constituents of the dry cleaning solvent used at the historic dry cleaning facility. Regardless of the original source of these VOCs, they are only detected at concentrations less than one percent of the PCE concentration and only in locations sourced via vapor intrusion.

Measured VOC Concentrations in Indoor Air

Indoor air measurements are being used to directly characterize indoor air levels and evaluate human exposures. EPA provided data summarizing indoor air sampling performed in spring (March-April 2003), fall (Oct 2003) and winter (Dec 2004). The sampling methods and results are presented in two reports (CH2M Hill, 2006; 2008). Most of the indoor air samples were collected as 24 hour time-integrated samples (eight samples from one location were collected as 8 hour time-integrated samples; CH2M Hill, 2006). The maximum and average indoor values are summarized in Table 1.[2] ATSDR health comparison values assume continuous exposure for 24 hours per day and 365 days per year. Contaminant concentrations from commercial buildings are adjusted for non-continuous, occupational exposures by multiplying measured air concentrations by 8/24 hours per day and 5/7 days per week. See Appendix D for more information.

The indoor air sample locations included five commercial buildings (with 10 sample locations) and four residential buildings (3 single family homes and one duplexes; with 7 sample locations). Two of the commercial buildings and one residence were sampled multiple times (October and December, 2014 sample events). Outdoor air samples were collected in March/April, 2003 and concurrently with the October and December, 2004 indoor samples (CH2M Hill, 2008).

PCE was the only VOC measured in residential buildings at concentrations above its comparison value (CV). PCE and TCE were measured above CVs in commercial buildings (Table 1). Overall, measured indoor PCE concentrations are at least 3 orders of magnitude greater than TCE or cis-1,2-DCE and vinyl chloride concentrations (Table 1). Vinyl chloride analyses were either below detection limits and/or screening values (Table 1). ATSDR and EPA have not established inhalation screening values for cis-1,2-DCE. However, the National Institute for Occupational Safety and Health has established a Recommended Exposure Limit for cis-1,2-DCE of 790,000 $\mu g/m^3$ for 8 hour occupational exposures (Table 1). As cis-1,2-DCE was not detected in any residential locations and the detections in commercial buildings were more than 200,000 times lower than the occupational limit, cis-1,2-DCE is not a contaminant of concern. Only PCE and TCE were detected at concentrations above their respective screening values. Consequently only exposures to PCE and TCE will be addressed in following sections.

Table 2 shows the relative PCE source contributions in different buildings (based on a 2004 tracer

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[2] The VOC concentrations listed in Tables 1 and 2 are presented in units of micrograms per cubic meter ($\mu g/m^3$). The VOC concentrations in the source documents (CH2MHill, 2006; 2008) are variously presented in units of $\mu g/m^3$ or as parts per billion by volume (ppbv). At standard temperatures and pressures the PCE conversion is 1 ppb = 6.78 $\mu g/m^3$; at the altitude of Roswell, NM, this conversion is 1 ppb = 6.13 $\mu g/m^3$.

study; CH2M Hill, 2006). Note that multiple analyses from different sample events or different rooms within the same building are averaged in Table 1. The commercial buildings with the highest PCE concentrations and the affected residential buildings are all sourced via fugitive emissions from the operating dry cleaner (Table 2). Two other commercial buildings with average PCE concentrations of 317 $\mu g/m^3$and 123 $\mu g/m^3$ were predominately sourced via vapor intrusion. Also, detectable concentrations of TCE were only present in those buildings sourced via vapor intrusion.

Seasonal effects have been observed in at least one commercial building, where indoor air PCE concentrations averaged 38.5 $\mu g/m^3$ on a warm fall day and 208 $\mu g/m^3$ on a cool winter day (overall building average was 123 $\mu g/m^3$). Subsurface vapor intrusion into a building can vary by season due to the effect of heating systems that reduce air pressure near the base of buildings and cause more subsurface vapor to enter the building (CH2M Hill, 2008). Indoor air PCE concentrations during warm weather may also be reduced because windows are open to increase ventilation.

In addition to indoor air samples, outdoor air monitoring was collocated with crawlspace sampling of select residences along Hahn Street and the residence and commercial building in the SE corner of the McGaffey and & Main block. The crawl space PCE measurements in the residential buildings are not elevated relative to indoor or outdoor air samples (CH2M Hill, 2008), which supports a fugitive emission source for those locations. Outdoor air concentrations of PCE during all rounds of sampling ranged from 0.2 to 293 $\mu g/m^3$ and appear to be entirely sourced via fugitive emissions from the operating dry cleaner (CH2M Hill, 2006).

Table 1. VOC Air Concentrations and Health Comparison Values

Contaminant	Maximum Indoor Air (µg/m^3)		Comparison Value (µg/m^3)
Tetrachloroethylene PCE	Commercial Bldg.	1,338^	3.8 CREG 40 EMEG-chronic, intermediate, acute
	Residential Bldg.*	27.3 22.1(Avg.)	
Trichloroethylene TCE	Commercial Bldg.	0.8^ 0.3 (Avg.)$^+$	0.24 CREG 2 EMEG-chronic
	Residential Bldg.	0.14$^\#$	
cis 1,2-Dichloroethene	Commercial Bldg.	0.8^	Not Available
	Residential Bldg.	<0.44	
Vinyl chloride	Commercial Bldg.	<0.1	1. 0.1 CREG 100 RfC
	Residential Bldg.	<0.05	

VOC: volatile organic compound
CREG: Cancer risk evaluation guide
RfC: Reference concentration
EMEG: Environmental media evaluation guide
Chronic: long term exposure (≥ year).
Acute: short term exposure (hours to 14 days).
^ Concentrations in commercial buildings are adjusted for occupational exposures by multiplying measured concentrations by 8/24 hours per day and 5/7 days per week (see Appendix D).
*The house with the highest indoor PCE concentration is identified as an abandoned rental property (two sample locations). A nearby occupied house had similar, albeit lower PCE concentrations.
$^+$ Four samples (2 locations, 2 sample events) from same building.
$^\#$ This is similar to ambient outdoor concentrations from outside study area.
See Appendix A for definitions of these health comparison values.
The indoor air samples were collected in October and December, 2004 (CH2M Hill, 2008).

Table 2. PCE Source Contributions in Indoor Air

	PCE concentration range; average ($\mu g/m^3$)	Indoor Air PCE from Dry Cleaner Exhaust	Indoor Air PCE from Vapor Intrusion
Commercial Buildings^	5,621	100%	0%
	394-419; 406*	100%	0%
	251-360; 317*	5%	95%
	38-223; 123*	3%	97%
	56-57; 57*	86%	14%
Residential Bldgs.	17-27; 22.1*	100%	0%
^ These are measured concentrations and not adjusted for occupational exposures. * Values listed are averages of multiple sample events or interior locations Source contributions based on tracer study (CH2M Hill, 2006).			

Groundwater Pathway

Groundwater from Private Wells

Following the 1994 discovery of PCE-contaminated groundwater at the intersection of McGaffey Street and South Main Street, NMED sampled private wells on several occasions. By 1996, NMED sampled 18 private wells and found 12 to be contaminated with PCE. Most of these private wells are located near the intersection of South Atkinson Avenue and Poe Street, which is 1 to 1.5 miles southeast of the main source of contamination (McGaffey Street and South Main Street) (Figure 2). Some of these private wells draw water from 65 feet below the surface while others draw water from as much as 200 feet below the surface (Appendix C; Table C-1).

PCE and TCE levels in these private wells (1995 to 2002 samples) are shown in Table C-1 (Appendix C; CH2M Hill, 2003; NMED, 1999/2001). It should be noted that in 1995 only one residence used a well for drinking water (EPA, 2008). This residence (SM-06 in Table C-1) had PCE levels of 105 µg/L in January 1995 and 260 µg/L in May 1995. According to NMED, this well was used by the residents for drinking and indoor uses until 1995. Five to ten other wells were used for irrigation or stock watering (EPA, 2008).

Vinyl chloride and cis-1,2-DCE were not analyzed in any of the private wells. However, these contaminants were analyzed but not detected in three rounds of sampling (Fall 2002; Spring 2003; and Fall 2003) in more than 30 monitor wells located within the PCE plume (EPA, 2008). Consequently, vinyl chloride and cis-1,2-DCE are not contaminants of concern for the groundwater pathway.

The highest PCE level detected in a private well during 1995 was 260 µg/L (a sample from this

well taken 5 months earlier had a PCE concentration of 105 µg/L; Table C-1). NMED reported that only 2 wells down-gradient from the source draw water from the deeper aquifer. One well located on South Cahoon contained PCE at 57 µg/L in 1995 and 151 µg/L in 2000. NMED reported that it is possible that this well on South Cahoon is not structurally sound and may be drawing water from the shallow aquifer. Therefore, the PCE from this well might be coming from the shallow aquifer rather than the deeper aquifer. The other well that draws water from the deeper aquifer (located on East Poe) did not contain PCE.

Low concentrations of TCE were measured in these same wells. However, TCE sampling in these wells did not begin until 2002 (these wells were not used for drinking water after 1995). Well SM-04 had the highest TCE concentration (10 µg/L). It is not known whether this well was used as a drinking source (Table C-1). As PCE concentrations in well SM-04 are similar to those in SM-06 (Table C-1) and no TCE measurements are available for well SM-06, the following assessment of TCE exposure is based on the maximum measured concentration of 10 µg/L.

By 2000, NMED identified a total of 25 private wells down-gradient from the intersection of McGaffey Street and South Main Street. NMED sampled 16 of these wells and 13 showed PCE contamination ranging from 1 to 193 µg/L (see Table C-1). The remaining 9 wells were no longer functional. In general, PCE levels in some private wells increased from 1995 to 2002, while PCE levels in other wells remained the same. NMED reported in 2001 that no residents were known to be drinking PCE-contaminated water and that all residences were receiving drinking water from either the City of Roswell or the Berrendo Water Cooperative.

Based on the 2002 well survey (CH2M Hill, 2003b), people living at several locations might have been previously exposed to contaminated groundwater from their private wells. One resident reported that the private well had plumbing to the house and that water from the private well was used for bathing. The well was constructed in 2000 and is about 165 feet deep. The well has been tested twice, did not contain PCE in 2002, but did contain 0.2 µg/L PCE in 2000. Two other residents reported that they use city water for potable uses but used their former private wells to fill swimming pools. Another residence has both a city water connection and a private well with plumbing that connects the well to the residence. The resident reports using city water for drinking water. It is uncertain if the private well was used bathing. The private well was tested in October 2000 and found to contain 1 µg/L PCE, which is below the EPA drinking water standard.

Other private wells contain small amounts of PCE, some at levels above the EPA drinking water standard. While several reports have stated that people do not use these wells for drinking water, ATSDR could not confirm these statements because survey results were not available or the residents did not participate in the EPA well survey. In addition, previous surveys did not ask if people used private well water for bathing or to fill pools. Wells with unknown usage or status are identified in Table C-1.

In summary, PCE and/or TCE were detected in at least 16 private water wells. Only one of these residences was known to have used their contaminated well as a source for drinking or bathing.

Water from 13 other contaminated wells could have been used for drinking or bathing. The well known to have been used as a drinking water source (until 1995) had the highest measured levels of PCE contamination. The following section evaluating the public health implications for "Exposure to PCE in Groundwater via private well" are based on the measured PCE concentrations from that well.

Groundwater from Public Water Supply Wells

The City of Roswell has 20 producing municipal wells around the city that provide drinking water for its residents. These municipal wells draw water from the deeper aquifer. According to the City of Roswell Water Department (personal communication with Roger Buckley, 3/28/13), one supply well is located within the PCE groundwater plume area and three other wells are within a mile of the plume. Because of required sampling of municipal wells by New Mexico Department of Health and by the federal Safe Drinking Water Act, these wells are regularly tested for a variety of groundwater contaminants including PCE and other VOCs.

City Well Number 12 is within the plume area but only has low and intermittent detections of PCE and TCE (Table 3). City Wells 13, 15, and 16 are near, but outside the plume area and also have had low concentrations of PCE and TCE. According to the City of Roswell Water Department (personal communication with Roger Buckley 3/28/13), the contamination in wells 15 and 16 is likely due to historic waste disposal activities near those wells and not related to the McGaffey and Main site. These detected levels of PCE and TCE are below the federal drinking water standard of 5 µg/L (Maximum Contaminant Level or MCL for both PCE and TCE) and no actions have been required of the City of Roswell.

Table 3. PCE and TCE Concentrations in Roswell Water System Wells near the Site

City Well ID	PCE (µg/L)	TCE (µg/L)
CV	**5 MCL** **17 CREG**	**5 MCL** **0.76 CREG**
Well 12	ND to 1.5	ND to 0.1
Well 13	ND to 2.3	ND to 0.2
Well 15	ND to 3.8	ND to 0.1
Well 16	ND to 2.2	ND to 0.1
Data from 1977 to present are from the New Mexico Drinking Water Watch website: https://dww.water.net.env.nm.gov/DWW/ MCL—maximum contaminant level (EPA). CREG—cancer risk evaluation guide (ATSDR)		

Public Health Implications

The discussion section of this report covers the public health implications for people who might have been exposed to contaminants from the McGaffey and Main Site. Specifically, this section will include an evaluation of the following questions:

- how people might be exposed to contaminants from the McGaffey and Main Site,
- how those exposures might affect their health,

The description will focus on past, present, and future exposures and will include specific discussion of children's health issues.

Table 4 summarizes the different elements of pathways of exposure. Specifically, Table 4 shows the following:

1) Site-related PCE and TCE contamination most likely occurred because of disposal from local dry cleaning businesses near the intersection of McGaffey Street and Main Street (the source),
2) Chemicals migrated in groundwater (environmental media) to private wells (point of exposure) where a few people were exposed by ingestion, inhalation, and skin contact when they used contaminated water for drinking water and other household uses.
3) Some portion of the PCE/TCE contamination migrates upward via vapor intrusion into buildings near the source area,
4) PCE air concentrations in the buildings affected by vapor intrusion are also subject to fugitive emissions from a nearby operating dry cleaner.
5) Until recent EPA building remediation, people working in those buildings were exposed by breathing the PCE/TCE from vapor intrusion; exposure to PCE from fugitive air emissions may be ongoing.

Several houses (including duplexes) near the McGaffey and Main source area have measurable concentrations of PCE and TCE in indoor air. Only PCE was measured at concentrations above screening values (Table 1) and is apparently sourced from the operating dry cleaning facility (Table 2).

As mentioned previously, only one family is known to have been exposed to PCE and TCE (via drinking water) in the past. This exposure is not occurring now, but similar exposure could occur in the future should someone install a private well that draws water from the contaminated portion of the aquifer. Information obtained during EPA's 2002 private well survey shows that water from some of the private wells may be used to irrigate garden and lawns. It is unclear at this time if some of the wells are used for swimming pools, might be used for bathing, or to provide drinking water for pets or livestock.

Table 4. Completed and Potential Pathways of Exposure at the McGaffey and Main Site

Pathway	Exposure Pathway Components							Comments
	Source	Environmental Media	Point of Exposure	Route of Exposure	Exposed Population	Time		
Ground water	PCE from former dry cleaners near McGaffey Street and South Main Street	Groundwater beneath 1107 South Main Street and southeast of source	Private wells up to 3.5 miles southeast of source	Ingestion Inhalation Skin Contact	People who used water from their private well for drinking or bathing.	Past	Completed	Contaminants may also be present in wells used for crop irrigation and livestock watering.
						Current	Not occurring via daily drinking water; incidental exposures may be occurring via inhalation/skin contact during irrigation.	
						Future	Potential if new wells are drilled in contaminant plume	
Indoor Air	Vapor Intrusion from groundwater plume and air emissions from operating dry cleaner	Indoor Air	Commercial buildings and homes near the source	Inhalation	People who work in commercial buildings or who live in homes near the source	Past Current Future.	Complete	Vapor intrusion affected buildings have been remediated; exposure via fugitive air emissions may be ongoing.

Analyses of groundwater samples from residential wells and indoor air at commercial buildings near the site have detected low concentrations of several VOCs. The specific compounds, their measured concentrations, and health comparison values (CVs) are listed in Tables 1, 3, and C-1. PCE and TCE were the only compounds detected at concentrations above their respective screening values and consequently, are the only contaminants discussed in this section.

The CVs are calculated concentrations of a substance in air, water, food, or soil that are unlikely to cause harmful (adverse) health effects in exposed people. The CV is used as a screening level during the public health evaluation process. Exposure to these compounds at concentrations greater than their comparison value does not necessarily mean that someone will get sick. Substances found in amounts greater than their CVs are subject to further evaluation by estimation of the doses and concentrations that people may be exposed to via drinking or direct absorption of the contaminants from water and breathing them in air. These estimated doses and concentrations are then compared with doses that have resulted in disease or sickness for people or laboratory animals. The health implications for each contaminant are presented in a discussion that relates the potential doses with the specific diseases or health effects caused by each contaminant.

It must be noted that these dose estimates and health determinations are based on the available measured VOC concentrations. Although unlikely (based on extrapolation of available concentration trends), it is possible that pre-1995 concentrations were higher than 1995 measured concentrations. If this had occurred, the resulting doses would have been commensurately higher.

Studies have shown that exposure to VOCs from routes other than direct ingestion might be as large as the exposure from ingestion alone. The inhalation dose due to volatization during a shower may equal the ingestion dose from 1.3 liters of water (Wan, et.al., 1990) and that 50% to 90% of VOCs in water may volatize during showering, laundering, and other activities (Moya et al, 1999; Giardino and Andelman, 1996). Similarly, the dermal dose has been estimated to equal 30% of the ingested dose (Maine DEP/DHS, 1992). Based on the results of these studies, combined VOC exposure pathways listed in Table 4 include an inhalation and dermal contact dose that is equal to and summed with the ingestion dose.

Tables 5 and 6 show the estimated doses and theoretical excess cancer risk for PCE and TCE (via indoor air at residential and commercial buildings and cumulative drinking water and air from a contaminated well, respectively). The comparison values used are defined and their derivation described in Appendix D.

A discussion of the estimated doses, cancer risks, and possible health effects from exposure to each to these contaminants is presented in the following sections.

Table 5. PCE and TCE concentrations in indoor air and associated CVs and 20 year excess cancer risks.

Contaminant	CV $\mu g/m^3$	Indoor Air Concentration $\mu g/m^3$		20 year Cancer Risk
PCE	3.8 CREG 40 EMEG a,i,c	Residential	22*	0.0000016 (1.6E-06)
		Commercial	1,338^	0.000095 (9.5E-05)
TCE	0.24 CREG 2 EMEGc	Commercial	0.8 **^	0.0000008 (8.0E-07)

PCE Inhalation Unit Risk is 2.6E-7.
TCE Inhalation Unit Risk is 4.1E-06.
*Average concentration (2 sample events, same house).
**Average of three sample locations within same building (same sampling event).
^The measured concentration is adjusted for occupational exposures by assuming an 8 hour workday for 5 days per week; the residential exposures assume 24 hours per day, 365 days per year.
CREG is cancer risk evaluation guide (ATSDR).
EMEG a,i,c is environmental media evaluation guide for acute, intermediate and chronic exposures.
20 year cancer risk = (Concentration [adjusted for 20 yr. exposure duration]) x (PCE or TCE inhalation unit risk; see Appendix D).

Table 6. The estimated doses and cancer risks from PCE and TCE exposures via a private water well.

PCE	GW conc. µg/L	MRL mg/kg/day	Dose mg/kg/day	Cancer Risk (16 yr.)	Child/Adult Cancer Risk (32 yr.)
Child (0-2 yrs.)	260 A,I 183 C	0.008 A,I,C	0.043 A,I 0.031 C	0.000009	0.000013 (1.3E-05)
Adult			0.013 A,I 0.010 C	0.000004	
TCE	GW conc. µg/L	MRL mg/kg/day	Dose mg/kg/day	Cancer Risk (16 yr.)	Child/Adult Cancer Risk (32 yr.)
Child (0-2 yrs.)	10 A,I,C*	0.0005 I,C	0.0013	0.00005	0.000055 (5.5E-05)
Adult			0.0006	0.000005	

GW conc. are measured groundwater concentrations from residential drinking water wells (see Appendix C).
PCE and TCE doses are calculated assuming that inhalation doses are 70% of the ingestion dose and dermal doses are 30% of the ingestion dose (see text for discussion).
Child doses for acute and intermediate duration exposures are calculated assuming body weight and intake rates for a child 0-2 years old (see Appendix D).
16 year cancer risks are calculated assuming either child or adult exposure factors (see Appendix D for discussion).
MRLs (minimal risk levels) are health comparison values in units of daily dose (see Appendix A).
A,I Acute and intermediate PCE doses are calculated using the maximum PCE concentration (260 µg/L).
C Chronic PCE doses and cancer risks are calculated using the average PCE concentration (183 µg/L).
*TCE was not analyzed in private wells during the potential period of exposure (1995 or earlier). The listed TCE concentration (10 µg/L) was measured in 2002 when the well was no longer in use.
Cancer risk = PCE or TCE dose x PCE or TCE cancer slope factor (respectively).
PCE cancer slope factor is 0.0021 (1/mg/kg/day) and the TCE cancer slope factor is 0.0046 (1/mg/kg/day). The cancer slope factors are from the EPA IRIS website (http://www.epa.gov/iris/).
See Appendix D for dose and risk calculation procedures.

Tetrachloroethylene (PCE)

Based on the available measured PCE concentrations and estimated doses, past exposure to PCE via vapor intrusion to indoor air in houses and commercial buildings adjacent to the McGaffey and Main site is unlikely to produce any harmful health effects, including cancer. Past exposure to a child via the contaminated residential water well near the McGaffey and Main site for 16 years would have no apparent increased risk of cancer. An adult at this location for 16 years (plus 16 years as a child) would have no increased risk of cancer. Non-cancer health effects are unlikely for children or adults at this location.

The following summary of PCE health effects is from the ATSDR Toxicological Profile of Tetrachloroethylene (ATSDR, 2014a) and from the EPA Integrated Risk Information System (EPA, 2012). PCE is a manufactured compound widely used for dry cleaning fabrics and as a metal degreaser. It is also used as an intermediate in the manufacturing of other products. Summaries of both cancer and non-cancer PCE health effects for humans and laboratory animals are discussed below, followed by a discussion of the site-related exposure doses in relation to those health effects.

Non-cancer Effects

"A number of targets of toxicity from chronic exposure to tetrachloroethylene have been identified in published animal and human studies. These targets include the central nervous system, kidney, liver, immune and hematologic system, and development and reproduction. In general, neurological effects were found to be associated with lower tetrachloroethylene inhalation exposures. The nervous system is an expected target with oral tetrachloroethylene exposures because tetrachloroethylene and metabolites produced from inhalation exposures will also reach the target tissue via oral exposure." (http://www.epa.gov/iris/subst/0106.htm)

Liver and kidney damage have been observed in laboratory animal studies after exposure to high doses of PCE. Liver weight/body weight ratios were significantly higher than controls for animals treated with 100 mg/kg/day of PCE. At higher doses, hepatotoxic effects were observed (ATSDR, 2014a; EPA, 2011).

Groups of 20 Sprague-Dawley rats of both sexes were administered doses from 14 to 1,400 mg/kg/day (424-42,400 times greater than those estimated for the SM-06 residents). Male rats in the high-dose group and females in the two highest groups exhibited depressed body weights. Equivocal evidence of hepatotoxicity (increased liver and kidney weight/body weight ratios) were also observed at the higher doses (ATSDR, 2014a; EPA, 2012)

ATSDR has established a minimal risk level (MRL) of 40 μg/m^{3}for chronic, acute, and intermediate inhalation exposures to PCE[3]. An MRL is an estimate of the daily human exposure to a hazardous substance that is likely to be without appreciable risk of adverse non-cancer health effects over a specified duration of exposure. The PCE inhalation MRL is based on measured decreases in color vision in dry cleaning workers at PCE air concentrations of 49,494 μg/m^3 (this concentration is 11,526 μg/m^3 when adjusted for continuous, residential exposures).

Cancer Effects

PCE is a common commercial chemical used in the dry cleaning industry which has prompted a number of human studies on workers in this industry. These studies suggest a possible association between long-term PCE exposure and an increased risk of cancer. The cancer types most consistently showing an increase are esophageal cancer, bladder cancer, cervical cancer, and non-Hodgkin's lymphoma. Unfortunately, dry cleaning workers are also exposed to other chemicals while working so it is difficult to determine whether PCE or some other chemical used in the dry cleaning industry is the cause of these cancers. A study of a community exposed to PCE through their drinking water showed increases in bladder cancer and leukemia (a cancer of the blood) in the exposed population (Aschengrau et.al.,1993; Webler, 1993). Smoking and other life-style variables add to the complexity of the PCE-cancer issue. A review of these studies has concluded that esophageal cancer might have been caused by cigarette smoking and alcohol consumption and that bladder cancer might have been caused by exposure to other solvents in the industry (Weiss 1995; ATSDR, 2014a).

Various case-control studies were evaluated for possible associations between exposure to PCE and cancer effects in human populations. Although some of these studies indicate a possible association between exposure to PCE and various cancers, including bladder cancer, kidney cancer, and leukemia, the studies had limitations which precluded definitive conclusions. Cancer has been reported in experimental animals after oral exposure to PCE. Statistically significant increases in hepatocellular carcinomas occurred in the treated mice of both sexes. A cancer effect level of 386 mg/kg/day was derived from a chronic mouse study (ATSDR, 2014a). The cancer effects in this study were hepatocellular carcinomas.

PCE in air has been shown to cause cancer in rats and mice following near lifetime exposure. In a 2-year study of rats, Mennear et al. (1986) showed an increase in mononuclear cell leukemia following exposure to 1,650,000 μg/m^3 PCE for 5 days a week, 6 hours a day (Figure 3). Mennear et al. (1986) also showed that PCE in air caused an increase in liver cancer in mice exposed to PCE at 600,000 μg/m^3 for 5 days a week, 6 hours a day for over 2 years.

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[3] Note that the ATSDR MRL for oral PCE exposure is 0.008 mg/kg/day. The oral MRL is based on the same PCE exposure study but the MRL is presented in units of dose (mg/kg/day) rather than PCE concentration in air (μg/m^3; ATSDR, 2014a).

The Environmental Protection Agency has concluded that PCE is "likely to be carcinogenic in humans by all routes of exposure…" (EPA, 2012). The National Toxicology Program (NTP) within the federal Department of Health and Human Services has reviewed the available cancer information and determined that there is sufficient evidence that PCE is "reasonably anticipated to be a human carcinogen". NTP's summary of PCE carcinogenicity can be found at this website: http://ntp.niehs.nih.gov/ntp/roc/twelfth/profiles/Tetrachloroethylene.pdf#search=tetrachloroethylene

All of the uncertainties and conservative exposure assumptions associated with the dose calculations are included in the risk estimation as well as the uncertainty in deriving the cancer slope factor (EPA, 2000). The risk estimates in Table 6 cannot be interpreted as evidence that people using the affected well will develop cancer as a result of PCE exposure. ATSDR uses the estimates of cancer risk to help determine whether additional exposure evaluation or other public health actions are warranted.

Exposure to PCE in Indoor Air from Vapor Intrusion

Figure 3 shows health effect levels for PCE exposure via inhalation. The chronic Minimal Risk Level (MRL) of 40 µg/m^3 is the lowest screening level for non-cancer effects from PCE exposure. However, actual health effects have not been observed in humans for long term exposures at less than 50,000 µg/m^3 (ATSDR, 2014a). The most sensitive non-cancer effect for PCE exposure for long term exposure is effects to the vision. EPA's modeled effect level (called a point of departure) for color vision is 15,000 µg/m^3 for long term exposure (EPA, 2012). Therefore, all indoor air levels detected at the McGaffey and Main Ground Water Plume site were well below the non-cancer effect levels for long-term exposures.

ATSDR's cancer screening value for PCE in air (called a Cancer Risk Evaluation Guide: CREG) is 3.8 µg/m^3. This CREG value represents an increased theoretical risk of 0.000001 (1E-06) for someone breathing PCE for 24 hours a day over a 78 year (lifetime) period. This value is derived from EPA modeling of a study where exposed mice developed indicators of liver cancer (EPA, 2012).

Figure 3 shows the relationship between the maximum measured PCE air concentration (adjusted for continuous exposure), PCE air concentrations associated with human health effects, and the different health comparison or screening values. The definitions and derivations of the health comparison values (CVs) are presented above and in Appendix D. The human health effect concentrations are based on numerous studies as summarized in The Toxicological Profile for Tetrachloroethylene (ATSDR, 2014a). The health effect concentrations are LOAELs which are the lowest concentrations associated with each health effect or NOAELs which are concentrations at which specific health effects were not detected.

Figure 3 also shows two occupational health standards. The Occupational Safety and Health Administration Permissible Exposure Limit [OSHA PEL] and the American Conference of

Governmental Industrial Hygienists Threshold Limit Value [ACGIH TLV] are time weighted concentrations deemed protective for workplace exposures and are the health standards applicable to workers in the active dry cleaning facility. Note that occupational limits may not be protective for community or residential exposures that include sensitive populations and longer duration exposures.

The highest adjusted indoor air PCE concentrations in the commercial buildings (1,338 $\mu g/m^3$) exceeds ATSDR's screening value for non-cancer exposures (MRL; 40 $\mu g/m^3$; Table 5 and Figure 3). This screening value is based on a LOAEL of 11,526 $\mu g/m^3$ for decreased color vision effects (ATSDR, 2014a). As the highest indoor air PCE concentration, was more than eight times lower than the health effect on which the MRL is based, non-cancer health effects are unlikely. The highest indoor air PCE concentration is also below levels set by the Occupational Safety and Health Administration (OSHA) including the Permissible Exposure Limit (PEL) of 690,000 $\mu g/m^3$ (8 hour time weighted average) and the level Immediately Dangerous to Life and Health (IDLH) of 1,000,000 $\mu g/m^3$ (NIOSH 1994).

Therefore, exposure to PCE from vapor intrusion in commercial buildings does not present a public health hazard at this site. Similarly, maximum inhalation concentrations of PCE (27 $\mu g/m^3$; Table 2) in houses near the site are below the MRL of 40 $\mu g/m^3$ and do not present a public health hazard.

Table 5 presents the calculated excess cancer risks associated with inhalation of PCE in nearby houses and of PCE and TCE in commercial locations. It should be noted that both the residential and commercial locations with the maximum concentrations listed in Table 5 are sourced via fugitive emissions (from the operating dry cleaner) and have undetectable contributions from vapor intrusion (Table 2). Regardless of source, the locations listed in Table 1 have the highest measured PCE/TCE concentrations and resulting cancer risks.

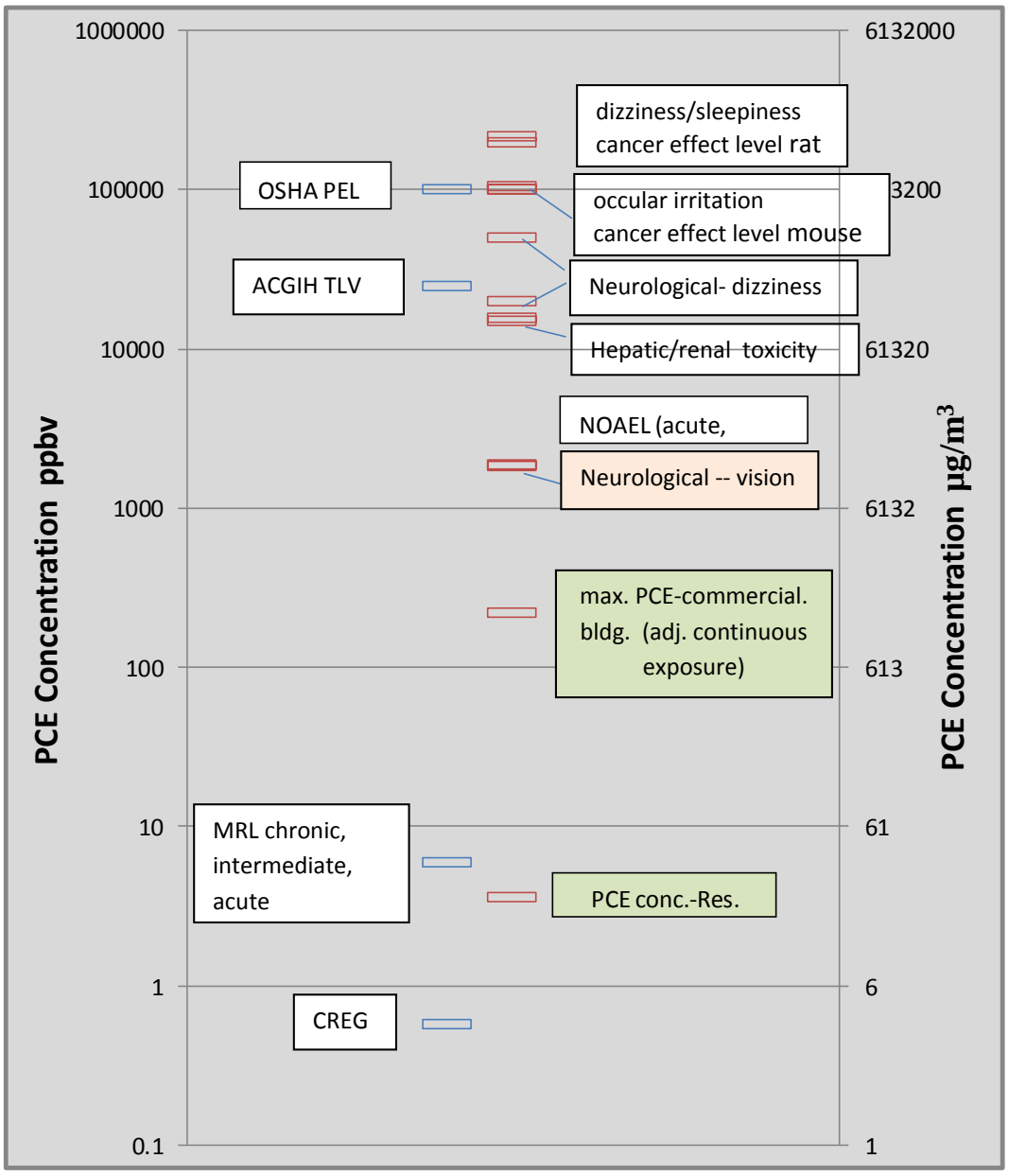

Figure 3. PCE air concentrations and health effects and comparison values. *Note that all health effect values except cancer effect levels are from human exposures. Cancer effect levels are the lowest concentrations for initiation of tumors or cancer precursors in rats and mice. All health effect values are from ATSDR (2014a). EMEG- environmental media evaluation guide, CREG- cancer risk evaluation guide, OSHA PEL- Occupational Safety Health Administration permissible exposure limit, ACGIH TLV- American Conference Governmental Industrial Hygienists threshold limit value; Res. is residential (see Appendix D for derivations).

Exposure to PCE in Groundwater via Private Well

Most people who lived southeast of the intersection of McGaffey Street and South Main Street were not exposed to PCE and other contaminants in groundwater because they used either city water for drinking and bathing or they obtained water from a private company. Therefore, the people living, working, or visiting the residences and businesses affected by vapor intrusion were not exposed via contaminated groundwater. City water was tested in the past and at times was found to contain small amounts of PCE. However, the levels of PCE in city water were below federal drinking water standards (MCL 5 µg/L for both PCE and TCE); therefore, the City did not need to take further action. A few residences, located further down-gradient, had private wells that were contaminated with PCE and other chemicals. If these wells were used only to irrigate lawns or gardens, these people were not exposed to PCE or other chemicals at levels above health screening values.

In 1995 when groundwater contamination was discovered, one household had a contaminated private well that was used for drinking water and for bathing or showering. The people at this house were exposed to PCE (maximum level was 260 µg/L) and TCE (maximum level was 10 µg/L) in their private well, possibly for 10 years or more prior to 1995 (see Table C-1, Appendix C, SM-06). Although the initial groundwater contamination probably occurred between 1955 and 1963, contamination of nearby private wells probably did not occur for several to many years later because of the time needed for PCE to migrate in groundwater from the McGaffey Street and South Main Street intersection to this private well southeast of the site.

The specific year that exposure might have started at this private well is unknown. However, it is important to note that two PCE analyses were conducted at this location in 1995. A January 1995 analysis had a PCE concentration of 105 µg/L and a May 1995 analysis had a PCE concentration of 260 µg/L (Table C-1). Consequently, short term exposures (acute and intermediate; Table 6) are evaluated using a PCE concentration of 260 µg/L, while long term exposures (chronic) are evaluated using an average PCE concentration of 183 µg/L (Table 6).

The duration of long term exposure is unknown due to the lack of sampling data prior to 1995. Consequently, this assessment will assume that that a person living at the affected residence could have been exposed for a total of 32 years (1963 to 1995). This includes 16 years as a child with age-appropriate intake rates and body weights and 16 years as an adult. As doses for children are higher than those of adults, this exposure scenario comprises the highest possible dose estimate. Details on the exposure calculations and factors are included in Appendix D.

While oral (ingestion) exposure is obvious from drinking PCE- and TCE-contaminated water, residents at this house were also exposed to PCE and TCE via inhalation and skin contact while bathing and showering. Based on the results of previously mentioned studies, the PCE and TCE exposure doses in Table 6 include an inhalation dose that is 70% of the ingestion dose, and a dermal contact dose that is 30% of the ingestion dose.

Exposure to PCE/TCE might be possible in several other homes with private wells; however,

information is not available to confirm exposure. From the well survey that EPA conducted in 2002, several homes have private wells with plumbing to the house. While EPA has confirmed that residents were not using their private wells for drinking water, some residents might be using their well for bathing and showering, filling swimming pools, or irrigating foodcrops or livestock. If PCE/TCE exposures occurred at these locations, they would have been at much lower concentrations (Table C-1) and for much shorter durations and frequencies, relative to location SM-06.

The maximum measured concentration of PCE in1995 was 260 µg/L in Well SM-06 which results in estimated combined (ingestion plus inhalation plus dermal contact) short term PCE doses of 0.013 mg/kg/day for adults and 0.043 mg/kg/day for children (aged 0 to 2 years; Table 6; chronic doses estimated using an average PCE concentration of 183 µg/L are slightly lower). Estimated doses from acute groundwater exposure are shown in relation to doses with adverse health effects in Figure 4. Figure 4 shows that the estimated cumulative doses from PCE exposure (via ingestion, inhalation, and dermal contact) are lower than doses associated with observed health effects, cancer effect levels, and the 1E-04 32-year cancer risk range but higher than the MRL and the dose associated with the 1E-06 32-year cancer risk.

ATSDR has established an MRL for ingestion of PCE of 0.008 mg/kg/day (for acute, intermediate, and chronic exposures; Figure 4; ATSDR, 2014a). The maximum PCE dose from cumulative exposure is 0.043 mg/kg/day (0-2 year old child), which is about five times greater than the MRL (Table 6). Although greater than the MRL, the maximum dose is more than 50 times lower than the dose on which the MRL is based (2.3 mg/kg/day for decreased color vision in dry cleaning workers; ATSDR, 2014a). As MRLs are established to be health protective to the most sensitive people, harmful non-cancer health effects from short to long term PCE exposure to the highest measured PCE concentration are unlikely.

For long term or chronic exposures there is some uncertainty from not knowing if PCE levels before 1995 were higher than the observed maximum and how long residents were exposed to PCE. One other sample taken from this well in January 1995 showed PCE levels of 105 ppb. Lower PCE levels in previous years would decrease the risk of cancer and non-cancer health effects while higher PCE levels would increase those risks. These estimated exposure doses and cancer risks do not mean that the people living at that residence will necessarily get cancer or any other harmful health effect. Using health protective estimates of exposure and risk, a 16 year exposure to a child (age 0 to 16 years), results in an increased cancer risk of 0.000009 (or 9E-06) and a 16 year adult exposure results in an increased cancer risk of 0.000004 (or 4E-06).

Based on the timing of the initial PCE release (1955 to 1963), the time required for PCE to migrate to the affected residence, and the time the well was removed from service (1995), a total duration of exposure at this location is probably much less than the 32 year exposure duration included in Table 6. If exposure occurred at the affected residence for a total duration of 32 years (16 years as a child, 16 years as an adult) the total excess cancer risk would be 0.000013 (1.3E-

05). All of these estimated excess risks are within the EPA "target risk range for Superfund cleanups" (http://www.epa.gov/oswer/riskassessment/baseline.htm) and interpreted by ATSDR to be "no increased risk".

In summary, based on available data, no harmful health effects, including cancer, are likely for residents who used the PCE-contaminated well. However, if PCE concentrations before 1995 were higher than 260 µg/L, such health effects are possible. Consequently, PCE exposure (prior to 1995) via the private well cannot be determined based on the available data. Since no other private wells are known to have been used by residents as drinking water or for bathing, other residents in the neighborhoods associated with the McGaffey and Main Site are not at increased risk of cancer or other harmful health effects from PCE contamination of the groundwater.

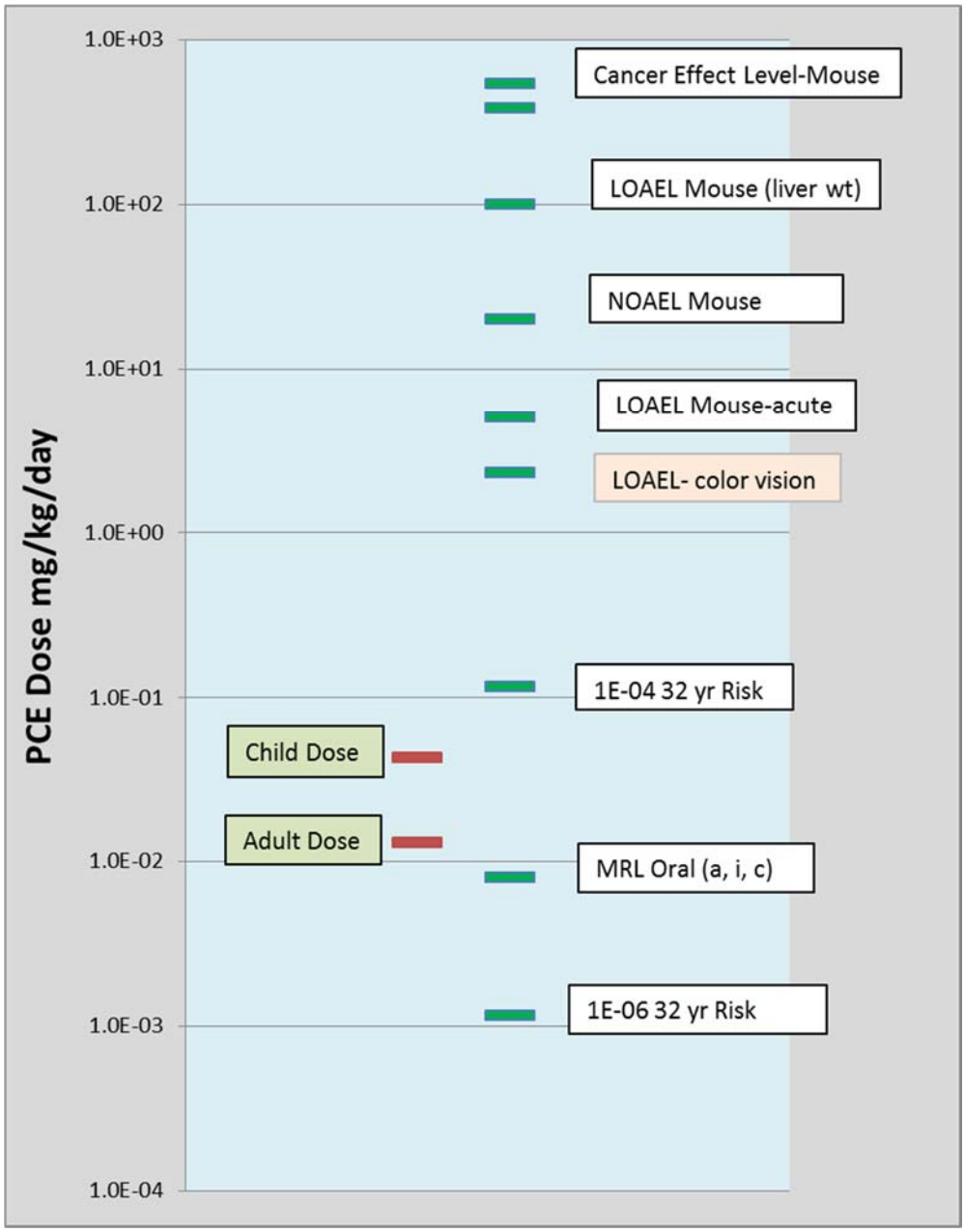

Figure 4. Child and adult PCE doses from contaminated water in relation to health effect and cancer risk levels. Doses are calculated using a PCE concentration of 260 µg/L and assume that inhalation and dermal exposures are equal to and added to the ingestion dose. Doses are in units of mg PCE per kilogram body weight per day (mg/kg/day). LOAEL is lowest observed adverse effect level; NOAEL is no observed adverse effect level; MRL is minimum risk level. 1E-04 and 1E-06 32-year risk levels are estimated PCE doses associated with 0.0001 and 0.000001 cancer risks for 32 year exposures (see Appendix D for derivations).

Trichloroethylene (TCE or trichloroethene)

Based on estimated doses to the maximum measured TCE concentration, adverse health effects from past exposure to TCE via exposure to contaminated water (via the private well) and indoor air (via vapor intrusion) are unlikely to produce any harmful health effects, including cancer.

The following summary of TCE health effects is from the ATSDR Toxicological Profile of Trichloroethylene (ATSDR, 2014b) and from the EPA Integrated Risk Information System (EPA, 2011). TCE is a nonflammable, oily, colorless liquid that has a sweet odor and a sweet, burning taste. Years ago, TCE was used as an anesthetic. It is now used as a solvent to remove grease from metal parts and to make other chemicals. It is heavier than water and has low solubility (up to one part TCE per thousand parts of water at room temperature; ATSDR, 2014b).

When present in groundwater, free-phase TCE tends to settle into a layer at the bottom of the aquifer and then continuously dissolves into the groundwater. This may result in high levels of TCE in the aquifer for years after the original release of contamination has ended. Alternatively, dissolved-phase TCE flows with groundwater. There is limited evidence of free-phase (or dense, non-aqueous phase PCE/TCE) at the source area such that most of the documented contaminant plume is present as a dissolved phase. Summaries of both cancer and non-cancer TCE health effects for humans and laboratory animals are discussed below.

Non-cancer Effects

Adverse non-cancer effects associated with oral TCE exposure include decreased body weight, liver and kidney effects, and neurological, immunological, reproductive, and developmental effects. The EPA (2011) has established:

> "an RfD of **0.0005 mg/kg/day** based on the critical effects of heart malformations (rats), adult immunological effects (mice), and developmental immunotoxicity (mice), all from oral studies. This RfD is further supported by results from an oral study for the effect of toxic nephropathy (rats) and route-to-route extrapolated results from an inhalation study for the effect of increased kidney weight (rats)."

ATSDR has accepted the EPA RfD as its chronic MRL and suggested that it may be applicable for intermediate and acute exposures (ATSDR, 2014b). The chronic MRL (and the underlying RfD) are based on reduced thymus weights in female mice (Keil, et.al., 2009) and fetal heart malformations in rats (exposed via pregnant female rats; Johnson, et.al., 2003). Both studies involved oral intake of TCE with the exposure doses converted to human equivalent concentrations via modeling approaches (ATSDR, 2014b).

Several studies of workers and community residents suggest a possible association between exposure to TCE (and other chemicals) and developmental outcomes (ATSDR, 2014b; Fagliano

et al., 1990; Bove et al., 1995). However, none of the studies provide conclusive evidence for a causal relationship, largely because information about TCE exposure was incomplete and exposure to other chemicals was likely (ATSDR, 2014b). Collectively, the scientific data indicate that the developing heart and nervous system in fetuses and young children may be sensitive to the toxic effects of TCE (ATSDR, 2014b), although the dose levels at which these effects occur has not been established.

Cancer Effects

TCE is characterized as carcinogenic to humans by all routes of exposure (EPA, 2011). This conclusion is based on convincing evidence of a causal association between TCE exposure in humans and kidney cancer. There are several reports of an increased occurrence of cancer from ingestion and inhalation of TCE by animals and humans (ATSDR, 2014b). Human health studies suggest an increased incidence of cancer of various types (e.g., bladder, lymphoma, kidney, respiratory tract, cervix, skin, liver, and stomach) from exposure to TCE; however, no studies provide clear, unequivocal evidence that exposure is linked to increased cancer risk in humans (ATSDR, 2014b). The available studies suffer from inadequate characterization of exposure, small numbers of subjects, and the fact that subjects were likely exposed to other potentially carcinogenic chemicals. There is, however, sufficient evidence that TCE exposure results in cancer development in animals, although animal studies may not be relevant for evaluating health hazard to humans (ATSDR, 2014b).

According to the EPA (2011)

> "Because the weight of evidence supports a mutagenic mode of action being operative for TCE carcinogenicity in the kidney (see Section 4.4.7), and there is an absence of chemical-specific data to evaluate differences in carcinogenic susceptibility, early-life susceptibility should be assumed and the ADAFs should be applied…"

Consequently, age dependent adjustment factors (ADAF) are used to calculate the TCE cancer risks to children via the groundwater pathway (Table 6; Appendix D). Exposures to TCE via vapor intrusion are based on adult occupational exposure factors such that ADAFs for child exposures are not used.

Exposure to TCE in Indoor Air from Vapor Intrusion

Table 5 shows the maximum measured (as adjusted) indoor air concentration of TCE and calculated cancer risk (commercial buildings only; TCE was not detected in the indoor air of residences at concentrations higher than ambient outdoor levels). The maximum indoor air TCE concentration adjusted for weekly occupational exposure was 0.8 $\mu g/m^3$ (Table 5) and is less than the chronic and intermediate inhalation EMEG (2.0 $\mu g/m^3$). However, multiple TCE air samples were collected in this building (two sample events at two locations; (CH2M Hill, 2008). The long

term whole building average adjusted concentration is 0.3 µg/m^3 (Table 5), which is also below the 2.0 µg/m^3 EMEG.

The ATSDR cancer risk evaluation guide (CREG) assumes a 78 year (lifetime) exposure duration. Because occupational exposures only occur for a portion of a 78 year lifetime, the average, adjusted exposure concentration of 0.3 µg/m^3 must be further adjusted for a 20 year occupational duration exposure in order to use the lifetime CREG. The resulting lifetime adjusted exposure concentration is 0.08 µg/m^3 (0.3 µg/m^3 x (20/78 years)) which is less than the lifetime CREG (0.24 µg/m^3) such that harmful health effects, including cancer, are unlikely from long term occupational exposures to indoor air.

Exposure to TCE in Groundwater via Private Well

TCE levels in the one contaminated private well where people used the well water for drinking and bathing were very low. Measurements of TCE ranged from 2.1 to 10 µg/L in Well SM-04, 1.3 to 3.2 µg/L in Well SM-05, and trace or non-detectable concentrations in all other residential wells (Table C-1). Using the highest measured TCE concentration (10 µg/L) results in estimated combined (ingestion plus inhalation plus dermal contact) TCE doses of 0.0006 mg/kg/day for adults and 0.0013 mg/kg/day for children (Table 6). The estimated doses of TCE from drinking water from this well are shown in Figure 5 along with doses associated with various health effects.

Both the child and adult TCE doses are above the chronic MRL and the 1E-06 cancer risk (for lifetime or 78 year exposure). However, the child/adult doses are less than the doses to laboratory animals (rats and mice) on which the MRL is based (LOAEL- thymus weight in mice and the lowest benchmark dose level [BMDL] - fetal heart malformations in rats). Because it is difficult to directly compare doses (and dose effects) in laboratory animals with humans, the EPA converted the doses to mice and rats to the 99th percentile human equivalent doses (HED 99%; EPA 2011). The cumulative TCE dose for a child is about 5 times lower than the 99th percentile human equivalent dose for the most sensitive health effect (fetal heart malformations in rats; Figure 5).

The excess cancer risks in Table 6 represent an estimate of the increase in cancer risk due to 16 and 32 years of exposure to TCE (at 10 µg/L). All of the uncertainties and conservative exposure assumptions associated with the dose calculations are included in the risk estimation as well as the uncertainty in deriving the cancer slope factor (EPA, 2000). The risk estimates in Table 6 cannot be interpreted as evidence that any of the Area B site neighbors will develop cancer as a result of TCE exposure. These estimates of excess risk fall within the range of no apparent increased risk (ATSDR, 1991). Note that the highest 16 year excess cancer risk (based on child intake rates and body weights) of about 0.000007 (7E-06) is "within the EPA target risk range." These low risk estimates indicate that TCE exposure from groundwater sources is not likely to cause an observable increase in cancer.

Based on the available information, cumulative child and adult exposures to TCE from the contaminated private well are unlikely to produce any harmful health effects, including cancer. However, there is the potential for adverse health effects if TCE concentrations in this well prior to 1995 were higher than 10 µg/L.

Child Health Considerations

In communities faced with air, water, or food contamination, the many physical differences between children and adults demand special emphasis. Children could be at greater risk than are adults from certain kinds of exposure to hazardous substances. Children play outdoors and sometimes engage in hand-to-mouth behaviors that increase their exposure potential. Children are shorter than are adults; this means they breathe dust, soil, and vapors close to the ground. A child's lower body weight and higher intake rate results in a greater dose of hazardous substance per unit of body weight. If toxic exposure levels are high enough during critical growth stages, the developing body systems of children can sustain permanent damage. Finally, children are dependent on adults for access to housing, for access to medical care, and for risk identification. Thus adults need as much information as possible to make informed decisions regarding their children's health.

In this PHA, doses to children have been estimated using child-specific intake rates and body weights. Additionally, because TCE is considered to have a mutagenic mode of action for induction of cancer and children may be especially susceptible, ADAFs have been used to adjust cancer risks to children (see above and Appendix D). As the estimated child-specific doses and cancer risks are somewhat greater than adult doses and risks, the resulting public health determinations are based on the doses and potential adverse health effects to children.

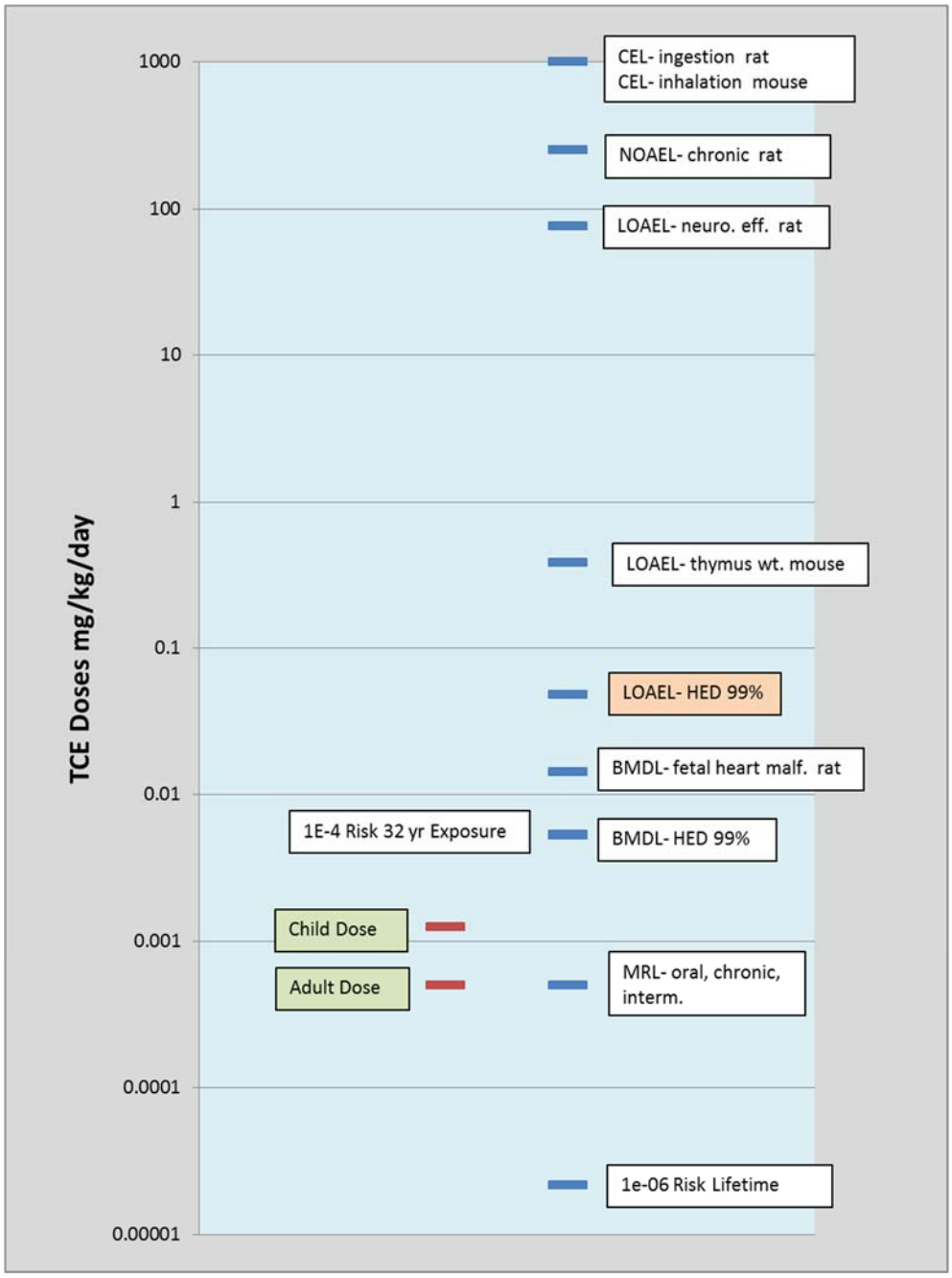

Figure 5. Cumulative TCE doses for a child and adult from contaminated water and health effect levels.

Exposure doses include ingestion of drinking water, inhalation of TCE in household air, and dermal absorption. CEL is cancer effect level, LOAEL is lowest observed adverse effect level, NOAEL is no observed adverse effect level, BMDL is lowest benchmark dose level, MRL is minimal risk level, and HED 99% is the 99th percentile human equivalent dose (adjusted from LOAEL and BMDL mouse/rate doses).

Adequacy of Available Data

The well survey and water and air sampling data which provide the basis for the following public health conclusions and recommendations were collected and analyzed with appropriate sampling and quality assurance procedures (e.g., CH2M Hill, 2006/2008; NMED, 2001). However, there are several potentially important gaps in the available dataset. These gaps are:

1. The initial onset, duration, and concentrations of PCE contamination in affected drinking water wells before 1995 (and TCE before 2002),
2. How long the residents lived in the affected houses and if they included children or pregnant women (as they may be more sensitive to PCE/TCE exposures),
3. If the highest measured and average of the highest measured, when available, indoor air PCE/TCE concentrations are representative of long term concentrations. Note that the maximum PCE concentration in a commercial location is based on a single measured value (5,621 $\mu g/m^3$).

The following public health determinations are based on the available measured contaminant concentrations and health protective assumptions regarding the potential exposures, i.e., 32 years of exposure to the highest average concentrations for residential exposures, that women and children lived in that house, and occupational exposures of 40 hours per week over a 20 year duration for inhalation exposure occurred in commercial buildings. Children are not assumed to be present on a continuous, long term basis for exposures at commercial locations. It should be noted that measured 24 hour PCE air concentrations may not be representative of long term average concentrations over all seasons and many years and that the highest measured PCE concentration in a commercial location is based on a single 24 hour value.

Conclusions, Recommendations, and Public Health Action Plan

Conclusions and Recommendations

Based on the available data and above findings ATSDR concludes that:

1. Past, present, and future inhalation exposures to PCE and TCE in indoor air in buildings adjacent to the McGaffey and Main source area are not a public health hazard.

   Basis for Conclusion: Inhalation of PCE and TCE-contaminated indoor air in buildings adjacent to the McGaffey and Main Site source area is a completed pathway of exposure. Sources of PCE include both vapor intrusion from a historic subsurface plume and fugitive emissions from a currently operating dry cleaner. TCE is only sourced from the subsurface contaminant plume. Based on the available measured PCE and TCE concentrations and estimated doses, occupational exposure via vapor intrusion and fugitive emissions to indoor air in commercial buildings adjacent to the McGaffey and Main site is unlikely to produce any harmful health effects, including cancer.

   Next Steps: Current and future PCE/TCE exposures from vapor intrusion have been mitigated by installation of a soil gas extraction and treatment system in the affected buildings. PCE exposures via fugitive emissions from the dry cleaning facility are ongoing. No additional public health actions are necessary.

2. Until 1995, at least one family used PCE and TCE-contaminated water from their private well for bathing and drinking. Based on available data (post-1995), the cumulative PCE and TCE exposures (via the private well) are unlikely to cause any harmful health effects, including cancer. However, because PCE and TCE concentrations before 1995 could have been higher than those measured post-1995, ATSDR cannot determine if these past exposures were at harmful levels.

   Basis for Conclusion: These past exposures for adults and children from this contaminated water (which stopped in 1995) occurred from drinking the water, breathing the contaminants released into indoor air (from the water), and absorbing the contaminants through their skin while bathing or showering. Past exposure to a child via the contaminated residential water well near the McGaffey and Main site for 16 years or more would have no apparent increased risk of cancer. An adult at this location for 16 years or more would have no increased risk of cancer. Non-cancer health effects from PCE exposure are unlikely for children or adults at this location.

   Next Steps: No additional actions are necessary with regard to the past drinking water exposures (which stopped in 1995). Three well surveys have determined that no current residents are exposed to contaminated groundwater. ATSDR recommends that appropriate restrictions are enacted to prevent new drinking water wells in the area of groundwater

contamination.

3. PCE and TCE contamination in public (or community) water systems do not present a public health hazard.

   Basis for Conclusion: Monitoring data from the public (community) water systems indicate that contaminant concentrations are below health screening values.

   Next Steps: Continue monitoring as required by the Safe Drinking Water Act.

Public Health Action Plan

As part of ATSDR's investigation at hazardous waste sites, ATSDR works with state, local, and other federal agencies to develop a public health action plan for a site. The intent of the public health action plan is to encourage actions be taken to protect public health. For this site the groundwater exposures of primary public health concern stopped in 1995 and vapor intrusion remedial actions were completed or are ongoing. Consequently, ATSDR does not anticipate any future public health actions at this site.

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Appendix A: Abbreviations and Glossary of Terms

List of Abbreviations

ADAF	Age dependent adjustment factor
ACGIH	American Congress Government Industrial Hygienists
ATSDR	Agency for Toxic Substances and Disease Registry
bgs	below ground surface
BMDL	Benchmark Dose (Lower Confidence Limit)
CREG	cancer risk evaluation guide
CV	comparison value
DCE	dichloroethylene
DNAPL	dense non-aqueous phase liquid
EMEG	environmental media evaluation guide
EPA	(U.S.) Environmental Protection Agency
HED	human equivalent dose
LOAEL	lowest observed adverse effect level
MCL	maximum contaminant level
MRL	minimal risk level
µg/L	micrograms per liter
$\mu g/m^3$	micrograms per cubic meter
mg/kg/day	milligrams per kilogram per day
NMED	New Mexico Environment Department
NOAEL	no observed adverse effect level
NPL	National Priority List
NTP	National Toxicology Program
OSHA	Occupational Safety and Health Administration
PCE	tetrachloroethylene (perchloroethylene)
PHA	public health assessment
ppb	parts per billion
RfD	reference dose
TCE	trichloroethylene or trichloroethene
VOC	volatile organic compound

ATSDR Glossary of Terms

Absorption
The process of taking in. For a person or an animal, absorption is the process of a substance getting into the body through the eyes, skin, stomach, intestines, or lungs.

Acute
Occurring over a short time [compare with chronic].

Acute exposure
Contact with a substance that occurs once or for only a short time (up to 14 days) [compare with intermediate duration exposure and chronic exposure].

Adverse health effect
A change in body function or cell structure that might lead to disease or health problems

Ambient
Surrounding (for example, ambient air).

Biodegradation
Decomposition or breakdown of a substance through the action of microorganisms (such as bacteria or fungi) or other natural physical processes (such as sunlight).

Biologic uptake
The transfer of substances from the environment to plants, animals, and humans.

Biota
Plants and animals in an environment. Some of these plants and animals might be sources of food, clothing, or medicines for people.

Cancer
Any one of a group of diseases that occur when cells in the body become abnormal and grow or multiply out of control.

Cancer risk
A theoretical risk for getting cancer if exposed to a substance every day for 78 years (a lifetime exposure). The true risk might be lower.

Carcinogen
A substance that causes cancer.

Central nervous system
The part of the nervous system that consists of the brain and the spinal cord.

Chronic
Occurring over a long time [compare with acute].

Chronic exposure
Contact with a substance that occurs over a long time (more than 1 year) [compare with acute exposure and intermediate duration exposure]

Comparison value (CV)
Calculated concentration of a substance in air, water, food, or soil that is unlikely to cause harmful (adverse) health effects in exposed people. The CV is used as a screening level during the public health assessment process. Substances found in amounts greater than their CVs might be selected for further evaluation in the public health assessment process.

Completed exposure pathway [see exposure pathway].

Concentration
The amount of a substance present in a certain amount of soil, water, air, food, blood, hair, urine, breath, or any other media.

Contaminant
A substance that is either present in an environment where it does not belong or is present at levels that might cause harmful (adverse) health effects.

Dermal
Referring to the skin. For example, dermal absorption means passing through the skin.

Dermal contact
Contact with (touching) the skin [see route of exposure].

Detection limit
The lowest concentration of a chemical that can reliably be distinguished from a zero concentration.

Disease registry
A system of ongoing registration of all cases of a particular disease or health condition in a defined population.

Dose (for chemicals that are not radioactive)
The amount of a substance to which a person is exposed over some time period. Dose is a measurement of exposure. Dose is often expressed as milligram (amount) per kilogram (a measure of body weight) per day (a measure of time) when people eat or drink contaminated water, food, or soil. In general, the greater the dose, the greater the likelihood of an effect. An "exposure dose" is how much of a substance is encountered in the environment. An "absorbed dose" is the amount of a substance that actually got into the body through the eyes, skin, stomach, intestines, or lungs.

Dose-response relationship
The relationship between the amount of exposure [dose] to a substance and the resulting changes in body function or health (response).

Environmental media
Soil, water, air, biota (plants and animals), or any other parts of the environment that can contain contaminants.

Environmental media and transport mechanism
Environmental media include water, air, soil, and biota (plants and animals). Transport mechanisms move contaminants from the source to points where human exposure can occur. The environmental media and transport mechanism is the second part of an exposure pathway.

Exposure
Contact with a substance by swallowing, breathing, or touching the skin or eyes. Exposure may be short-term [acute exposure], of intermediate duration, or long-term [chronic exposure].

Exposure assessment
The process of finding out how people come into contact with a hazardous substance, how often and for how long they are in contact with the substance, and how much of the substance they are in contact with.

Exposure pathway
The route a substance takes from its source (where it began) to its end point (where it ends), and how people can come into contact with (or get exposed to) it. An exposure pathway has five parts: a source of contamination (such as an abandoned business); an environmental media and transport mechanism (such as movement through groundwater); a point of exposure (such as a private well); a route of exposure (eating, drinking, breathing, or touching), and a receptor population (people potentially or actually exposed). When all five parts are present, the exposure pathway is termed a completed exposure pathway.

Exposure registry
A system of ongoing follow-up of people who have had documented environmental exposures.

Groundwater
Water beneath the earth's surface in the spaces between soil particles and between rock surfaces [compare with surface water].

Hazard
A source of potential harm from past, current, or future exposures.

Health consultation
A review of available information or collection of new data to respond to a specific health question or request for information about a potential environmental hazard. Health consultations are focused on a specific exposure issue. Health consultations are therefore more limited than a public health assessment, which reviews the exposure potential of each pathway and chemical [compare with public health assessment].

Indeterminate public health hazard
The category used in ATSDR's public health assessment documents when a professional judgment about the level of health hazard cannot be made because information critical to such a decision is lacking.

Ingestion
The act of swallowing something through eating, drinking, or mouthing objects. A hazardous substance can enter the body this way [see route of exposure].

Inhalation
The act of breathing. A hazardous substance can enter the body this way [see route of exposure].

Intermediate duration exposure
Contact with a substance that occurs for more than 14 days and less than a year [compare with acute exposure and chronic exposure].

Lowest-observed-adverse-effect level (LOAEL)
The lowest tested dose of a substance that has been reported to cause harmful (adverse) health effects in people or animals.

Metabolism
The conversion or breakdown of a substance from one form to another by a living organism.

Metabolite
Any product of metabolism.

Minimal risk level (MRL)
An ATSDR estimate of daily human exposure to a hazardous substance at or below which that substance is unlikely to pose a measurable risk of harmful (adverse), noncancerous effects. MRLs are calculated for a route of exposure (inhalation or oral) over a specified time period (acute, intermediate, or chronic). MRLs should not be used as predictors of harmful (adverse) health effects [see reference dose].

Mutagen
A substance that causes mutations (genetic damage).

Mutation
A change (damage) to the DNA, genes, or chromosomes of living organisms.

National Priorities List for Uncontrolled Hazardous Waste Sites (National Priorities List or NPL)
EPA's list of the most serious uncontrolled or abandoned hazardous waste sites in the United States. The NPL is updated on a regular basis.

National Toxicology Program (NTP)
Part of the Department of Health and Human Services. NTP develops and carries out tests to predict whether a chemical will cause harm to humans.

No apparent public health hazard
A category used in ATSDR's public health assessments for sites where human exposure to contaminated media might be occurring, might have occurred in the past, or might occur in the future, but where the exposure is not expected to cause any harmful health effects.

No-observed-adverse-effect level (NOAEL)
The highest tested dose of a substance that has been reported to have no harmful (adverse) health effects on people or animals.

No public health hazard
A category used in ATSDR's public health assessment documents for sites where people have never and will never come into contact with harmful amounts of site-related substances.

NPL [see National Priorities List for Uncontrolled Hazardous Waste Sites]

Plume
A volume of a substance that moves from its source to places farther away from the source. Plumes can be described by the volume of air or water they occupy and the direction they move. For example, a plume can be a column of smoke from a chimney or a substance moving with groundwater.

Point of exposure
The place where someone can come into contact with a substance present in the environment [see exposure pathway].

Population
A group or number of people living within a specified area or sharing similar characteristics (such as occupation or age).

Public comment period
An opportunity for the public to comment on agency findings or proposed activities contained in draft reports or documents. The public comment period is a limited time period during which comments will be accepted.

Public health action
A list of steps to protect public health.

Public health advisory
A statement made by ATSDR to EPA or a state regulatory agency that a release of hazardous substances poses an immediate threat to human health. The advisory includes recommended measures to reduce exposure and reduce the threat to human health.

Public health assessment (PHA)
An ATSDR document that examines hazardous substances, health outcomes, and community concerns at a hazardous waste site to determine whether people could be harmed from coming into contact with those substances. The PHA also lists actions that need to be taken to protect public health [compare with health consultation].

Public health hazard
A category used in ATSDR's public health assessments for sites that pose a public health hazard because of long-term exposures (greater than 1 year) to sufficiently high levels of hazardous substances or radionuclides that could result in harmful health effects.

Public health hazard categories
Public health hazard categories are statements about whether people could be harmed by conditions present at the site in the past, present, or future. One or more hazard categories might be appropriate for each site. The five public health hazard categories are no public health hazard, no apparent public health hazard, indeterminate public health hazard, public health hazard, and urgent public health hazard.

Receptor population
People who could come into contact with hazardous substances [see exposure pathway].

Reference dose (RfD)
An EPA estimate, with uncertainty or safety factors built in, of the daily lifetime dose of a substance that is unlikely to cause harm in humans.

Registry
A systematic collection of information on persons exposed to a specific substance or having specific diseases [see exposure registry and disease registry].

Remedial investigation
The CERCLA process of determining the type and extent of hazardous material contamination at a site.

Risk
The probability that something will cause injury or harm.

Route of exposure
The way people come into contact with a hazardous substance. Three routes of exposure are breathing [inhalation], eating or drinking [ingestion], or contact with the skin [dermal contact].

Safety factor [see uncertainty factor]

Sample
A portion or piece of a whole. A selected subset of a population or subset of whatever is being studied. For example, in a study of people the sample is a number of people chosen from a larger population [see population]. An environmental sample (for example, a small amount of soil or water) might be collected to measure contamination in the environment at a specific location.

Sample size
The number of units chosen from a population or an environment.

Solvent
A liquid capable of dissolving or dispersing another substance (for example, acetone or mineral spirits).

Source of contamination
The place where a hazardous substance comes from, such as a landfill, waste pond, incinerator, storage tank, or drum. A source of contamination is the first part of an exposure pathway.

Special populations
People who might be more sensitive or susceptible to exposure to hazardous substances because of factors such as age, occupation, sex, or behaviors (for example, cigarette smoking). Children, pregnant women, and older people are often considered special populations.

Substance
A chemical.

Surface water
Water on the surface of the earth, such as in lakes, rivers, streams, ponds, and springs [compare with groundwater].

Survey
A systematic collection of information or data. A survey can be conducted to collect information from a group of people or from the environment. Surveys of a group of people can be conducted by telephone, by mail, or in person.

Toxic agent
Chemical or physical (for example, radiation, heat, cold, microwaves) agents that, under certain circumstances of exposure, can cause harmful effects to living organisms.

Toxicological profile
An ATSDR document that examines, summarizes, and interprets information about a hazardous substance to determine harmful levels of exposure and associated health effects. A toxicological profile also identifies significant gaps in knowledge on the substance and describes areas where further research is needed.

Toxicology
The study of the harmful effects of substances on humans or animals.

Tumor
An abnormal mass of tissue that results from excessive cell division that is uncontrolled and progressive. Tumors perform no useful body function. Tumors can be either benign (not cancer) or malignant (cancer).

Uncertainty factor
Mathematical adjustments for reasons of safety when knowledge is incomplete. For example, factors used in the calculation of doses that are not harmful (adverse) to people. These factors are applied to the lowest-observed-adverse-effect-level (LOAEL) or the no-observed-adverse-effect-level (NOAEL) to derive a minimal risk level (MRL). Uncertainty factors are used to account for variations in people's sensitivity, for differences between animals and humans, and for differences between a LOAEL and a NOAEL. Scientists use uncertainty factors when they have some, but not all, the information from animal or human studies to decide whether an exposure will cause harm to people [also sometimes called a safety factor].

Urgent public health hazard
A category used in ATSDR's public health assessments for sites where short-term exposures (less than 1 year) to hazardous substances or conditions could result in harmful health effects that require rapid intervention.

Vapor intrusion
Migration of volatile chemicals from contaminated ground water or soil into an overlying building.

Volatile organic compounds (VOCs)
Organic compounds that evaporate readily into the air. VOCs include substances such as benzene, toluene, methylene chloride, and methyl chloroform.

Volatization
The process of evaporation of a liquid into the air; VOCs such at PCE and TCE readily evaporate into air at normal ambient or room temperatures.

Other glossaries and dictionaries:
Environmental Protection Agency (http://www.epa.gov/OCEPAterms/)
National Library of Medicine (NIH) (http://www.nlm.nih.gov/medlineplus/mplusdictionary.html)

Appendix B: Timeline of Investigations at the McGaffey and Main Site

November 1994	NMED learns of contaminated groundwater while investigating a leaking underground storage tank. Groundwater samples detect benzene, toluene, ethylbenzene and xylene (BTEX) from a leaking storage tank and PCE from another source. Suspected PCE source is the Highland Center located at 1107 South Main Street, which housed a local dry cleaning business from 1955 to 1963. Other dry cleaning businesses are also identified in the area (NMED 1996).
December 1994 and early 1995	NMED tests 15 privately owned domestic and irrigation wells near the site. Some are found to be contaminated with PCE and some houses are connected to city water (NMED 1996). NMED installs one groundwater monitoring well (ED95-1) on Buena Vista Street 0.6 miles down-gradient from the site to test the shallow aquifer for contamination (NMED 1996). No PCE was detected.
September 1995	NMED installs 13 additional groundwater monitoring wells.
November 1995	NMED tests soil vapor beneath the parking lot at 1107 South Main Street and finds significant VOCs vapors in soil gas. Additional soil vapor tests are planned for 1996 (NMED 1996).
July 1996	NMED reports on a test pilot to determine if PCE can be removed from soil gas using a soil vapor extraction system. The conclusion is that a system of closely positioned extraction wells is possible, but expensive.
June 1998	NMED verifies PCE levels from municipal wells 12, 15, and 16 by checking with city personnel. NMED also verified status of existing monitoring wells and discovered that wells (3 up-gradient and 2 cross-gradient) were no longer functional.
September 1998	NMED collects groundwater samples from 21 monitoring wells and reports finding significant PCE contamination in the groundwater.
April 2000	NMED collects groundwater water samples from monitoring wells near the site
October 2000	NMED collects water samples from residential wells near the site.

July 2001	NMED releases report showing PCE in residential wells from 1994 to 2000, in monitoring wells from 1995 to 2000, and in certain city wells for Roswell from 1993 to 2000.
August/Sept 2003	EPA collects soil samples from the 35 boring locations in the parking lot at the intersection of McGaffey and Main. EPA also collects soil gas samples from the locations.
February 2003	EPA releases round 1 data report after collecting the following information: residential well survey, soil and groundwater samples to better characterize the source of contamination, and hydrogeologic investigation.
March 2003	EPA prepares draft report for sampling ambient air and soil gas at the McGaffey and Main Groundwater Plume Site. The purpose of the plan is to sample ambient (outdoor) air and soil gas at 1107 South Main Street and at nearby commercial businesses and one residence. The data was used to for run the Johnson and Ettinger vapor intrusion model.
November 2007	EPA held an Open House Public Meeting at the Roswell Adult Center on November 29, 2007 to report on the remedial investigation and risk assessment findings.
September 2008	The Record of Decision (ROD), which documents the preferred selected remedy, was issued on September 30, 2008. The ROD is contained in the Administrative Record File; it is available at the Roswell Public Library and the New Mexico Environment Department in Santa Fe, NM.
August 2010	NMED initiated and received the Groundwater Plume Remedial Design Investigation Results Report (CH2M Hill, Technical Memo-Draft; McGaffey and Main Ground water Plume Superfund Site: Groundwater Plume Area Remedial Design Investigation Results, Roswell, NM.)
June 2012	NMED initiated and received an Expanded Well Survey of groundwater wells down-gradient of the PCE/TCE plume(s).
October 2012	EPA completed the construction and initiated operation of a vapor intrusion mitigation system at six building locations that exhibit site-related PCE concentrations in indoor air.

Appendix C

Table C- 1. Summary of Private Well Usage, Status, and PCE and TCE Concentrations.

Table C- 1. Summary of Private Well Usage, Status, and PCE and TCE Concentrations.

Private Well *	Well Depth (feet) Groundwater Zone	Date	PCE conc. (µg/L)	TCE conc.† (µg/L)	Operating Status (2002)	Used for Drinking/ Bathing	Garden and Lawn Irrigation	Comments
SM-01	Unknown‡	12/14/94 4/3/95 4/00 10/00 2002	11 6.1 5.9 4.0 ND§	1.6	Unknown	Unknown	Unknown	Not surveyed in 2002
SM-02	Unknown	1/30/95 2/13/95	79 60		Unknown	Unknown	Unknown	Not surveyed in 2002
SM-03	Unknown	2/21/95 10/00	69 187		Unknown	Unknown	Unknown	Not surveyed in 2002

* The specific addresses are omitted because of privacy issues.
† TCE was not sampled in private wells until 2002.

‡ The groundwater aquifers in this area are not well characterized so the P zone designation is uncertain. In addition, while the depth of the well is known for some addresses, the depth of the screen for withdrawing water is not known. For these reasons the depth from which groundwater is withdrawn is uncertain for most of the private wells.
§ ND is not detected.

Private Well *	Well Depth (feet) Groundwater Zone	Date	PCE conc. (µg/L)	TCE conc.† (µg/L)	Operating Status (2002)	Used for Drinking/ Bathing	Garden and Lawn Irrigation	Comments
SM-04	Unknown	2/21/95 4/00 10/00 2002 12/10 7/11 8/12	40 154 193 214 30 82 89	6.2 10 2.1	Unknown	Unknown	Unknown	Not surveyed in 2002
SM-05	97	2/21/95 10/00 2002 5/09	18 97 147 133	3.2 1.3	Unknown	Unknown	Unknown	Not surveyed in 2002
SM-06**	Unknown	1/27/95 5/18/95 9/26/00	105 260 1.7		Yes	Yes/Yes	Yes	Not surveyed in 2002. NMED reported that resident used well until 1995.
SM-07	Deep aquifer††	4/28/95 6/08/95 10/00	57.3 53 151		No	No/No	No	Well is in disrepair and is not used.

** The house at this address was connected to city water in 1995.

†† A 1996 NMED report states that this well draws water from the deeper aquifer; however, the water chemistry for this well indicates that water from this well could be a mix of water from the deep and shallow aquifer. Therefore, it may be possible that PCE in this well is coming from PCE contamination of the shallow aquifer (NMED 1996).

Private Well *	Well Depth (feet) Groundwater Zone	Date	PCE conc. (µg/L)	TCE conc.† (µg/L)	Operating Status (2002)	Used for Drinking/ Bathing	Garden and Lawn Irrigation	Comments
SM-08	Unknown	12/14/94	1.6		Unknown	Unknown	Unknown	Not surveyed in 2002
SM-09	125	12/15/94	ND		Yes	No/No	Yes	Private well is not connected to house
SM-10	Unknown	1/30/95 2/13/95 10/00	ND ND ND		Yes	No/No	Yes	Residence is connected to city water; private well is not connected to house
SM-11	80	10/00 2002	ND ND	ND	Yes	No/No	Yes	Residence is connected to city water; well is not connected to house
SM-12	Unknown	10/00 12/10	1 1.4	ND	Yes	No/?	Yes	Residence is connected to city water; private well is connected to house; unclear is private well might be used for bathing
SM-13	Unknown	10/00	0.1		Unknown	Unknown	Unknown	Not surveyed in 2002
SM-14	Unknown	1/30/95 7/15/96 10/00 2002 5/09	0.5 ND 0.5 2.5 2.1	ND ND	Yes	No/No	Yes	Private well not connected to house

Private Well *	Well Depth (feet) Groundwater Zone	Date	PCE conc. (µg/L)	TCE conc.† (µg/L)	Operating Status (2002)	Used for Drinking/ Bathing	Garden and Lawn Irrigation	Comments
SM-15	Unknown	4/28/95 12/14/95 7/15/96 2002	ND ND ND ND	ND	Yes	No/No	Yes	Private well is not connected to house;
SM-16	Unknown	12/994 5/18/96 10/00 2002	2.2 2.8 3.0 ND	ND	Unknown	Unknown	Unknown	Not surveyed in 2002
SM-17	170	2/21/95 10/00 2002	36 16 10.6	2.4	Unknown	Unknown	Unknown	Not surveyed in 2002
SM-18	Unknown	2/13/95	ND		Unknown	Unknown	Unknown	Not surveyed in 2002
SM-19	165	10/00 2002 12/10	0.2 ND 0.7	ND ND	Yes	No/Yes	Yes	This well was constructed in 2000, the residence is not connected to city water; private well is operational and is used for bathing.
SM-20	Unknown	1/27/95 10/00 2002	ND 0.4 ND		Unknown	Unknown	Unknown	Not surveyed in 2002
SM-21	Shallow aquifer	10/00	46		Unknown	Unknown	Unknown	Not surveyed in 2002

Private Well *	Well Depth (feet) Groundwater Zone	Date	PCE conc. (µg/L)	TCE conc.† (µg/L)	Operating Status (2002)	Used for Drinking/ Bathing	Garden and Lawn Irrigation	Comments
SM-22	Deep aquifer	4/28/95 10/00 2002 5/09 12/10	ND ND ND 1.4 ND	ND ND ND	Yes	No/No	Yes	Private well not connected to house
SM-23	Unknown	12/14/94	2.2		Unknown	Unknown	Unknown	Not surveyed in 2002
SM-24	Unknown	7/25/95 7/24/95	2.5 1.3		Unknown	Unknown	Unknown	Not surveyed in 2002
SM25	Unknown	Not sampled			No	No/No	Yes	Residence is connected to city water; Resident reports using private well for filling pools. Comment: a well in disrepair appears to be at this property but is not used.
SM26	Unknown	Not sampled			No	No/No	Yes	Residence is connected to city water, Resident reports using private well for swimming pool but well was recently covered with dirt.

Private Well *	Well Depth (feet) Groundwater Zone	Date	PCE conc. (µg/L)	TCE conc.† (µg/L)	Operating Status (2002)	Used for Drinking/ Bathing	Garden and Lawn Irrigation	Comments
SM27	Unknown	Not Sampled			No	No/?	Yes	Resident reports using private well for garden and lawn; residence is connected to city. Well appears in disrepair.

Appendix D: Health Comparison Values and Dose Calculation Procedures

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 specific toxicological effects of the substance, determine the health effects that may result. The following summary of ATSDR's procedure for developing health comparison values and calculating exposure doses is derived from the ATSDR Public Health Assessment Guidance Manual (ATSDR, 2005).

ATSDR considers these physical and biological characteristics when developing health guidelines. Health guidelines provide a basis for evaluating exposures estimated from concentrations of contaminants in different environmental media (soil, air, water, and food) depending on the characteristics of the people who may be exposed and the length of exposure. Health guideline values are in units of dose such as milligrams (of contaminant) per kilogram of body weight per day (mg/kg/day).

ATSDR reviews health and chemical information in documents called toxicological profiles. Each toxicological profile covers a particular substance; it summarizes toxicological and adverse health effects information about that substance and includes health guidelines such as ATSDR's minimal risk level (MRL), EPA's reference dose (RfD) and reference concentration (RfC), and EPA's cancer slope factor (CSF). ATSDR uses these guidelines to determine a person's potential for developing adverse non-cancer health effects and/or cancer from exposure to a hazardous substance.

An MRL is an estimate of daily human exposure to a contaminant that is likely to be without an appreciable risk of adverse non-cancer health effects over a specified duration of exposure for sensitive individuals and children (acute, less than 15 days; intermediate, 15 to 364 days; chronic, 365 days or more). Oral MRLs are expressed in units of milligrams per kilogram per day (mg/kg/day); inhalation MRLs are expressed in micrograms per cubic meter ($\mu g/m^3$). MRLs are not derived for dermal exposure.

For nonradioactive chemicals, ATSDR uses comparison values like environmental media evaluation guides (EMEGs), cancer risk evaluation guides (CREGs), reference dose (or concentration) media evaluation guides (RMEGs), and others. EMEGs, since they are derived

from MRLs, apply only to specific durations of exposure. They depend on the amount of a contaminant ingested or inhaled. Thus, EMEGs are determined separately for children and adults, and for various durations of exposure. A CREG is an estimated concentration of a contaminant that would likely cause, at most, one excess cancer in a million people exposed over a 78 year lifetime. CREGs are calculated from CSFs. Reference dose (or concentration) media evaluation guides (RMEGs) are media guides based on EPA's RfDs and RfCs.

EPA's maximum contaminant levels (MCLs) are maximum contaminant concentrations of chemicals allowed in public drinking water systems. MCLs are regulatory standards set as close to health goals as feasible and are based on treatment technologies, costs, and other factors.

Health comparison values, such as EMEGs and MCLs, are derived using standard intake rates for inhalation of air and ingestion of water, soil, and biota. These intake rates are derived from the ATSDR Public Health Assessment Guidance Manual (ATSDR 2005) or from the EPA Exposure Factors Handbook (EPA 2011b). Doses calculated using health protective exposure factors and environmental concentrations are considered "health protective doses" because it is unlikely that any real community exposures are greater than the calculated doses and are most likely to be less than the health protective doses.

After estimating the potential exposure at a site, ATSDR identifies the site's "contaminants of concern" by comparing the exposures of interest with health guidelines, or contaminant concentrations with comparison values. As a general rule, if the guideline or value is exceeded, ATSDR evaluates exposure to determine whether it is of potential health concern. Sometimes additional medical and toxicological information may indicate that these exposures are not of health concern. In other instances, exposures below the guidelines or values could be of health concern because of interactive effects with other chemicals or because of the increased sensitivity of certain individuals. Thus additional analysis is necessary to determine whether health effects are likely to occur.

For cancer-causing substances, EPA established the cancer slope factor (CSF; http://www.epa.gov/iris/help_ques.htm#cancersf). A CSF is used to estimate the theoretical excess cancer risks expected from maximal exposure for a lifetime. Cancer risk evaluation guides (CREGs) are estimated contaminant concentrations that would be expected to cause an estimated excess theoretical cancer risk less than 1.0E-06 (or 0.000001). The CREGs and CSFs represent statistical estimates of risk and are not indicative of actual health effects. Specifically, a one in a million risk does not mean that one person (out of a million exposed) will get cancer, but rather that one person exposed has a theoretical cancer risk probability of 1.0E-06.

The CREG values are derived assuming continuous (24 hours per day) long-term exposure to the chemical at the maximum detected values, which likely overestimates the occupational exposures to indoor air at this site. In order to adjust a continuous exposure (24 hours per day, 7 days per week, 78 year lifetime) to exposures likely in a business or commercial setting, the USEPA

recommends the calculation of an adjusted **Exposure Concentration** (Equation 1; EPA 2009). Excess cancer risk is then calculated by multiplying the adjusted Exposure Concentration by the Inhalation Unit Risk (IUR; or excess risk per unit of concentration; Equation 2, below). Note that because the vapor intrusion exposures occurred only in commercial buildings the exposures are based on adult occupational exposure factors. While children might occasionally be in those buildings, it would be infrequent relative to full-time adult workers.

Using the highest measured PCE air concentration (5,621 $\mu g/m^3$) as a starting point and assuming that workers are present in the affected buildings for 8 hours per day for 5 days per week, the resulting weekly Exposure Concentration is 1,338 $\mu g/m^3$. For comparison with a lifetime (78 year) cancer risk evaluation guide the weekly exposure concentration is further adjusted to account for a 20 year occupational exposure* (1,338 $\mu g/m^3$ x 20/78 = 367 $\mu g/m^3$). Multiplying the adjusted 20 year exposure concentration times the PCE inhalation unit risk results in an excess cancer risk of 0.00009; Table 6). Excess cancer risks between 0.0001 and 0.000001 are within the EPA "target risk range" and defined as "low to no apparent increased risk" by ATSDR. As the theoretical cancer risk for the building with the highest measured PCE air concentration is less than a low increased cancer risk, PCE inhalation exposures at this site do not represent a public health hazard.

Exposure Concentration = (CA x EF) (Equation 1)

Where: EC ($\mu g/m^3$) = exposure concentration;
CA ($\mu g/m^3$) = contaminant concentration in air;
EF (Exposure Factor) = F (exposure frequency- 40 hours/week) x ED (duration- 1 week)/AT (averaging time (ED x 168 hours/week)

Risk = IUR x EC (Equation 2)

Where: IUR $(\mu g/m^3)^{-1}$ = Inhalation Unit Risk; and
EC ($\mu g/m^3$) = exposure concentration (See Equation 1).

Above equations are from: (USEPA, 2009)
http://www.epa.gov/oswer/riskassessment/ragsf/pdf/partf_200901_final.pdf

Exposure doses via ingestion are calculated on the basis of the following equation:

Dose (Ingestion) = (Chemical Conc. x IR x EF x ED) / (BW x AT) [equation 3]

Where:

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* The EPA Exposure Factors Handbook indicates that the median (50th percentile) occupational tenure for all US workers is 6.6 years (EPA, 2011b; Table 16-103). This may over-estimate location specific employment as it includes worker tenure for multiple employers and intermittent time periods. A twenty year occupational tenure is a health protective estimate of site-specific occupational tenure.

Chemical Conc.	=	concentration of each contaminant (in µg/L)
IR	=	ingestion rate (in liters/day)
EF	=	exposure frequency in days per year
ED	=	exposure duration in years
BW	=	body weight in kilograms
AT	=	averaging time in days

As previously discussed, exposure to VOCs such as PCE and TCE from contaminated drinking water occurs via inhalation of vapors and direct dermal absorption as well as from ingestion of water. Consequently, the ingestion doses as calculated by the above equation are multiplied by two to account for the cumulative ingestion/inhalation/dermal uptake dose. The doses calculated using the above equation are presented in units of milligrams (PCE or TCE) per kilogram of body weight per day (mg/kg/day; Table 6; Figures 4 and 5). Note that inhalation exposures and health comparison values for the vapor intrusion pathway (Table 5, Figure 3) are presented in concentration units ($\mu g/m^3$).

The various exposure factors used in calculating exposures for the groundwater pathway are summarized in Table D-1. The groundwater pathway occurs in a residential setting and children are likely to have the highest exposure. This is particularly important for TCE exposures. Because TCE is a carcinogen with a potential mutagenic mode of action, infants and young children may be especially susceptible to cancer effects. Consequently, TCE cancer risk calculations use age dependent adjustment factors (ADAFs) to account for this susceptibility. TCE cancer risks for infants (age 0 to 2) are multiplied by the ADAF of 10, and risks for children (age 2 to 16) are multiplied by the ADAF of 3 (Table D-1).

Table D-1. Parameters Used to Calculate Doses and Risks for Groundwater Exposures

Age (yrs)	Intake Rate (L/day)	Body weight (kg)	ED (yrs)	EFc (unitless)	ADAF (unitless)
0--<2	1	12	2	2	10
2--<6	1	17	4	2	3
6--<16	1.7	44	10	2	3
≥16	2	80	16	2	1

Doses are calculated using equation 3 assuming that exposure occurs every day (EF x ED)/AT = 1.
All ingestion doses multiplied by exposure factor (EFc) of 2 to account for inhalation and dermal uptake.
Sixteen year child cancer risks are sum of risks for 0-<2, 2-<6, and 6-<16 year age groups x 16/78 (portion of 78 year lifetime).
TCE cancer risks multiply age group specific doses x ADAFs before summing as above.