Health Consultation

FOSTER WHEELER ENERGY CORPORATION/
CHURCH ROAD TCE SITE

MOUNTAIN TOP, LUZERNE COUNTY, PENNSYLVANIA

EPA FACILITY ID: PAD003031788

Prepared by the
Pennsylvania Department of Health

JULY 27, 2010

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

Health Consultation: A Note of Explanation

A health consultation is a verbal or written response from ATSDR or ATSDR's Cooperative Agreement Partners to a specific request for information about health risks related to a specific site, a chemical release, or the presence of hazardous material. In order to prevent or mitigate exposures, a consultation may lead to specific actions, such as restricting use of or replacing water supplies; intensifying environmental sampling; restricting site access; or removing the contaminated material.

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

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Summary

Introduction

At the request of concerned community members, the Pennsylvania Department of Health (PADOH) and the Agency for Toxic Substances and Disease Registry (ATSDR) prepared this Health Consultation (HC) document for the former Foster Wheeler Energy Corporation (FWEC) and Church Road trichloroethylene (TCE) site (collectively called the FWEC/Church Road TCE site or ‘the site’) in Mountain Top, Wright Township, Pennsylvania. Since 2004, PADOH and ATSDR have been reviewing environmental sampling data for the site and providing consultation to the local community, the Pennsylvania Department of Environmental Protection (PADEP), and the Environmental Protection Agency (EPA). The purpose of this HC is to determine if exposure to site-related TCE could harm people’s health. To that end, PADOH and ATSDR reviewed the residential well sampling data, surface water sampling data and limited residential indoor air/vapor intrusion sampling data. In addition, PADOH and ATSDR analyzed the cancer incidence data for the Mountain Top area to determine if the rates of cancer, that are associated with exposures to TCE, are elevated compared to the Commonwealth of Pennsylvania as a whole. In April 2009, the site was proposed to the EPA National Priorities List (NPL). In communities where hazardous chemicals exist, PADOH and ATSDR’s primary goal for the community is to evaluate whether a community is being exposed, has been exposed or will be exposed to levels of contaminants that may harm their health, as well as to ensure that the community has the best information possible to safeguard their health. PADOH worked under a cooperative agreement with ATSDR to complete this Health Consultation document.

Conclusions

Upon reviewing the available environmental sampling data and cancer incidence data, PADOH and ATSDR conclude the following for the FWEC/Church Road TCE site:

Conclusion 1 ***Exposure to TCE in residential well water is not expected to harm people's health.***

Basis for conclusion Residential well water samples collected in 2004 showed levels of TCE above the current Environmental Protection Agency's (EPA) maximum contaminant level (MCL), for drinking water. However, since 2004 the residents have been supplied with alternative drinking water and subsequently hooked up to public water supply, with the exception of one residential property, and are not a current pathway of exposure. Ingestion of residential well water represents a past pathway of exposure but long-term well sampling data are not available. Guardian wells, installed and monitored by EPA and FWEC, are located outside the current area of TCE contamination and do not show that the plume has migrated to other residential areas.

Next Steps PADOH and ATSDR will review additional well sampling data if requested and issuing a public health conclusion.

Conclusion 2 ***The levels of TCE detected in surface water samples are not expected to harm people's health.***

Basis for conclusion TCE was detected in surface water samples collected by EPA above the EPA MCL for drinking water but PADOH and ATSDR do not anticipate the public would be using the surface water for drinking purposes. The public, however, could breathe TCE volatilizing from the surface water. However, given the measured TCE, the processes of diffusion, volatilization, and the likely exposure durations by the public; the resulting levels in the ambient air are not expected to be at levels that could harm people's health.

Next Steps PADOH and ATSDR will consider reviewing additional surface water sampling data, if requested and issuing a public health conclusion.

Conclusion 3 ***Breathing the TCE levels detected in the residential indoor air samples is not expected to harm people's health***.

Basis for conclusion EPA and FWEC conducted indoor air/vapor intrusion sampling at ten residences along Church Road. The highest detected indoor air level of TCE was 7 $\mu g/m^3$. This TCE level is more than 70 times lower than MRL/Intermediate EMEG CV for TCE of 500 $\mu g/m^3$ and more than 1400 times lower than the ATSDR acute minimum risk level (MRL) CV for TCE of 10,000 µg/m^3. This residence had a plumbing leak, which was later repaired resulting in non-detectable levels of TCE. However, since the residential vapor intrusion sampling data is very limited and can fluctuate over time due to various conditions, *a****dditional vapor intrusion sampling is recommended if multiple lines of evidence suggest a vapor intrusion potential is exist at homes along Church Road*** to determine if VI is occurring and if exposure to any detected VOCs could harm people's health.

Next Steps PADOH and ATSDR will review additional vapor intrusion sampling and issue a public health conclusion.

Conclusion 4 Based on estimated exposure dose calculations for inhalation, dermal and ingestion, ***exposure to TCE at the site is not expected to cause an observable increase in cancers and non-cancer health effects.***

Basis for conclusion PADOH and ATSDR calculated an estimated exposure dose based on the residential well sampling data for TCE collected along Church Road. The exposure doses, using EPA's cancer slope factor (CSF) for TCE, were used to compute a theoretical excess cancer risk. The highest theoretical excess cancer risk (calculated using the highest EPA cancer slope factor and assuming 30 years of exposure) of approximately 5.72E-04, falls within the range of low increased cancer risk. Since the residents do not currently use the groundwater for drinking, the theoretical cancer level is likely much lower. In addition, the estimated exposure dose for both adults and children were below levels documented in the scientific literature to show non-cancerous health effects in laboratory animals.

Next Steps PADOH and ATSDR will review sampling data and issue a public health conclusion.

Conclusion 5 Based on a review of the cancer incidence data, ***rates of cancers within the Mountaintop ZIP code were generally not statistically elevated compared to the Commonwealth population as a whole.***

Basis for conclusion PADOH and ATSDR reviewed the Pennsylvania Cancer registry data for the Mountaintop community for the reporting years 1996 to 2007 for all reportable cancers. The analysis indicated no statistically elevated rates for specific cancers that are thought (based on current scientific literature and sources) to be potentially associated with TCE exposure (e.g. hepatic or liver cancer, renal or kidney cancer). Overall, the rates of all reportable cancers for the community were less than the expected rates, based on Commonwealth population as a whole. The analysis did indicate that the rates for one specific cancer types (Thyroid cancer in females only) were statistically elevated for the Mountaintop ZIP code when compared to the Commonwealth population as a whole. However, this cancer is not thought or known, based on current scientific literature and sources, to be potentially associated with TCE exposure or toxicity.

Next Steps

PADOH and ATSDR will consider updating the cancer incidence data, with additional reporting years, in the future and issuing a public health comment.

For More Information

If you have concerns about your health, you should contact your health care provider. For questions or concerns about the Foster Wheeler Energy Corporation/Church Road TCE site, please contact the Pennsylvania Department of Health, Division of Environmental Health Epidemiology at (717) 346-3285.

Background and Statement of Issues

Background

TCE contamination was first discovered at the former Foster Wheeler Energy Corporation (FWEC) located in Mountaintop in 1985, as a result of a real estate transaction. In 1988, FWEC entered into a consent order with EPA and PADEP requiring additional site investigation activities and implementation of remedial measures. Since 1993, FWEC has implemented remedial measures consisting of a groundwater treatment system. On-site groundwater samples are collected on an annual basis from groundwater monitoring wells on the former FWEC property. TCE levels have decreased from a high of 180,000 µg/L, prior to the installation of the groundwater treatment system, to an on-site maximum concentration of 17,000 µg/L in 2008. [1]

In 1986, residential well sampling data collected from seven residential wells located along Church Road by the Pennsylvania Department of Environmental Resources (now called the Pennsylvania Department of Environmental Protection) did not detect volatile organic compounds (VOCs), including TCE, above the EPA maximum contaminant level (MCL). A 2004 groundwater sampling event, however, detected TCE in 14 residential wells along Church Road, exceeding the EPA MCL for TCE of 5 µg/L. Community members and elected officials have expressed their concerns that exposure to TCE in the Mountaintop community could cause adverse health effects. [6]

Based on the 2004 TCE residential well results, EPA and FWEC offered affected residents alternative water supplies (i.e. bottled water and installation of whole house carbon filter in the residences) to mitigate human exposure to VOCs in groundwater. In November 2004, to ensure the carbon filters were properly removing TCE, FWEC and EPA collected tap samples from 34 homes. The results indicated that the filters were effective in removing TCE. [8] In August 2005, Foster Wheeler and EPA entered into an Administrative Settlement Agreement and Order on Consent requiring Foster Wheeler to connect 36 impacted residents to a public water line to eliminate ingestion exposure to TCE. All residents except one were connected to the public water line in July 2007 and the impacted wells were later disconnected and abandoned. [1] One resident who refused to agree to be connected to the public water line and continues to utilize a carbon filtration system for drinking water. [8]

In April 2009, The FWEC site/Church Road TCE site (collectively called the site) was proposed to the EPA National Priorities List (NPL). [2] The NPL is a national list of sites where hazardous substances could impact human health and/or the environment. Listing the site on the NPL assists with determining the magnitude and extent of the groundwater contamination and ensures long-term monitoring of the site. NPL sites first undergo a thorough investigation to determine the full nature and extent of contamination. Next, EPA or the parties responsible for the contamination then address the potential risks the sites might pose to human health and the environment. Once a site has been proposed for the NPL, there is a 60-day comment period, with a final decision on the site made after EPA evaluates and responds to all the public comments in writing. [3]

In 2010, the EPA began a remedial investigation and feasibility study (RI/FS) for the site, which consists of groundwater monitoring well installation and sampling, regional and local geological evaluations, soil sampling, vapor intrusion testing, and pilot-scale testing of remedial technologies. Residential vapor intrusion sampling should be completed in 2011, and, depending on the sampling results the RI/FS should be finished between 2012 and 2013. In addition, a baseline risk assessment will also be developed to identify the existing or potential risks to human health and the environment at the site.

Site Description

The Foster Wheeler Energy Corporation/Church Road TCE Site consists of the former Foster Wheeler facility and the Church Road TCE site located in Mountain Top, Wright Township, Luzerne County, Pennsylvania (Figure 1). FWEC site is a 105 acre site located in the Crestwood Industrial Park and is surrounded by residences and business (Figure 2). This facility was active from 1953 until 1984 and manufactured large pressure vessels utilizing Trichloroethylene (TCE) as a degreaser and machine parts oil remover. The facility has been inactive since 1984. The Church Road TCE site has residential homes, located approximately 1 mile south of the former Foster Wheeler facility, and is referred to as the Affected Area. The Affected Area of homes extends from east to west along Church Road and Watering Run, encompassing approximately 295 acres. [1] There are 29 residential wells near the site that are contaminated with TCE above EPA's Safe Drinking Water MCL. There are an additional 7 wells that contain TCE but are not above the EPA MCL. [6]

Site Visit

In January 2010, the PADOH Health Assessment Program (HAP) personnel met with the PADEP Northeast Regional Office to discuss site background information, environmental sampling and community concerns. In addition, a site visit of the former facility and tour of the surrounding community was conducted.

Public Health Involvement

Since September 2004, ATSDR and PADOH have been collaborating with PADEP and EPA to evaluate environmental sampling data and community concerns related to the FWEC/Church Road TCE site. [4] PADOH and ATSDR has reviewed, evaluated and provided comment on residential sampling data collected by PADEP, EPA, and FWEC. The following is a summary of ATSDR and PADOH involvement with the site:

1. October 2004 – ATSDR Region 3 evaluated groundwater sample data from 14 residential wells in Mountaintop, and found that the TCE levels found in the samples exceeded the MCL of 5 µg/L. The sample levels ranged from 7.6 to 160 µg/L. ATSDR concurred with EPA that alternate water supplies are necessary to mitigate human exposures to contaminated groundwater at this site. In addition, ATSDR recommended that the residents limit the length of showers and baths to less than five minutes until whole-house treatment units are installed and evaluated for effectiveness. ATSDR recommended that EPA conduct a well survey in the area to determine if any other wells were contaminated with TCE;

2. June 2005 - ATSDR Region 3 evaluated one surface water sample from a spring on a residential property. The reported TCE level in the surface water was 26 µg/L, which is between 1,000 and 10,000 times below levels shown to cause health effects via ingestion. ATSDR concluded that exposure to the reported level posed no apparent public health hazard;

3. May 2006 – ATSDR Region 3 evaluated indoor air sampling data results collected from a residence, and found that the reported TCE levels (1.3 ppb or 7 $\mu g/m^3$) were more than 1000 times below levels shown to cause adverse effects in animals (either cancerous or non-cancerous) and approximately 100 times below the levels that are estimated to cause harm in humans via inhalation. The minimum risk level (MRL) for acute exposure to TCE is 2000 ppb (10,000 $\mu g/m^3$) and for intermediate exposure is 100 ppb (500 $\mu g/m^3$). ATSDR concluded that exposure to the reported level posed no apparent public health hazard. [4]

4. August 2008 – PADOH prepared a technical assist (TA) document and factsheet (FS) for the site. The TA and FS documents included: (1) A review of residential sampling data and previous health assessment activities; (2) A review of cancer incidence data for the Mountaintop community for all reportable cancer from 1996 to 2005; and (3). A review of general health information of the community, collected by an area citizen. The cancer incidence analysis did not detect statistically elevated rates of cancer know to be potentially associated with TCE exposure. The community-collected health information listed a tally of certain diseases (cancer, liver damage, heath disease, and autoimmune disease); however, this data could not be quantitatively evaluated nor correlated to exposure and disease, because the information provided lacked sufficient detail. Such details include specific time frames, location of resident, distance of residence from site; possible exposure pathway , environmental sampling or exposure levels, length of time residing at residence; past occupational exposures, heredity and medical history and residence at time of diagnosis. [4]

Exposure Pathway Analysis

An exposure pathway is how a person comes in contact with contaminants originating from a site. A completed pathway requires that all five elements be present: 1) a source of contamination, 2) an environmental medium that transports contaminants, 3) a point of exposure, 4) a route of human exposure, and 5) a receptor population. PADOH and ATSDR consider the air pathway to be a completed pathway, as described in the table below. The presence of a completed exposure pathway does not, however, necessarily mean that adverse health effects will occur or have occurred in the past as a result of such exposure.

Currently, because the groundwater for drinking and showering has been essentially eliminated via filters/public water, the primary current exposure pathway of concern for residents is inhalation of TCE via vapor intrusion from contaminated groundwater (see the Exposure Pathway tables on the following). The public could also be exposed to TCE volatilizing from surface water. However, given the processes of diffusion and volatilization the resulting levels of TCE in ambient air would be low. The likely exposure duration by the public from surface water is expected to be limited; therefore, this is not the pathway of greatest concern at the site.

Current Exposure Pathways

Source of Contamination	***Transport via Environmental Medium***	***Point of Exposure***	***Route of Exposure***	***Receptor Population***
FWEC/Church Road TCE site	Contaminated groundwater via vapor intrusion	Ambient indoor air	Inhalation or breathing	Residents with impacted water near Church Rd.
FWEC/Church Road TCE site	Surface water	Ambient outdoor air	Inhalation or breathing	Residents with impacted water near Church Rd.

In addition, ingestion of residential well water represents a past exposure pathway for residents. The residents along Church Road have been hooked up to public water supply, with the exception of one residential property. Based on the 2004 TCE residential well sampling results, affected residents were provided alternative water (i.e. bottle water and installed carbon filter in the residences) and residents (except one) were later connected to the public water supply. Therefore, ingestion of residential drinking water is not a current exposure pathway for the site, but represents a past exposure for most residents. In 2004, ATSDR recommended the affected resident limit showers and baths to 5 minutes, to reduce any potential inhalation exposures from TCE.

Past Exposure Pathways

Source of Contamination	***Transport via Environmental Medium***	***Point of Exposure***	***Route of Exposure***	***Receptor Population***
FWEC/Church Road TCE site	Contaminated groundwater with volatilization during household use	Ambient indoor air	Inhalation or breathing	Residents with impacted water near Church Rd.
FWEC/Church Road TCE site	Ingestion via contaminated groundwater	Residential drinking water	Drinking or ingestion	Residents with impacted water near Church Rd.

ATSDR Comparison Values

To evaluate whether the public might be exposed to contaminant concentrations that could cause adverse health effects, PADOH and ATSDR evaluated the sampling data against ATSDR Comparison Values (CVs). ATSDR has developed CVs to determine the likelihood of possible health effects from exposure to site-specific contaminants. [10] CVs are health guidelines or environmental guidelines set well below levels that are known or anticipated to result in adverse health effects. ATSDR developed these values to help make consistent decisions about what substance concentrations or dose levels associated with site exposures might require further assessment and evaluation. CVs are not thresholds of toxicity and cannot be used to predict adverse health effects. Although concentrations at or below the relevant CV may reasonably be considered safe, it does not automatically follow that any environmental concentration that exceeds a comparison value would be expected to produce adverse health effects. In general, CVs are derived for substances for which adequate toxicity data exist, based on route of exposure. CVs are typically available for three specified exposure periods: acute (14 days or less), intermediate (15 to 364 days), and chronic (365 days or more). [10]

How Are ATSDR Comparison Values Used?

Comparison values are doses (health guidelines) or substance concentrations (environmental guidelines) set well below levels that are known or anticipated to result in adverse health effects. ATSDR and other government agencies have developed these values to help assess substance concentrations or dose levels associated with site exposures that might require a closer look. Comparison values are derived for substances for which adequate toxicity data exist for the exposure route of interest, if available. However, *comparison values are not thresholds of toxicity and are not used to predict adverse health effects.* These values serve only as guidelines to provide an initial screen of human exposure to substances. Although concentrations at or below the relevant comparison value may reasonably be considered safe, it does not automatically follow that any environmental concentration that exceeds a comparison value would be expected to produce adverse health effects.

PADOH and ATSDR compared the sampling data to the available CVs and these are described in detail in the table on the following page. For cancerous effects, ATSDR has established Cancer Risk Guides (CREGs). For non-cancerous effects, ATSDR utilizes several CV's including Environmental Media Evaluation Guide (EMEG), Minimum Risk Level (MRL), and Reference Dose Media Evaluation Guides (RMEGs). [10]

Type of CV	Description
Environmental Media Evaluation Guides (EMEGs)	EMEGs are estimated contaminant concentrations that are not expected to result in adverse noncarcinogenic health effects based on ATSDR evaluation. EMEGs are based on ATSDR MRLs and conservative assumptions about exposure, such as intake rate, exposure frequency and duration, and body weight.
Cancer Risk Guides (CREGs)	CREGS are estimated contaminant concentrations that would be expected to cause no more than one excess cancer in a million (10^{-6}) persons exposed during their lifetime (70 years). ATSDR's CREGs are calculated from EPA's cancer slope factors (CSFs) for oral exposures or unit risk values for inhalation exposures. These values are based on EPA evaluations and assumptions about hypothetical cancer risks at low levels of exposure.
Reference Dose Media Evaluation Guides (RMEGs)	RMEGs are values derived from EPA's oral reference doses, which are developed based on EPA evaluations. RMEGs represent the concentration in water or soil at which daily human exposure is unlikely to result in adverse noncarcinogenic effects.
Minimal Risk Levels (MRLs)	MRL is an estimate of daily human exposure to a substance that is likely to be without noncarcinogenic health effects during a specified duration of exposure based on ATSDR evaluations. MRLs are set well below toxic effect levels in order to provide an added measure of safety.

TCE is considered to be the contaminant of greatest concern at the site. However, ATSDR does not currently have a CREG value for inhalation exposure or a CV for ingestion exposure. Other ATSDR CVs including intermediate EMEG CV/MRL for inhalation exposure, as well as EPA and California EPA screening levels were used for comparison of the environmental sampling data. PADOH and ATSDR summarized the available CVs in the table below.

ATSDR Comparison Values and EPA Reference Values used for evaluating TCE exposure at the FWEC/Church Road TCE site

Route of Exposure	Source	Type	Level
Ingestion/Drinking			
	EPA	MCL	5 µg/L
Inhalation/Air			
	ATSDR	Intermediate EMEG CV and MRL	500 µg/m^3
	ATSDR	Acute MRL	10,000 µg/m^3
	EPA	RfC (proposed)	40 µg/m^3
	CalEPA	Chronic REL	600 µg/m^3

Sampling Data and Results

Residential Well Sampling and Results

In 2004, EPA and FWEC sampled residential well water for volatile organic compounds (VOCs), semi-volatile organic compounds (SVOCs) and metals. TCE was discovered in 15 residential wells along Church Road above the EPA MCL and PADEP Act 2 Medium Specific Concentrations (MSC) of 5 µg/L with an average level of 59 µg/L. In the 2004 and subsequent additional sampling events by FWEC and EPA, the TCE levels in the residential wells ranged from non-detect to 270 µg/L (Table 1). The concentrations of TCE vary from east to west along Church Road with concentrations in the east east ranging from non-detect to approximately 76 µg/L, wells in the center of the Affected Area from approximately 100 µg/L to 270 µg/L, and wells in the western portion of the Affected Area from non-detect to 30 µg/L. A background well sample was collected approximately 2 miles north to northeast and up-gradient of the FWEC. Sampling results of the background well sample showed a TCE level of 1 µg/L. [16] In total, there are 29 residential wells near the site that are contaminated with TCE above EPA's Safe Drinking Water Act maximum contaminant level (MCL). There are an additional seven wells that contain TCE but are not above the EPA MCL. [6]

Other VOCs were detected in the residential well samples including bromodichloromethane, bromoform, carbon tetrachloride, chloroform, dibromochloromethane, 1,2-dichloroethane, 1,2-dichloropropane, dichlorodifluoromethane, methylene chloride, tetrachlorothene, and trichlorofluoromethane (Table 2). Methylene chloride (maximum concentration of 41 µg/L) and 1,2-dichloropropane (maximum concentration of 5.9 µg/L) were the only other VOCs detected above EPA MCLs and PADEP Act 2 medium specific concentration (MSC) of 5 µg/L for each compound. Some of the VOCs (bromodichloromethane, bromoform, carbon tetrachloride, and 1,2-dichloroethane) were above their respective ATSDR CREG CV. Arsenic was the only metal detected above the ATSDR CV (Table 2). The maximum arsenic level of 10 µg/L exceeded the ATSDR CREG of 0.02 µg/L. However, the residents are not currently utilizing the well water for drinking, as described further in the Background Section. Residential groundwater and exposure via ingestion to VOCs, SVOCs, and metals is not a current pathway of exposure and thus is not a current public health concern. [16]

Guardian Wells Sampling and Results

EPA and FWEC have selected six private residential well locations outside the current affected area, to serve as guardian wells that ensure the plume has not migrated to other residential wells. [7] Since October 2004, EPA and FWEC have conducted quarterly monitoring of the guardian wells. TCE levels in the guardian wells have been below the corresponding laboratory method detection limits at the time of the laboratory analysis (i.e. 0.18, µg/L, 0.4 µg/L, and 1.0 µg/L). [9] Based on this data, it does not appear the TCE contamination plume has migrated beyond the currently known affected areas and homes. EPA and FWEC will continue to monitor the guardian wells to detect and identify if the TCE plume migrates.

Surface Water Sampling and Results

During 2004 and 2005, EPA collected surface water samples in the affected residential areas and in the Watering Run Watershed for VOCs. TCE was detected in 20 of the 65 surface water samples collected ranging from non-detect to 87 µg/L. In November 2007 and March 2008, EPA collected surface water samples at 11 locations for VOCs (table 3). During both sampling events, the maximum TCE concentration in the surface water samples was 160 µg/L. [17] Generally, the highest levels of contamination correspond with highest well sample results. It is unlikely that the general public would utilize surface water for drinking. It is possible people could be directly exposed to the surface water and could breathe TCE volatilizing or evaporating from the surface water. However, given the processes of diffusion, volatilization, and the likely limited exposure frequency by residents, the resulting levels of TCE in the ambient air would be very low. Exposure to the reported levels of TCE in the surface water is not expected to cause adverse health effects in children or adults. In addition, since TCE has a low tendency to bioaccumulation (or build up) in fish, ingestion of fish in surface water is not a likely exposure pathway for the site. PADOH and ATSDR are not aware of people swimming in the streams. PADOH and ATSDR will evaluate results of additional environmental sampling if/when they become available to verify the validity of the above conclusions. [12]

Indoor Air Sampling and Results

In addition to residential well contamination, there is also the potential for VOCs vapors from the contaminated groundwater to enter the overlying residents, in a process called vapor intrusion (VI). Additional details on the VI pathway are discussed in the Contaminant Evaluation Section. Since November 2004, EPA has collected soil gas samples at nine (9) homes. TCE vapors were detected at trace concentrations at two (2) residences and at a higher concentration at a third (1.3 ppb or 7 µg/m^3), in an unfinished basement of the home The higher vapor concentration in the third home was determined to be attributable to a plumbing leak, which was later repaired. TCE contaminated groundwater was also found in a resident's sump pump water in the basement. An investigation led by EPA determined that TCE-contaminated groundwater was getting into the basement's sump pump due to a cracked waterline. The waterline was repaired to mitigate the release of impacted groundwater into the basement. All other homes sampled were non-detect for TCE vapors. [8]

In 2006, EPA and FWEC collected indoor air sampling which was performed to confirm that the repairs at the residence stopped the TCE contaminated groundwater from getting into the basement and to confirm that the residents are not being exposed to TCE at levels of concern at this residence. EPA collected 24-hour indoor air samples (SUMMA canisters) from four locations (two from the unfinished basement, one from the finished basement and one in the first floor dining room) at a single residence and in December 2006, FWEC collected seven indoor air samples at the same residence (Table 4). The highest level of TCE detected was 1.8 µg/m^3. In addition to TCE, the air samples were analyzed for several VOCs, including acetone, benzene, carbon disulfide, dichlorodifluoromethane, ethylbenzene,4-ethyltoluene, 2-butanone, styrene, toluene, trichlorofluoromethane, 1,2,4-trimethylbenzene, and xylenes. The levels of VOCs did not exceed ATSDR's available CV levels.

During the residential air sampling event, the highest detected TCE level was 7 µg/m^3. This TCE level is more than 70 times lower than MRL/Intermediate EMEG CV for TCE of 500 µg/m^3 and more than 1400 times lower than the ATSDR acute MRL CV for TCE of 10,000 µg/m^3. In addition, indoor TCE levels are well below the proposed EPA RfC value of 40 µg/m^3 and CalEPA Chronic REL value of 600 µg/m^3. The maximum reported levels of the above mentioned other VOCs did not exceed their respective chronic EMEG/MRL values. Exposure to reported levels of TCE and other VOCs in indoor air at this site is not expected to cause adverse health effects in children or adults. [12, 14]

The movement of VOCs from subsurface (soil and/or groundwater) into an enclosed structure can vary by season. Factors that can influence such VOC migration include temperature, barometric pressure, and precipitation. Because of these and other variables, VOC concentrations in samples collected during a single sampling event may not accurately represent year-round conditions. [14] Therefore, PADOH and ATSDR recommend that additional indoor air samples, in both the basement and first floor, of the homes along Church Road at varying seasons be collected to better determine the potential exposure levels. It is the understanding of PADOH and ATSDR that EPA and FWEC are planning to collect additional indoor air samples during the RI/FS. Additional information on the indoor air and the vapor intrusion pathway are discussed in the Contaminant Evaluation Section below.

Exposure Scenarios and Dose Calculations

Estimating an exposure dose requires identifying how much, how often, and how long a person may come in contact with some chemical in a specific medium (air, water, soil). PADOH and ATSDR calculated estimated exposure doses for the site.   The equation used to estimate exposure doses from ingestion of TCE in water is below:

Equation 1: Exposure Dose Equation for Ingestion

$$D = \frac{C \times IR \times EF \times CF}{BW}$$

Where: D = exposure dose (mg/kg/day); C = chemical concentration (µg/L); IR = intake rate in liters per day (L/day); EF = exposure factor (unitless = 2); CF = conversion factor 1×10^{-3} (mg/µg); and BW = body weight in kilograms (kg)

Doses from ingestion, inhalation, and dermal contact with TCE at residential locations for both adults and children for past exposures are shown in Table 5 below. The doses are presented in units of milligrams of TCE per kilogram of body weight (mg/kg/day), based on the average TCE concentration (58.4 µg/L) during the 2004 residential well sampling data. The exposure dose calculations assume that exposure occurred in a residential setting that exposure was continuous (24 hours per day, 365 days per year) and all water consumed was from affected wells. Exposures are assumed to have occurred over a 30 year timeframe. Adult doses are estimated assuming a person drinks 2 liters of water per day (from the household source) and weighs 70 kg (154 pounds). Doses for children assume 1 liter per day and a weight of 16 kg (35 pounds). In addition to the oral dose from affected drinking water, a person may also absorb TCE directly from contaminated water through the skin (dermal dose) and breathe the compound in the air (inhalation dose). These secondary exposures to the TCE in drinking water essentially represent a doubling of the ingestion dose (or an exposure factor of 2).

Table 5 – Past exposure past dose for ingestion, inhalation and dermal contact to TCE

	Body Weight (kg)	Ingestion Rate (L/day)	Exposure Factor	Average TCE (µg/L)	Exposure Duration	Dose (mg/kg/day)
Adult	70	2	2	58.4	30 years	0.0033
Child	16	1	2	58.4	30 years	0.0073

Public Health Implications

Cancer Effects

There are several reports of an increased occurrence of cancer from ingestion and inhalation of TCE by animals and humans. [12] Human health studies *suggest* an increased incidence of cancer of various types (e.g., bladder, lymphoma, kidney, respiratory tract, cervix, skin, liver, and stomach) from exposure to TCE; however, no studies provide clear, unequivocal evidence that exposure is linked to increased cancer risk in humans.[12] The available studies suffer from inadequate characterization of exposure, small numbers of subjects, and the fact that subjects were likely exposed to other potentially carcinogenic chemicals. There is, however, sufficient evidence that TCE exposure results in cancer development in animals, although animal studies may not be relevant for evaluating health hazard to humans. [12]

In 1989, EPA withdrew its cancer assessment for TCE, which was based primarily on animal studies, because more recent pharmacokinetic and mechanistic data for TCE became available. [22] An updated approach to TCE cancer assessment using existing animal data and state-of-the-science papers has been proposed. [22] This approach, which is supported by high-dose animal studies, does not appear entirely relevant for evaluating the health hazard of low-dose human environmental exposures for several reasons. First, cancer in animals appears to result from species-specific mechanisms that are not entirely relevant to humans. [12] Second, the animals used in these studies were exposed to very high doses of TCE, compared to those at the FWEC/Church Rd. TCE and the overall death rate in the animal studies was high. The surviving animals were not likely to have been in good health and, therefore, would have been more susceptible to adverse effects from TCE exposure (like infections and illnesses) than healthy animals. Third, the overall findings from animal studies are inconsistent: some studies report an increased incidence of cancer, while an equal number do not report an increase at similar levels of exposure. Fourth, the studies used pure TCE and did not evaluate the effect of exposure to stabilizers and impurities in TCE; these things may also be carcinogenic. [12]

Based on the exposure doses calculated presented in the previous section, PADOH and ATSDR computed a theoretical excess cancer risk based on EPA's cancer slope factor (CSF). A CSF is an estimate of possible increases in cancer cases in a population. A CSF is expressed in dose units [$(mg/kg/day)^{-1}$] to allow for comparison with calculated oral doses. Only adult doses are used to estimate cancer risk because the risks are based on lifetime exposures. Child exposures and intakes occur over a small portion of the assumed lifetime. *It should be noted that the theoretical cancer risk does not predict if an exposed person will get cancer*. [9] Again, because the user of a TCE contaminated water supply would also have exposure through inhalation as TCE volatilizes into the air, inhalation exposure must be included as part of the exposure. Most of this inhalation exposure takes place during and after showering as time spent in the bathroom. To consider both pathways of exposure and their additive effect, we double the (x 2) ingestion. For potential carcinogenic health effects (from past exposure), the doses are multiplied by the contaminant-specific cancer slope factor (Table 6) to determine the theoretical excess cancer risk:

Equation 2: Estimation of Theoretical Excess Cancer Risk

$$CR = D \times CSF \times EY/70 \text{ years}$$

Where: CR= Theoretical cancer risk; D= dose (mg/kg/day); CSF = Cancer Slope Factor $(mg/kg/day)^{-1}$; and EY= Exposure years (30 years)

The theoretical excess cancer risks in Table 6 below represents the expected increase in cancer risk due to (past) exposure to TCE. Note that the TCE cancer risks in Table 6 include estimated excess risk calculated with two different cancer slope factors, a lower bound value (0.4 $(mg/kg/day)^{-1}$) or more conservative, and an upper bound (0.02 $(mg/kg/day)^{-1}$) or less conservative value. The EPA TCE Health Risk Assessment has identified several cancer slope factors, with most between $2x10^{-2}$ and $4x10^{-1}$ per mg/kg-d. [21] As there is no scientific consensus on a specific CSF, the EPA recommends using a range of CSFs and presenting a range of estimated excess cancer risks. Consequently, the highest estimated excess cancer risks due to 30 years of TCE exposure range from about 5.72E-04 or 5.72 excess cancers in 10,000 exposed to about 7.80E-05 or 7.8 excess cancers in 100,000 exposed.

Table 6 – Excess theoretical cancer risk calculations for ingestion, inhalation and dermal exposure.

Cancer Slope Factor $(mg/kg/day)^{-1}$	Adult Dose (mg/kg/day)	Theoretical Excess Cancer Risk
0.4 (Lower bound)	0.0033	5.72E-04
0.02 (Upper bound)	0.0033	7.80E-05

The theoretical cancer risk can never be zero (i.e., any exposure to a carcinogen could potentially have some cancer risk), so exposures are described in terms such as low, moderate or high risks. For example, exposures that could cause one additional case of cancer in a population of one million are considered to have a slight cancer risk, while exposures that could cause one additional case in 10,000 have a low cancer risk. All of the uncertainties and conservative exposure assumptions associated with the dose calculations are included in the risk estimation as well as the uncertainty in deriving the cancer slope factor. [21] The risk estimates in Table 7 cannot be interpreted as evidence that any of the "site" receptors will develop cancer as a result of TCE exposure. The highest theoretical excess cancer risk (calculated using the highest cancer slope factor and assuming 30 years of exposure) of approximately 5.72E-04 *risk fall within the range of low increased risk*, as presented in the table of risk category definitions on the following page. [22] These low risk estimates indicate that TCE exposure in the Affected Area, currently, is not likely to cause an observable increase in cancers. In addition, since the residents do not currently use the groundwater for drinking, the theoretical cancer level is likely much lower.

Risk Category Definitions	
Category	**Theoretical Excess Cancer Risk**
No Increased Risk	<1E-05
No Apparent Increased Risk	1.00E-06
Low Increased Risk	1.00E-04
Moderate Increased Risk	1.00E-03
High Increased Risk	1.00E-02
Very High Increased Risk	>1E-02

Non-cancer Effects

ATSDR has derived a health guideline of 0.2 mg/kg/day for ingestion of TCE based on an acute-duration (less than 14 days) study showing developmental and behavioral changes in mouse pups administered 50 mg/kg/day of TCE. In this study, the TCE was dissolved in oil and administered by stomach tube. [22] The findings of this study are not entirely relevant for evaluating health hazard for Area B site neighbors exposed to TCE in well water for several reasons. First, gavage doses in the animal study were administered as one large dose per day, while residents in Affected area were likely to have been exposed to TCE in drinking water several times a day. The body handles a single large dose much differently than it does a series of small estimated TCE doses, as described in the previous section. Second, the total dose entering the body is higher and maintained for a longer time when TCE is dissolved in oil than when it is dissolved in water. Lastly, exposure to TCE in the animal study lasted less than 14 days, while maximum exposures at the site may have occurred over a period of many years. For ingestion, EPA has set a maximum contaminant level (MCL) of 5µg/L in drinking water. The World Health Organization (WHO) recommended drinking water limit for TCE is 30 µg/L. [14] Although, residential well sampling data did exceed the current EPA MCL, consumption of residential well water is not a current pathway of exposure. Despite these limitations, the estimated exposure doses in the Affected area from past exposure to well water (0.00333 mg/kg/day for adults and 0.0073 for children mg/kg/day) are much lower than doses documented in the literature for adverse non-cancerous health effects and in the above referenced ATSDR health guideline.

For non-cancerous health effects from inhalation, PADOH and ATSDR compared the air sampling data to the ATSDR MRL, EPA Inhalation reference concentrations (RfCs) and California EPA (CalEPA) reference exposure level (REL) for TCE. The EPA RfC of 40 µg/m^3 is based on the critical effects in the central nervous system, liver, and secretion system and is the concentration in air below which non-cancerous harmful effects are unlikely over a lifetime exposure (i.e. 70 years). The REL is a concentration at or below which adverse health effects are not likely to occur. [15] The highest TCE level observed in the residential vapor intrusion was 7 µg/m^3 in the residence with a broken sewer pipe, and 1.8 µg/m^3 the next highest concentration. Therefore, based on the limited vapor intrusion air sampling, at this time PADOH and ATSDR do not expect these levels to harm people's health. Additional sampling data will better delineate exposure levels and the potential for adverse health effects associated with vapor intrusion of TCE.

Contaminant Evaluation

This section provides more information on the chemicals detected during the sampling events. The majority of information summarized below, including context for how the levels for the various CVs were developed, has been extracted from ATDSR's chemical-specific Toxicological Profile for TCE. For more information about each chemical, please refer to these online profiles at http://www.atsdr.cdc.gov/toxprofiles/tp19.html

As explained in the above sections, based on the sampling data evaluated by PADOH and ATSDR, exposure to the levels of TCE in indoor air, groundwater and surface water would not be expected to cause adverse health effects. That being said, the adverse health effects documented in the toxicological literature and summarized here are based on much higher levels than were observed in communities along Church Road, and are usually based on occupational exposures in humans or laboratory animal studies. Some studies with mice and rats have suggested that high levels of TCE may cause liver, kidney, or lung cancer. Some studies of people exposed over long periods to high levels of trichloroethylene in drinking water or in workplace air have found evidence of increased cancer. Although, there are some concerns about the studies of people who were exposed to TCE, some of the effects found in people were similar to effects in animals.

An overview of the vapor intrusion pathway/indoor air and the potential health effects associated with inhalation of TCE is discussed. Although, the levels of TCE in indoor air are not at levels that would result in health effects, the vapor intrusion sampling events are very limited, and additional samples from indoor air and sub-slab soil would better delineate the TCE levels and the potential residential exposures. Lastly, it is important to note, that simply being exposed to a hazardous substance does not make it a hazard. The magnitude, frequency, timing, and duration of exposure and the toxicity characteristics of individual substances affect the degree of hazard, if any.

TCE

Trichloroethylene (TCE) is a nonflammable, colorless liquid at room temperature with a somewhat sweet odor and a sweet, burning taste. TCE is mainly used as a solvent to remove grease from metal parts, to make other chemicals, and can be found in some household products, including wood stains, typewriter correction fluid, varnishes, lubricants paint removers, adhesives, and cleaners. TCE can also be found in industrial settings, in homes undergoing renovations, and in homes using private wells located near TCE disposal or contamination sites, in which TCE vapors seep through basement structures and cracks. [10] TCE was once used as an anesthetic for surgery. [12]

TCE has been found in ambient air, surface water, and groundwater. TCE is heavier than water and therefore is likely to move downward through the subsurface into groundwater, and can persist in groundwater, due to high solubility. Biodegradation of TCE under anaerobic conditions is slow, making TCE relatively persistent in subsurface waters. In settings where groundwater charges surface water, contaminated groundwater can lead to contaminated surface water and sediment. TCE can volatilize from water at a rate that depends on temperature, water movement, and aeration. When released into the air, TCE may be moderately degraded through reaction with photochemically-produced hydroxyl radicals to then form phosgene, dichloroacetyl chloride, and formyl chloride. The half-life of TCE in air is approximately 7 days.

An EPA Groundwater Supply Survey of 945 drinking water systems nationwide using groundwater sources found TCE in 91 water systems, or 10%, with a median level of approximately 1 µg/L. [12] TCE has been detected in outdoor air at concentrations of 0.16 µg/m^3 in rural/remote areas, 2.48 µg/m^3 in urban/suburban areas, and 6.47µg/m^3 in areas near industrial emission sources of TCE. [12] In addition, a survey of indoor air showed median concentrations of TCE as high as 27 µg/m^3 in a North Carolina office building, 0.74 µg/m^{3}in a Washington, D.C. school, and 0.82 µg/m^3 in a Washington, D.C. home for the elderly. [12]

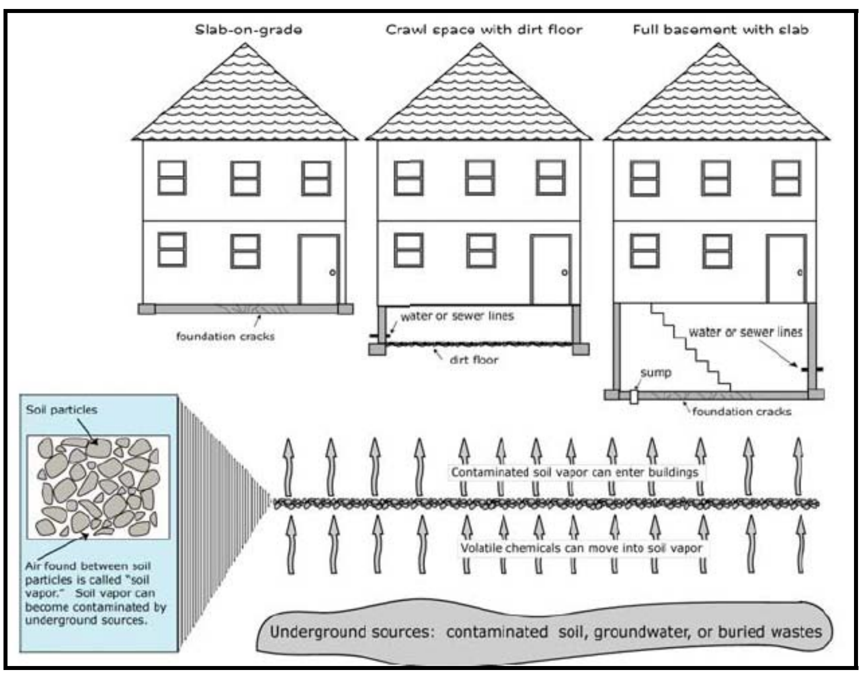
Exposure to TCE is associated with a number of potential health effects, including neurotoxicity, immunotoxicity, developmental, liver, kidney, and endocrine effects. In experimental rodent studies, high doses of TCE administered to mice resulted in tumors of the lungs, liver, and testes. The effects reported at high levels include liver and kidney damage and changes in heart beat. The levels at which these effects occur in humans are not well characterized. Animals that were exposed to moderate levels of TCE had enlarged livers, and high-level exposure caused liver and kidney damage. [12]

Based on the limited data in humans regarding TCE exposure and cancer, and evidence that high doses of TCE can cause cancer in animals, the International Agency for Research on Cancer (IARC) and the WHO have determined that TCE is probably carcinogenic to humans. Many studies reviewed by the IARC examined the relationship between TCE exposure and kidney and liver cancer mortality or incidence. [11] The EPA Integrated Risk Information System (IRIS) is currently producing an assessment of TCE and will include analysis of noncancerous and cancer effects. IRIS is a human health assessment program that evaluates quantitative and qualitative risk information on effects that may result from exposure to environmental contaminants. [17]

Vapor Intrusion

Vapor intrusion (VI), a rapidly developing field of science and policy, is the migration of volatile organic compounds (VOCs) from the subsurface into overlying buildings through the basement foundation and cracks (see the figure below). VOCs in buried wastes and/or contaminated groundwater can emit vapors that may migrate through subsurface soil and into air spaces of overlying building or homes through volatilization, and move to other levels and areas of the home. Inhalation exposures are not limited to buildings with basements, but can also occur for slab-on-grade and suspended floor (or crawl-space) designs as well. In most cases, chemical concentrations of VOCs are low, or depending on site-specific conditions, vapors may not be present at detectable concentrations. VI of TCE can be a public health concern because TCE is readily absorbed by the lungs. [13] It is important to note that exposure to VOCs due to vapor intrusion does not necessarily mean that adverse health effects will occur. Whether or not a person experiences health effects depends on several factors, including the length of exposure (short-term or acute versus long-term or chronic), the amount of exposure (i.e., dose), the frequency of exposure, the toxicity of the chemical and the individual's sensitivity to the chemical. [14]

Overview of Vapor Intrusion Pathway



A complicating factor in evaluating the potential risk from vapor intrusion is the potential presence of some of the same chemicals at or above background concentrations. In addition to contaminated groundwater, indoor air can be contaminated with VOCs due to the wide use in household chemicals and other products, such as cigarette smoke, paint, wood stoves, scented candles, floor wax, perfumes, pesticides, off-gassing from new furniture, and automotive exhaust from an attached garage. Similarly, VOCs can be in the outdoor air that enters a home or place of business from certain commercial and industrial facilities, such as gasoline stations and dry cleaners. Vehicle exhaust is another example of VOCs in outdoor air. Indoor and outdoor sources of VOCs can make evaluating VI migration complicated. [14]

Soil vapor, also referred to as soil gas, is the air found in the pore spaces between soil particles. Soil vapor, containing VOCs, can enter a building through cracks or perforations in slabs or basement floors and walls. Underground conduits, such as sewer lines and utility pipes, fractures in bedrock, or cracks in the basement slab can serve as preferential pathways for vapor migration into the home. In addition, heating, ventilation or air-conditioning (HVAC) systems and/or the operation of large mechanical appliances (e.g., exhaust fans, dryers, etc.) may create a negative pressure that can draw soil vapor into the building. Predicting the extent of soil vapor contamination from soil or groundwater contamination, as well as the potential for human exposure from soil vapor intrusion into buildings, is complicated by factors that can affect soil vapor migration and intrusion. For example, soil vapor contaminant plumes may not mimic groundwater contaminant plumes since different factors affect the migration pattern of each medium. [18]

Indoor air sampling provides the most direct estimate of potential inhalation exposures to residents. [14] However; indoor air sampling data are subject to vast variation due to seasonal variations, changes in the HVAC, contaminant biodegradation processes, contaminant migration, and the presence of VOCs in indoor air from home products. Other types of sampling data that can be used to address VI issues include using sub- slab (i.e., soil vapor samples immediately beneath the foundation or slab of a building), soil vapor samples (i.e., soil vapor samples not beneath the foundation or slab of a building) or groundwater to indoor air attenuation models/factors. These sampling data types provide an indirect measurement of potential indoor air level and can depend on building characteristics, chemical type, soil type, depth of the source, time of year, and a host of other factors. Potential vapors can be reduced or eliminated through engineered controls and containment systems (sub-slab de-pressurization, soil vacuum extraction, vapor barriers) ventilation systems (building pressurization, indoor air purifiers), avoidance (temporary or permanent receptor relocation), removal actions to reduce the concentrations of subsurface chemicals, sealing preferential pathways, and installation of sub-slab vapor mitigation system similar to the type used for radon gas control. [14]

In November 2002, the EPA Office of Solid Waste and Emergency Response (OSWER) issued a draft guidance for Evaluating the Vapor Intrusion to Indoor Air Pathway from Groundwater and Soils (Subsurface Vapor Intrusion Guidance). The draft provides technical and policy recommendations and guidance on conducting a screening evaluation to determine if the vapor intrusion pathway poses an unacceptable risk to human health at cleanup sites. In the document, EPA recommends a site-specific evaluation be performed simultaneously with the subsurface assessment, such as the collection of sub-slab soil vapor, if indoor air concentrations exceed target levels. [14] To evaluate carcinogenic effects of TCE for site-specific assessments, a TCE level in air of 1.2µg/m^3 would represent an upper bound life-time cancer risk to an individual of 10^{-6} (or no apparent increased risk) and a TCE level in air of 120 µg/m^3 would correspond to the lower bound life-time cancer risk to an individual of 10^{-4} (or low increased risk) The TCE air levels were derived based on residential scenario of exposure (24 hours/day, 350 days/year), for 30 years averaged over a 70 year lifetime. In addition to the proposed EPA Rfc value of 40 μg/m^3 for TCE, the OSWER also identified two values in evaluating systemic toxicity at a site from vapor intrusion/inhalation exposure. [14] The first developed by the New York State Department of Health (NYSDOH) of 10 μg/m^3 is based on laboratory data and reports in occupational exposures. The second value is a chronic reference exposure level (REL) of 600 μg/m^3 developed by CalEPA, based on self-reported neurological effects from occupational exposures. [14] The limited site-specific indoor air sampling for TCE (maximum value of 7 μg/m^3) was below the above mentioned comparison values (i.e. EPA Rfc of 40 μg/m^3, NYSDOH value of 10 μg/m^3 and CalEPA REL of 600 μg/m^3).

Cancer Incidence Data Analysis

Due to community concerns about potential elevated disease rates in the community, PADOH and ATSDR reviewed the available cancer incidence data for the community to determine if there are an excess number of cancer cases, currently associated with exposure to TCE.

Sources of Data

The Commonwealth of Pennsylvania maintains health outcome databases including vital statistics and cancer registries. [19] For this HC, The Pennsylvania Cancer Registry (PCR) provided PADOH Bureau of Epidemiology, Division of Community Epidemiology with cancer incidence data for a twelve-year period (1996-2007) for all reportable cancer sites (Table 7). Multiple-year data are generally used in analyses because cases collected in a single year are subject to a large amount of chance variation from year-to-year. The health outcome analysis centers the population data around the available U.S. Census data, which occurs every ten years with the most recent Census occurring in 2000.

The PCR relies on coding sites and histology using International Classification of Disease (ICD) system specifically using the ICD Oncology Code, 3rd edition (ICD0-3). This data is submitted to the PADOH Bureau of Health Statistics and Research. [20] The Registry receives monthly reports from all acute care hospitals and pathology laboratory electronically and represents cancer incidence rates. The PCR also incorporates cancer mortality, using ICD 10th revision codes (ICD-10) when the underlying cause of death is determined to be cancer. For this HC, the number of cancers refers to the number of primary sites reported, not the number of people. Although some individuals may have more than one cancer during the period of interest, in general the number of primary sites is expected to be relatively similar to the number of persons with cancer.

To detect increases in cancer risk for a relatively rare cancer, the population of a ZIP Code needs to be large enough to reliably calculate and compare the relevant cancer incidence rates, and to rule out fluctuations in cancer rates due to chance variation. The 18707 ZIP code has an approximate population of 16,000 persons, and geographically includes but is not limited to in whole or in part to the neighborhoods, areas, and communities of Albert, Fairview Heights, Glen Summit, Lindbergh, Mountain Top, Nuangola, Nuangola Station, Penobscot, Pine View, Reilly, Rippletown, Rita, Slocum, Slocum Corners, Welch Corners, and Wright. The "observed cases" (or study area) for the entire ZIP code represents the number of cancers reported for Mountaintop. The "expected cases" (or comparison area) represent the number of expected cases if the study area had experienced rates of cancer similar to the rest of Commonwealth of Pennsylvania, as a whole, during the same timeframe.

Methods

A statistical analysis was conducted for all cancers combined and for specific cancer sites for males, females, and total (males & females combined). To determine whether there is an excess of cancer in the community, the observed number of cancers in the Mountain Top ZIP code were compared to the "expected" number of cases, based on the Commonwealth of Pennsylvania data during the same timeframe. The cancer rate for the Commonwealth as a whole was used to calculate an expected number of cancer rates that would have hypothetically occurred in the study ZIP codes over the same period of time. The observed cases, theoretically, should not vary significantly from the Commonwealth data as a whole. The information was further standardized to eliminate possible effects due to differences in race, gender, and age between the study area and the rest of the Commonwealth. Statewide sex, gender, and site-specific incidence rates were multiplied by age-groups (0-4, 5-14, 15- 24, … 85+ years). The statistical significance of the indirectly age-adjusted incidence rates was calculated in accordance with the methodology recommended by Selven, et. al [20]

The evaluation of cancer incidence was performed using the Standard Incidence Ratio (SIR), or the ratio of the observed number of cancer incidence divided by the expected number (O:E).
A SIR ratio of 1.0 indicates that the number of cases observed in the population is the same as the expected or Commonwealth rate. A SIR ratio greater than 1.0 indicates that more cases occurred than expected; and a ratio less than 1.0 indicates that fewer cases occurred than expected. Accordingly, a ratio of 1.5 is interpreted as 50% more cases than expected; and a ratio of 0.9 indicates 10% fewer cases than would be expected. [20]

The Z-score, a tool used to determine statistical significance, indicates how far, and in what direction, the observed rates deviate from the mean, expressed in units of standard deviation. Z-scores are utilized to help rule out the possibility that the results are due to chance variation. A Z-score of 1.96 equates to a 95% level of statistical significance, or a 1 in 20 chance that the results are due to random variation alone ($p<0.05$). The 95% CI is the range of estimated ratio values that has a 95% probability of including the true ratio for the population and is a statistical measure of precision. "Statistically significant" means there is less than 5% chance that the observed difference is merely the result of random fluctuation. If a confidence interval Z-score is above + 1.96, it implies there is a statistically significantly higher rate than would be expected. Similarly, if the confidence interval Z-score is below - 1.96, then the number of cases is statistically significantly lower than expected. [20]

Results

PADOH and ATSDR reviewed the cancer incidence health outcome data analysis for the Mountaintop community, for all reportable cancers from 1996-2007 (Tables 5). The cancers most likely, based on current scientific knowledge, related to or associated with exposure to TCE in drinking water are hepatic (liver) and renal (kidney) cancer. However, the toxicological profile for TCE carcinogenicity and specific target organs is still somewhat less well-developed than those for certain other compounds.

Mountaintop had zero reported cases of liver/intrahepatic bile duct cancer, compared to 11.22 expected cases based on Commonwealth as a whole data. In other words, the incidence rate of liver cancer in the Mountaintop community was less than expected and statistically significant less than expected, with a Z-score of -3.38. A Z-score of 1.96 equates to a 95% level of statistical significance, or a 1 in 20 chance that the results are due to random variation alone ($p<0.05$). A Z-score greater than 1.96, the difference between the observed and expected values, is probably due to some other set of factors and unlikely a result of chance variation. For kidney and renal pelvis cancers, there were 31 cases in Mountaintop compared to an expected 31.2 cases, based on the Commonwealth population. Therefore, the number of kidney and renal pelvis cancers in the community is not elevated and is similar to rates observed in the Commonwealth as whole.

Overall, during the 1996-2007 reporting years, the total number of cases for all reportable cancers in Mountaintop (952) was significantly less than the expected number of cases (1071.42), based on Commonwealth as a whole data. In other words, the number of cancer cases in the community was less than expected and statistically less than expected with a Z-score of -4.04. Thyroid cancer was the only cancer that was statistically elevated in the community, with 25 observed cases compared to 23.25 expected cases with a resulting Z-score of +3.16 (statistically elevated in females and total population, but not in males). However, based on the current scientific literature and data, thyroid cancer is not believed to be associated with exposure to TCE. Lastly, several other cancers were statistically less than expected in the Mountaintop community, including larynx, bronchus/lung, breast, cervix, prostate, non-Hodgkin lymphoma, and multiple myeloma. Therefore, based on *a review of the available cancer incidence data, PADOH and ATSDR did not observe elevated levels of cancer in the Mountaintop ZIP code* with the exception of the above-mentioned thyroid cancer data. However, cancer incidence data is not without limitations and a summary of such limitations is discussed in detail in the next section.

Limitations of Cancer Incidence Data

Cancer is a common illness and its occurrence in a population increases with age. Depending on the type of cancer, a population with no known environmental exposure could be expected to have a substantial number of cancer cases. Approximately 1 in 3 people living in the United States will develop cancer at some point in their lives. [21]

Health outcome data evaluations are measures of disease occurrence in a defined population. Such evaluations can help to provide an overall picture of community health, and can potentially identify or confirm excess disease in a community. However, there are many limitations, like any statistical analysis, to using the existing data to examine the relationship between environmental exposures and chronic diseases such as cancer. Most of these limitations would make it less likely (as opposed to more likely) that this health outcome data analysis would identify any potentially elevated rates due to TCE exposure. Such limitations include:

- The cancer incidence analysis was based on an analysis of the entire Mountaintop ZIP code. However, in actuality, only a small percentage of the community (in this case 32 homes) were potentially exposed to TCE. It is difficult to perform cancer incidence analysis when the sample size is small (i.e. the population of given area is small) or when the cancer type is very rare. Therefore, a larger sample size is used and increases the overall confidence in the analysis. This issue of adequate sample size is a common problem faced in epidemiology and statistical analysis, also referred to as low statistical power.

- The quality of the information is directly related to the accuracy of the reporting system, and under reporting of cases is very possible. However, in general Pennsylvania is considered to have a highly reliable cancer registry. A contaminant can contribute to illness or disease without being reflected in the available health outcome data;

- Cancer incidence data can only determine whether there is an increased rate of cancer in the ZIP code area. Cause and effect relationships cannot be established because other factors that may contribute to the observation, such as heredity, lifestyle, environmental exposures from other sources, and occupational exposures, are unable to be accounted for;

- The cancer registry uses only the residence of the individual at the time of diagnosis. Information on previous residence and length of residency are not included in the cancer registry. Population mobility and changes in population can greatly affect the results of this analysis;

- Cancer can have a long latency period (i.e. 30 years). Any elevated incidence rates detected by the analysis provide information on historical exposures potentially related to the site but do not reflect current site conditions or exposure levels; and,

- The interpretation of the SIR has inherent limitations. Any conclusions drawn from the ratios depend on both the ratio value and the total number of observed and expected cases. Two ratios can have the same value but be interpreted differently. For example, a ratio of 1.5 based on 2 expected cases and 3 observed cases indicates a 50% excess in cancer, but the excess is actually only a single case. However, a ratio of 1.5 based on 200 expected cases and 300 observed cases represents the same 50% excess in cancer, but because it is based upon a greater number of cases, the estimate is less likely to be attributable to chance.

Child Health Considerations

ATSDR and PADOH recognize that children are especially sensitive when exposed to many contaminants. 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. PADOH and ATSDR have taken into account the unique vulnerabilities of infants and children to environmental contaminants during the evaluation of this public health issue and the preparation of this health consultation, specifically to through possible inhalation, to children off-site.

There is some data on potential health effects from ingestion of TCE but as discussed further above, the levels of TCE are not at levels expected to cause adverse health effects. PADOH and ATSDR would not expect health effects in children from the reported level of TCE. However, further research needs to be conducted to better define potential health effects from exposure to TCE in children. [12]

Studies suggest that birth defects may occur when mothers drink water containing TCE. People who used water for several years from two wells that had high levels of TCE may have had a higher incidence of childhood leukemia than other people, but these findings are not conclusive. In another study of TCE exposure from well water, increased numbers of children were reported to be born with heart defects, which are supported by data from some animal studies showing developmental effects of TCE on the heart. However, other chemicals were also in the water from this well and may have contributed to these effects. One study reported a higher number of children with a rare defect in the respiratory system and eye defects. Children listed in the National Exposure Subregistry of persons exposed to TCE were reported to have higher rates of hearing and speech impairment. There are many questions regarding these reports, due to the small number of children in the study. [12]

Developmental experimental animal studies have focused on TCE exposure during pregnancy. Observed effects include increased fetal death, altered glucose metabolism in brain or behavior in offspring, delayed organ and bone development, and altered cardiac development. Changes in the immune system have been observed in rat offspring exposed to TCE during pregnancy and lactation, changes included decreased B cell responses, increased delayed-type hypersensitivity responses (involving T lymphocytes), and thymocyte changes in the thymus. [12] PADOH and ATSDR evaluated potential childhood exposure to TCE, as discussed in the Exposure Scenarios and Dose Calculations Section. Based on the dose calculations and the available residential sampling data, PADOH and ATSDR would not expect current exposures to harm children’s health.

Conclusions

Based on a review of the sampling and cancer incidence data, PADOH and ATSDR conclude the following:

1. ***Exposure to TCE in residential well water is not expected to harm people’s health.*** Residential well water samples collected in 2004 showed levels of TCE above the current Environmental Protection Agency’s (EPA) maximum contaminant level (MCL), for drinking water. However, since 2004, the residents have been supplied with alternative drinking water and subsequently hooked up to public water supply, with the exception of one residential property, and are not a current pathway of exposure. Ingestion of residential well water represents a past pathway of exposure but long-term well sampling data are not available. Guardian wells, installed and monitored by EPA and FWEC, are located outside the current area of TCE contamination and do not show that the plume has migrated to other residential areas.

2. ***Exposure to TCE levels detected in surface water samples are not expected to harm people’s health*** TCE was detected in surface water samples collected by EPA above the EPA MCL for drinking water but PADOH and ATSDR do not anticipate the public would be using the surface water for drinking purposes. The public, however, could breathe TCE volatilizing from the surface water. However, given the measured TCE, the processes of diffusion, volatilization, and the likely exposure durations by the public the resulting levels in the ambient air are not expected to be at levels that could harm people’s health.

3. ***Breathing the TCE levels detected in the residential indoor air samples (to date) is not expected to harm the publics' health.*** EPA and FWEC conducted indoor air/vapor intrusion sampling at ten residences along Church Road. The highest detected indoor air level of TCE was 7 µg/m3. This TCE level is more than 70 times lower than MRL/Intermediate EMEG CV for TCE of 500 µg/m3 and more than 1400 times lower than the ATSDR acute MRL CV for TCE of 10,000 µg/m3. This residence had a plumbing leak, which later repaired resulted in non-detect levels of TCE. However, since the residential vapor intrusion sampling data is very limited and can fluctuate over time due to various conditions, ***Additional vapor intrusion sampling is recommended if multiple lines of evidence suggest a vapor intrusion potential be***

performed at homes along Church Road to determine if VI is occurring and if exposure to any detected VOCs could harm people's health.

4. Based on ***estimated exposure dose calculations for inhalation, dermal and ingestion, exposure to TCE at the site is not expected to cause an observable increase in cancers and non-cancer health effects.*** PADOH and ATSDR calculated an estimated exposure dose, based on the residential well sampling data for TCE collected along Church Road. The exposure doses, using EPA's cancer slope factor (CSF) for TCE, were used to compute a theoretical excess cancer risk. The highest theoretical excess cancer risk (calculated using the highest EPA cancer slope factor and assuming 30 years of exposure) of approximately 5.72E-04 (or 5.72 excess cancers in 10,000 exposed), falls within the range of low increased cancer risk. The estimated exposure dose for both adults and children were below levels documented in the scientific literature to show non-cancerous health effects in laboratory animals. In addition, the since the residents do not currently use the groundwater for drinking, the theoretical cancer level is likely much lower.

5. Based on a review of the cancer incidence data, ***rates of cancers within the Mountaintop ZIP code were generally not statistically elevated compared to the Commonwealth population as a whole.*** PADOH and ATSDR reviewed the Pennsylvania Cancer registry data for the Mountaintop community for the all reportable cancers for the years 1996 to 2007. The analysis indicated no statistically elevated rates for specific cancers that are thought, based on current scientific literature and sources, to be potentially associated with TCE exposure (e.g. hepatic - liver, renal - kidney). Overall, the rates of all reportable cancers for the community were less than, and statistically significant, the expected rates, based on Commonwealth population as a whole. The analysis did indicate that the rates for one specific cancer types (Thyroid cancer in females only) were statistically elevated for the Mountaintop ZIP code when compared to the Commonwealth population as a whole. However, this cancer is not thought or known, based on current scientific literature and sources, to be potentially associated with TCE exposure or toxicity.

Recommendations

PADOH and ATSDR recommend that additional indoor air samples, in both the basement and first floor, of the homes along Church Road at varying seasons, be collected to evaluate potential exposure via vapor intrusion.

PADOH and ATSDR recommend continued groundwater monitoring, to ensure the TCE plume has not migrated to other residential areas

PADOH and ATSDR recommend that EPA and PADEP continue to educate and reach out to the community and local officials on the TCE sampling in the community and residential homes.

Public Health Action Plan

The public health action plan for the proposed site contains a description of actions that have been or will be taken by PADOH, ATSDR and other government agencies at the site. The purpose of the public health action plan is to ensure that this health consultation both identifies public health hazards and provides a plan of action designed to mitigate and prevent harmful human health effects resulting from exposure to, or breathing of, hazardous substances. Included is a commitment on the part of PADOH and ATSDR to follow up on this plan to ensure that it is implemented.

Public health actions that have been taken include:

- PADOH conducted a site visit of the former facility and the surrounding community.
- PADOH met with the PADEP Northeast Regional staff to discuss site background information, environmental sampling and community concerns.
- PADOH attended a meeting with the concerned community members.
- PADOH and ATSDR produced a fact sheet, which was posted on the EPA Foster Wheeler website.
- PADOH and ATSDR updated the cancer incidence data analysis for Mountaintop, PA with data from additional reporting years.
- PADOH and ATSDR completed this health consultation.

Public health action that is currently being implemented or will be implemented:

- PADOH and ATSDR will provide and discuss this HC with the PADEP, EPA, and other community members, as feasible and appropriate.
- PADOH and ATSDR will continue to be available to discuss concerns associated with the FWEC and Church Road TCE site with community members and local authorities as appropriate.

- PADOH and ATSDR will provide review and consultation to environmental sampling data, if requested and deemed appropriate, and issue an appropriate public health response

- PADOH and ATSDR will provide education and outreach to the local community and physicians.

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Certification

This health consultation for the Foster Wheeler Energy Corporation/Church Road TCE site was prepared by the PADOH under a cooperative agreement with the ATSDR. It is in accordance with approved methodology and procedures existing at the time the health consultation were initiated. Editorial review was completed by the cooperative agreement partner.

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The Division of Health Assessment and Consultation (DHAC), ATSDR, has reviewed this health consultation and concurs with its findings.

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Figures

Figure 1 – Arial view of the former FWEC facility and Church Road TCE Site

The former Foster Wheeler Energy Corporation facility

Church Road TCE site

Figure 2- Sample location of residential private wells along Church Road and up-gradient background well sample location

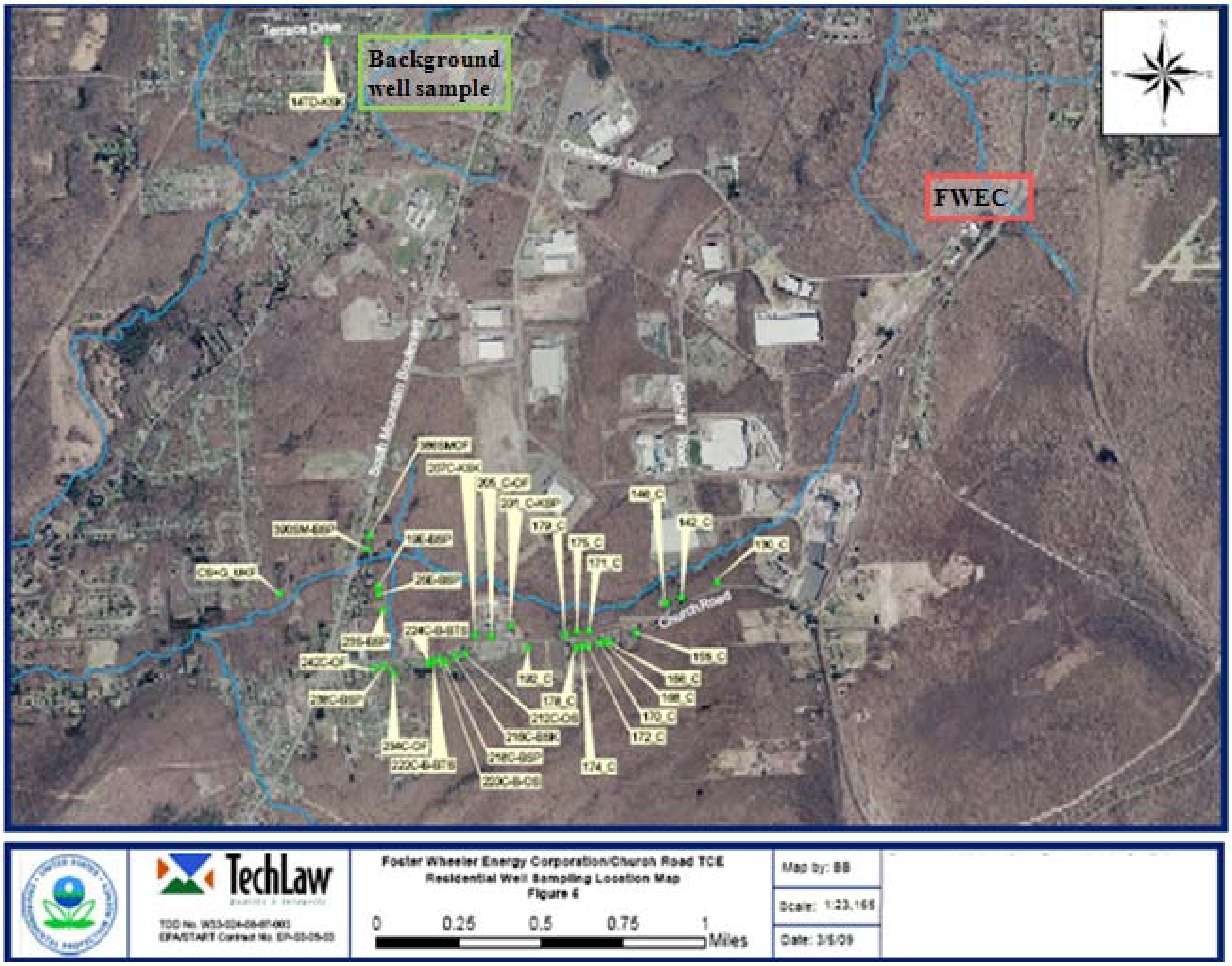
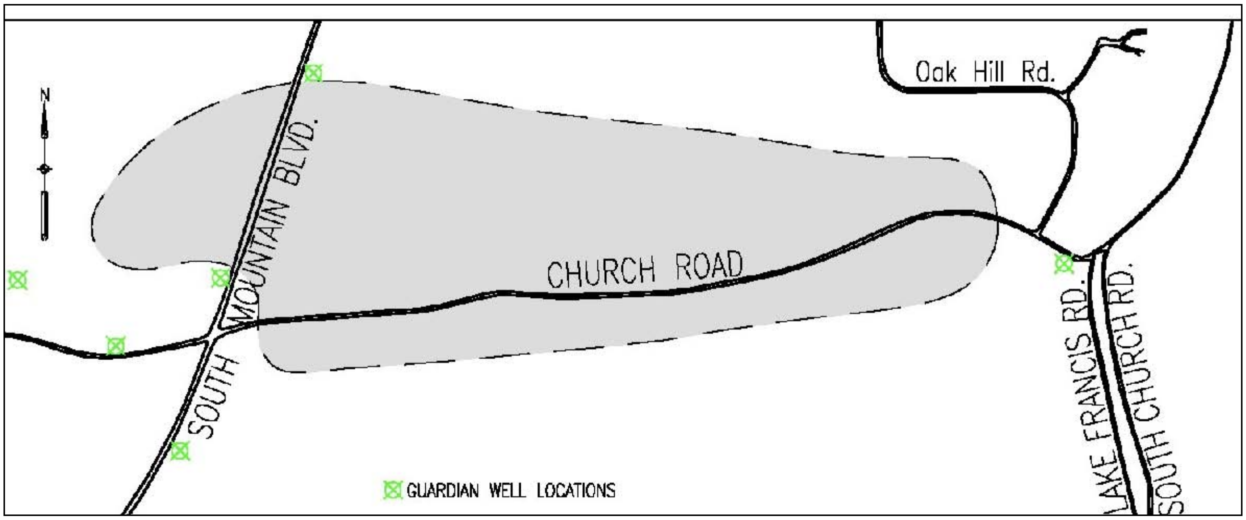


Figure 3 - Location of guardian wells, outside the Church Road TCE plume



Tables

Table 1- Residential private well (Church Rd.) and background sampling results for TCE

Sample ID	Date Collected	TCE (µg/L)	Notes
14TD-KSK	11/4/2004	1.0	Background sample
130_C	9/14/2004	6.3	
142_C	9/14/2004	20.0	
146_C	9/14/2004	42.0	
158_C	9/14/2004	22.0	
166_C	9/14/2004	7.6	
168_C	9/14/2004	60.0	
170_C	9/14/2004	130.0	
171_C	9/14/2004	150.0	ML
172_C	9/14/2004	140.0	
174_C	9/14/2004	100.0	
175_C	9/14/2004	160.0	
178_C	9/14/2004	54.0	
179_C	9/14/2004	82.0	
192_C	9/14/2004	110.0	
201C-KSP	10/16/2004	19.0	
205C-OF	10/18/2004	110.0	ML
207C-KSK	10/18/2004	61.0	
212C-OS	10/16/2004	100.0	
216C-BSK	10/21/2004	77.0	
218C-BSP	10/21/2004	63.0	
220C-B-OS	10/16/2004	82.0	
222C-B-BTS	10/16/2004	52.0	
224C-B-BTS	10/18/2004	82.0	
234C-OF	10/22/2004	3.2	
238C-BSP	10/16/2004	14.0	
242C-OF	11/1/2004	3.7	
23S-BSP	10/18/2004	26.0	
19E-BSP	10/18/2004	27.0	
25E-BSP	10/18/2004	21.0	
386SMOF	10/25/2004	22.0	
390SM-BSP	11/17/2004	15.0	
CS+G_UKF	10/29/2004	6.5	
Average (minus background)		58.4	

ML= estimated value due to low % recovery in matrix spike duplicated and reported concentration may be low

Table 2 - Residential well sampling results for VOCs, SVOCs, and metals

	Contaminant	Detection Limit or Minimum (µg/L)	Maximum (µg/L)	CV (µg/L)
VOCs	Bromodichloromethane	<0.2	4.8	EMEG=700, CREG=0.6
	Bromoform	<0.2	2.1	EMEG=700, CREG=4
	2-Butanone	<0.2	170	
	Carbon tetrachloride	<0.3	2.9	CREG = 0.3
	Chloroform	<0.2	44	EMEG=400
	Chloromethane	<0.3	3	
	Dibromochloromethane	<0.2	0.9	EMEG=3,000, CREG=0.4
	1,1-Dichloroethene	<0.3	0.8	
	1,2- Dichlorobenzene	<0.3	0.9	EMEG = 10,000
	1,4-Dichlorobenzene	<0.4	0.17	EMEG=2,000
	1,2-Dichloroethane	<0.3	3.8	CREG=0.4
	Dichlorodifluoromethane	<0.3	4.7	RMEG= 7,000
	cis-1,2-Dichloroethene	<0.3	1.1	Intermediate EMEG= 10,000
	trans-1,2-Dichloroethene	<0.3	0.2	Intermediate EMEG= 7,000
	1,2-Dichloropropane	<0.3	5.9	EMEG=900
	cis-1,3-Dichloropropene	<0.1	0.16	
	Ethylbenzene	<0.3	0.8	Intermediate EMEG=20,000
	2-Hexanone	<1	0.31	
	Methyl tert-butyl ether	<0.5	0.23	Intermediate EMEG= 10,000
	Methylene Chloride	<0.4	41	EMEG=2,000, CREG=5
	Tetrachlorethene	<0.4	1.8	RMEG=400
	Toluene	<0.3	1	Intermediate EMEG=700
	Trihalomethanes (total)	<0.2	49	MCL = 80
	1,1,1,-Trichloroethane	<0.3	2	Intermediate EMEG= 700,000
	Trichloroethylene	<0.4	270	MCL=5
	Trichlorofluoromethane	<0.2	22	RMEG=10,000
	1,1,2- Trichloro-1,2,2-trifluoroethane	<0.5	1.1	RMEG=1,000,000
	Xylene (total)	<0.2	1.9	EMEG=7,000
SVOCs	Bis (2-ethylhexyl)phthalate	<5	1.1	
	Caprolactam	<5	1.1	RMEG=20,000
Metals	Arsenic	<3.2	10	EMEG=10, CREG=0.02
	Barium	<1.7	26.9	EMEG=7,000
	Cadmium	<0.4	1	EMEG=400
	Calcium	<43	12,900	
	Chromium (total)	<1.6	0.22	EMEG=40 (Chromium VI)
	Cobalt	<1	0.09	Intermediate EMEG=400
	Copper	8.2	163	Intermediate EMEG=400
	Iron	<39.2	41.4	
	Lead	<1.2	7.9	MCL=15
	Magnesium	<41.6	2,200	
	Manganese	<1.2	26.5	RMEG=2,000
	Nickel	<2.4	5.3	RMEG=700
	Potassium	<315	672	
	Selenium	<2.2	0.44	EMEG=200
	Silver	<1	1.5	RMEG=200
	Sodium	2,350	34,800	
	Vanadium	<1	3.8	Intermediate EMEG=100
	Zinc	<5.8	82.6	EMEG=10,000

EMEG = Environmental Media Evaluation Guide,
RMEG= Reference Dose Media Evaluation Guide
MCL = Maximum Contaminant Level (EPA)

Table 3 – Surface water sampling results

Contaminant	Detection Limit or Minimum (µg/L)	Maximum (µg/L)
Acetone	<5	9.4
Bromodichloromethane	<0.2	0.73
Bromoform	<0.2	2.5
2-Butanone	<1.3	1.7
Carbon disulfide	<0.5	19
Carbon tetrachloride	<0.3	0.39
Chloroform	<0.2	2.9
Chloromethane	<0.3	0.41
Dibromochloromethane	<0.12	0.12
1,1-Dichlorethane	<0.3	0.44
1,1-Dichlorethene	<0.3	0.5
1,2-Dichloro-1,1,2,2-tetrafluoroethane	1.3	12
cis-1,2-Dichloroethene	<0.3	8.9
Dichlorofluoromethane	<0.3	4.7
Methyl tert-butyl ether	<0.5	0.21
Methylene Chloride	<0.4	3.7
Tetrachlorethene	<0.4	0.22
Toluene	<0.3	12
1,1,1,-Trichloroethane	<0.3	1.2
1,1,2- Trichloro-1,2,2-trifluoroethane	<0.5	1.5
Trichloroethylene	<0.4	160
Trichlorofluoromethane	<0.2	8.8
Vinyl chloride	<0.2	2.2

Table 4 – Residentail indoor air (vapor intrusion) sampling resutls

Contaminant	Minimum (μg/m^3)	Maximum (μg/m^3)	CV (μg/m^3)
Acetone	12	36	Chronic EMEG/MRL=30,000
Benzene	1	1.7	Chronic EMEG/MRL=10, CREG=0.1
Carbon Disulfide	2.7	2.7	Chronic EMEG/MRL= 900
Dichlorodifluoromethane	2.1	2.8	
Ethylbenzene	0.69	0.9	Chronic EMEG/MRL=1,000
4-Ethyltoluene	1	1.1	
2-Butanone	1.3	4.7	Rfc = 5,000
Styrene	0.74	2	Chronic EMEG/MRL = 900
Toluene	0.68	1.8	Chronic EMEG/MRL = 300
Trichloroethylene	0.42	7 (1.8 next highest since pipe repaired)	Rfc = 40
Trichlorofluoromethane	1.8	2.1	Intermediate EMEG/MRL=500
1,2,4-Trimethylbenzene	0.99	2.3	
Xylenes (Total)	1.5	4.5	Chronic EMEG/MRL=200

Table 5 – Cancer incidence data analysis for Mountaintop, 1996-2007

	POP	CASES	EXPECTED	SIR	ST RATE	CR RATE	ADJ RATE	Z-SCORE
All CANCER SITES								
MALE	7824	483	572.67	.84	620.38	514.44	523.24	-4.15 -
FEMALE	8147	469	498.76	.94	548.73	479.73	515.99	-1.48
TOTAL	15971	952	1071.42	.89	583.32	496.73	518.30	-4.04 -
BUCCAL CAVITY AND PHARYNX								
MALE	7824	16	15.79	1.01	16.31	17.04	16.52	.05
FEMALE	8147	8	6.58	1.22	7.18	8.18	8.73	.54
TOTAL	15971	24	22.37	1.07	11.59	12.52	12.43	.33
ESOPHAGUS								
MALE	7824	9	9.46	.95	10.12	9.59	9.63	-.15
FEMALE	8147	3	2.42	1.24	2.81	3.07	3.49	.38
TOTAL	15971	12	11.88	1.01	6.34	6.26	6.41	.04
STOMACH								
MALE	7824	6	10.44	.57	11.44	6.39	6.58	-1.86
FEMALE	8147	7	5.53	1.27	6.69	7.16	8.47	.66
TOTAL	15971	13	15.97	.81	8.98	6.78	7.31	-.89
COLON AND RECTUM								
MALE	7824	66	65.79	1.00	72.12	70.30	72.35	.03
FEMALE	8147	60	58.19	1.03	68.78	61.37	70.92	.27
TOTAL	15971	126	123.98	1.02	70.40	65.74	71.54	.20
LIVER/INTRAHEPATIC BILE DUCT								
MALE	7824	0	8.26	.00	8.68	.00	.00	-2.85 -
FEMALE	8147	0	2.96	.00	3.41	.00	.00	-1.83
TOTAL	15971	0	11.22	.00	5.95	.00	.00	-3.38 -
PANCREAS								
MALE	7824	7	12.94	.54	14.07	7.46	7.61	-2.29 -
FEMALE	8147	13	11.60	1.12	13.74	13.30	15.40	.45
TOTAL	15971	20	24.54	.81	13.90	10.44	11.33	-1.10
LARYNX								
MALE	7824	3	8.21	.37	8.64	3.20	3.16	-2.97 -
FEMALE	8147	1	2.05	.49	2.16	1.02	1.05	-1.09
TOTAL	15971	4	10.26	.39	5.29	2.09	2.06	-3.09 -
BRONCHUS AND LUNG								
MALE	7824	62	89.04	.70	97.26	66.04	67.72	-3.52 -
FEMALE	8147	34	63.25	.54	70.80	34.78	38.05	-5.49 -
TOTAL	15971	96	152.30	.63	83.57	50.09	52.68	-6.04 -
MELANOMA OF THE SKIN								
MALE	7824	16	19.10	.84	20.24	17.04	16.96	-.77
FEMALE	8147	18	14.18	1.27	14.78	18.41	18.76	.92
TOTAL	15971	34	33.28	1.02	17.42	17.74	17.79	.12
BREAST								
MALE	7824	0	1.49	.00	1.60	.00	.00	-1.22
FEMALE	8147	121	146.01	.83	154.41	123.77	127.96	-2.35 -
TOTAL	15971	121	147.50	.82	80.62	63.14	66.14	-2.52 -
CERVIX UTERI								
FEMALE	8147	6	14.47	.41	18.54	6.14	7.69	-4.33 -
CORPUS/UTERUS, NOS								
FEMALE	8147	46	34.75	1.32	36.44	47.05	48.24	1.70
OVARY								
FEMALE	8147	15	16.31	.92	17.52	15.34	16.11	-.35
PROSTATE								
MALE	7824	134	164.19	.82	177.04	142.72	144.49	-2.64 -
TESTIS								
MALE	7824	6	5.39	1.11	5.94	6.39	6.61	.26

	POP	CASES	EXPECTED	SIR	ST RATE	CR RATE	ADJ RATE	Z-SCORE
URINARY BLADDER								
MALE	7824	43	41.18	1.04	45.64	45.80	47.67	.29
FEMALE	8147	9	13.16	.68	15.41	9.21	10.54	-1.59
TOTAL	15971	52	54.34	.96	30.01	27.13	28.72	-.34
KIDNEY AND RENAL PELVIS								
MALE	7824	21	19.67	1.07	20.77	22.37	22.18	.29
FEMALE	8147	10	11.54	.87	12.69	10.23	11.00	-.52
TOTAL	15971	31	31.20	.99	16.59	16.18	16.48	-.04
BRAIN/OTHER NERVOUS SYSTEM								
MALE	7824	5	7.98	.63	8.47	5.33	5.31	-1.33
FEMALE	8147	6	6.30	.95	6.75	6.14	6.43	-.13
TOTAL	15971	11	14.28	.77	7.58	5.74	5.84	-1.01
THYROID								
MALE	7824	4	5.50	.73	5.66	4.26	4.12	-.72
FEMALE	8147	41	17.86	2.30	17.80	41.94	40.86	3.52 +
TOTAL	15971	45	23.35	1.93	11.94	23.48	23.00	3.16 +
NON-HODGKIN LYMPHOMA								
MALE	7824	14	23.21	.60	25.03	14.91	15.09	-2.49 -
FEMALE	8147	18	19.28	.93	21.79	18.41	20.34	-.33
TOTAL	15971	32	42.50	.75	23.36	16.70	17.59	-1.95
HODGKIN LYMPHOMA								
MALE	7824	2	3.44	.58	3.79	2.13	2.20	-1.06
FEMALE	8147	3	2.83	1.06	3.03	3.07	3.21	.11
TOTAL	15971	5	6.27	.80	3.39	2.61	2.71	-.59
MULTIPLE MYELOMA								
MALE	7824	7	6.24	1.12	6.81	7.46	7.64	.29
FEMALE	8147	0	5.22	.00	6.05	.00	.00	-2.43 -
TOTAL	15971	7	11.46	.61	6.42	3.65	3.92	-1.81
LEUKEMIAS								
MALE	7824	23	14.89	1.54	16.28	24.50	25.14	1.73
FEMALE	8147	9	10.54	.85	11.98	9.21	10.23	-.57
TOTAL	15971	32	25.43	1.26	14.06	16.70	17.69	1.23
MESOTHELIOMIA								
MALE	7824	3	2.44	1.23	2.77	3.20	3.41	.35
FEMALE	8147	0	.54	.00	.63	.00	.00	-.78
TOTAL	15971	3	2.97	1.01	1.66	1.57	1.68	.02
KAPOSIS SARCOMA								
MALE	7824	0	.66	.00	.70	.00	.00	-.81
FEMALE	8147	0	.08	.00	.10	.00	.00	-.31
TOTAL	15971	0	.75	.00	.39	.00	.00	-.87
ALL OTHER SITES								
MALE	7824	32	36.12	.89	39.66	34.08	35.13	-.75
FEMALE	8147	39	37.54	1.04	43.60	39.89	45.30	.27
TOTAL	15971	71	73.66	.96	41.70	37.05	40.19	-.34
2001-2007 Reporting Years								
POLYCYTHEMIA VERA (2001-2007)								
MALE	7824	2	.96	2.07	1.76	3.65	3.66	.73
FEMALE	8147	1	.60	1.67	1.20	1.75	2.01	.46
TOTAL	15971	3	1.56	1.92	1.47	2.68	2.83	.87
CHRONIC MYELOPROLIFERATIVE DISEASE (2001-2007)								
MALE	7824	2	.27	7.41	.52	3.65	3.86	1.29
FEMALE	8147	1	.18	5.68	.38	1.75	2.13	1.00
TOTAL	15971	3	.45	6.73	.45	2.68	3.00	1.65

Cases = Number of newly diagnosed cases during the reporting period.
Expected = Number of expected cases if study area had experienced average PA state rates during 1996-2007
SIR = Standard Incidence Ratio (observed/expected cases).
ST Rate = Average annual state rate per 100,000 population during reporting period.
CR Rate = Average annual crude rate per 100,000 population for study area during reporting period.\
ADJ Rate = Average annual age-adjusted per 100,000 population for study area during reporting
Z-score = Statistical significance of study area compared to state during reporting period (a Z-score of 1.96 equates to a 95 % level of statistical significance or a 1 in 20 chance that the results are due to random variation).

\+ Screened Higher Rate (Z-Score greater than or equal to 1.96)

\- Screened Lowerer Rate (Z-Score less than or equal to- 1.96)