ATSDR
AGENCY FOR TOXIC SUBSTANCES AND DISEASE REGISTRY

Public Health Assessment for

NORTH SHORE DRIVE GROUNDWATER CONTAMINATION
UNINCORPORATED ELKHART COUNTY, INDIANA

EPA FACILITY ID: INSFN0507828

APRIL 6, 2017

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

Comment Period Ends:

MAY 18, 2017

THE ATSDR PUBLIC HEALTH ASSESSMENT: A NOTE OF EXPLANATION

This Public Health Assessment-Public Comment Release 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. This document represents the agency's best efforts, based on currently available information, to fulfill the statutory criteria set out in CERCLA section 104 (i)(6) within a limited time frame. To the extent possible, it presents an assessment of potential risks to human health. Actions authorized by CERCLA section 104 (i)(11), or otherwise authorized by CERCLA, may be undertaken to prevent or mitigate human exposure or risks to human health. In addition, ATSDR will utilize this document to determine if follow-up health actions are appropriate at this time.

This document has previously been provided to EPA and the affected state in an initial release, as required by CERCLA section 104 (i)(6)(H) for their information and review. Where necessary, it has been revised in response to comments or additional relevant information provided by them to ATSDR. This revised document has now been released for a 45-day public comment period. Subsequent to the public comment period, ATSDR will address all public comments and revise or append the document as appropriate. The public health assessment will then be reissued. This will conclude 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.

Use of trade names is for identification only and does not constitute endorsement by the Public Health Service or the U.S. Department of Health and Human Services.

Please address comments regarding this report to:

Agency for Toxic Substances and Disease Registry
Attn: Records Center
1600 Clifton Road, N.E., MS F-09
Atlanta, Georgia 30333

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PUBLIC HEALTH ASSESSMENT

NORTH SHORE DRIVE GROUNDWATER CONTAMINATION
UNINCORPORATED ELKHART COUNTY, INDIANA

EPA FACILITY ID: INSFN0507828

Prepared by:

Eastern Branch
Division of Community Health Investigations
U.S. Department of Health and Human Services
Agency for Toxic Substances and Disease Registry

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SUMMARY

Introduction

The North Shore Drive Groundwater site is located in unincorporated Elkhart County, Indiana. The North Shore Drive area is a mix of residential and commercial properties. The site was discovered by the Indiana Department of Environmental Management (IDEM) around 1997 after a concerned citizen contacted IDEM and complained of skin irritation, headaches, and rashes. The citizen believed the health effects were caused by the water from their private well. Water from the private wells in the area are used for household purposes, including drinking and bathing.

As a result of the complaint, the IDEM began sampling the groundwater in the area for contamination. The sampling results revealed that residential wells in the area were contaminated with VOCs, primarily trichloroethylene (TCE). TCE was detected in wells at levels that exceeded EPA's Maximum Contaminant Level (MCL) of 5 ppb for TCE.

The IDEM is currently supplying and maintaining point of entry (POE) carbon filtration systems to 9 homes in North Shore area identified as having well contamination levels exceeding the Maximum Contaminant Level (MCL) of 5 ppb for TCE. IDEM is also investigating potential sources of the groundwater contamination. The source(s) of the contamination has not yet been determined.

The site is listed on the NPL as is undergoing the remedial process under the Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA). EPA is the lead agency for the remedial process and will be conducting a site investigation to characterize the site to determine appropriate remedial actions.

Conclusions

ATSDR evaluated the available environmental data and reached the following conclusions regarding the North Shore Drive site:

Conclusion 1

TCE is the main contaminant of concern in groundwater at this site. People who used TCE-contaminated well water for household purposes (e.g., drinking, showering, bathing, etc.) in the past may be at risk for harmful non-cancer health effects associated with TCE exposure.

Basis for Conclusion ATSDR evaluated whether children and adults exposed to different levels of TCE (ranging from 19 to 85 ppb) detected in private wells might experience harmful health effects. Based on this evaluation, children and adults exposed to TCE in private wells in the past, at concentrations evaluated in this assessment, may be at an increased risk for harmful immunological effects. Additionally, if a pregnant woman was exposed to TCE during the first trimester of pregnancy, her baby may be at increased risk for a heart birth defect.

Next Steps Use of TCE-contaminated water is not a current exposure pathway. The Indiana Department of Environmental Management (IDEM) is supplying and maintaining point of use (POU) carbon filtration systems to 9 homes in the North Shore area identified as having well contaminant levels exceeding the Maximum Contaminant Level (MCL) of 5 ppb for TCE.

ATSDR recommends continued monitoring of the filtration systems to ensure that the systems are operating properly.

ATSDR recommends routine monitoring of private wells until the groundwater reaches remedial goals or until residents are supplied with another permanent source of water.

ATSDR also recommends monitoring of the groundwater contaminant plume to determine if the plume is migrating to additional residential wells.

ATSDR recommends full characterization of the site to ensure the conclusions and recommendations in this evaluation are appropriate.

Conclusion 2 Children and adults exposed to the highest measured TCE concentration (85 ppb) may be at an increased risk for cancer.

Basis for Conclusion ATSDR estimated the increased cancer risk for individuals who were exposed to TCE in their well water via the ingestion, inhalation, and dermal routes of exposure. We assumed that children were exposed for 21 years (from birth to 21 years of age) and that adults were exposed for a total of 33 years. The estimated risks indicate that exposure to the maximum concentration (85 ppb) of TCE–contaminated water could have increased the risk to 1 in 10,000 for adults and 2 in 10,000 for children. We interpret this as an elevated cancer risk for children and adults. However, ATSDR assumed many years of exposure in estimating the

cancer risks, so the actual cancer risks could be much lower. Without historical data or knowledge about the source of the contamination, ATSDR recognizes that there is some uncertainty in estimating cancer risk at this site.

Next Steps

The IDEM is currently supplying and maintaining point of entry (POE) carbon filtration systems to homes in North Shore area identified as having well contamination levels exceeding the Maximum Contaminant Level (MCL) of 5 ppb for TCE.

ATSDR recommends that currently installed filtration systems be monitored for proper operation and that steps be taken to reduce exposures to TCE or other VOCs, as appropriate.

ATSDR recommends further investigation of the nature and extent of contamination (including the source of contamination) at the site.

Conclusion 3

A potential current exposure pathway to consider at this site is the vapor intrusion pathway. Vapor intrusion is the migration of volatile organic compounds (VOCs) from the subsurface-contaminated groundwater and soil through the pore spaces of soil into the indoor air above buildings. ATSDR could not evaluate this pathway because the pathway has not yet been investigated.

Basis for Conclusion

Vapor intrusion warrants consideration because of the volatile nature (i.e., easily evaporates) of TCE and because the plume runs underneath residential homes and other structures. More data are needed to determine if residents could be exposed to VOCs at levels of potential health concern in the indoor air if vapor intrusion is occurring at this site.

Next Steps

ATSDR recommends a prioritized investigation of the vapor intrusion pathway to determine the significance of this potential pathway at this site.

STATEMENT OF ISSUES

On September 16, 2014, the United States Environmental Protection Agency (EPA) added the North Shore Drive Site, unincorporated Elkhart County, Indiana, to the National Priorities List (NPL) of Superfund sites. Pursuant to the Comprehensive Environmental Response, Compensation and Liability Act of 1980 (CERCLA) and the Superfund Amendments and Reauthorization Act (SARA) of 1986, the Agency for Toxic Substances and Disease Registry (ATSDR) is required to conduct public health assessment activities for sites listed or proposed to be added to the NPL.

In this PHA, ATSDR evaluates the concentrations of toxic substances present at this site and the pathways by which people living on or near the site may be exposed to those substances. If site-related toxic substances are present at concentrations of health concern, ATSDR determines if such exposures are at levels likely to cause harm to human health.

This PHA does not address past exposures to workers at commercial establishments who could have been exposed when or soon after these toxic substances were released. There is no information that would allow an evaluation of these workers and ATSDR does not typically evaluate work-related exposures.

BACKGROUND

Site Description and History

The North Shore Drive site is located in unincorporated Elkhart County, Indiana. The approximately 70-acre site is a groundwater contaminant plume bounded by the St. Joseph River to the south, Old U.S. 20 to the north, Sheridan Boulevard to the east, and Corwin Drive to the west. The area includes approximately 35 residential properties and a few commercial establishments. The commercial establishments are located along U.S. 20, north of the residential area in question. The North Shore Drive area has been an area of mixed usage for approximately 45 years or longer [IDEM 2003].

The first indication of groundwater contamination occurred in 1997 when a concerned citizen contacted the Indiana Department of Environmental Management (IDEM) to complain of skin irritation, headaches, and rashes. The citizen believed their health effects were caused by the water from their private well that was used for drinking and other household purposes. Most residences along North Shore Drive and the surrounding area use private wells as a source of drinking water [IDEM 2003]. After the citizen's complaint, the IDEM began investigating the North Shore Drive area for potential groundwater contamination. During their investigations, the IDEM collected water samples from private wells and limited soil samples from nearby commercial properties that were determined to be the most likely for potential contamination to exist. The samples were tested for volatile organic compounds (VOCs), semi-volatile organic

compounds (SVOCs), metals and pesticides.[1] The early investigations revealed that private residential wells, and thus the groundwater, in the area were contaminated with VOCs, primarily trichloroethylene (TCE). TCE was detected in wells at levels that exceeded EPA's Maximum Contaminant Level (MCL)[2] of 5 ppb for TCE. Other VOCs were also detected, including tetrachloroethylene (PCE) and *cis*-1,2-dichloroethylene (cis-1,2-DCE), but at levels below EPA's MCL for these chemicals [IDEM 2003].

In 2002, IDEM collected soil samples from the footprint of a former junkyard, and from nearby residential wells, in an effort to determine the source of the groundwater contamination in the area. A total of 6 soil samples were collected at a depth of approximately 4 feet below ground surface. The soil sample analytical results revealed the presence of low levels of inorganic compounds and two VOCs - acetone and methylene chloride. The VOCs, however, were likely present due to laboratory contamination, and are therefore, not considered to be site-related. All soil samples were collected at a depth of approximately 4 feet below ground surface [IDEM 2003]. Because people are unlikely to come into contact with these subsurface soils, ATSDR did not evaluate the soil samples in this PHA.

IDEM is currently supplying and maintaining point of entry (POE) carbon filtration systems[3] to nine homes in the North Shore Drive area identified as having well contaminant levels exceeding the MCL of 5 ppb for TCE. IDEM also investigated potential sources of the groundwater contamination. The source(s) of the contamination has not yet been determined.

The site is listed on the NPL and is undergoing the remedial process under CERCLA. EPA is the lead agency for the remedial process and will be conducting a site investigation to characterize the site to determine appropriate remedial actions.

Demographics

ATSDR examines demographic data to identify sensitive populations – such as young children, the elderly, and women of childbearing age – to determine whether these sensitive populations are exposed to contaminants at levels that may pose a health risk. Demographics also provide details on population mobility and housing statistics for a particular area. According to the 2010 U.S. census, 5,418 people live within one mile of the North Shore Drive site boundary – 1,077 of whom are women of childbearing age, 566 are children aged 6 and younger, and 600 are adults aged 65 and older (See Figure 1).

[1] As the investigation progressed, more sample testing was limited to VOCs, as it became apparent that VOCs were the contaminants of potential concern at this site.
[2] The Maximum Contaminant Level (MCL) is the highest level of a chemical that EPA allows in public drinking water systems. MCLs are enforceable standards as required by the Safe Drinking Water Act.
[3] The IDEM initially provided bottled water to homes with contaminants in wells that ed an MCL. Around June 1997, IDEM selected point of use (POU) filtration systems to replace the bottled water service. Previously, connection to the municipal water service was not feasible because the closest municipal water main to any property was more than a mile away [IDEM memo from I. Ewusi-Wilson to H. Atkinson, dated May 16, 1997].

Groundwater in the area

The Indiana Department of Natural Resources indicates that the North Shore Drive area is underlain by layers of sand and gravel. Locally, groundwater flow is to the south towards the St. Joseph River [IDEM 2003]. Most of the drinking water within a 4-mile radius of the site is believed to be served by private wells, although some commercial establishments may get their water from the municipal water supply. Approximately 150 people in the immediate North Shore Drive area are served by private wells. Additional information on drinking water sources is needed. The municipal wells for the City of Elkhart are approximately 2.5 miles east of the site [IDEM 2003].

Figure 1. General Site Demographic Profile

North Shore Drive Area

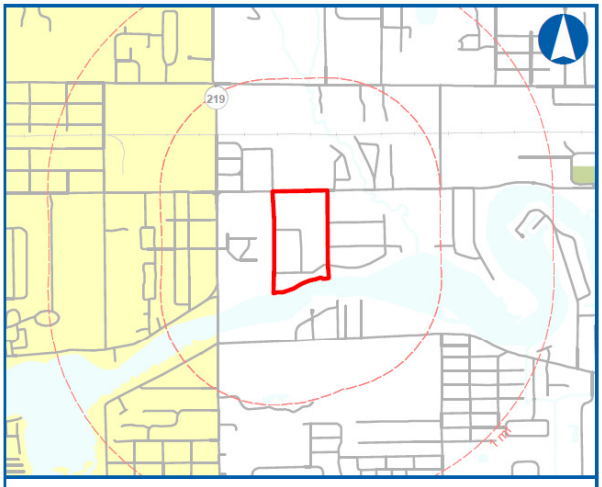
Elkhart, Elkhart County, IN

GENERAL SITE PROFILE

EPA FACILITY ID INSFN0507828

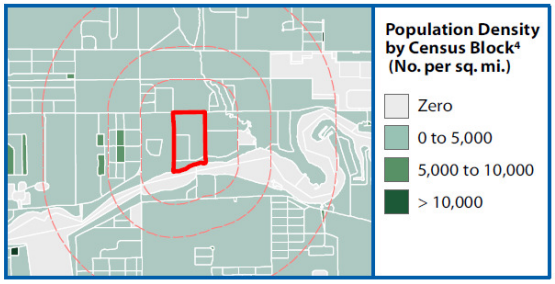
INTRODUCTORY MAP SERIES

Site Vicinity Map

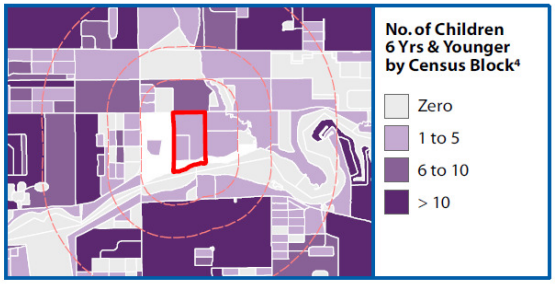


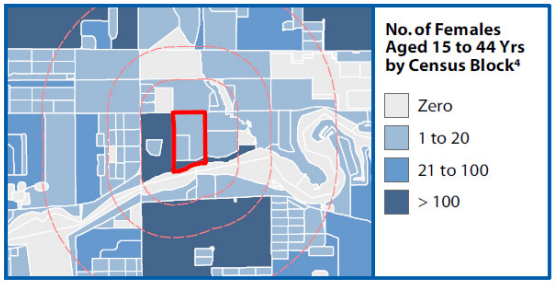
The **General Site Profile Map** depicts the hazardous waste site of interest, highlights locations of other environmental hazards and community gathering points, and provides community demographic and housing statistics.

General Population Density



Sensitive Populations





Demographic Statistics[4,5]

Within 1 mile buffer of site boundary

Measure	2000	2010	Change
Total Population	4,347	5,418	+24%
White Alone	4,186	4,885	+16%
Black Alone	35	251	+617%
Am. Indian & Alaska Native Alone	5	12	+140%
Asian Alone	55	54	-1%
Native Hawaiian & Other Pacific Islander Alone	0	12	N/A
Some Other Race Alone	27	68	+151%
Two or More Races	39	133	+241%
Hispanic or Latino[6]	83	205	+146%
Children Aged 6 and Younger	443	566	+27%
Adults Aged 65 and Older	454	600	+32%
Females Aged 15 to 44	895	1,076	+20%
Housing Units	1,595	2,063	+29%
Housing Units Pre 1950	102	145	+42%

Data Sources: [1]ATSDR GRASP Hazardous Waste Site Boundary Database. [2]ATSDR GRASP. [3]TomTom International BV (2012). [4]US Census 2010. **Notes:** [5]Calculated using area-proportion spatial analysis method. [6]Individuals identifying origin as Hispanic or Latino may be of any race. **Projection:** Projection used for all map panels is NAD 1983 StatePlane Indiana East FIPS 1301 Feet.

PRJ 04576 IWE7 3/19/15

GRASP

Agency for Toxic Substances and Disease Registry

Division of Toxicology and Human Health Sciences

ATSDR

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Community Concerns

ATSDR has not yet gathered community concerns at this site, but intends to do so in the near future. ATSDR plans to conduct a site visit and perform public health activities at the site, as needed. ATSDR has communicated with other involved federal, state and local agencies in an effort to understand the concerns of the community. The only documented concern has been that some community members are concerned about potential health effects from using the contaminated water for household purposes. That community concern is addressed in this document.

ENVIRONMENTAL CONTAMINATION

Environmental sampling data are critical inputs to the public health assessment process. ATSDR uses available environmental sampling data collected on or near the site for our evaluation. In most cases, the environmental data are collected by EPA, other federal/state/local governmental agencies, or involved third parties. Environmental data indicate the levels of chemicals found in water, soil, air or the food chain (biota). ATSDR determines whether the available environmental data are adequate to determine if people could have been (a past scenario), are currently (a current scenario), or could be (a future scenario) exposed to site-related contaminants. If the data are not adequate, then ATSDR will request additional data to fill critical data gaps, as needed.

The Screening Analysis - Identifying Chemicals of Concern

During the screening analysis, ATSDR sorts through the environmental data in a consistent manner to identify chemicals within completed and potential exposure pathways that may need to be evaluated more closely. ATSDR selects chemicals for further evaluation by comparing them against health-based (or technology-based such as the MCL) screening values. These screening values are developed from the available scientific literature on the magnitude of exposure and health effects. Screening values are derived for each of the different environmental media (e.g. air, water, soil), and each reflects an estimated contaminant concentration that is *not expected* to cause adverse health effects for a given chemical, assuming a standard daily contact rate (e.g., amount of water or soil consumed or amount of air breathed) and body weight. To be conservative and protective of public health, screening values are generally based on contaminant concentrations *many times lower than levels at which no effects were observed* in experimental animals or human studies. ATSDR does not use screening values to predict the occurrence of adverse health effects, but rather to serve as a health protective first step in the evaluation process.

The first screening analysis involves ATSDR selecting the chemicals for further evaluation by comparing the chemical concentrations detected to *comparison values* (CVs) ATSDR has developed CVs for substances in drinking water, soil, and air. ATSDR's comparison values CVs include: environmental media evaluation guides (EMEGs), reference dose media evaluation guides (RMEGs), and cancer risk evaluation guides (CREGs). CREGs, EMEGs, and RMEGs are non-enforceable, health-based CVs developed by ATSDR for screening environmental contamination for further evaluation. When no ATSDR CV is available, the CV from other sources, such as EPA's Maximum Contaminant Levels (MCLs), may be used. MCLs are enforceable drinking water regulations developed to protect public health, taking feasibility and cost into consideration. Other factors that become important in deciding which chemicals to evaluate further include the frequency of detection and a chemical's inherent toxicity. See Appendix B for more information on CVs.

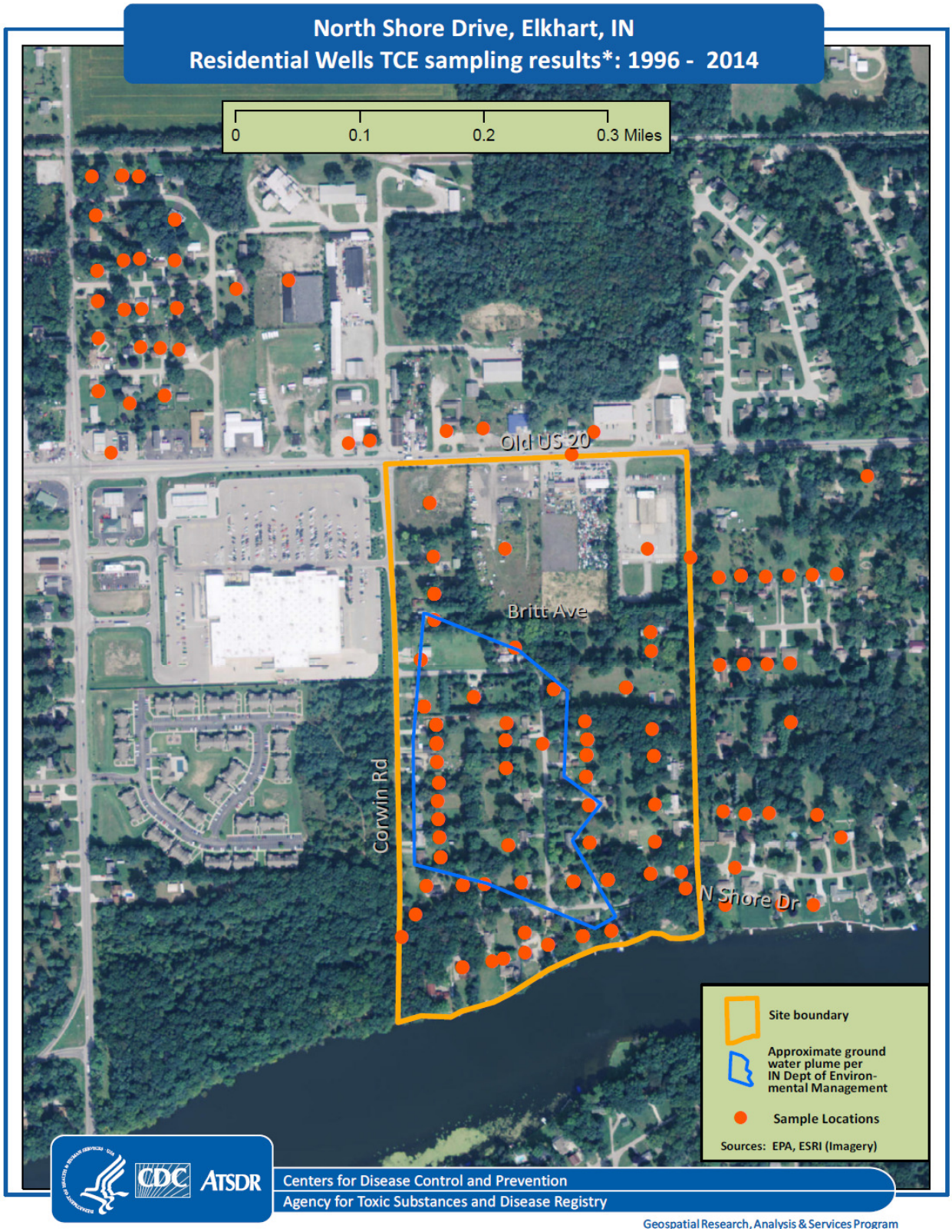
ATSDR defines a comparison value (CV) as a calculated concentration of a substance in air, water, food, or soil that is unlikely to cause harmful (adverse) health effects in exposed people. The CV is used as a screening tool during the public health assessment process. Substances found in amounts greater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

If contaminant concentrations are above these environmental screening values (or CVs), ATSDR analyzes exposure variables (for example, duration and frequency), the toxicology of the contaminant, and epidemiology studies for possible health effects. During this part of the public health assessment process, ATSDR estimates site-specific exposure doses and compares them to health guideline values. This health guideline comparison allows health assessors to study possible public health implications of site-specific conditions. Health guidelines are derived based on data drawn from the epidemiologic and toxicological literature with many uncertainty or safety factors applied to ensure that they are amply protective of human health. ATSDR's minimal risk level (MRL) and EPA's reference doses (RfDs) and reference concentrations (RfCs) are the health guidelines most commonly used in the public health assessment screening process. Estimated doses that are below health guidelines are not expected to cause adverse health effects.

Data Used in this Evaluation – Nature and Extent of Contamination

The major sources of data for this evaluation are reports prepared by the IDEM and information contained on EPA's website for the NPL listing for the site. A number of environmental investigations have been completed for this site. Particularly, in 2003, the IDEM prepared a Preliminary Assessment/Site Inspection Report for the North Shore Drive area that documents the results of the investigation of the TCE plume until 2002 [IDEM 2003]. The IDEM also prepared and submitted to ATSDR various field and analytical reports, dating from 1995 to 2014, detailing their groundwater investigation in the North Shore Drive and surrounding areas [IDEM 1995-2014]. Over these years of investigation, IDEM has collected many residential well samples in and around the North Shore Drive area (See Figure 2).

Figure 2. Sampling Locations in and around the North Shore Drive Area



TCE is the main contaminant of potential concern at the North Shore Drive Site. The highest TCE concentration detected in a private well was 85.4 ppb. TCE was the only chemical detected at levels above an EPA MCL (5 ppb) or an ATSDR CV (0.43 ppb). In addition to TCE, other related chlorinated solvents, or VOCs, including PCE and dichloroethene (DCE), were detected in some private wells. However, none of the other VOCs (including some not listed below[4]) were detected at levels that exceeded EPA's MCL or ATSDR's CV for the chemicals. Table 1 shows the maximum detected concentrations of each of these VOC contaminants of potential concern in groundwater. As shown in Table 1, TCE is the only contaminant detected at concentrations above applicable comparison values. Therefore, the remainder of this document will focus only on TCE as a contaminant of concern.

Table 1. Maximum Concentration of VOCs of Potential Concern Detected in Private Wells, 1996 - 2014

Chemical	*Units*	*Maximum Concentration Detected*	*EPA MCL*	*ATSDR CV*
TCE (trichloroethylene)	ppb	**85.4**	5	0.43 CREG
PCE (tetracholorethylene)	ppb	3.5	5	12 CREG
cis-1,2-DCE (dichloroethylene)	ppb	2.7	70	14 (Child RMEG)

ppb - parts per billion
NA – not available
MCL – EPA's maximum contaminant level (national primary drinking water standard)
ATSDR CV – ATSDR comparison value for drinking water
CREG – ATSDR cancer risk evaluation guide
RMEG – ATSDR reference dose media evaluation guide
Bolded text means the value is greater than the comparison value (CV)

Between 1996[5] and 2014, the IDEM collected private wells samples in and around the North Shore Drive area to determine if chemicals were in groundwater at concentrations above the drinking water standard, or the MCL. The sampling results for TCE over the years are presented in Table 2. Some private wells were re-tested in subsequent years or at different times within the same year. The IDEM bypassed any previously-installed filtration system when taking additional samples at a residence.

[4] Other VOCs were detected but not listed here because either the chemical was not frequently detected, is believed to be a laboratory contaminant, or was not considered to be site-related. In all instances, the VOC concentration detected was less than the applicable drinking water standard for the chemical.

[5] The IDEM submitted analytical results for groundwater samples collected in December 1995; the results are presented in Table 2. ATSDR was unable to match the samples with residential wells because no corresponding addresses were provided. Although the results are presented herein (Table 2), ATSDR cannot confirm that the sampling addresses are within the area that is generally described as the North Shore Drive groundwater investigation area. It is worth noting that TCE was not detected in any samples collected in December 1995.

The maximum concentration of TCE detected in any well is 85.4 ppb, which is greater than the MCL of 5 parts per billion (ppb) for TCE. Table 2 shows only those wells where the TCE concentrations exceeded the MCL. The first TCE contamination was detected in 1996. As the sampling expanded, additional wells were identified that also had TCE contamination above the drinking water standard. The IDEM supplied bottled water to these homes until the point of use (POU) filtration systems were installed in some homes around June 1997. Currently, IDEM provides filtration systems to 9 homes where the wells contain TCE concentrations that exceed the MCL. Until a long-term remedy is implemented, additional homes may need filtration systems in the future if more private wells are found to have TCE concentrations that exceed the MCL.

Table 2. Summary of TCE in Residential Wells at the North Shore Drive Site, 1995 to 2014

Date Sample Collected	Trichloroethylene (TCE) ppb	No. of TCE Detects greater than CV (out of total number of samples tested for TCE)
CV	**5 MCL 0.76 CREG**	
12/1995	ND	0 / 8
03/1996	ND - **19**	1 / 1
06/1996	ND - **21**	1 / 4
09/1996	ND	0 / 5
02/1997	ND - **71**	2 / 5
10/1997	ND – **85.4**	4 / 18
06/1998	ND - **3**	6 / 24
03/2001	ND – **19.5**	1 / 1
09/2002	ND - **58**	6 / 18
10/2003	ND	0 / 3
06/2005	ND	0 / 2
01/2010	ND - **19**	6 / 10
01/2010	ND - **13**	3 / 12
02/2010	ND – **5.8**	1 / 25
05/2010	ND	0 / 13
06/2010	ND	0 / 16
05/2011	ND – **20**	1 / 14
09/2011	ND – 0.33	0 / 19
12/2012	ND - **18**	15/46
04/2014	ND - **21**	5/8

CV = comparison value
ppb = parts per billion
ND – Not Detected
MCL = Maximum Contaminant Level (EPA)
CREG = Cancer Risk Evaluation Guide (ATSDR)
Bolded text means the value is greater than the CV

Figure 3 graphically depicts the approximate location of wells that have had at least one TCE concentration above the MCL of 5 ppb. (The wells are assigned a Well ID number for ease of

identification only.) Little quantitative information is available about the levels of contamination residents may have been exposed to in the past. ATSDR is aware that it is possible that TCE concentrations could have been higher in the past, before 1996, when the first contamination was discovered. Further characterization of the contaminant plume and knowledge about the source(s) of the contamination are needed for more accurate analyses.

Table 3. Residential Wells where at least one TCE Level Exceeded the MCL

*Well ID**	*Year Sampled*	*TCE Concentration (ppb)*
Well A	1997	71
	2003	ND
	2012	ND
	2014	ND/ND (duplicate)
Well B	2010	11
	2012	11
	2014	10
Well C	1997	36
	2002	30/31 (duplicate)
	2010	19
	2012	17/18 (duplicate)
Well D	1996	21
	2002	13
	2010	3.3
	2012	ND
	2014	ND
Well E	2010	5.8
	2012	8.7
	2014	8.1
Well F	1996	19
	2001	19.5
	2010	2.8
	2012	4.2
	2014	4.8
Well G	2002	3
	2011	19/20 (duplicate)
	2012	ND/ND (duplicate)
	2012	ND
	2014	21
Well H	2010	13
	2012	12/12 (duplicate)
Well I	1997	85.4
	2002	57/58 (duplicate)
	2010	15/15 (duplicate)
	2012	11/11 (duplicate)
	2014	9.6

ND = Not Detected
*Wells are identified alphabetically for illustrative purposes only.

Data Limitations

ATSDR made every attempt to accurately assess the potential impact that the TCE contamination had on the community's health, but there were limitations in the environmental data used to make the assessment. When limitations existed, ATSDR chose to be more conservative in an effort to be protective of the community's health. Therefore, actual exposures may have been different from those described in this document. The major limitations are:

- The contaminant dose that a person receives depends on the concentration of TCE in the well at a given time. However, we have no, or limited, sampling data about wells that may have been contaminated in the past, or may become contaminated in the future if the plume migrates. (Characterization of the groundwater contaminant plume is ongoing.) Therefore, it is difficult to accurately estimate the contaminant levels people might have been exposed to in the past, or may be exposed to in the future (assuming chronic exposure). ATSDR assumed that the data taken beginning in 1996 are reflective of prior years of exposure; however, the actual exposures may have been higher or lower. We also selected a range of TCE concentrations that residents may have been potentially exposed – a high, middle, and low concentration – based on the TCE concentrations in wells in the past (before installation of the filtration systems).

- Another major limitation is that the exact duration of exposure to contaminated water is unknown. The VOC contamination was first detected in 1996 (although the site was not officially "discovered" until 1997). However, the wells could have been contaminated for many years before being discovered. By 1997, wells known to have contaminated water were being fitted with filtration systems. As more contamination was discovered, filtration systems were introduced to other homes, as needed. However, the source of the

What is TCE?

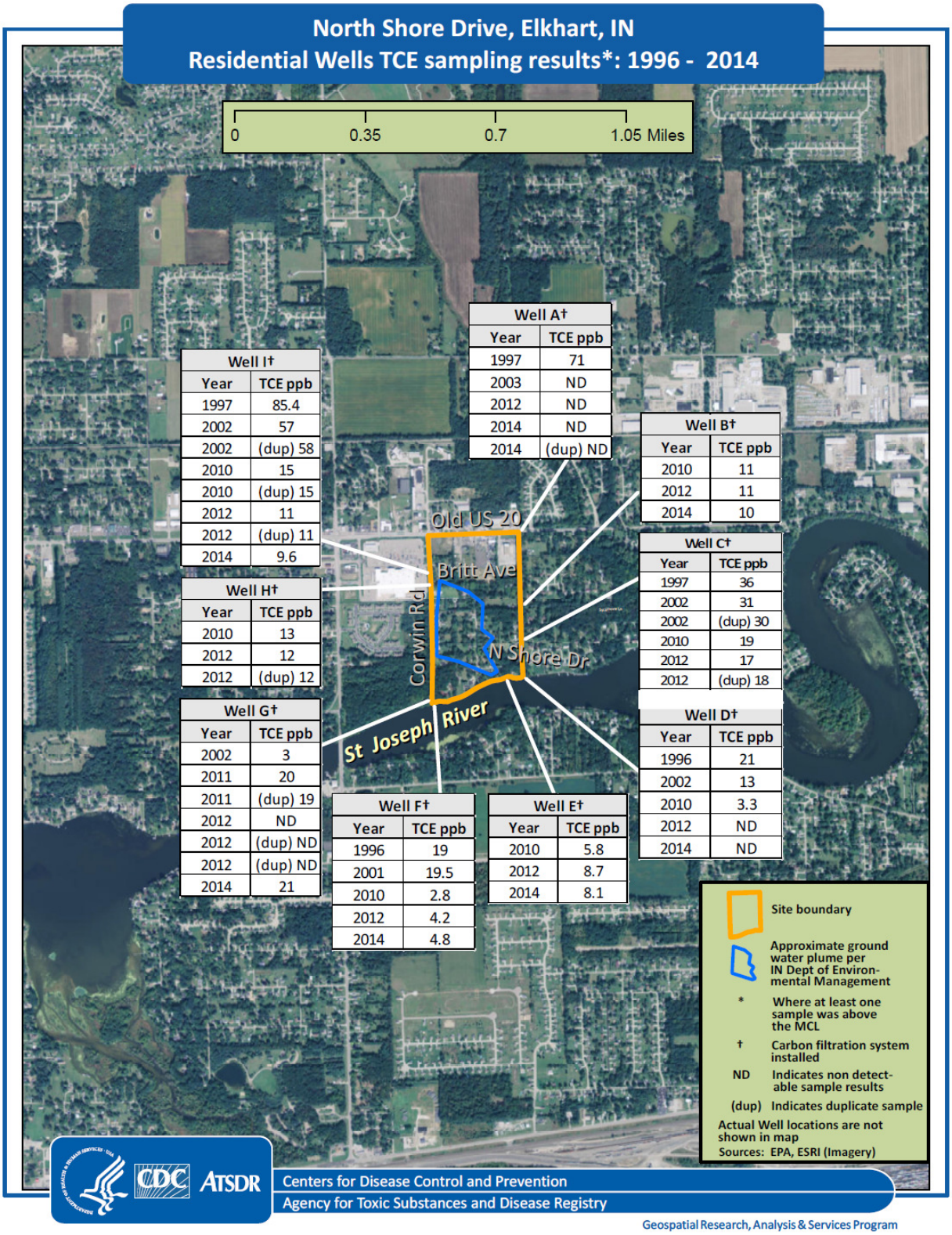
The primary industrial use of TCE has been degreasing metal parts. TCE use has been closely associated with the automotive and metal-fabricating industries from the 1950s through the 1970s. It is an excellent solvent for removing greases, oils, fats, waxes, and tars. As a solvent, TCE was used alone or blended with other solvents. These solvents were also added to adhesives, lubricants, paints, varnishes, paint strippers, pesticides, and cold metal cleaners.

TCE is known to be only slightly soluble in water, but there is ample evidence that dissolved TCE remains in groundwater for a long time. Studies show that TCE in water will rapidly form a gas when it comes into contact with air. 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 into groundwater. Likely, most TCE in a groundwater contaminant plume is present as a dissolved phase.

contamination has not yet been identified. Many of the industrial operations that could be potential sources of the contamination have operated in the area since the mid-1950s [IDEM 2003]. Public records[6] indicate that some of the homes in the area were constructed in the 1950s, 1960s, or early 1970s. Assuming that people were exposed to the contamination in their wells beginning in the mid-1960s, and that the exposures continued until 1997, it is reasonable to assume 33 years of exposure, which is the default time a person lives at a single residence. However, because the exact length of time people might have been exposed cannot yet be determined, the actual duration of exposure could be greater or less.

[6] Source: http://www.elkhartcountyindiana.com/departments/GIS/GIS.htm

Figure 3. TCE in Wells, Measured over Time, at Concentrations Greater than MCL



EXPOSURE PATHWAY ANALYSIS

A critical step in ATSDR's evaluation process is to assess exposure pathways. ATSDR uses exposure pathways to evaluate the specific ways in which people come into contact with environmental contamination. An exposure pathway is the link between an environmental release and the people that come into contact with, or are exposed to, the environmental contamination. The goal of exposure pathway analysis is to identify likely site-specific exposure situations and answer the questions:

- Is anyone exposed to environmental contamination?
- Under what conditions does this exposure occur?

People can only be exposed to a contaminant if they come in contact with it; this is, if they breathe, eat, drink, or come into skin contact with the contaminant. If a person is not exposed to a contaminant, no harmful health effects can occur.

ATSDR identifies an exposure pathway as completed, potential, or eliminates the pathway from further evaluation.

An exposure pathway has five elements: (1) a source of contamination, (2) an environmental media, (3) a point of exposure, (4) a route of human exposure, and (5) a receptor population. The *source* is the place where the chemical or radioactive material was released. The *environmental media* (such as groundwater, soil, surface water, or air) transport the contaminants. The *point of exposure* is the place where people come into contact with the contaminated media. The *route of exposure* (for example, ingestion, inhalation, or dermal contact) is the way the contaminant enters the body. The people actually exposed are the *receptor population*.

- *Completed exposure pathways* exist for a past, current, or future exposures if contaminant sources can be linked to a receptor population. All five elements of the exposure pathway must be present. In other words, people contact or are likely to come into contact with site-related contamination at a particular exposure point. As stated above, a release of a chemical or radioactive material into the environment does not always result in human exposure. For an exposure to occur, a completed exposure pathway—contact with the contaminant—must exist.

- *Potential exposure pathways* indicate that exposure to a contaminant might have occurred in the past, might be occurring currently, or might occur in the future. It exists when one or more of the elements are missing but available information indicates possible human exposure. A potential exposure pathway is one that ATSDR cannot rule out, even though not all of the five elements are identifiable.

- An *eliminated exposure pathway* exists when one or more of the elements are missing. Exposure pathways can be ruled out if the site characteristics make past, current, and

future human exposures extremely unlikely. If people are not exposed to contaminated areas, the pathway is eliminated from further evaluation. Also, an exposure pathway is eliminated if site monitoring reveals that media in accessible areas are not contaminated.

Exposure Pathways at the North Shore Drive Site

ATSDR evaluated exposure pathways to determine if people might come into contact with VOC contamination in private wells in the North Shore Drive area. Past, current and future exposure conditions were considered and ATSDR determined that a past completed pathway and current and future potential pathways apply to the site. The pathways findings are summarized in Table 4.

Drinking Water Pathway

As early as 1996, private wells on North Shore Drive were found to be contaminated with TCE above the drinking water standard. The date these wells first became contaminated is not known, but ATSDR conservatively estimates that people could have been exposed for 33 years. Since that time, a total of 9 homes have been supplied with point of use filtration systems because the TCE levels exceeded the drinking water standard of 5 ppb for TCE.

Prior to the installation of the filtration systems, people were exposed to TCE through the drinking water because their private wells were contaminated. Since the private wells supplied water to the households, the residents who had contaminated wells could have been exposed to TCE in several ways:

- *Ingestion*: Residents may have drunk the contaminated water or eaten food prepared using the water;
- *Inhalation*: Residents may have breathed in volatilized chemicals while shower, bathing, or other household uses such as dishwashing and laundering; and
- *Dermal contact*: Residents may have absorbed TCE though their skin during showering, bathing, or other use.

The drinking water pathway is a past completed exposure pathway. Currently, all households known to have well TCE levels exceeding the MCL have been supplied with filtration systems. Therefore, current exposure to TCE from known contaminated wells has been eliminated or reduced. The drinking water pathway is also a potential future completed exposure pathway if any of the following conditions were to ever exist: 1) if the installed filtration systems fail or are not maintained or 2) if the contaminant plume migrates and spreads the contamination to other (currently uncontaminated) residential wells.

Vapor Intrusion Pathway

A potential pathway to consider at this site is the vapor intrusion pathway. Vapor intrusion warrants consideration because of the volatile nature of TCE and because the plume runs underneath homes under certain conditions. Residents could be exposed to TCE in the indoor air. Vapor intrusion is the migration of VOCs from the subsurface-contaminated groundwater and

soil through the pore spaces of soil into above buildings. The concentrations of contaminants entering the indoor air from subsurface are dependent upon site and building-specific factors such as building construction, number and spacing of cracks and holes in the foundation, and the impact of the heating and air conditioning system on increasing or decreasing flow from the subsurface. Low confidence is generally attributed to decisions based on one sampling event, unless there is clear evidence that this will result in a health protective decision. Indoor air monitoring that reflects seasonal variations for the site should provide a better basis for an exposure estimate [ATSDR 2016].

The California Environmental Protection Agency (CalEPA) establishes two basic criteria for determining if it is necessary to evaluate vapor intrusion. First, volatile contaminants must be present in the subsurface, and second, the existing or future buildings at a site must be close to subsurface contamination so that vapor migration into indoor air is possible [CalEPA 2005]. EPA recommends that any building within 100 feet laterally or vertically of the contamination plume should be considered a candidate for vapor intrusion [EPA 2015]. The 100 foot distance assumes that no preferential pathways are present and other factors such as fluctuations in groundwater levels are minimal [ATSDR 2016]. The contaminant plume at North Shore Drive has not been fully characterized. Future investigations are needed to determine the significance of the vapor intrusion pathway for this site.

Figure 4: Exposure Pathways Table for North Shore Drive Groundwater Contamination

Completed Exposure Pathways

Medium	*Source*	*Exposure Point*	*Exposure Route*	*Time Frame*	*Comments*
Groundwater	Private wells	Drinking water	Ingestion	Past	Past household use of groundwater from contaminated wells has been documented and was, therefore, a completed exposure pathway. Currently, households with well TCE levels exceeding the drinking water standard (MCL) have been supplied with point-of-use filtration systems. Exposure occurred for an unknown duration, but assumed to be 33 years.
Indoor Air Skin Contact (Groundwater)	Private wells	Showering and other household uses	Inhalation Dermal (skin contact)	Past	Residents were exposed to TCE as it volatilized during showering and other household uses such as dishwashing and laundering. Residents also were exposed to TCE when it was absorbed though their skin during showering, bathing, or other uses.

Potential Exposure Pathways

Medium	*Source*	*Exposure Point*	*Exposure Route*	*Time Frame*	*Comments*
Groundwater	Private wells	Drinking water Showering and other household uses	Ingestion Dermal (skin contact) Inhalation	Future	Failure of current filtration systems or discovery of newly-impacted wells could lead to a completed pathway in the future.
Indoor Air (Vapor Intrusion)	Vapor intrusion	Affected rooms in residence Basements above the plume	Inhalation	Future	Vapor intrusion is a potential pathway because of the volatile nature of TCE and because the plume runs underneath homes.

HEALTH EFFECTS EVALUATION

This section of the public health assessment evaluates the health effects that could possibly result from exposures to TCE in private wells at the North Shore Drive site. For a public health hazard to exist, people must contact contamination at levels high enough and for long enough to affect their health.

To evaluate resident's exposure to TCE in drinking water, ATSDR calculated exposure doses and estimated non-cancer and cancer risks. Calculated exposure doses are then compared to the available health guidelines to determine whether the potential exists for adverse non-cancer health effects. In the event that calculated exposure doses exceed established health guidelines (e.g., ATSDR's Minimal Risk Levels [MRLs] or EPA's Reference Doses [RfD]), an in-depth toxicological evaluation is necessary to determine the likelihood of harmful health effects. ATSDR also may compare the estimated doses directly to human and animal studies, which are reported in ATSDR's chemical-specific Toxicological Profiles or other sources.

The equations to calculate exposure doses and the exposure assumptions used to estimate exposure doses are in Appendix C.

Public Health Implications of TCE in Private Well Water

People using private wells near the North Shore Drive site were exposed to TCE at levels above the drinking water standard, or Maximum Contaminant Level (MCL), of 5 ppb for TCE for an unknown period of time, assumed to be 33 years for purposes of this evaluation. These past exposures to TCE occurred through ingestion (drinking the water), inhalation (breathing in TCE evaporating from the water during showering, bathing, or other household use), and dermal absorption (skin contact with contaminated water). Wells with TCE levels greater than the MCL have been fitted with POU filtration systems. The filters, when properly maintained and operated, remove TCE and other VOCs to below detection limits.

For this health assessment, ATSDR derived exposures doses for several different age groups, ranging from a child to an adult, including pregnant women. Exposure doses help determine the extent to which the ingestion, inhalation and dermal absorption of contaminated well water might be associated with harmful health effects. In the absence of completed site-specific exposure information, ATSDR used conservative, health protective exposure assumptions. (Exposure assumptions are summarized in Appendix C.) ATSDR then compared the site-specific exposure doses to the observed effect levels reported in critical published studies on TCE.

ATSDR estimated doses using the equations in Appendix C and by applying the following conservative exposure assumptions:

- ATSDR assumed the duration of exposure to be 33 years, which is plausible, but not certain, based on the length of time potential commercial sources have/had been

operating in the area and when the residential homes were built. ATSDR will refine this assumption as more information becomes available as the site is fully characterized, including information on the release source and the fate and transport of chemicals in the environment.

- ATSDR selected a low, middle and maximum concentration of TCE detected in wells at the site to calculate the exposure doses. ATSDR selected these concentrations to evaluate a range of possible exposures because it is difficult to know what TCE concentrations people may have been exposed to over time. The sample size for any given well is not large enough to apply many useful statistical methods. Additionally, the data are limited on TCE levels in private wells before 1996 (the year the contamination was first detected), when the TCE levels may have been higher or lower. Beginning in 1997, wells with contamination levels above or approaching the drinking water standard were fitted with filtration systems.

- ATSDR used an exposure factor of 1 to represent being exposed daily. The exposure factor is an expression of how often and how long a person may be exposed to a substance. The exposure factor is calculated by multiplying the frequency of exposure by the exposure duration and dividing by the averaging time.

- ATSDR used average body weights, inhalation rates, breathing rates, surface area, and shower times for calculating exposure doses. ATSDR used above-average (i.e., high-end, 95th percentile) water intake rates to be especially health protective.

- ATSDR assumed the bioavailability of TCE was 100% - that is, all of the contaminant that a person ingested was assumed to enter the bloodstream.

Evaluating Non-cancer Health Effects from Past TCE Exposure in Well Water

Methods

ATSDR attempted to evaluate contaminant levels that people may have been exposed to in the past, with emphasis on wells where the TCE contamination exceeded the MCL. In the past, residents used the contaminated water from their private wells for household purposes, including drinking, cooking, and showering. These exposures are assumed to not be currently occurring because affected wells have been fitted with a point of use (POU) carbon filtration system.

To better estimate past exposures, ATSDR selected a high (85.4 ppb), middle (36 ppb) and low (19 ppb) TCE concentration measured in residents' private wells in the past. ATSDR was able to determine, using available data, that nine residential wells had TCE concentrations that exceeded the MCL at least once (See Table 3). The TCE concentrations detected at or above the MCL of 5 ppb ranged from 5.8 ppb to 85.4 ppb.

Often, ingestion exposure is the most significant source of exposure to hazardous substances from a site. However, in the case of VOC contamination, inhalation and dermal exposures can make a significant contribution to the total exposure dose. Studies have shown that exposure to VOCs from routes other than direct ingestion might be as large as the exposure from ingestion alone. Showering is considered a major contributor to overall exposure because TCE evaporates quickly from hot water into the air, and showering is typically done in a small, enclosed space where TCE concentrations might build up. The inhalation dose due to volatization during a shower may equal to the ingestion dose and 50% to 90% of VOCs in water may volatize during showering, laundering, and other activities [Moya *et. al*. 1999; Giardino and Andelman 1996]. In addition to breathing in the TCE from the air, people can absorb the chemical through their skin. The dermal dose has been estimated to equal 30% of the ingested dose [Maine DEP/DHS 1992]. Therefore, ATSDR added an inhalation and dermal contact dose to the ingestion dose to estimate total TCE exposures.

ATSDR used the inhalation model developed by Andelman and dermal exposure methods documented by EPA to estimate exposures from inhalation and dermal exposure, respectively. The Andelman method is used to estimate the exposure that would occur due to volatilization of TCE during the showering process and the subsequent inhalation of TCE-contaminated air. The model includes time spent in the enclosed bathroom after showering [Andelman 1990]. The EPA dermal exposure calculation is used to estimate skin intake of TCE during the showering process. ATSDR evaluated exposures using an average shower time of 15 minutes. ATSDR combined the drinking (oral), inhalation, and dermal exposures to derive a total exposure dose. Table E in Appendix C presents the combined oral, inhalation and dermal exposure doses for children and adults, including pregnant women. (It should be noted that ATSDR did not estimate inhalation and dermal exposures from showering for children less than 1 year of age because these very young children are more likely to take baths than showers.) ATSDR paid special attention to the exposure doses for young children and pregnant women because the scientific data indicate that the developing heart and nervous system in fetuses and young children may be especially sensitive to the toxic effects of TCE [ATSDR 2014a].

ATSDR compared the effect levels in key studies to the estimated exposure doses (from ingestion, inhalation, and dermal exposure) for children and adults to evaluate the potential for adverse non-cancer health effects.

Health 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. Previous epidemiological studies of women living in areas where the drinking water was contaminated with TCE, as well as other VOCs, have suggested an increased risk of several types of birth defects. Studies in Arizona and New Jersey suggested an association between TCE contamination in public drinking water wells and cardiac defects, and the New Jersey study also found an increased risk of oral clefts and neural tube defects [Bove *et. al*., 1995, Goldberg *et. al*, 1990]. Studies of women exposed to TCE-contaminated drinking water have shown some

evidence of increased risks of low or very low birth weight, term low birth weight, and small for gestational age. In laboratory animals, exposure to high levels of TCE has damaged the central nervous system, immune system, liver and kidneys, and adversely affected reproduction and development of offspring [ATSDR 2014a].

Using the highest measured TCE concentration (85.4 ppb) results in estimated doses ranging from 0.02 to 0.0052 mg/kg/day. The middle TCE concentration (36 ppb) results in estimated doses ranging from 0.0085 to 0.0022 mg/kg/day. The low evaluated TCE concentration (19 ppb) results in estimated doses ranging from 0.0045 to 0.0012 mg/kg/day. Generally, the estimated doses are higher for young children (1 to <2 years) than for older children (16 to <21 years) and adults (21+ years). ATSDR compared the estimated exposure doses to ATSDR's health guideline, or MRL, of 0.0005 mg/kg/day (5.0E-04 mg/kg/day). All of the exposure doses for children and adults exceed the MRL; therefore, ATSDR next compared the estimated exposure doses with effect levels from available studies.

ATSDR adopted EPA's RfD of 0.0005 mg/kg/day as its chronic oral MRL in January 2013 [ATSDR 2013]. The most sensitive observed adverse effects, which were used as the primary basis for the RfD, were based on the critical effects of heart malformations (rats), adult immunological effects (mice), and developmental immunotoxicity (mice), all from oral studies. The RfD is further supported by studies showing adverse effects in the kidney (an oral study for the effect of toxic nephropathy [rats]) and route-to-route extrapolated results from an oral study for the effect of increased kidney weights (rats).

Three principal toxicological studies used in developing the RfD are detailed below:

- Johnson showed increased rates of heart defects in newborn rats born to mothers who were exposed to TCE in drinking water during gestation [Johnson *et al*., 2003]. EPA applied Physiologically Based Pharmacokinetics (PBPK) models of TCE metabolism in rats and humans to the study results to obtain a 99th percentile human equivalent dose (HED_{99})[7] of 0.0051 mg/kg/day. At 0.0051 mg/kg/day ingested TCE, a 1% response rate is expected for fetal heart malformations in humans [EPA 2011; Johnson *et. al*. 2003].

- A study in female adult mice showed immune system effects (decreased thymus weight) after exposure to TCE in a thirty week drinking water study [EPA 2011; Keil *et. al*. 2009]. EPA converted the study findings to obtain a HED_{99} of 0.048 mg/kg/day.

- A study of mice exposed during gestation and following birth to TCE in drinking water showed problems with immune system development [EPA 2011; Peden-Adams *et. al*. 2006]. EPA used the lowest study effect level of 0.37 mg/kg/day as a point of departure.

[7] The HED_{99} can be interpreted as the applied dose in humans for which there is 99% likelihood that a randomly selected individual will have an internal dose less than or equal to the internal dose derived in the animal study.

Additional support for the RfD was based on adverse effects in the kidney [Woolhiser *et al.* 2006, increased kidney weights, a sign of stressed function; and NTP 1988, kidney effects, toxic nephropathy].

Based on estimated doses, newborns born to mothers who were exposed to TCE during pregnancy may be at increased risk for heart defects. One of the studies supporting the RfD is based on the critical effect of fetal heart malformations in rats. The estimated exposure doses for pregnant women are 0.0064 (high), 0.0023 (middle), and 0.0013 (low) mg/kg/day. The estimated doses for pregnant women approach or exceed the HED_{99} of 0.005 mg/kg/day for cardiac birth defects; therefore, babies born to mothers who were exposed to TCE during pregnancy may be at increased risk for a heart malformations. Some TCE-associated adverse health effects have been documented after short-term exposures. For example, fetal cardiac malformations have been shown to occur in rats after only 3 weeks of exposure at a level that would be equivalent to human ingesting a dose of 0.005 mg/kg/day. Thus, the concern exists for developmental effects if a woman was exposed even for a fairly short period of time during the three week window of critical fetal heart development in the first trimester of pregnancy. Therefore, even short-term exposures to any of the TCE concentrations during pregnancy may have resulted in cardiac effects. The risk to the fetus is greater among pregnant women who took longer showers (longer than 15 minutes).

Based on estimated doses for all levels of TCE evaluated, children and adults exposed to TCE in private wells may be at increased risk for harmful immunological effects. In addition to heart defects, EPA based the RfD on immune system toxicity. Harmful effects related to the immune system have been associated with TCE exposure in both human and animal studies. A relationship between systemic autoimmune diseases, such as scleroderma, and occupational exposure to TCE has been reported in several recent studies. Human evidence for the immunological effects of TCE includes studies reporting TCE-associated changes in levels of inflammatory cytokines in occupationally-exposed workers and infants exposed via indoor air at air concentrations typical of such exposure scenarios; a large number of case reports of a severe hypersensitivity skin disorder, distinct from contact dermatitis and often accompanied by hepatitis; and a reported association between increased history of infections and exposure to TCE contaminated drinking water. Immunotoxicity has also been reported in experimental studies of TCE in animals. Numerous studies have demonstrated accelerated autoimmune responses in autoimmune-prone mice, including changes in cytokine levels similar to those reported in human studies, with more severe effects, including autoimmune hepatitis, inflammatory skin lesions, and alopecia, manifesting at longer exposure periods. Developmental immunotoxicity in the form of hypersensitivity responses have been reported in TCE-treated guinea pigs and mice via drinking water pre- and postnatally. Evidence of localized immunosuppression has also been reported in mice and rats. Overall, the human and animal studies of TCE and immune-related effects provide strong evidence for a role of TCE in autoimmune disease and in a specific type of generalized hypersensitivity syndrome, while there are less data pertaining to immunosuppressive effects [EPA 2011].

The key animal studies upon which the RfD is based derived an HED_{99} of 0.048 mg/kg/day for decreased thymus weight, an indication of immune toxicity, and a lowest observed adverse effect level (LOAEL) of 0.37 mg/kg/day for developmental immunotoxicity. The estimated doses for young children (1 to <2 years), representing the highest estimated doses for any age group, are only 2 (high), 7 (middle), and 11 (low) times lower than the dose that caused decreased thymus weights in animal studies. Other estimated doses range from 4 to 40 times lower. The estimated doses for children and adults are 19 to 300 times lower than the dose that caused developmental immunotoxicity in animals. However, an uncertainly factor of 1,000 was applied to account for, among other factors,[8] the uncertainty in extrapolating from a LOAEL rather than from a no observed adverse effect level (NOAEL). Also, when we consider the limitations of the methods used to estimate exposures, an immunological health risk to children and adults cannot be ruled out. ATSDR used the Andelman model to estimate inhalation exposures while showering and in the enclosed bathroom after the shower, but the model does not account for VOC exposures that occur in the rest of the house throughout the day. It is estimated that the model does not account for up to 30% of the exposures that might occur during the rest of the day [personal communication with D. Mellard, May 2016]. The potential for the aforementioned immunological health effects increases as the shower time increases.

Potential Cancer Health Effects from TCE Exposure in Well Water

TCE exposures can cause cancer, with increased susceptibility for early-life exposures. The occupational studies of relatively high TCE exposures have shown increased risks for several types of cancer. The most consistent evidence has been for kidney, liver, and esophageal cancers and non-Hodgkin's lymphoma [ATSDR 2014a]. Additional evidence from occupational studies points to possible relationships between TCE exposure and increased risk of Hodgkin's disease, cervical cancer, multiple myeloma, bladder cancer, female breast cancer, and prostate cancer [Krishnadasan *et al*., 2007; Sung *et al*., 2007; Siegel Scott and Chiu, 2006; Zhao *et al*., 2005; Hansen *et al*., 2001; Wartenberg *et al*., 2000; ATSDR 2014a]. Many of these studies have strong limitations including unknown exposure levels and small sample sizes. In addition, many of these studies were unable to adequately separate the effects of TCE from other solvents present in the workplace.

The National Toxicology Program (NTP) classifies TCE as reasonably anticipated to be a human carcinogen based on limited evidence of carcinogenicity from studies in humans, sufficient evidence of carcinogenicity from studies in experimental animals, and information from studies on mechanisms of carcinogenesis [NTP 2011]. The human studies were epidemiological studies that showed increased rates of liver cancer and non-Hodgkin's lymphoma, primarily in workers who were exposed to TCE on the job. The animal studies showed increased numbers of liver, kidney, testicular, and lung tumors by two different routes of exposure. EPA characterizes TCE as "carcinogenic to humans" by all routes of exposure [EPA 2011d]. This conclusion is based on

[8] Other uncertainty factors were applied to account for toxicokinetic and toxicodynamic differences between mice and humans (10x) and for human variability (10x).

human epidemiology studies showing associations between human exposure to TCE and kidney cancer, non-Hodgkin's lymphoma, and liver cancer.

In 2011, EPA published an oral cancer slope factor for TCE of 0.046 $(mg/kg/day)^{-1}$ and an inhalation unit risk of 4.1 x 10^{-6} $(\mu g/m^3)^{-1}$ reflecting total incidence of kidney, non-Hodgkin's lymphoma, and liver cancers [EPA 2011d]. EPA used a PBPK model-based route-to-route extrapolation of the inhalation unit risk estimate for kidney cancer, with a factor of 5 applied to include non-Hodgkin's lymphoma and liver cancer risks, to obtain an oral slope factor for combined cancer risk of 0.046 $(mg/kg/day)^{-1}$, or 4.6 x 10^{-2} $(mg/kg/day^{-1})$. The combined cancer slope factor can be split into individual component slope factors as follows: for kidney cancer, the oral slope factor is 9.33 x 10^{-3} $(mg/kg/day)^{-1}$; for non-Hodgkin's lymphoma, the oral cancer slope factor is 2.16 x 10^{-2} $(mg/kg/day)^{-1}$; and for liver cancer, the oral cancer slope factor is 1.55 x 10^{-2} $(mg/kg/day)^{-1}$.

EPA also concluded, by weight of evidence evaluation, that TCE is carcinogenic by a mutagenic mode of action for induction of kidney tumors. As a result, increased early-life susceptibility is assumed for kidney cancer, and age-dependent adjustments factors (ADAFs) should be used for the kidney cancer component of the total cancer risk when estimating age-specific cancer risks. The ADAFs are factors by which cancer risk is multiplied to account for increased susceptibility to mutagenic compounds early in life. Standard ADAFs are 10 (for ages below 2 years old), 3 (for ages 2 up to 16 years old), and 1 (for ages greater than 16).

For a given age group, the estimated increased risk of developing cancer resulting from exposure to the contaminants was calculated by multiplying the site-specific estimated exposure dose, by an appropriate cancer slope factor or inhalation unit risk (EPA values can be found at http://www.epa.gov/iris), the appropriate ADAF, and the fraction of a 78-year lifetime under consideration. Using the above factors, ATSDR calculated the lifetime excess cancer risk from exposure to a range of concentrations of TCE in well water. (See Appendix D for detailed explanation and calculations.) The excess cancer risk is the number of increased cases of cancer in a population over a lifetime above background that may result from exposure to a particular contaminant under the assumed exposure conditions. For example, an estimated cancer risk of 1E-06 represents a possible one excess cancer case in a population of one million. Because of the uncertainties and conservatism inherent in deriving the cancer slope factors, this is only an estimate of risk; the true risk is unknown.

Appendix D summarizes the estimated increased cancer risk for potential past exposures for individuals who were exposed to TCE in their well water via ingestion, inhalation, and dermal routes of exposure. ATSDR calculated the excess cancer risk for people exposed to 85 ppb, 36 ppb, or 19 ppb TCE in water. We assumed that children were exposed for 21 years (from birth to >21 years of age) and that adults were exposed for a total of 33 years.

Table 5. Estimated Increased Risk of Cancer from Past Exposure to TCE in Drinking Water from Private Wells at the North Shore Drive Site, Unincorporated Elkhart County, Indiana

Age Group	Estimated Cancer Risk		
	19 ppb TCE	36 ppb TCE	85 ppb TCE
Children: birth to <21 years (21 years exposure)	4E-05 (4 in 100,000)	7E-05 (7 in 100,000)	2E-04 (2 in 10,000)
Adults: +21 years (33 years exposure)	2E-05 (2 in 100,000)	5E-05 (5 in 100,000)	1E-04 (1 in 10,000)

Based on the calculated increased cancer risks for long-term exposure, children and adults that were exposed to the highest (85 ppb) levels of TCE in private well water in the past could be at increased risk for cancer health effects. The estimated risks indicate that exposure to the maximum concentration (85.4 ppb) of TCE–contaminated water could have increased the risk of cancer from 1 in 10,000 (1E-04) for adults and 2 in 10,000 for adults (highlighted cells in the table). Stated another way, the calculated excess cancer risks are 1 to 2 extra cases of cancer for every 10,000 exposed adults and children, respectively. We interpret this as an elevated lifetime cancer; therefore, ATSDR considers children and adults exposed to the most highly TCE-contaminated water (85 ppb) in the past to be at increased risk for cancer.

Based on the calculated increased cancer risk for long-term exposure, children and adults that were exposed to the middle (36 ppb) and low (19 ppb) levels of TCE in private well water in the past are at a lower risk for cancer health effects. The middle (36 ppb) TCE concentration would result in 5 to 7 extra cases of cancer for every 100,000 exposed adults and children, respectively; and the low (19 ppb) TCE concentration, 2 to 4 extra cancers for every 100,000 exposed adults and children, respectively. We interpret the lifetime cancer risks for the middle and low TCE concentrations as a low increased risk and, therefore, not likely to harm people's health. Without historical data or knowledge about the source of the contamination, ATSDR recognizes that there is significant uncertainty in estimating cancer risk at this site.

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 adults from certain kinds of exposure to hazardous substances. Children are shorter than 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. TCE

exposures are a particular concern during the development of the fetus [Johnson *et al* 2003, EPA 2011]. Exposures during the critical fetal heart developmental period in the first trimester are of special concern. Collectively, the scientific data indicate that the developing heart and nervous system of fetuses and young children may be sensitive to the toxic effects of TCE. Our toxicity discussion above addresses these exposure concerns.

CONCLUSIONS

The purpose of this public health assessment (PHA) is to determine if the North Shore Drive Groundwater Plume site is a public health hazard for people who live near the site. The public health determination is based on an evaluation of the concentrations of toxic substances present at or released to the area and the pathways by which people living or around the site may be exposed to those substances.

Based on an evaluation of identified exposure pathways and available sampling data, ATSDR determines whether exposures to contaminants in groundwater are likely to cause harm to human health. An investigation of the site is ongoing. These conclusions may change as additional data become available or site conditions change.

Conclusion 1

Trichloroethylene (TCE) is the main contaminant of concern in groundwater at this site. People who used TCE-contaminated well water for household purposes (e.g., drinking, showering, bathing, etc.) in the past may be at risk for harmful non-cancer health effects associated with TCE exposure.

ATSDR evaluated whether children and adults exposed to different levels of TCE (ranging from 19 to 85 ppb TCE) detected in private wells might experience harmful health effects. Based on this evaluation, children and adults exposed to a range of TCE concentrations in private wells in the past may be at increased risk for harmful immunological effects. Additionally, newborns born to mothers who were exposed to TCE during pregnancy may be at increased risk for fetal heart malformations. The baby may be at risk for cardiac health defects even if the pregnant women was exposed for a fairly short period of time (i.e., under three weeks) if the exposure occurred when the fetal heart was developing (during the first trimester).

Currently, the Indiana Department of Environmental Management (IDEM) is supplying carbon filtration systems to 9 homes in the North Shore area identified as having well contaminant levels exceeding the Maximum Contaminant Level (MCL) of 5 ppb for TCE.

Conclusion 2

Children and adults exposed into the highest measured TCE concentration (85 ppb) may be at an increased risk for cancer.

ATSDR estimated increased cancer risk from potential past exposures for individuals who were exposed to TCE in their well water via the ingestion, inhalation, and dermal routes of exposure. We assumed that children were exposed for 21 years (from birth to >21 years of age) and that adults were exposed for a total of 33 years. The estimated risks indicate that exposure to the maximum concentration (85 ppb) of TCE–contaminated water could have increased the risk of cancer to 1 in 10,000 for adults and 2 in 10,000 for

children. We interpret this as an elevated cancer risk for children and adults. However, ATSDR assumed many years of exposure in estimating the cancer risks, so the actual cancer risks could be much lower. Without historical data or knowledge about the source of the contamination, ATSDR recognizes that there is some uncertainty in estimating cancer risk at this site.

Conclusion 3

A potential current exposure pathway to consider at this site is the vapor intrusion pathway. Vapor intrusion is the migration of volatile organic compounds (VOCs) from the subsurface-contaminated groundwater and soil through the pore spaces of soil into indoor air above buildings. ATSDR could not evaluate this pathway because the pathway has not yet been investigated.

Vapor intrusion warrants consideration because of the volatile nature (i.e., easily evaporates) of TCE and because the plume runs underneath residential homes and other enclosed structures. More data are needed to determine if residents are exposed to VOCs in the indoor air at levels that could pose a potential health risk.

RECOMMENDATIONS

ATSDR recommends that the IDEM, in conjunction with appropriate federal, state or local agencies:

- investigate the nature and extent of subsurface contamination at the site for potential vapor intrusion;
- conduct soil gas and indoor air VOC sampling as soon as is practicable at residences to characterize vapor intrusion, giving priority to homes nearest the groundwater plume;
- continue to monitor the private wells in the area to ensure filtration systems are operating properly; consider collecting samples pre- and post-filtration water samples;
- continue to monitor the groundwater contaminant plume to assess whether additional private wells are currently impacted or might become impacted in the future;
- consider a long-term solution (e.g., connection to the public water supply) for residents in the potential path of the plume who use private well water for household purposes;
- characterize the nature and extent of contamination at the site, including soil contamination, if any;
- determine the source(s) of groundwater contamination in the area; and
- take proper actions, as needed, to reduce exposure to TCE.

ATSDR will refine the exposure assessment and/or conclusions as more information becomes available about the site.

PUBLIC HEALTH ACTION PLAN

The North Shore Drive site is an active CERCLA (Superfund) site. We anticipate additional investigations and characterization studies to occur at this site. ATSDR will evaluate additional data and revise the conclusions and recommendations of this PHA as appropriate. ATSDR will distribute this PHA document to the community and other interested parties and solicit feedback. We will assess what additional actions, if any, are needed based upon the feedback.

Given the current actions by IDEM to eliminate and/or reduce exposures at this site, ATSDR believes it is unlikely that people continue to be exposed to TCE at harmful levels. However, people who are concerned about past exposures should discuss their concerns with their physicians.

Primary Author:

Teresa Foster, MPH
Environmental Health Scientist
ATSDR, Eastern Branch
Division of Community Health Investigations

ATSDR Regional Representative:

Motria Caudill, Ph.D.
Regional Representative
Region 5

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APPENDIX A:

ATSDR Fact Sheet for TCE

Trichloroethylene - ToxFAQs™

CAS # 79-01-6

This fact sheet answers the most frequently asked health questions (FAQs) about trichloroethylene. For more information, call the CDC Information Center at 1-800-232-4636. This fact sheet is one in a series of summaries about hazardous substances and their health effects. It's important you understand this information because this substance may harm you. The effects of exposure to any hazardous substance depend on the dose, the duration, how you are exposed, personal traits and habits, and whether other chemicals are present.

HIGHLIGHTS: Trichloroethylene is used as a solvent for cleaning metal parts. Exposure to very high concentrations of trichloroethylene can cause dizziness, headaches, sleepiness, incoordination, confusion, nausea, unconsciousness, and even death. The Environmental Protection Agency (EPA) and the International Agency for Research on Cancer (IARC) classify trichloroethylene as a human carcinogen. Trichloroethylene has been found in at least 1,045 of the 1,699 National Priorities List sites identified by the EPA.

What is trichloroethylene?

Trichloroethylene is a colorless, volatile liquid. Liquid trichloroethylene evaporates quickly into the air. It is nonflammable and has a sweet odor.

The two major uses of trichloroethylene are as a solvent to remove grease from metal parts and as a chemical that is used to make other chemicals, especially the refrigerant, HFC-134a. Trichloroethylene was once used as an anesthetic for surgery.

What happens to trichloroethylene when it enters the environment?

- Trichloroethylene can be released to air, water, and soil at places where it is produced or used.
- Trichloroethylene is broken down quickly in air.
- Trichloroethylene breaks down very slowly in soil and water and is removed mostly through evaporation to air.
- It is expected to remain in groundwater for long time since it is not able to evaporate.
- Trichloroethylene does not build up significantly in plants or animals.

How might I be exposed to trichloroethylene?

- Breathing trichloroethylene in contaminated air.
- Drinking contaminated water.
- Workers at facilities using this substance for metal degreasing are exposed to higher levels of trichloroethylene.
- If you live near such a facility or near a hazardous waste site containing trichloroethylene, you may also have higher exposure to this substance.

How can trichloroethylene affect my health?

Exposure to moderate amounts of trichloroethylene may cause headaches, dizziness, and sleepiness; large amounts may cause coma and even death. Eating or breathing high levels of trichloro¬ethylene may damage some of the nerves in the face. Exposure to high levels can also result in changes in the rhythm of the heartbeat, liver damage, and evidence of kidney damage. Skin contact with concentrated solutions of trichloroethylene can cause skin rashes.

There is some evidence exposure to trichloroethylene in the work place may cause scleroderma (a systemic autoimmune disease) in some people. Some men occupationally-exposed to trichloroethylene and other chemicals showed decreases in sex drive, sperm quality, and reproductive hormone levels.

How likely is trichloroethylene to cause cancer?

There is strong evidence that trichloroethylene can cause kidney cancer in people and some evidence for trichloroethylene-induced liver cancer and malignant lymphoma. Lifetime exposure to trichloroethylene resulted in increased liver cancer in mice and increased kidney cancer and testicular cancer in rats.

The IARC and the EPA determined that there is convincing evidence that trichloroethylene exposure can cause kidney cancer. The National Toxicology Program (NTP) is recommending a change in cancer classification to "known human carcinogen" http://ntp.niehs.nih.gov/ntp/roc/monographs/finaltce_508.pdf.

Agency for Toxic Substances and Disease Registry
Division of Toxicology and Health Human Sciences

ATSDR

CS256651A

Trichloroethylene

CAS # 79-01-6

How can trichloroethylene affect children?

It is not known whether children are more susceptible than adults to the effects of trichloroethylene.

Some human studies indicate that trichloroethylene may cause developmental effects such as spontaneous abortion, congenital heart defects, central nervous system defects, and small birth weight. However, these people were exposed to other chemicals as well.

In some animal studies, exposure to trichloroethylene during development caused decreases in body weight, increases in heart defects, changes to the developing nervous system, and effects on the immune system.

How can families reduce the risk of exposure to trichloroethylene?

- Avoid drinking water from sources that are known to be contaminated with trichloroethylene. Use bottled water if you have concerns about the presence of chemicals in your tap water. You may also contact local drinking water authorities and follow their advice.
- Discourage your children from putting objects in their mouths. Make sure that they wash their hands frequently and before eating.
- Prevent children from playing in dirt or eating dirt if you live near a waste site that has trichloroethylene.
- Trichloroethylene is used in many industrial products. Follow instructions on product labels to minimize exposure to trichloroethylene.

Is there a medical test to show whether I've been exposed to trichloroethylene?

Trichloroethylene and its breakdown products (metabolites) can be measured in blood and urine. However, the detection of trichloroethylene or its metabolites cannot predict the kind of health effects that might develop from that exposure. Because trichloroethylene and its metabolites leave the body fairly rapidly, the tests need to be conducted within days after exposure.

Has the federal government made recommendations to protect human health?

The EPA set a maximum contaminant goal (MCL) of 0.005 milligrams per liter (mg/L; 5 ppb) as a national primary drinking standard for trichloroethylene.

The Occupational Safety and Health Administration (OSHA) set a permissible exposure limit (PEL) of 100 ppm for trichloroethylene in air averaged over an 8-hour work day, an acceptable ceiling concentration of 200 ppm provided the 8 hour PEL is not exceeded, and an acceptable maximum peak of 300 ppm for a maximum duration of 5 minutes in any 2 hours.

The National Institute for Occupational Safety and Health (NIOSH) considers trichloroethylene to be a potential occupational carcinogen and established a recommended exposure limit (REL) of 2 ppm (as a 60-minute ceiling) during its use as an anesthetic agent and 25 ppm (as a 10-hour TWA) during all other exposures.

References

This ToxFAQs™ information is taken from the 2014 Toxicological Profile for Trichloroethylene (Draft for Public Comment) produced by the Agency for Toxic Substances and Disease Registry, Public Health Service, U.S. Department of Health and Human Services.

Where can I get more information?

For more information, contact the Agency for Toxic Substances and Disease Registry, Division of Toxicology and human Health Sciences, 1600 Clifton Road NE, Mailstop F-57, Atlanta, GA 30329-4027.

Phone: 1-800-232-4636.

ToxFAQs™ Ion the web: www.atsdr.cdc.gov/toxFAQs.

ATSDR can tell you where to find occupational and environmental health clinics. Their specialists can recognize, evaluate, and treat illnesses resulting from exposure to hazardous substances. You can also contact your community or state health or environmental quality department if you have any more questions or concerns.

APPENDIX B: Derivation and Use of ATSDR Health Comparison Values

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.

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 public health professionals use 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 (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.

RfDs and RfCs are estimates of daily human exposure, including exposure to sensitive subpopulations that are likely to be without appreciable risk of adverse non-cancer health effects during a lifetime (assumed to be 78 years). These guidelines are derived from experimental data and lowest-observed-adverse-effect levels (or no-observed-adverse-effect levels), adjusted downward using uncertainty factors. The uncertainty factors are used to make the guidelines adequately protective for all people, including susceptible individuals. RfDs and RfCs should not be viewed as strict scientific boundaries between what is toxic and what is nontoxic.

For cancer-causing substances, EPA established the cancer slope factor. A CSF is used to determine the number of excess cancers expected from maximal exposure for a lifetime. Health comparison values (CVs) are estimated contaminant concentrations that are unlikely to cause adverse health outcomes when these concentrations occur in specific media. CVs are used to select site contaminants for further evaluation. CVs are calculated from health guidelines and are presented in media specific units of concentration, such as micrograms/liter (µg/l) or ppm. CVs are calculated using conservative assumptions about daily intake rates by an individual of standard body weight. Because of the conservative assumptions and safety factors, contaminant concentrations that exceed comparison values for an environmental medium do not necessarily indicate a health hazard.

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. Also, they depend on the amount of a contaminant ingested or inhaled. Thus, EMEGs are determined separately for children and adults, and also separately 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 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, 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 or from the EPA Exposure Factors Handbook. Doses calculated using health protective exposure factors and environmental concentrations are considered "health protective doses" because it is unlikely that any community exposures are greater than the calculated doses and are most likely to be less than the health protective doses.

After estimating the potential for 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, the contaminant is considered to be of concern and requires further evaluation. 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.

APPENDIX C:

ATSDR's Exposure Dose Equations

And

Exposure Assumptions

To estimate past exposure to TCE, ATSDR used mathematical models and default exposure assumptions to estimate exposure doses. An exposure dose (usually expressed as milligrams of chemical per kilogram of body weight per day, or “mg/kg/day”) is an estimate of how much of a substance a person may contact based on their actions and habits. Estimating an exposure dose requires identifying how much, how often, and how long a person or population may come in contact with a concentration of a substance in a specific medium.

To estimate exposure doses at this site, ATSDR used default exposure assumptions about weight and other body characteristics of children and adults exposed, how they may have been exposed, and how often they may have been exposed. The following section details the exposure assumptions and calculation of exposure doses for the drinking water, inhalation, and dermal contact pathways evaluated in this document.

Ingestion of contaminated water is one of the most significant exposure pathways at this site. ATSDR used the following equation and assumptions to estimate exposure to TCE from the ingestion of contaminated well water:

Table A. Exposure Dose Equation and Assumptions for Ingestion of Contaminated Water

Water Ingestion Exposure Dose Equation:

$$D = \frac{C\ x\ IR}{BW}$$

D = exposure dose in milligrams per kilogram per day, mg/kg/day
C = chemical concentration in milligrams per liter, ($\frac{mg}{L}$)
IR = ingestion rate in liters per day, ($\frac{L}{day}$)
BW = body weight in kilograms, kg

Age Group	Body Weight in kg	Ingestion of Water in Liters Per Day
		High-end Water Intake Rate (L/day)
Birth to <1 year	7.8	1.113
1 to <2 years	11.4	0.893
2 to <6 years	17.4	0.977
6 to <11 years	31.8	1.404
11 to <16 years	56.8	1.976
16 to <21 years	71.6	2.444
21+ years	80	3.1
Pregnant Women (16 to 45 years)	73	2.589

[ATSDR 2014b]. Agency for Toxic Substances and Disease Registry. 2014. Exposure Dose Guidance for Water Ingestion. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service. November 2014.

High-end water intake rate represents the water intake for a person who consumes more than the average amount of water per day (the 95th percentile values).

EXAMPLE OF INGESTION CALCULATION: -

Use the equation and values in Table A to calculate the amount of TCE ingested from drinking contaminated well water

Example using: Adult (21+ years old) using High-End Water Intake Containing the Maximum TCE - Concentration in Well Water (85 ppb) -

Ingestion Exposure Dose

High-End Water Intake

$$\frac{0.0854\,\frac{mg}{L}\,x\,3.1\,\frac{L}{day}}{80\,kg} = 3.3E-03\,mg\,/kg/day$$

The results of these calculations are reported in Table E. -

Private drinking water wells in the North Shore Drive area were contaminated with TCE. The TCE-contaminated water was used for household purposes, including showering. Volatile organic compounds (VOCs) such as TCE can escape, or volatilize, from water used in the home. Breathing in (inhaling) the TCE vapors in air that occurs when using contaminated water for showering can be a significant source of exposure. Because inhalation and skin absorption of TCE during showering can be significant, ATSDR evaluated those exposures separately. To evaluate inhalation and dermal exposures, ATSDR computed the 24-hour TCE concentration and then compared that value with available health guidelines. There are several steps, discussed below, in estimating the equivalent 24-hour air concentration.

Note: We recognize that very young children (>1 year) are likely to take more baths than showers, therefore, we did not estimate showering exposures for this age group. While we recognize that bathing would not likely result in exposures as great as showering because showering has a high flow rate and more volatilization of VOCs, we still likely underestimated the total exposures to very young children. ATSDR used several equations and exposure assumptions to estimate how much TCE a person would inhale while showering.

Table B. Exposure Assumptions for Inhalation of TCE while Showering

Age Group	Average Shower time (minutes)	Average Bathroom Stay after Shower (minutes)	Average Short-term Breathing Rates While Showering (m^3/min)	Average Long-term Breathing Rates (m^3/day)
1 to <2 years	15	5	0.012	8.0
2 to <6 years	15	5	0.011	9.8
6 to <11 years	15	5	0.011	12.0
11 to <16 years	15	5	0.013	15.2
16 to <21 years	15	5	0.012	16.3
21+ years	15	5	0.012	15.2
Pregnant Women (16 to 45 years)	15	5	0.016	22

Average shower time and bathroom stay after shower derived using professional judgment with input from Table 16-32: Time spent (minutes) Showering and in Shower Room Immediately After Showering, EPA Exposure Factors Handbook (2011)
Table 6-2: Recommended Short-Term Exposure Values for Inhalation(males and females combined), Light Intensity, EPA Exposure Factors Handbook (2011)
Average represents the mean (50^{th} percentile) value

EXAMPLE OF INHALATION CALCULATION(S): -

Use the equations below and values in Table B to calculate the amount of TCE inhaled while showering

Example using: Adult (21+ years old) Showering for 15 Minutes with Maximum Concentration of TCE (85 ppb) in Water

Estimating the inhalation exposure dose is a 2-step process:

1) Calculate the TCE concentration in the bathroom
2) Calculate the amount of TCE inhaled

ATSDR used a model developed by Andelman [Andelman 1990] to estimate the TCE concentration occurring in the bathroom as a result of showering. The equation is as follows:

Equation for Concentration of TCE in Air:

$$C_a = \frac{k \; x \; F_w \;\; x \; T_s \; x \; C_w \; x \; CF}{V_a}$$

C_a = air concentration in bathroom/shower, in milligrams per cubic meter, $\frac{mg}{m^3}$

k = volatile mass transfer coefficient, unitless (default is 0.6)

F_w = flow rate of water through shower, in liters per min, L/min (default is 8 $\frac{L}{min}$)

T_s = time in shower, in minutes (varies with age, See Table B)

C_w = VOC concentration in water, in milligrams per liter, $\frac{mg}{L}$

CF = conversion factor (1,000 $\frac{L}{m^3}$)

V_a = bathroom air volume, in liters, L (default is 10,000 L)

Step 1. Calculate the concentration of TCE in the bathroom

Concentration TCE in Air

$$\frac{0.6\ x\ 8\ \frac{L}{min}\ x\ 15\ \text{min}\ x\ 0.0854\ \frac{mg}{L}\ x\ 1000\ \frac{L}{m^3}}{10{,}000L} = 0.615\ \frac{mg}{m^3}$$

The TCE concentration in air will be breathed in during the shower and during any time stayed in the bathroom after the shower. Next is to calculate how much of the contaminant in the air will be inhaled by the average person, using the following equation and the average short-term breathing rates found in Table B.

Step 2. Calculate the amount of TCE inhaled

Equation for Intake of TCE in Air:

$Intake_{inhalation}$ (µg) = peak concentration ($\frac{\mu g}{m^3}$) x IR_{st} $\frac{m^3}{min}$ x (T_s + T_b)(min)

Peak conc = concentration calculated in Step 1, in $\frac{\mu g}{m^3}$

IR_{st} = short-term inhalation rate ($\frac{m^3}{min}$)

T_s = time in shower, in minutes (varies with age, See Table B) -

T_b = time in bathroom after shower, in minutes (varies, See Table B) -

TCE Intake Due to Inhalation

615 $\frac{\mu g}{m^3}$ x 0.012 $\frac{m^3}{min}$ x (15 + 5) min = 147.6 µg TCE

Dermal (skin contact) absorption of contaminants in water occurs while showering or bathing. ATSDR used the equation and exposure assumptions in Table C to estimate how much dermal exposure to TCE would occur while showering.

ATSDR estimated skin intake using the general methods of EPA's Risk Assessment Guidance for Superfund, Part E [EPA 2004].

Table C. Exposure Equation and Estimates for Dermal Absorption of TCE while Showering

Dermal Absorption Equation:

$$2\ x\ FA\ x\ K_p\ x\ C_w\ x\ \frac{1L}{1000\ cm^3}\ x\ SA\ x\ \sqrt{\frac{6\ x\ \tau(hr) x\ T_s}{60\ \frac{min}{hr}\ x\ \pi}}$$

$Intake_{skin}$ = absorbed dose (µg)
FA = fraction absorbed water (assumed to be 1)
K_p = dermal permeability coefficient for TCE (0.012 cm/hr)
C_w = chemical concentration in water (µg/L)
SA = total skin surface area in cm^2 (varies with age, See Table C)
τ_{event} = lag time per event (0.58 hr)
T_s = time in shower (varies with age, See Table B)
π = pi, 3.14

Age Group	Total Body Surface Area in cm^2 (Average surface area)
1 to <2 years	5,300
2 to <6 years	7,775
6 to <11 years	10,800
11 to <16 years	15,900
16 to <21 years	18,400
21+ years	19,780
Pregnant Women (16 to 45 years old)	19,375

Agency for Toxic Substances and Disease Registry. 2015. Exposure Dose Guidance for Dermal Exposures to Soil and Sediment. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service. October 2015.

EXAMPLE OF DERMAL ABSORPTION CALCULATION

Example using: Adult (21+ years old) Showering for 15 Minutes with Maximum Concentration of - TCE (85.4 ppb) in Water -

TCE Intake Due to Skin Absorption

$$2\ x\ 1\ x\ 0.012\ \frac{cm}{hr}\ x\ 85.4\ \frac{\mu g}{L}\ x\ \frac{1L}{1000\ cm^3}\ x\ 19{,}780\ cm^2\ x\ \sqrt{\frac{6\ x\ 0.58\ (hr) x\ 15\ min}{60\ \frac{min}{hr}\ x\ 3.14}}$$

= 21.3 µg TCE

To estimate the total (inhalation + dermal) intake of TCE from showering, ATSDR summed the inhalation and skin intakes and converted to a 24-hour equivalent air concentration.

EXAMPLE OF CONVERTING TO 24-HOUR EQUIVALENT AIR CONCENTRATION

Exposures that occurs through skin contact need to be converted to a 24-hour equivalent air concentration to compare to inhalation guidelines.

Example using: Adult (21+ years old) with an average breathing rate of ($15.2\ \frac{m^3}{day}$)

To convert to a 24-hour air concentration, the total intake is divided by the daily average long-term breathing rate for an adult using the following equation:

$$24\ hour\ Equivalent\ Concentration = \frac{Intake\ (\mu g)}{Inhalation\ Rate\ (\frac{m^3}{day})}$$

$$24\ hour\ Equivalent\ Concentration = \frac{(147.6 + 21.3)(\mu g)}{15.2\ (\frac{m^3}{day})} = \ 11.1\ \frac{\mu g}{m^3}$$

Table D. Estimated Equivalent 24 Hour Air Equivalent Concentrations from Inhalation and Dermal Exposures to TCE while Showering at the North Shore Drive Site

TCE Concentration (ppb)	Age Group	Average (15 Minute) Shower Time Inhalation + Dermal Exposure ($\mu g/m^3$)	ATSDR Minimal Risk Level (MRL) Health Guideline Exceeded? (2.1 $\mu g/m^3$)
85 (maximum value)			
	1 to <2 years	19.2	**Yes**
	2 to <6 years	14.7	**Yes**
	6 to <11 years	12.2	**Yes**
	11 to <16 years	11.3	**Yes**
	16 to <21 years	10.3	**Yes**
	21+ years	11.1	**Yes**
	Pregnant Women (16 to 45 years)	9.9	**Yes**
36 (middle value)			
	1 to <2 years	8.1	**Yes**
	2 to <6 years	6.2	**Yes**
	6 to <11 years	5.2	**Yes**
	11 to <16 years	4.8	**Yes**
	16 to <21 years	4.3	**Yes**
	21+ years	4.7	**Yes**
	Pregnant Women (16 to 45 years)	4.2	**Yes**
19 (low value)			
	1 to <2 years	4.3	**Yes**
	2 to <6 years	3.2	**Yes**
	6 to <11 years	2.7	**Yes**
	11 to <16 years	2.5	**Yes**
	16 to <21 years	2.3	**Yes**
	21+ years	2.5	**Yes**
	Pregnant Women (16 to 45 years)	2.2	**Yes**
	Note: ATSDR did not estimate exposures from showering for children less than 1 year of age because these very young children are more likely to take baths rather than showers.		

Table E. Total Oral, Inhalation and Dermal Exposures to TCE in Private Wells at the North Shore Drive Site

TCE Concentration (ppb)	Age Group	Doses for High-End Water Consumer (Ingestion) (mg/kg/day)	Average (15 Minute) Shower Time Exposure Expressed as a Dose (Inhalation + Dermal) (mg/kg/day)	TOTAL DOSE Ingestion + Inhalation + Dermal (mg/kg/day)
85 (maximum value)	Birth to <1 year	1.2E-02	N/A	1.2E-02*
	1 to <2 years	6.7E-03	1.3E-02	2.0E-02
	2 to <6 years	4.8E-03	8.3E-03	1.3E-02
	6 to <11 years	3.8E-03	4.6E-03	8.4E-03
	11 to <16 years	3.0E-03	3.0E-03	6.0E-03
	16 to <21 years	2.9E-03	2.3E-03	5.2E-03
	21+ years	3.3E-03	2.1E-03	5.4E-03
	Pregnant Women (16 to 45 years)	3.0E-03	3.0E-03	6.4E-03
36 (middle value)	Birth to <1 year	5.1E-03	N/A	5.1E-03*
	1 to <2 years	2.8E-03	5.7E-03	8.5E-03
	2 to <6 years	2.0E-03	3.5E-03	5.5E-03
	6 to <11 years	1.6E-03	1.9E-03	3.5E-03
	11 to <16 years	1.3E-03	1.3E-03	2.6E-03
	16 to <21 years	1.2E-03	9.8E-04	2.2E-03
	21+ years	1.4E-03	8.9E-04	2.3E-03
	Pregnant Women (16 to 45 years)	1.3E-03	1.3E-03	2.6E-03
19 (low value)	Birth to <1 year	1.2E-03	N/A	1.2E-03*
	1 to <2 years	1.5E-03	3.0E-03	4.5E-03
	2 to <6 years	1.1E-03	1.8E-03	2.9E-03
	6 to <11 years	8.4E-04	1.0E-03	1.8E-03
	11 to <16 years	6.6E-04	6.8E-04	1.3E-03
	16 to <21 years	6.5E-04	5.2E-04	1.2E-03
	21+ years	7.3E-04	4.8E-04	1.2E-03
	Pregnant Women (16 to 45 years)	6.7E-04	6.5E-04	1.3E-03
	An average shower time is assumed to be a 15 minute shower per day *The ingestion dose was used as the total exposure dose for the birth to <1 year age group; does not include inhalation or dermal doses.			

APPENDIX D:

Evaluating Cancer Risks

EVALUATING CANCER HEALTH EFFECTS

In 2011, EPA published an oral cancer slope factor for TCE of 0.046 $(mg/kg/day)^{-1}$ and an inhalation unit risk of 4.1×10^{-6} $(\mu g/m^3)^{-1}$ reflecting total incidence of kidney, non-Hodgkin's lymphoma, and liver cancers [EPA 2011d]. EPA used a PBPK model-based route-to-route extrapolation of the inhalation unit risk estimate for kidney cancer, with a factor of 5 applied to include non-Hodgkin's lymphoma and liver cancer risks, to obtain an oral slope factor for combined cancer risk of 0.046 $(mg/kg/day)^{-1}$, or 4.6×10^{-2} $(mg/kg/day^{-1})$. The combined cancer slope factor can be split into individual component slope factors as follows:

- For kidney cancer: 9.33×10^{-3} $(mg/kg/day)^{-1}$
- For non-Hodgkin's lymphoma: 2.16×10^{-2} $(mg/kg/day)^{-1}$
- For liver cancer: 1.55×10^{-2} $(mg/kg/day)^{-1}$

The methods used to calculate cancer slope factors rely upon several assumptions. The method assumes that high-dose animal data can be used to estimate the risk for low dose exposures in humans. The methods also assume that no safe level exists for exposure. Little experimental evidence exists to confirm or refute those two assumptions. Lastly, most methods compute the upper 95^{th} percent confidence limit for the risk. The actual cancer risk can be lower, perhaps by several orders of magnitude.

If a substance causes cancer by a mutagenic mode of action, there is a greater risk for exposures that occur in early life. For these substances, age-dependent adjustment factors (ADAFs) are applied to the risks estimated as follows: An ADAF of 10 is applied for exposures taking place from birth up to 2 years old, and an ADAF of 3 is applied for exposures taking place from age 2 up to age 16. No adjustment is applied for exposures at age 16 or above. The EPA has concluded that TCE is carcinogenic by a mutagenic mode of action for induction of kidney tumors. As a result, increased early-life susceptibility is assumed for kidney cancer, and age-dependent adjustment factors (ADAFs) are used for the kidney cancer component of the total cancer risk when estimating age-specific cancer risks.

The lifetime excess cancer risk indicates the cancer potential of contaminants. The cancer estimates are usually expressed in terms of excess cancer cases in an exposed population in addition to the background rate of cancer. For remedial decision, the EPA considers estimated cancer risks of less than one additional cancer case among one million persons exposed as insignificant or no increased risk (expressed exponentially as 10^{-6}).

To calculate the lifetime excess cancer risk, ATSDR multiplied the component oral cancer slope factor by the daily exposure dose (oral, inhalation, and dermal), the appropriate ADAF, and the fraction corresponding to the fraction of a 78-year lifetime under consideration.

CANCER RISK CALCULATIONS:

Table F. Calculation of Excess Cancer Risk for Residents Exposed to TCE in Private Well Water via Drinking, Inhalation, and Dermal Contact – at 85 ppb - North Shore Drive Site

Age Group	Estimated Total Exposure Dose (mg/kg/day)	Duration (years)	Fraction of Lifetime	Unadjusted Kidney Lifetime Cancer Slope Factor $(mg/kg/day)^{-1}$	ADAF	Adjusted Kidney Cancer Risk	NHL and Liver Lifetime Cancer Slope Factor $(mg/kd/day)^{-1}$	NHL and Liver Cancer Risk	Total Cancer Risk: Adjusted Kidney and Unadjusted NHL and Liver
Birth to <1 year	1.2E-02	1	1/78	9.3E-03	10	1.4E-05	3.7E-02	5.7E-06	2.0E-05
1 to <2 years	2.0E-02	1	1/78	9.3E-03	10	2.4E-05	3.7E-02	9.5E-06	3.3E-05
2 to <6 years	1.3E-02	4	4/78	9.3E-03	3	1.9E-05	3.7E-02	2.5E-05	4.3E-05
6 to <11 years	8.4E-03	5	5/78	9.3E-03	3	1.5E-05	3.7E-02	2.0E-05	3.5E-05
11 to <16 years	6.0E-03	5	5/78	9.3E-03	3	1.1E-05	3.7E-02	1.4E-05	2.5E-05
16 to <21 years	5.2E-03	5	4/78	9.3E-03	1	3.1E-06	3.7E-02	1.2E-05	1.5E-05
Total years exposed - children		21						Total cancer risk - children	1.7E-04
Adult 21+ years	5.4E-03	33	33/78	9.3E-03	1	2.1E-05	3.7E-02	8.5E-05	1.1E-04
Total years exposed - adults		33						Total cancer risk - adults	1.1E-04

Birth to <1 year total exposure dose does not include shower exposures (inhalation and dermal contact) because very young children are assumed to take baths instead of showers. The ingestion dose was used as the total exposure dose, which might underestimate total exposures to this age group.
ADAF = age-dependent adjustment factor
NHL = non-Hodgkin's lymphoma

Table G. Calculation of Excess Cancer Risk for Residents Exposed to TCE in Private Well Water via Drinking, Inhalation, and Dermal Contact – at 36 ppb - North Shore Drive Site

Age Group	Estimated Total Exposure Dose (mg/kg/day)	Duration (years)	Fraction of Lifetime	Unadjusted Kidney Lifetime Cancer Slope Factor $(mg/kg/day)^{-1}$	ADAF	Adjusted Kidney Cancer Risk	NHL and Liver Lifetime Cancer Slope Factor $(mg/kd/day)^{-1}$	NHL and Liver Cancer Risk	Total Cancer Risk: Adjusted Kidney and Unadjusted NHL and Liver
Birth to <1 year	5.1E-03	1	1/78	9.3E-03	10	6.1E-06	3.7E-02	2.4E-06	8.5E-06
1 to <2 years	8.5E-03	1	1/78	9.3E-03	10	1.0E-05	3.7E-02	4.0E-06	1.4E-05
2 to <6 years	5.5E-03	4	4/78	9.3E-03	3	7.9E-06	3.7E-02	1.0E-05	1.8E-05
6 to <11 years	3.5E-03	5	5/78	9.3E-03	3	6.3E-06	3.7E-02	8.3E-06	1.5E-05
11 to <16 years	2.6E-03	5	5/78	9.3E-03	3	4.7E-06	3.7E-02	6.2E-06	1.1E-05
16 to <21 years	2.2E-03	5	4/78	9.3E-03	1	1.3E-06	3.7E-02	5.2E-06	6.5E-06
Total years exposed - children		21						Total cancer risk - children	7.3E-05
Adult 21+ years	2.3E-03	33	33/78	9.3E-03	1	9.1E-06	3.7E-02	3.6E-05	4.5E-05
Total years exposed - adults		33						Total cancer risk - adults	4.5E-05

Birth to <1 year total exposure dose does not include shower exposures (inhalation and dermal contact) because very young children are assumed to take baths instead of showers. The ingestion dose was used as the total exposure dose, which might underestimate total exposures to this age group.
ADAF = age-dependent adjustment factor
NHL = non-Hodgkin's lymphoma

Table H. Calculation of Excess Cancer Risk for Residents Exposed to TCE in Private Well Water via Drinking, Inhalation, and Dermal Contact – at 19 ppb - North Shore Drive Site

Age Group	Estimated Total Exposure Dose (mg/kg/day)	Duration (years)	Fraction of Lifetime	Unadjusted Kidney Lifetime Cancer Slope Factor $(mg/kg/day)^{-1}$	ADAF	Adjusted Kidney Cancer Risk	NHL and Liver Lifetime Cancer Slope Factor $(mg/kd/day)^{-1}$	NHL and Liver Cancer Risk	Total Cancer Risk: Adjusted Kidney and Unadjusted NHL and Liver
Birth to <1 year	1.2E-03	1	1/78	9.3E-03	10	2.3E-06	3.7E-02	9.0E-07	3.2E-06
1 to <2 years	4.5E-03	1	1/78	9.3E-03	10	5.4E-06	3.7E-02	2.1E-06	7.5E-06
2 to <6 years	2.9E-03	4	4/78	9.3E-03	3	4.2E-06	3.7E-02	5.5E-06	9.7E-06
6 to <11 years	1.8E-03	5	5/78	9.3E-03	3	3.2E-06	3.7E-02	4.3E-06	7.5E-06
11 to <16 years	1.3E-03	5	5/78	9.3E-03	3	2.3E-06	3.7E-02	3.1E-06	5.4E-06
16 to <21 years	1.2E-03	5	4/78	9.3E-03	1	7.2E-07	3.7E-02	2.8E-06	3.6E-06
Total years exposed - children		21						Total cancer risk - children	3.5E-05
Adult 21+ years	1.2E-03	33	33/78	9.3E-03	1	4.7E-06	3.7E-02	1.9E-05	2.4E-05
Total years exposed - adults		33						Total cancer risk - adults	2.4E-05

Birth to <1 year total exposure dose does not include shower exposures (inhalation and dermal contact) because very young children are assumed to take baths instead of showers. The ingestion dose was used as the total exposure dose, which might underestimate total exposures to this age group.
ADAF = age-dependent adjustment factor
NHL = non-Hodgkin's lymphoma