

# Health Consultation

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CTS OF ASHEVILLE, INC SITE  
SKYLAND, NORTH CAROLINA

EPA FACILITY ID: NCD003149556

**Prepared by**  
**North Carolina Department of Health and Human Services**

JULY 24, 2013

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

## **Health Consultation: A Note of Explanation**

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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North Carolina Department of Health and Human Services  
Division of Public Health  
Occupational and Environmental Epidemiology Branch  
under Cooperative Agreement with  
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## Acronyms

AF	Attenuation factor
AT	Averaging time
ATSDR	Agency for Toxic Substances and Disease Registry
CDC	Centers for Disease Control and Prevention
CF	Conversion factor
cm	Centimeter
CREG	ATSDR Cancer Risk Evaluation Guide
CR	Contact rate
CV	Comparison Value
DAF	Dermal absorption efficiency
DPH	N.C. DHHS Division of Public Health
DWM	N.C. DENR Division of Waste Management
DWQ	N.C. DENR Division of Water Quality
ED	Exposure duration
EF	Exposure frequency
EMEG	ATSDR Environmental Media Evaluation Guide
EPA	U.S. Environmental Protection Agency
HG	Health Guideline value
IR	Inhalation rate
IUR	Inhalation Unit Risk factor
Kg	Kilogram
L	Liter
LOAEL	Lowest Observed Adverse Effect Level
MCLG	EPA Maximum Contaminant Level Goal
MCL	EPA Maximum Contaminant Level
M	Meter
mg	milligram
$\mu\text{g}/\text{m}^3$	micro-gram per cubic meter
$\mu\text{g}$	microgram
ng	nano-gram
NA	Not applicable
NC DENR	North Carolina Department of Environment and Natural Resources
NC DHHS	North Carolina Department of Health and Human Services
NIOSH	National Institute for Occupational Safety and Health
NOAEL	No Observed Adverse Effect Level
ppm	Parts per million
ppb	Parts per billion
RfC	Reference Concentration
RfD	Reference Dose
RSL	EPA Regional Screening Level
SVOC	Semi-volatile organic compound
VOC	Volatile organic compound

- **These acronyms may or may not be used in this report**

## SUMMARY

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**INTRODUCTION** The North Carolina Division of Public Health (DPH) understands the community’s concerns about potential exposure to chemicals from the CTS of Asheville, Inc National Priorities List (NPL) site. The facility was bought and operated by CTS from 1959 until 1986. CTS manufactured electronic parts at the facility. Trichloroethylene (TCE) was used to clean or degrease metal parts in the electroplating process.

Chlorinated solvent and metals contamination were identified at the site in 1991. Chlorinated solvent and petroleum contamination was identified in a spring and residential well in 1999. Subsequent North Carolina Department of Environment and Natural Resources (DENR) and U. S. Environmental Protection Agency (EPA) investigations included the collection of samples of groundwater, private drinking water wells, surface water, sub-surface soils, residential crawlspace air, residential sub-slab soil vapor, and outdoor air. The DPH evaluated this data in a Public Health Assessment which was made available for public comment in January 2010.

EPA began collecting quarterly samples from private drinking water wells in a 1 mile radius of the site in September 2008. The quarterly sampling is on-going. The well water data collected through January 2010 was evaluated in a Health Consultation that was completed in July 2010. Substances evaluated in the report included trichloroethylene, metals, and polycyclic aromatic hydrocarbons.

This evaluation includes more than 800 drinking water samples that were collected in 2010 and 2011. Drinking water samples were collected 8 times (quarterly) from approximately 100 homes during the two year period. Stream sediment and surface water samples collected near the CTS site by EPA are also evaluated in this report.

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**CONCLUSION 1** **The NC DPH concludes that drinking water containing elevated lead or copper could harm people’s health.**

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**Basis for decision** Elevated lead levels were identified in drinking water at 7 locations. Six of these locations had more than one drinking water sample with elevated lead. The source of the lead contamination is not known but “first draw” water samples had higher lead levels at 3 locations. Therefore, household plumbing may be a contributing

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	<p>factor.</p> <p>The total blood lead levels calculated exceeded CDC’s reference value of 5 µg/dL at 6 locations. The basis of CDC’s recommendation is evidence of adverse impact on children’s cognitive, behavior, cardiovascular, immunological, and endocrine systems at blood lead levels above 5 µg/dL.</p> <p>Elevated copper levels were identified at 5 locations. Multiple drinking water samples collected at these locations had elevated copper levels. Children drinking water from these wells could experience temporary gastrointestinal health effects.</p>
<b>Next Step</b>	<p>The DPH will contact persons using wells with elevated copper and lead. We will provide information on ways to reduce exposures. We will also have a physician available to provide guidance on appropriate medical follow-up.</p>
<b>Conclusion 2</b>	<p><b>The NC DPH cannot conclude if drinking water containing arsenic could harm people’s health.</b></p>
<b>Basis for decision</b>	<p>The highest concentration of arsenic in drinking water exceeded health guidelines. However, inconsistencies with the data prevent DPH from reaching a conclusion on whether arsenic could have harmed people’s health. The data inconsistencies include:</p> <ul style="list-style-type: none"> <li>• Arsenic was only identified in more than one drinking water sample at 2 of the 19 locations.</li> <li>• The arsenic levels were reported as an estimate in 21 of the 24 drinking water samples.</li> <li>• Arsenic was identified in the laboratory blank at a concentration above ATSDR’s cancer risk evaluation guide (CREG) in the June 2010 quarterly samples.</li> </ul>
<b>Next Steps</b>	<p>The DPH will contact persons using wells with elevated arsenic. We will provide information on ways to reduce exposures. We will also have a physician available to provide guidance on appropriate medical follow-up.</p>
<b>Conclusion 3</b>	<p><b>The NC DPH cannot conclude if drinking water containing polycyclic aromatic hydrocarbons (PAHs) or cyanide could harm people’s health.</b></p>
<b>Basis for decision</b>	<p>The highest concentration of PAHs in drinking water exceeded health guidelines. However, consistency and data quality issues exist with the drinking well water samples. PAHs were only detected at a concentration above EPA’s Regional Screening Level</p>

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in 1 quarterly sample at most locations. In addition, PAHs were identified in field blanks submitted with one set of quarterly drinking water samples.

The highest concentration of cyanide in drinking water exceeded health guidelines. However, consistency and data quality issues exist with the drinking well water samples. Most of the cyanide detections occurred in two of the quarterly sampling events. Cyanide was measured above ATSDR's Comparison Values in the laboratory blanks.

The uncertainty in concentration, length of exposure, and presence of contaminants in the field blanks impact our ability to characterize the risk.

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**Conclusion 4**

**Adverse health impacts are not anticipated for drinking well water containing trichloroethylene, pentachlorophenol, 1,4-dioxane, di-ethylhexyl phthalate, antimony, manganese, or selenium.**

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**Basis for decision**

Trichloroethylene (TCE) was only detected in drinking well water samples at one location. The home was connected to the municipal water system prior to 2010. Therefore, no additional exposure occurred during the timeframe addressed in this report.

The metals antimony, manganese, and selenium were only present in one sample at concentrations above the comparison value. The calculated exposure dose based on the highest concentration was less than ATSDR's Minimal Risk Level.

The estimated cancer risk based on the highest concentration of 1,4-dioxane was less than 1 case per million people exposed. This is considered no increased risk.

The cancer risk estimate based on the highest concentration of pentachlorophenol was 9 to 27 cases per million people exposed. This is considered a low risk.

The excess cancer risk calculated for di-ethylhexyl phthalate (DEHP) was 5 to 16 cases per million people exposed. This is considered a low cancer risk. The actual risk is likely lower because DEHP was detected in some field blanks indicating the possibility of field or laboratory contamination

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**Conclusion 5**

**Adverse health impacts are not anticipated for incidental exposure to sediment or surface water containing trichloroethylene, benzene, or vinyl chloride.**

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**Basis for decision**

No contaminants were detected in the 4 sediment samples or surface water samples collected near the Oaks residential area.

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Contaminants were detected in the samples collected from 2 springs within the fenced area southeast of the CTS site. The highest concentration of each chemical was evaluated to determine incidental exposure (ingestion) from children playing in the creek. The assumptions used in the calculation were that children 6 years old ingest 50 ml (1.5 ounces) of water once a week, six months a year, and for 10 years while playing in the creek.

The calculated exposure doses did not exceed ATSDR's Minimal Risk Level (MRL) for benzene or vinyl chloride. The calculated dose did exceed ATSDR's Minimal Risk Level for trichloroethylene in one of the spring samples. Adverse health impacts are not anticipated because:

- the springs are enclosed in a fenced area
- the sample with the highest concentration was collected in December when children are unlikely to play in the downstream creek.

The excess cancer risk calculated for benzene and vinyl chloride were less than 1 case per million people exposed. This is considered "no increased" risk of cancer. The excess cancer risk for trichloroethylene was one case per hundred thousand people exposed. This is considered a "low" cancer risk.

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### **Information**

If you have concerns about your health as it relates to this site you should contact your health care provider. You can also call the NC Division of Public Health at (919) 707-5900, or send an e-mail to [nchace@dhhs.nc.gov](mailto:nchace@dhhs.nc.gov), and ask for information on the CTS/Mills Gap Road NPL Site Health Consultation.

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## **Background**

### **Site Description and History**

The former CTS Site is located off Mills Gap Road, approximately one mile east of Skyland, North Carolina. International Resistance Company (IRC) built and operated an electroplating facility at the site beginning in 1952. The facility was bought and operated by Chicago Telephone Supply Company (CTS) from 1959 until 1986. IRC and CTS used trichloroethylene (TCE) to clean or degrease metal parts in the electroplating process. After CTS closed, Arden Electroplating leased a portion of the facility for approximately one year. Mills Gap Road Associates (MGRA) purchased the property in 1987. A 46 acre parcel was sold and residential units constructed. The core 9 acre manufacturing area was retained by Mills Gap Road Associates. The facility was vacant after the mid-1990s and the building was demolished in December 2011.

Contamination was identified in a private drinking water well in 1999. Subsequent North Carolina Department of Environment and Natural Resources (DENR) and U.S. EPA investigations included the collection of samples of groundwater, drinking water, surface water, sub-surface soils, residential crawlspace air, residential sub-slab soil vapor, and outdoor air. EPA began collecting quarterly samples from private drinking water wells within a 1 mile radius of the site in September 2008. The quarterly sampling is on-going.

EPA proposed that the CTS of Asheville, Inc (CTS) site be added to the National Priorities List (NPL) in March 2011. In January 2012, EPA and CTS entered into a consent agreement for a Remedial Investigation and Feasibility Study which establishes tasks CTS is responsible for completing. EPA finalized the CTS site on the NPL in March 2012. CTS recently offered whole house water filtration systems for homes within a one mile radius of the site. CTS' contractor began installation of filtration systems in September 2012. As of February, 2013, filtration systems have been installed in 85 homes. In June 2012, Buncombe County Commissioners approved a resolution to seek a loan to pay for the extension of municipal water to homes within a one mile radius of the site.

### **Community Health Concerns**

Community members have expressed concerns regarding the potential for adverse health effects from exposure to chemicals associated with the CTS site. The concerns include cancer and non-cancer related health effects. There is also concern that all cancer related illnesses in the community are not captured by existing tracking systems. The community also raised concerns about vapor intrusion in nearby homes. A vapor intrusion study is currently underway but is outside the scope of this report.

### **Previous NC DPH Involvement**

The North Carolina Division of Public Health (DPH) evaluated the sampling data collected through August 2008 and issued a Public Health Assessment for public comment on January 19, 2010. The *CTS/Mills Gap Road Public Health Assessment Final Release* was issued on January 20, 2011.

The DPH evaluated approximately 520 drinking well water samples that were collected from June 2008 through January 2010. DPH prepared a Health Consultation: *Private Well Waters from the Mills Gap Road Area* that was issued in July 2010.

## Demographics Update

According to Census 2010 data, approximately 3,418 people live within one mile of the CTS site. Census figures show a predominantly White population (89% compared to 68% in the state and 72% in the U.S.). Other ethnic groups include 6% African-Americans, 3% Hispanics, and 2% Asians. Approximately 19% of the population in the area is under 18 years of age. There are 1,725 housing units occupied, with 59% occupied by the owner, and 41% occupied by renters.

Complete 2010 Census data is not yet available. However, the American Community Survey estimates from 2006-2010 are available. The summary report identifies the education level of the population in this area is higher than the rest of the state with 95% of the population having earned a high school diploma.

## Site Geology and Hydrology

The geology and hydrology of the CTS site have been investigated by the North Carolina Geological Survey, U.S. Geological Survey, EPA and its contractors. The CTS site is underlain by a layer of clay, silt and weathered rock that rests on bedrock. The depth of the overburden ranges from approximately 28 to 81 feet thick (MACTEC 2009). The bedrock is comprised of metasedimentary and metavolcanic rocks. Contaminants from the site migrate downward through the porous material to the impervious bedrock. Once at the bedrock, the contaminants can pool and move into bedrock fractures. The fractures function as interconnections that can transmit water to springs, streams, or wells.

An EPA memorandum on the CTS site geology/hydrology (EPA 2011) concluded:

- Groundwater with up to 35,000 µg/L of TCE exists in fractured bedrock beneath and near the CTS plant. Private drinking water wells within the Oaks Subdivision have a higher TCE concentration at greater depths. This is consistent with a flow path from a distant source that traveled to the wells via deep fractures in bedrock.
- Geologic mapping of exposed rock and subsurface borehole measurements document a pattern of joints and fractures which form a pathway for contaminated groundwater near the CTS site to flow toward wells located on Concord Road, Chapel Hill Road, and the Oaks Subdivision.

## DISCUSSION

### Exposure Pathway Analysis

An exposure to a chemical requires persons to come into contact with the chemical through:

- ingestion (eating or drinking the chemical),
- inhalation (breathing the chemical), or
- dermal (absorbing the chemical through the skin)

How people may come into contact with substances (the exposure pathway) is evaluated to determine if people have come into contact with site contaminants, or if they may in the future. A **completed exposure pathway** is one that includes the following elements:

- a **source** of chemical of concern (contamination), such as a chemical release or a hazardous waste site
- movement (**transport**) of the contaminant through **environmental media** such as air, water, or soil

- a **point of exposure** where people come in contact with contaminated drinking water, soil in a garden, or in the air
- a **route of exposure**, or how people come into physical contact with the chemical, such as drinking contaminated well water, eating contaminated soil on homegrown vegetables, or inhaling contaminated air
- a **population** of persons that can come into contact with the contaminants

The elements of an exposure pathway may change over time, so the time frame of potential exposure (contact) is also considered. Exposure may have happened in the past, may be taking place at the present time, or may occur in the future. A completed pathway is one in which all five pathway components exist in the selected time frame (the past, present, or future). If one of the five elements is not present, it is considered an **incomplete exposure pathway**. The length of the exposure period, the concentration of the contaminants at the time of exposure, and the route of exposure (skin contact, ingestion, and inhalation), are all critical elements considered in defining a particular exposure event.

To result in adverse health effects, the chemical must be present at concentrations high enough and for long enough to cause harm. Knowing or estimating the frequency and length of time with which people have contact with hazardous substances is essential to assessing the public health importance of these contaminants. Responses of persons to potentially harmful substances may vary with the individual or particular groups of individuals, such as children, the elderly, or persons with weakened immune responses, or other chronic health issues.

## **Completed Exposure Pathways**

### *Drinking Water*

The population of concern is people living near the former CTS site and using private drinking water wells. The possible exposure routes investigated are ingestion, inhalation, and skin contact. Inhalation of contaminants is a concern for volatile contaminants that may out-gas from the water into the air. Absorption of well water contaminants through the skin is possible but generally considered minor when compared to ingestion or inhalation. The exposure pathway was considered complete for ingestion and inhalation because chemicals were detected in drinking water wells. As part of a Consent Agreement, CTS has offered to install whole house filtration systems for homes located within 1 mile of the site that rely on wells for their drinking water source (EPA 2012). The installation and proper maintenance of filtration systems should eliminate the future exposure pathway until a permanent solution is implemented.

### *Surface Water*

EPA also collected surface water samples were from area springs, creeks, and a pond. A number of contaminants were detected in two springs located southeast of the CTS site. The springs are located in a fenced area designed to prevent public access. However, the springs feed into a creek that exits the fenced area. The surface water from the springs was considered a completed pathway and the potential for incidental ingestion of contaminated surface water evaluated as part of this report. Exposure to the spring water could occur after the water exits the fenced area. No sampling data was available in 2010-2011 to evaluate inhalation exposures from ambient air or inside homes.

Table 1 Completed Exposure Pathways

Source	Medium	Exposure point	Exposure Route	Exposed Population	Time Frame
Ground-water	Groundwater	Private well water	Ingestion Inhalation	People with contaminated well water	Past Current Future
Surface water from springs	Surface water	Surface water	Ingestion Dermal	People with access	Past Current Future

### Evaluation Process

The DPH examined the concentration of chemicals found in the well water samples for potential negative health effects related to drinking the water. Breathing contaminants volatilizing out of the water during activities such as showering and bathing were also considered. It is not known when the contaminants may have first appeared in the wells, or the concentration of the substances over the total time period of contamination. Health-protective values and processes were utilized for all aspects of the evaluation.

A two step process is used to evaluate chemicals’ potential for producing adverse health effects. The first step is to screen each chemical against comparison values (CVs). The comparison values are concentrations of chemicals in the environment (air, water, or soil) below which no adverse human health effects are expected to occur. If a contaminant is present at a level higher than the corresponding CV, the contaminant of concern is retained for the next step of evaluation.

The second step of evaluation focuses on identifying which chemicals and exposure situations could be a health hazard. To identify the greatest potential for negative health effects, the highest concentration of a substance detected in a well was used to evaluate potential health effects. We estimate amounts of a contaminant that people come in contact with and may get into their bodies on an equivalent body weight basis (the “exposure dose”). Each calculated exposure dose is compared against the corresponding health guideline, typically an ATSDR minimal risk level (MRL) or EPA reference dose (RfD). Health guidelines are considered to be below levels where adverse health effects would be expected.

Estimated increased numbers of cancers are calculated for known or suspected cancer-causing contaminants using the estimated site-specific exposure dose and cancer slope factor (CSF) provided in ATSDR health guideline documents. This calculation is based on the assumption that there is no safe level of exposure to a chemical that causes cancer. A 33-year exposure period was used to evaluate cancer risks. This time period approximates the maximum time (95<sup>th</sup> percentile) a person is expected to live at one location. Age specific water consumption rates and body weight were used to calculate exposure doses (EPA EF 2011). Additional information on calculations is provided in Appendix D.

### *Surface Water Contaminants of Concern*

EPA collected 4 stream sediment samples and 7 surface water samples near the Oaks neighborhood in 2010 (Figure 3) to determine if other contaminant sources exist in the area. No volatile organic

chemicals (VOCs) were detected in the stream sediment or spring samples collected north of the Oaks residential area or the water sample collected east of the Oaks residential area at Robinson Creek. Water samples were also collected from a spring east of Mills Gap Road and a retention pond east of Chapel Hill Church Road. Low concentrations of VOCs were detected in the sample collected in the spring and methyl tert-butyl ether (MTBE) was detected in the retention pond sample. The contaminant levels were well below ATSDR's comparison values.

Samples were collected from 2 springs on the west side of Mills Gap Road within a fenced area southeast of the CTS site (Figures 2 and 3). Thirteen chemicals (see Table 1) were detected in samples from Spring-02 and Spring-04 that were collected in June 2011, September 2011, and December 2011. A minimum of four springs feed into a creek which exits the fenced area approximately 100 feet downstream of Spring-04.

The highest concentration of each chemical was evaluated to determine incidental exposure (ingestion) from children playing in the creek. The assumptions used in the calculation were that children 6 years old ingest 50 ml (1.5 ounces) of water once a week, six months a year, for 10 years while playing in the creek. The actual exposure may be less because the highest concentration was selected for the analysis and the sample was collected inside the fenced area. Some dilution will occur as the springs combine into the creek and travel outside the fenced area. A total of 6 chemicals were identified as contaminants of concern because their concentration exceeded comparison values.

**Table 2: Surface Water**

Surface Water Contaminants of Concern
Benzene
Cis-1,2-Dichloroethylene
1,1-Dichloroethane
1,1,1-Trichloroethane
Trichloroethylene
Vinyl chloride

#### *Drinking Water Contaminants of Concern*

DPH evaluated the quarterly well water data and associated quality control samples that were collected from January 2010 through December 2011. Drinking water samples collected prior to January 2010 were discussed in previous DPH reports. The quarterly drinking water samples were collected by EPA and their consultants from more than 100 homes. The samples were analyzed for volatile, semi-volatile compounds, and cyanide. Some well water samples were also analyzed for metals. EPA reviewed the quarterly sampling data when results became available from the analytical laboratory and sent letters to individual home owners informing them of the sampling results.

In 2010-2011, more than 800 drinking water samples were analyzed and approximately 50 different substances were detected. However, not all of the substances can be assumed to be associated with the CTS site. For instance, some of the metals (arsenic, selenium, and manganese) that were detected can also be naturally occurring. The potential health effects of all of the substances identified in the samples were evaluated regardless of the potential source. The highest concentration of a substance detected in the samples was compared to ATSDR's comparison value [ATSDR 2012]. If a comparison value did

not exist, EPA's maximum contaminant level (MCL) or regional screening levels (RSL) were used to identify contaminants of concern [EPA RSL 2012].

Eleven substances were identified at levels above the comparison value and are identified as contaminants of concern. Concentrations of some of the substances exceeded the comparison value in more than 10 samples. Selenium, manganese, and 1, 4-dioxane levels exceeded the comparison value in a single sample out of the 800 drinking water samples analyzed.

Trichloroethylene (TCE) was detected in one drinking water well. However, the well is used for monitoring purposes only. This well was disconnected in 2009 and the home was connected to municipal water. TCE was not detected in drinking water samples collected at other locations. The exposure pathway was incomplete for TCE for the timeframe (2010-2011) addressed in this report.

Inhalation of volatile contaminants can also occur as a result of activities such as showering and bathing. Inhalation exposure to contaminants volatilizing from tap water can be as high as ingestion, effectively doubling the exposure dose. However, the contaminants of concern have a low (or no) volatility and would not be expected to present an inhalation hazard.

**Table 3 Drinking Water Contaminants of Concern**

<b>Drinking Water Contaminants of Concern</b>
Lead
Copper
Arsenic
Polycyclic aromatic hydrocarbons (PAHs)
Cyanide
Pentachlorophenol
1,4 Dioxane
Di-ethylhexyl phthalate (DEHP)
Manganese
Selenium
Antimony

## **Public Health Implications**

The following information describes health effects that have been observed or are thought to be associated with elevated ingestion exposures to the specified chemicals. This information is not intended to be a list of health effects that are expected to occur for all persons consuming contaminated well water.

### **Surface/Spring Water**

The TCE concentration in spring #2 increased from 2,000 µg/L in June 2011 to 7,800 µg/L in December 2011. Similarly, the TCE concentration in spring #4 increased from 1,600 µg/L in June 2011 to 7,000 µg/L in December 2011. The exposure dose calculated using the highest TCE concentration (December

2011) exceeded ATSDR's Minimal Risk Level (MRL). The MRL is an estimate of human exposure below which non-cancer health effects are unlikely to occur. The exposure scenario used to evaluate the springs is based on incidental ingestion of water for children playing in the water. No (non-cancer) adverse health effects are anticipated because the highest concentration occurred in December when children are unlikely to be in the water. The exposure dose calculated for the highest TCE concentration (3,900 µg/L) measured in June, 2011 or September, 2011 was less than ATSDR's Minimal Risk Level (MRL). In addition, the area is fenced to prevent access to the springs.

None of the calculated contaminant exposure doses for the other contaminants exceeded ATSDR's Minimal Risk Level (MRL). Therefore, no (non-cancer) adverse health effects are anticipated from incidental ingestion of water among children playing in the creek.

The excess cancer risk was calculated for benzene, 1,1-dichloroethane, trichloroethylene, and vinyl chloride based on 10 years of incidental ingestion of creek water by a child beginning at age six. Age dependant adjustment factors were applied to the vinyl chloride and trichloroethylene calculations. The calculated excess cancer risk for benzene, 1,1-dichloroethane, and vinyl chloride were less than 1 case per million children exposed. This is considered "no increased" risk of cancer. The excess cancer risk for trichloroethylene was 1 case per hundred thousand people exposed. This is considered a "low" cancer risk.

## **Drinking Water**

### *Lead*

Lead is a naturally occurring metal. It is found in its pure form or in combination with other minerals. Lead is used in the production of batteries, solder, ammunition, sheet metal, and other metal alloys. Lead may leach from plumbing and water line components installed before 1998. Lead was also used as a paint pigment and pottery glazing. Since 1978, paint sold for residential use can contain no more than 600 parts per million lead. Lead paint may be present in a home built before 1978. The Safe Drinking Water Act required all pipes, fixtures, and solder be "lead free" after 1998.

Lead adversely affects the nervous system, kidneys, blood formation system, immune system, and reproductive system [ATSDR Lead 2007]. Elevated lead exposures can also decrease testosterone and thyroid hormone levels. Lead has also been shown to decrease vitamin D levels. Long-term lead exposure for working adults is associated with decreased performance in some tests that measure functions of the nervous system. Lead exposure may also cause weakness in fingers, wrists, or ankles and cause anemia. In pregnant women, high levels of exposure to lead may cause miscarriage. Children are more sensitive to the effects of lead than adults, and studies show that even low lead levels may impair children's cognitive function. The U.S. Department of Health and Human Services and the EPA have determined that lead is a probable human carcinogen. However, EPA stated that the underlying studies are not adequate to allow the accurate calculation of cancer risk [EPA IRIS].

Lead was detected at low levels in many of the drinking water samples tested. At 7 locations (a total of 14 samples) lead levels exceeded the 15 µg/L action level for public water systems. It is not known if the source of the lead is household plumbing or an environmental source. First draw samples were collected at some locations to determine if household plumbing may have contributed to the elevated lead levels. First draw samples are collected after the water has been allowed to remain in the plumbing for at least 6 hours without using the water. A second (flushed) sample was collected after the water



was allowed to run and the well pump starts. If the first draw sample results are significantly higher than the flushed sample result, household plumbing is the most likely source of the lead contamination. The highest value based on a first draw sample was (800 µg/L). The lead levels from “first draw” samples collected at 3 locations were higher than the respective post purge samples. Lead was not detected in the “first draw” or flushed water sample at a fourth location. This indicates that the household plumbing may contribute to lead levels at these locations. The “first draw” samples are not representative of daily household exposure but could result in significant lead exposure if the water lines are not adequately purged before use. The exposure was calculated using the highest first draw and purged drinking water samples.

ATSDR has not adopted a Minimal Risk Level (MRL) for lead because there is no known safe level of exposure for children. ATSDR recommends the use of site specific data to estimate blood lead levels. The CDC Advisory Committee on Childhood Lead Poisoning Prevention recently recommended that the reference value be lowered to 5 µg/dL (micrograms per deciliter) in blood because of increasing evidence of adverse impact on children’s cognitive, behavior, cardiovascular, immunological, and endocrine systems at blood lead levels less than 10 µg/dL [CDC Lead 2012]. The draft National Toxicology Program (NTP) Monograph on Lead supports CDC’s conclusions regarding low level childhood exposure to lead [NTP Lead 2011]. The NTP report also concludes adverse health effects such as hypertension and decreased renal function are associated with blood lead levels less than 10 µg/dL in adults.

Two methods were used to calculate the impact of consuming contaminated drinking water on blood lead levels. It should be noted that uncertainties exist with any mathematical model. The most reliable method to determine an individual’s exposure is blood lead analysis. The highest values measured in well water were used in the calculations. ATSDR developed an integrated exposure regression analysis approach which integrates lead exposures for all pathways [ATSDR Lead 2007]. The total blood lead level calculated using this approach exceeded 5 µg/dL for children and adults at 2 locations.

A second method, EPA’s Integrated Exposure Uptake Biokinetic (IEUBK) Model for Lead in Children, was used to calculate the total blood level [EPA IEUBK Model]. The highest lead levels measured in the well water and ATSDR recommended average water ingestion rates were used in the calculation. The IEUBK model system default values were accepted for other routes of exposure. The total blood lead calculated by this method exceeded 5 µg/dL for children at 6 locations. Therefore, drinking the water with elevated lead levels could harm people’s health.

#### *Polycyclic Aromatic Hydrocarbons (PAHs)*

Polycyclic aromatic hydrocarbons are a group of chemicals that form during the incomplete burning of coal, oil, gas, wood, garbage, or other organic substances. PAHs are a group of more than 100 structurally related compounds that generally occur as a complex mixture. They can occur naturally or as a result of human activity. PAHs may be used in medicines and to make dyes, plastics, and pesticides. They are found in asphalt used in road construction, coal tar pitch, creosote, and roofing tar. They are found throughout the environment but the most common release is to air from volcanoes, forest fires, residential wood burning, and exhaust from automobiles and trucks. PAHs in soils can contaminate groundwater, but most do not easily dissolve in water. In soils, PAHs are most likely to stick tightly to particles.

Animals fed high concentrations of PAHs during pregnancy had difficulty reproducing and so did their offspring. These offspring also had higher rates of birth defects and lower body weights. It is not known whether these effects also occur in people. Animal studies have also shown that PAHs can cause harmful effects on the skin, body fluids, and ability to fight disease after both short- and long-term exposure. The National Toxicology Program lists PAHs as reasonably expected to be human carcinogens. Some people who have breathed or touched mixtures of PAHs and other chemicals for long periods of time have developed lung or skin cancer. PAHs have caused lung cancer in laboratory animals when they breathed air containing PAHs, stomach cancer when PAHs were ingested in food, or skin cancer when PAHs were applied to their skin [ATSDR PAH 1995,].

Up to 20 individual PAHs were identified in the drinking water samples. One or more PAHs were present at levels above EPA's Regional Screening Level (RSL) for tap water in 11 drinking water samples collected from 9 different wells. The PAH levels were estimated values in 6 of these drinking water samples. At 7 of the well locations PAHs exceeded the RSL in only one quarterly sample. At the other 2 well locations, PAHs exceeded the RSL in 2 of the quarterly samples.

Most of the PAHs detections were in the June 2010 and September 2010 drinking water samples. In the June 2010 sampling event only one water sample exceeded the comparison value but PAHs were detected in another 42 drinking water samples. Naphthalene and phenanthrene were the specific PAHs most frequently detected. Naphthalene was also detected in 3 of the field water blanks collected in the June 2010 quarterly samples.

In the Sept 2010 sampling event, PAHs exceeded the regional screening level in 2 wells but were detected in another 84 samples. Six PAHs were detected in the field blanks for the Sept 2010 quarter drinking water samples. None of the contaminants in the field blanks exceeded the regional screening level. One or more of the PAHs detected in the field blanks were the only PAHs detected in more than 70 of the field samples. The presence of PAHs in the field blanks and the pattern of PAH detections indicate the well water samples may have been contaminated in the field or laboratory. The number of samples with PAHs detected decreased dramatically for the remaining quarterly drinking water samples.

Non-cancer health guidelines do not exist for many PAHs. The calculated exposure dose was less than the minimal risk level (MRL) for the individual PAHs with health guidelines.

The individual PAHs present in a drinking water sample were converted to a benzo(a)pyrene equivalent to more accurately estimate the overall cancer risk (see Appendix D). This method provides a more accurate description of the potential risk. The calculated benzo(a)pyrene equivalent values were added together to evaluate the cancer risk associated with the mixture. One drinking water sample was eliminated from consideration because the laboratory quality control indicated the reported concentrations were not valid. The highest benzo(a)pyrene equivalent concentration from the remaining drinking water samples was used to evaluate the potential adverse health effects.

The highest benzo(a)pyrene equivalent concentration for total PAHs in any one sample was 1.71 µg/L. This exceeds ATSDR's Cancer Risk Evaluation Guide (CREG) of 0.005 µg/L for benzo(a)pyrene. No PAHs were detected at this location in any of the other quarterly samples. ATSDR and EPA do not have non-cancer health guidelines for benzo(a)pyrene.

The cancer risk was calculated using the age specific water consumption rates and the benzo(a)pyrene equivalent concentration [EPA EF 2011]. Age-dependent adjustment factors were applied because PAHs are classified as having a mutagenic mode of action and the cancer potency may be increased for persons less than 16 years old [EPA 2005}. Mutagens are substances that can change genetic material in cells. The cancer rates for children were calculated based on 21 years exposure beginning at birth. The cancer rates were calculated based on average and a reasonable maximum age specific water intake rates. The excess cancer risk for children exposed from birth to 21 years was calculated as 3 to 7 cases per 10,000 people exposed. The cancer rate for adults was calculated based on 33 years of exposure. The excess cancer risk calculated for adults was 8 to 20 cases per 100,000 people exposed. The child and adult cancer risk are considered “moderate”.

The actual cancer risk is likely less because the calculated cancer risk is based on 21 and 33 years of constant exposure. The quarterly drinking water sampling results showed PAHs were not consistently detected in any specific wells over the 2 year sampling period.

### *Arsenic*

Arsenic is a naturally occurring element that is present in soil and minerals. Arsenic was used as a preservative in pressure treated wood and in some pesticides. Arsenic is also used as an alloy in lead-acid batteries and the manufacturing process for semiconductors and light emitting diodes. Some areas of North Carolina are known to have elevated levels of arsenic in well water due to naturally occurring geological formations.

Inorganic arsenic has been recognized as a human poison for thousands of years. Although the amount of arsenic required varies, most of the body’s organs can be adversely impacted by arsenic. Chronic exposures can result in skin lesions, high blood pressure, decreased lung function, and circulatory problems. Nausea, vomiting, and diarrhea may also occur following ingestion of arsenic. Chronic exposure of humans to inorganic arsenic in drinking water has been associated with excess incidence of miscarriages, stillbirths, preterm births, and infants with low birth weights. Animal data suggest that arsenic may cause changes to male and female reproductive organs as well as other developmental changes [ATSDR Arsenic 2007].

Ingestion of arsenic has also been reported to increase the risk of cancer in the liver, bladder, and lungs. There is convincing evidence that skin cancer may also develop from ingestion of arsenic. The Department of Health and Human Services (DHHS) has determined that inorganic arsenic is known to be a human carcinogen. The International Agency for Research on Cancer (IARC) has determined that inorganic arsenic is carcinogenic to humans. EPA also has classified inorganic arsenic as a known human carcinogen.

Children who are exposed to elevated levels of inorganic arsenic may have many of the same effects as adults, including irritation of the stomach and intestines, blood vessel damage, skin changes, and reduced nerve function. There is also some evidence that suggests that long-term exposure to inorganic arsenic in children may result in lower IQ scores. We do not know if absorption of inorganic arsenic from the gut in children differs from adults. There is some evidence that exposure to arsenic in early life (including gestation and early childhood) may increase mortality in young adults.

Arsenic levels above ATSDR's comparison value were identified at 19 locations. Most of the well water samples with elevated arsenic levels occurred in the June 2010 sampling event. The arsenic concentration in 16 samples was reported as an estimated value that was above ATSDR's Cancer Risk Evaluation Guide (CREG) of 0.02 µg/L. A laboratory quality control blank for this quarter also identified arsenic at an estimated value which is higher than ATSDR's CREG. The pattern of detections suggests that laboratory contamination may be a factor in the number of samples identified with elevated arsenic concentrations. The actual number of drinking water wells with elevated arsenic levels is not known but it is likely less than the 19 locations identified. Elevated arsenic levels were only identified in 3 locations after the June 2010 sampling event.

Arsenic levels at one drinking water well exceeded the comparison value in four samples that were collected in 2010. Two of these samples were identified as post filter samples. ATSDR's chronic non-cancer comparison value (3 µg/L child) was exceeded in 2 of the samples. Arsenic was not detected in the drinking well water at this location in the 2011 quarterly samples. Arsenic levels did not exceed the chronic (non-cancer) comparison value at any other well locations.

The exposure dose was calculated using the highest arsenic concentration (17 µg/L) and age specific water consumption rates. The average and reasonable maximum water intake rates were used to calculate the exposure dose. The exposure dose did not exceed the acute MRL. The calculated dose based on the reasonable maximum water intake rate exceeded the chronic exposure MRL for children and adults. The calculated dose for children less than 6 years old based on the average water intake rate exceeded the chronic exposure MRL but did not exceed it for other age categories. Human health studies indicate that drinking the well water at this concentration for more than a year could cause adverse health effects [ATSDR Arsenic 2007]. ATSDR determined the skin is the most sensitive system to ingestion of water containing arsenic. The estimated exposure dose exceeded the value some studies found could cause skin pigmentation or lesions.

There is debate in the scientific community about the model and cancer slope factor used to assess the cancer risk associated with low level arsenic exposures. The National Academy of Sciences and EPA Science Advisory Board evaluated arsenic animal data and epidemiological studies. EPA is reportedly considering increasing the cancer slope factor for arsenic. If this occurs, the result will be an increase in the calculated cancer risk for arsenic exposures.

The excess cancer risk was also calculated using the highest exposure level and age specific water consumption rates. The cancer rates were calculated based on average and reasonable maximum age specific water intake rates. The cancer rates for children were calculated based on 21 years exposure beginning at birth. The cancer risk for children was 1 to 3 cases per 10,000 people exposed. The cancer rates for adults were calculated based on 33 years of exposure. The cancer risk calculated for adults was 2 to 4 cases per 10,000 people exposed. The child and adult cancer risk are considered "moderate". The actual risk is anticipated to be lower because arsenic was not detected in samples collected at this location before or after 2010.

### *Copper*

Copper is a reddish metal that occurs naturally in rock, soil, water, and sediment. At low levels, it is an essential element for all known living organisms but when high levels are ingested toxic effects can occur. Drinking water that contains higher than normal levels of copper may cause nausea, vomiting, stomach

cramps, or diarrhea. Higher intakes of copper can cause liver and kidney damage. It is not known if copper can cause cancer in humans. EPA has not classified the potential for copper to cause cancer because there are not adequate human or animal cancer studies.

Exposure to high levels of copper will result in the same types of effects in children and adults. Studies in animals suggest that children may have more severe effects than adults but we do not know if this would also be true in humans. There are a very small percentage of infants and children who are unusually sensitive to copper. We do not know if copper can cause birth defects or other developmental effects in humans. Studies in animals suggest that ingestion of high levels of copper may cause a decrease in fetal growth [ATSDR Copper 2004].

The copper level exceeded the comparison value in the drinking water samples collected from 5 wells. Multiple samples from each location exceeded the comparison value. The highest copper concentration (3300 µg/L) was a “first draw” sample. The “first draw” sample was collected to determine if household plumbing may have contributed to the elevated copper levels. The copper level decreased to 31 µg/L at this location after the water line was purged and re-sampled. This suggests the most likely source of the copper is the household water line.

The exposure dose was calculated using the “first draw” sample (3300 µg/L) and age specific water consumption rates. The average and reasonable maximum water intake rates were used to calculate the dose. The exposure dose calculated using the first draw sample exceeded ATSDR’s MRL for all ages of children and adults. Health studies indicate that children and adults drinking water with this copper concentration may experience nausea and vomiting. The first draw sample may not be representative of daily exposure but reinforces the need to adequately purge water lines before use or investigate the use of a filtration system. The exposure dose at this location did not exceed the MRL if the 31 µg/L (purged sample) was used in the calculation.

The exposure dose was also calculated on the highest copper level (250 µg/L) excluding “first draw” samples. The calculated exposure dose based on the reasonable maximum water intake rates exceeded ATSDR’s MRL for children less than 6 years old. The exposure dose was below the no observed adverse effect level (NOAEL) identified from health studies. However, children who are sensitive to copper may experience gastrointestinal symptoms. The adult exposure dose was less than the MRL and no adverse health effects are anticipated.

### *Cyanide*

Cyanides can occur naturally or be man-made. The major man-made source of cyanide is discharges from industrial processes such as metal-finishing industries, iron and steel mills, and organic chemical industries. Other cyanide sources include vehicle exhaust, releases from certain chemical industries, burning of municipal waste, tobacco smoke, and use of cyanide-containing pesticides. Cyanide-containing substances also occur naturally in the fruits, seeds, roots, and leaves of numerous plants. [ATSDR cyanide]

Exposure to cyanide can be harmful. The severity of the harmful effects depends on the amount, route of exposure, and the form of cyanide. Exposure to high levels of cyanide for a short time harms the brain and heart and can even cause coma and death. Some of the first indications of cyanide poisoning

are rapid, deep breathing and shortness of breath, followed by convulsions (seizures) and loss of consciousness.

Chronic dose-response studies of human exposure to cyanide were not identified. However, studies of occupationally exposed workers indicate that chronic exposure to low concentrations of cyanide can cause alterations of thyroid function and neurological symptoms.

Animal studies have been performed to determine the effects of low level exposure to cyanide. Statistically significant male reproductive effects were observed in mice and rats. The effects include decreased weights of the testes, decreased weight of the epididymis, and altered sperm parameters. Other studies identified thyroid changes and decreased body weight in the animals. [IRIS Cyanide].

The EPA has determined that there is inadequate information to assess the potential of cyanide to cause cancer. The National Toxicology Program and International Agency for Research on Cancer (IARC) have not classified the potential of cyanide to cause cancer.

Cyanide was identified at concentrations above ATSDR's Comparison Value (CV) in four of the quarterly sampling events. No cyanide was detected in 2 of the quarterly sampling events. Most of the drinking water samples that were above the CV occurred in the September 2010 and December 2011 sampling events. The presence of cyanide was confirmed but the concentration was estimated in many of these samples. Laboratory quality control issues including the presence of cyanide in laboratory blanks were identified by EPA.

The exposure dose was calculated using the highest cyanide concentration (32J  $\mu\text{g/L}$ ). The exposure dose calculated using the reasonable maximum water consumption rates exceeded the MRL for all age groups. The exposure dose calculated using the average water consumption rates exceeded the MRL for children under 6 years of age. The exposure dose calculated using the highest confirmed (23  $\mu\text{g/L}$ ) concentration and reasonable maximum water consumption rates also exceeded the MRL for all age categories. The calculated exposure dose is less than 1 per cent of the bench mark dose level identified by EPA. However, the uncertainty in concentration, length of exposure, and presence of contaminants in the laboratory blanks impact our ability to characterize the risk of adverse health effects.

### *Selenium*

It is a naturally occurring mineral found in rocks and soil. Commercial uses of selenium include photographic equipment, gun bluing agent, plastics, paints, anti-dandruff shampoos, vitamin and mineral supplements, fungicides, and certain types of glass. Selenium is also used to prepare drugs and as a nutritional feed supplement for poultry and livestock [ATSDR Selenium 2003].

Selenium is an essential nutrient for humans and animals. However, selenium can be harmful when regularly taken in amounts higher than those needed for good health. Chronic oral intake of very high levels of selenium (10–20 times more than normal) can produce selenosis in humans, the major effects of which are dermal and neurological. Populations exposed to chronic dietary excess levels of selenium had diseased nails and skin, hair loss, and neurological problems, including unsteady gait and paralysis. There is no evidence to support an association between selenium compounds and cancer in humans.

The selenium level in drinking water exceeded the comparison value in one well. The selenium levels in the other quarterly samples at this location were less than the comparison value. The exposure dose was calculated using age specific water consumption rates. The exposure dose calculated using the reasonable maximum water consumption rates exceeded the MRL for children under 2. The exposure dose was less than the MRL for other age groups. The exposure dose was less than the MRL for all age groups using the average water consumption rates. Adverse health effects are not anticipated because selenium levels did not exceed the comparison value in the other quarterly drinking water samples at this location.

### *Manganese*

Manganese is a naturally occurring metal found in rocks and soil. Manganese is used in a variety of products including metal alloys, paints, batteries, cosmetics, and fireworks. It has also been used as a gasoline additive to improve the octane rating.

Manganese is an essential nutrient so consumption of small amounts is important to good health. However, animal studies show ingestion of larger doses can affect the nervous system, decrease fertility, and lead to inflammation of the kidneys. The nervous system impacts include tremors and behavioral changes. EPA has determined there is not adequate data to determine if ingestion of manganese can cause cancer [IRIS Manganese].

The manganese concentration exceeded the drinking water comparison value in one well. The manganese levels in the other quarterly samples at this location were less than the comparison value. The exposure dose calculated using the reasonable maximum water consumption rates exceeded the MRL for children under the age of 1 year. The exposure dose was less than the MRL for other age groups. The exposure dose was also less than the MRL for all age groups when the average water consumption rates were used to calculate dose. Chronic adverse health effects are not anticipated because elevated manganese levels were only detected in one of the quarterly samples.

### *Antimony*

Antimony is a naturally occurring metal. It is typically mixed with other metals to form alloys. The ore is not mined in the United States but antimony can be released to the environment from the combustion of coal. Acute exposure of workers to antimony has been shown to result in gastrointestinal effects including vomiting and diarrhea. Animal studies support the gastrointestinal effects of ingesting antimony. Animal studies also show changes in the ability to regulate arterial blood pressure following pre/post-natal ingestion of antimony. High exposures to antimony in animals also reduce the number of red blood cells. A study of women working in an antimony smelter found they had a higher incidence of spontaneous abortion and premature deliveries. No data is available on whether antimony can cause cancer [ATSDR antimony 1992].

Antimony was detected in a “first draw” drinking water well sample at a concentration just below ATSDR’s comparison value for child exposure. Antimony was not detected in the post purge sample or the other quarterly drinking water samples at this location.

The exposure dose calculated using the reasonable maximum water consumption rates exceeded the EPA’s reference dose (RfD) for children under the age of 1 year. The exposure dose was less than the MRL for all other age groups. The exposure dose was less than EPA’s RfD for all age groups when the

average water consumption rates were used to calculate dose. Chronic adverse health effects from antimony are not anticipated.

Copper, arsenic, and lead levels were also elevated in the “first draw” drinking water sample at this location. The elevated antimony re-enforces the importance of adequately purging the water system and/or installation of a filtration system at this location.

#### *Di(2-ethylhexyl) phthalate*

Di(2-ethylhexyl) phthalate (DEHP) is a man made chemical that is added to plastics to enhance pliability. It is found in a variety of products including hoses, wall covering, upholstery, rainwear, shower curtains and toys. DEHP is a wide-spread environmental contaminant and has been found at almost half of the EPA’s National Priority List Sites. It is also a contaminant sometimes associated with sample collection and analytical processes because of its presence in PVC gloves and a variety of other materials.

Little data is available on the long term impact of DEHP on humans. Animal studies of long-term exposures have shown that high oral doses of DEHP caused adverse health effects in the liver and testes. Toxicity of DEHP in other tissues is less well characterized, although effects in the thyroid, ovaries, kidneys, and blood have been reported in a few animal studies [ATSDR DEHP 2002].

The U. S. Department of Health and Human Services (DHHS) has determined that DEHP may reasonably be anticipated to be a human carcinogen. The EPA has determined that DEHP is a probable human carcinogen. These determinations were based on the development of liver cancer in rats and mice. The International Agency for Research on Cancer (IARC) has stated that the study data is not adequate to determine if DEHP causes cancer in humans.

The DEHP levels measured in 17 drinking water samples exceeded EPA’s Maximum Contaminant Level (MCL). The calculated exposure dose using the highest concentration of DEHP did not exceed EPA’s chronic oral exposure reference dose. Therefore, non-cancer adverse health effects are not anticipated. However, 24 samples did exceed ATSDRs Cancer Risk Evaluation Guide. DEHP was detected in only one quarterly drinking water sample at twenty two of the twenty three wells where DEHP levels exceeded the comparison value. Sampling methods or analytical processes may have contributed to the number of samples with elevated DEHP levels.

The highest DEHP drinking water concentration was used to estimate the cancer risk. The cancer risk for children exposed from birth to 21 years of age was 5 to 13 excess cancers per 1,000,000 children exposed. The adult cancer risk is 7 to 16 excess cancers per 1 million adults exposed to DEHP. This is considered a “low to very low” cancer risk. The actual risk is lower because DEHP was not consistently detected in drinking water samples at any of the sample locations

#### *Pentachlorophenol*

Pentachlorophenol is a man made chemical that was used for wood treatment. It was also used as an herbicide and pesticide. Long-term exposure to low levels such as those that occur in the workplace can cause damage to the liver, kidneys, blood, and nervous system. Animal studies suggest that the endocrine (hormone) system and immune system can also be damaged following long-term exposure to low levels of pentachlorophenol. Decreases in the number of newborn animals, harmful effects on



reproductive organs of the mothers, decreases in the number of successful pregnancies, and increases in the length of pregnancy were observed in animals exposed to pentachlorophenol while they were pregnant.

An increased risk of cancer has been shown in some laboratory animals given large amounts of pentachlorophenol orally for a long time. EPA has classified pentachlorophenol as likely to be carcinogenic to humans. This is based on epidemiology studies of workers in the sawmill industry and animal studies. The International Agency for Research on Cancer (IARC) has determined that pentachlorophenol is possibly carcinogenic to humans.

Pentachlorophenol levels measured in the drinking water wells did not exceed ATSDR's non-cancer comparison value. One sample was rejected because there was more than a 100 fold difference between the original and duplicate samples for the December 2011 sampling event. Non-cancer related adverse health effects are not anticipated. Twenty eight drinking water samples were reported with estimated concentrations of pentachlorophenol above the ATSDR's CREG. Twenty four of these drinking water samples were collected in the December 2011 quarterly sampling event. A single location had pentachlorophenol levels above the CREG more than once.

The highest pentachlorophenol concentration was used with age specific water intakes rates to calculate the cancer risk. The excess cancer risk for children exposed from birth to 21 years was 9 to 22 excess cancers per 1 million children exposed. This is considered a "low" cancer risk. The adult cancer risk is 9 to 27 cases per 1 million adults exposed to pentachlorophenol. This is also considered a "low" cancer risk. The actual cancer risk is likely lower because of the inconsistent pattern of detections.

#### *1,4 Dioxane*

1,4-dioxane is a chemical used as a solvent, laboratory reagent, and chemical intermediary. Exposure to 1,4-dioxane can occur by inhalation, ingestion, or absorption through the skin. Human and animal exposure data identify the target organs for adverse effects as the liver and kidneys. There is limited data available on the carcinogenicity of 1,4-dioxane in humans [ATSDR 1,4-Dioxane 2007]. However, animal studies have demonstrated ingestion of 1,4-dioxane can cause cancer in animals. Based on the animal (ingestion) data EPA considers 1,4-dioxane a probable human carcinogen. IARC lists 1,4-dioxane as a possible human carcinogen. An increase in the number of cancers was not observed for inhalation exposures in occupational or animal studies. EPA has determined that current inhalation studies are not adequate to assess the cancer risk from inhalation.

The 1,4 dioxane levels measured in the drinking water wells did not exceed the (non-cancer) comparison value. However, the level estimated in one drinking water well did exceed ATSDR's CREG. Although the presence of 1,4 dioxane was confirmed, the concentration was estimated in the sample. The cancer risk estimated for the highest concentration of 1,4-dioxane was less than 1 cancer per 1 million people exposed for both children and adults. This is considered "no" increased risk for cancer.

#### *Multiple Chemical Exposures*

The presence of multiple contaminants in drinking water may increase residents' overall risk of adverse health effects if the contaminants target the same organ or the interaction between the contaminants increases the toxicity of the mixture. The type, number, and concentration of contaminants varied dramatically between drinking water wells during the 2 year sampling period. The data were also

inconsistent between sampling events at most individual wells. Three contaminants of concern (pentachlorophenol, DEHP, and 1, 4-dioxane) can adversely impact the liver. The contaminants were not identified in the same drinking water sample. To be health protective, the highest concentration of each contaminant and average water consumption rates were used to calculate the additive effect of the contaminants. The hazard index was less than 1 which means adverse (non-cancer) liver effects are not expected.

## **Child Health Considerations**

Children can be at greater risk of developing illnesses from exposure to some hazardous substances. The reasons include their smaller height, lower body weight, and developing body systems. Children are typically more active than adults and may breathe more air and drink more water per body weight than adults. The nature of children's play/activities may also increase the risk of exposure to contaminated soil, dust, or surface water.

Children are more vulnerable than adults to lead poisoning. High levels of lead exposure may increase the risk of children developing anemia, kidney damage, colic, muscle weakness, and brain damage. Lower levels of lead exposure may affect development and behavior, or a child's cognitive abilities and physical growth. Children between the ages of six months to six years are in the greatest danger for lead poisoning. The most accurate way to determine the amount of lead exposure is to have children's blood tested.

Some infant and animal studies indicate that manganese absorption is higher for children than adults. Animal studies also suggest the distribution of manganese within the body may differ between children and adults. However, there is not adequate information to determine if children are more sensitive to the adverse effects of manganese.

Pre- and post-natal exposure to polycyclic aromatic hydrocarbons (PAHs) can produce adverse reproductive and developmental effects in human fetuses. Fetuses are susceptible to the toxic effects produced by maternal exposure to PAHs because of an increased permeability in the blood-brain barrier and a decrease in liver function [ATSDR PAH 1995]. Animal studies have shown an increase in the incidences of fetus death and abnormalities of the kidney and bladder. Animal studies have also shown delayed effects in the offspring including sterility, depressed immune system, changes to the endocrine system, and cancer.

## **Data Limitations**

The presence of arsenic was confirmed but the concentration was an estimate in some drinking water samples and a laboratory quality control blank. The estimated concentration exceeded ATSDR's cancer risk evaluation guide. In addition, PAHs were detected in the field blanks for one of the quarterly sampling events. These issues impacted our ability to characterize the health risks.

Variations in the type and concentration of contaminants occurred in drinking water samples over the 2 year sampling period. In many instances, the contaminant levels exceeded comparison values in one or

two of the quarterly samples. The drinking water wells are geographically dispersed, the hydrology of the area is complex, and the type/levels of contaminants varied with drinking water well locations.

To protect public health, the health risks were based on the highest concentration of a contaminant measured. If the contaminant concentrations used in this evaluation are significantly higher than the actual exposure or the water was consumed for significantly less than 33 years, the health risks may be overestimated. Conversely, if the actual time of exposure was longer or the contaminant concentration was higher, the health risks described in this report could be underestimated.

A second limitation is that some residents installed water filtration units. EPA collected drinking water samples at the well head whenever possible. If the well head was not accessible the drinking water sample was collected from the closest tap. If EPA was aware of the presence of a filtration system, they collected drinking water samples before and after the filtration units. Little or no information was available on the type, date of installation, or efficiency of the home owner installed systems. Properly selected and maintained filtration systems could reduce the actual exposure levels.

## Conclusions

EPA and their contractors collected more than 800 private drinking water samples from more than 100 homes near the CTS site in 2010 and 2011. DPH evaluated the data and reached the following conclusions:

- The trichloroethylene (TCE) levels in one well were consistently above ATSDR's drinking water comparison values. The drinking water well at this location was disconnected in 2009 and the home was connected to municipal water. The TCE exposure at this location was addressed in the previous health consultation. Residents were not in contact with contaminated water during the 2 year sampling period covered in this report.
- Elevated lead levels were identified in drinking water at 7 locations. Six of these locations had multiple quarter drinking water samples with elevated lead levels. The total blood lead level calculated exceeded 5 µg/dL for children at 6 drinking water wells. Children with long term exposure to lead from drinking water from 6 wells could experience adverse health effects.
- Children drinking water from 5 locations with elevated copper levels could have experienced temporary gastrointestinal effects. Copper levels were elevated in multiple quarterly drinking water samples at each location.
- Data are not adequate to determine if drinking well water containing arsenic at one location could cause non-cancer related adverse health effects. The highest exposure dose exceeded the chronic MRL. Arsenic was present in 4 of the 5 drinking water samples collected in 2010. Arsenic was not detected in the drinking water samples collected at this location in 2007, 2008, 2009, or 2011. Data are not adequate to conclude if drinking well water containing arsenic could cause cancer.
- Data are not adequate to conclude if polycyclic aromatic hydrocarbons (PAHs) present in drinking water could cause adverse health effects. The excess cancer risk based on the highest benzo(a)pyrene equivalent level is considered a "moderate" risk. However, PAHs were not consistently present above screening levels in the quarterly sampling data. The uncertainty in concentration, length of exposure, and presence of contaminants in field blanks impact our ability to accurately characterize the risk.

- Data are not adequate to conclude if cyanide that was present in drinking water could cause adverse health effects. The exposure dose exceeded the minimal risk level for children and adults. However, cyanide was not consistently present in drinking water samples and cyanide was identified in laboratory blanks.
- Adverse (non-cancer) health effects are not expected for people drinking water from wells containing 1,4-dioxane, pentachlorophenol, or di-ethylhexyl phthalate (DEHP). The exposure dose calculated using the highest exposure level was less than the MRL for each chemical.
- No increase in the risk of cancer is anticipated for 1,4-dioxane, pentachlorophenol, and di-ethylhexyl phthalate (DEHP).
- Adverse health impacts are not anticipated for people drinking water from wells that contained antimony, manganese, or selenium. In each case, only 1 drinking water sample exceeded the comparison value. In addition, the exposure dose was less than the Minimal Risk Level (MRL) for all age categories when average water consumption rates were used in the calculations.

EPA collected 4 stream sediment samples and 7 surface water samples in 2010. DPH evaluated the data and reached the following conclusions:

- Incidental exposure of children playing in the springs or creek near the Oaks neighborhood is not expected to cause adverse health effects.
- Incidental exposure of children playing in the springs or retention pond near Mills Gap Road is not expected to cause non-cancer adverse health effects.
- The excess cancer risk for incidental exposure of children playing in the springs on the west side of Mills Gap Road is 1 case per hundred thousand people exposed. This is considered a low risk. The samples were collected inside a fenced area. No increased cancer risk for incidental exposure is anticipated outside the fenced area.

## Recommendations

- The DPH should inform persons that lived at the residences served by the 2 wells with elevated copper of the potential health effects, especially those to children, and provide recommendations to follow-up with their personal physicians.
- The DPH and local health department should inform persons that live at the residences with lead detections greater than the health guideline value (15 µg/L) of the potential health effects to children, provide a contact for blood lead testing, and provide recommendations for follow-up with their personal physicians.
- The DPH should inform persons that live at the residence with elevated arsenic levels of the potential health effects and provide recommendations to follow-up with their physician.
- The DPH should inform persons that live at the residences with elevated antimony, manganese, and selenium of the potential health effects associated with the contaminants. The DPH will provide additional guidance if children are present and potentially exposed to the contaminants.
- The DPH and local health department should provide assistance to residents exposed to the elevated copper and lead to determine if the water pipes are a possible source. Provide information on alternatives for reducing their exposure and the importance of flushing the water lines for several minutes prior to collecting water to be used for drinking or cooking if other alternatives are not implemented.

- The DPH and EPA should inform residents with filtration systems if contaminant breakthrough was observed in post-filtration samples. Provide information to residents on proper selection and maintenance of water filtration systems.
- Residents concerned about metal and/or chemical exposures should consider the installation of a whole house filtration system until a permanent solution can be implemented. The cost of installation and maintenance of the system for residents within a 1 mile radius of the site will be borne by CTS Corp.

## **Public Health Action Plan**

The purpose of the Public Health Action Plan (PHAP) is to ensure that this Public Health Assessment provides a plan of action designed to mitigate or prevent potential adverse health effects.

- The EPA is conducting a Remedial Investigation and Feasibility Study at the site to identify and evaluate remediation alternatives.
- The DPH will contact persons using wells that could present possible adverse health effects related to elevated copper, lead, and arsenic exposures. We will provide information on possible health effects, ways to reduce exposures, and guidance on appropriate medical follow-up.
- The DPH will contact persons using filtration systems if contaminant breakthrough was identified in the post-filter samples. We will provide information on selection and maintenance of water filtration units.
- A summary factsheet for the HC will be prepared by DPH and be made available to the public and government agencies. Print copies will be available at Buncombe County locations selected as document repositories and electronic copies will be available from the HACE web site.
- The DPH will prepare a fact sheet for health hazards associated with the site contamination and make the fact sheet available to the community through the local health department and HACE web site.

## **REPORT PREPARATION**

This Health Consultation for the CTS/Mills Gap Road NPL Site was prepared by the North Carolina Department of Public Health under a cooperative agreement with the federal Agency for Toxic Substances and Disease Registry (ATSDR). It is in accordance with the approved agency methods, policies, procedures existing at the date of publication. Editorial review was completed by the cooperative agreement partner. ATSDR has reviewed this document and concurs with its findings based on the information presented.

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## **Appendix A**

### Figures

Figure 1: One Mile Radius of CTS Site

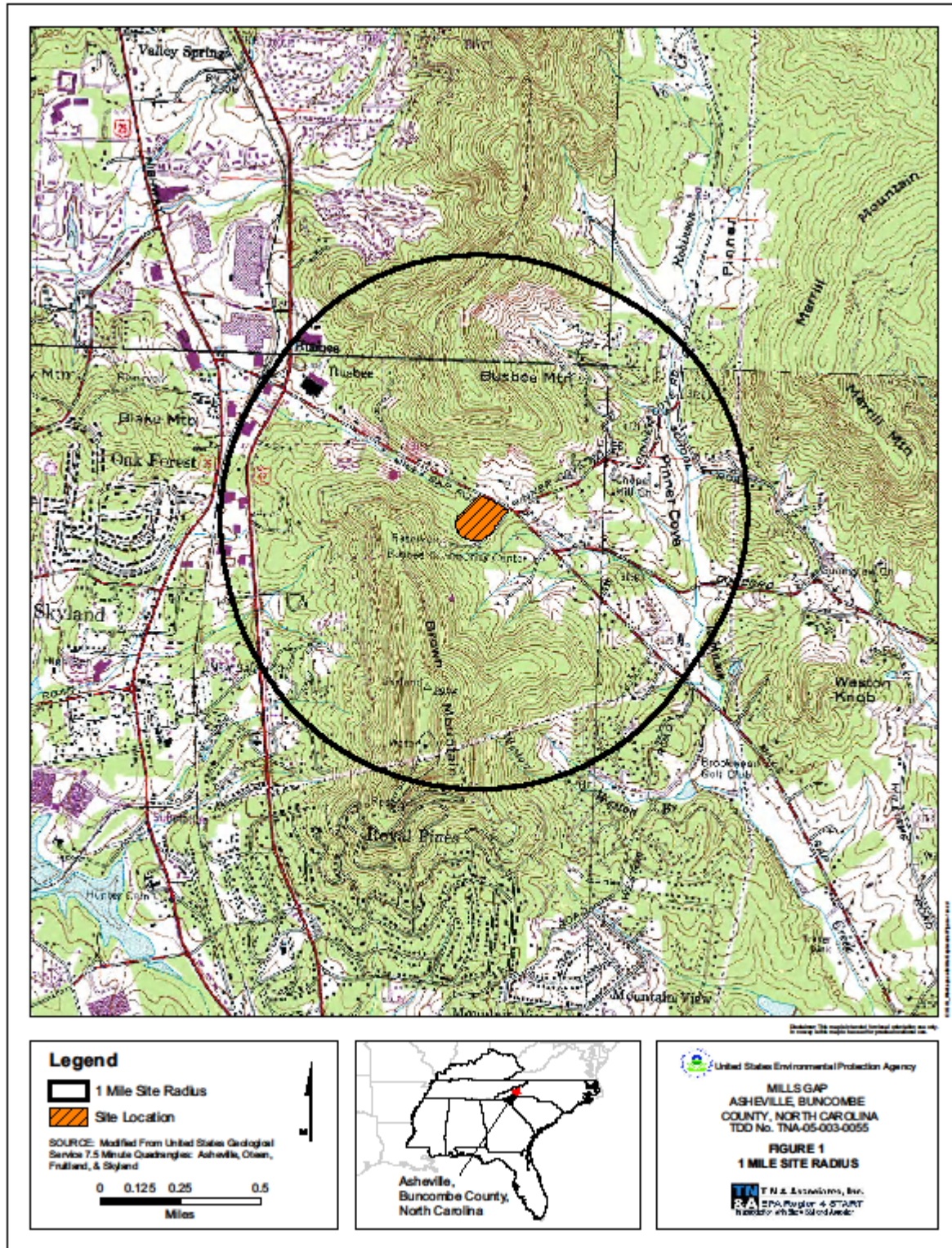
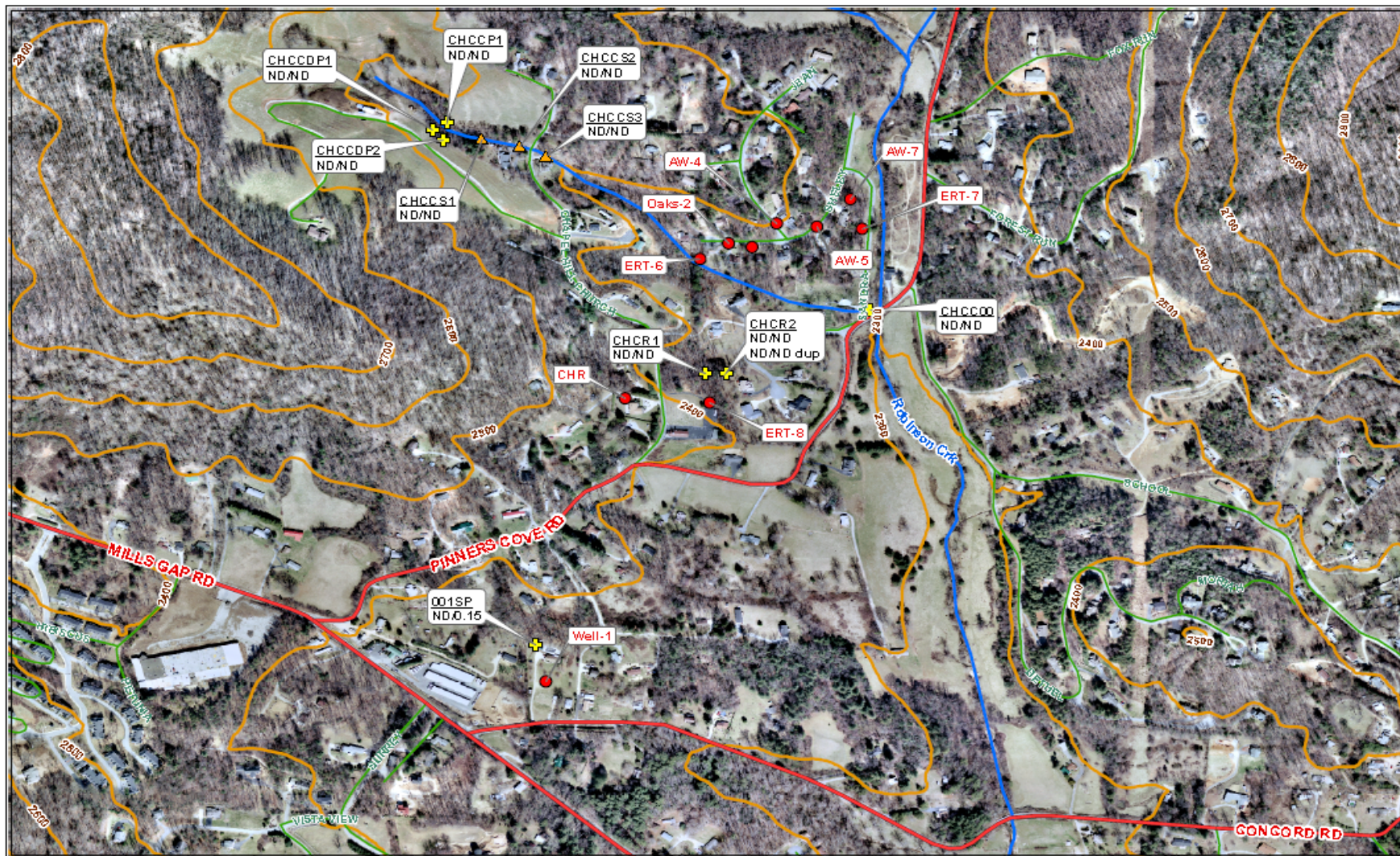


Figure 2: Spring #2 and Spring #4



Figure 3: Surface Water Sample Locations



Map created using 2006 high resolution (0.25 feet) color orthoimagery supplied by North Carolina State, contour line derived from DEM data.

Map Creation Date: 23 August 2010

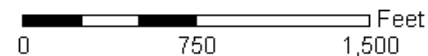
Coordinate system: North Carolina State Plane  
 FIPS: 3200  
 Datum: NAD83  
 Units: Feet

**Legend**

- + Surface Water Sample Location
- ▲ Stream Sediment Sample Location
- Monitor Well Locations

Note:

ND = Not Detected  
 TCE/Toluene Concentration in µg/L

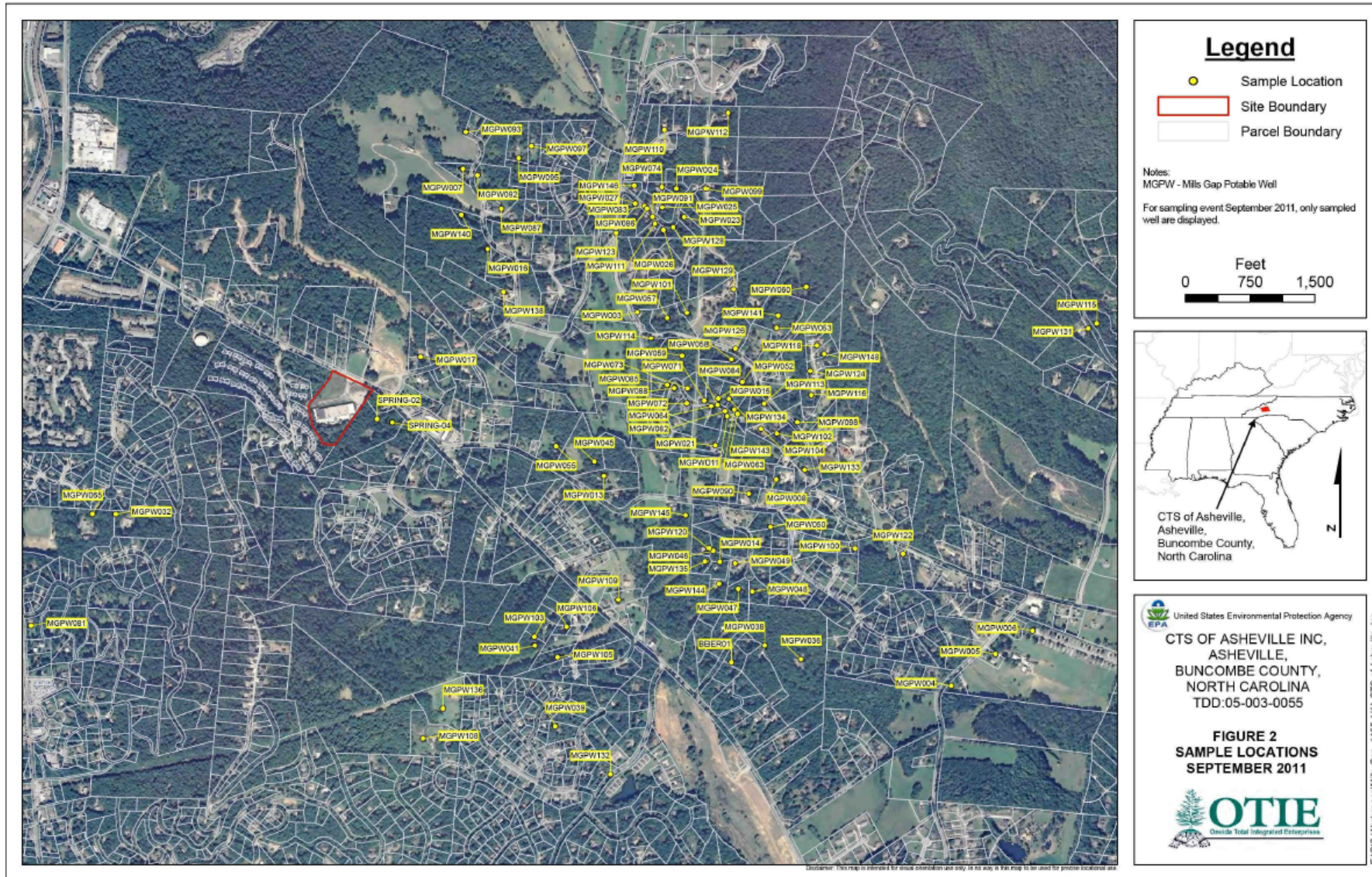


U.S. EPA Environmental Response Team  
 Scientific Engineering Response and Analytical Services  
 EP-W-09-031  
 W.A.# 0-052

Figure 3  
 Surface Sample Location  
 Mills Gap Road  
 Asheville, North Carolina

data: g:\arcview\projects\ERAG01\00-052  
 \MD file: g:\arcinfo\projects\ERAG01\ERAG0052 Mills Gap Road\052 SurfaceWater\_SampleLocation f3rev002

Figure 4: Drinking Water Sample Locations



Appendix B  
Demographic Data



## EJView Census 2010 Summary Report



Location: -82.506402,35.493067

Study Area: 1 mile around the point location

Summary	Census 2010
Population	3,418
Population Density (per sq. mile)	1,413
Minority Population	428
% Minority	13%
Households	1,725
Housing Units	1,931
Land Area (m <sup>2</sup> )	6,263,645
% Land Area	100%
Water Area (m <sup>2</sup> )	0
% Water Area	0%

Population by Race	Number	Percent
Total	3,418	-----
Population Reporting One Race	3,340	98%
White	3,042	89%
Black	200	6%
American Indian	9	0%
Asian	55	2%
Pacific Islander	0	0%
Some Other Race	34	1%
Population Reporting Two or More Races	78	2%
Total Hispanic Population	92	3%
Total Non-Hispanic Population	3,326	97%
White Alone	2,990	87%
Black Alone	198	6%
American Indian Alone	7	0%
Non-Hispanic Asian Alone	55	2%
Pacific Islander Alone	0	0%
Other Race Alone	8	0%
Two or More Races Alone	67	2%

Population by Sex	Number	Percent
Male	1,576	46%
Female	1,842	54%

Population by Age	Number	Percent
Age 0-4	160	5%
Age 0-17	633	19%
Age 18+	2,785	81%
Age 65+	655	19%

Households by Tenure	Number	Percent
Total	1,725	
Owner Occupied	1,010	59%
Renter Occupied	714	41%

Data Note: Detail may not sum to totals dues to rounding. Hispanic population can be of any race.

Source: U.S. Census Bureau, Census 2010 Summary File 1.



## EJView ACS Summary Report



Location: -82.506402,35.493067

Study Area: 1 mile around the point location

	2006 - 2010 ACS Estimates	Percent	MOE (±)
<b>Population 25+ by Educational Attainment</b>			
Total	2,448	100%	250
Less than 9th Grade	12	0%	132
9th - 12th Grade, No Diploma	69	3%	149
High School Graduate	591	24%	156
Some College, No Degree	748	31%	213
Associate Degree	318	13%	96
Bachelor's Degree or more	1,029	42%	201
<b>POPULATION AGE 5+ YEARS BY ABILITY TO SPEAK ENGLISH</b>			
Total	3,168	100%	414
Speak only English	3,100	98%	438
Non-English at Home <sup>1+2+3+4</sup>	69	2%	212
<sup>1</sup> Speak English "very well"	41	1%	149
<sup>2</sup> Speak English "well"	22	1%	172
<sup>3</sup> Speak English "not well"	6	0%	138
<sup>4</sup> Speak English "not at all"	0	0%	127
<sup>3+4</sup> Speak English "less than well"	6	0%	138
<sup>2+3+4</sup> Speak English "less than very well"	28	1%	181
<b>POPULATION AGE 5+ YEARS BY LANGUAGE SPOKEN AT HOME</b>			
Total	N/A	N/A	N/A
Speak only English	N/A	N/A	N/A
Non-English Speaking	N/A	N/A	N/A
<b>Population by Place of Birth for the Foreign-Born</b>			
Total	N/A	N/A	N/A
Europe	N/A	N/A	N/A
Asia	N/A	N/A	N/A
Africa	N/A	N/A	N/A
Oceania	N/A	N/A	N/A
Americas	N/A	N/A	N/A
<b>Households by Household Income in 1999</b>			
Household Income Base	1,705	100%	171
< \$15,000	235	14%	134
\$15,000 - \$25,000	142	8%	82
\$25,000 - \$50,000	423	25%	164
\$50,000 - \$75,000	381	22%	98
\$75,000 +	524	31%	181
<b>Occupied Housing Units by Tenure</b>			
Total	1,705	100%	171
Owner Occupied	983	58%	128
Renter Occupied	721	42%	166

**Data Note:** Detail may not sum to totals dues to rounding. Hispanic population can be of any race. N/A means not available.

**2006-2010 ACS 5-year Estimates:** The American Community Survey (ACS) summary files provide nation-wide population and housing characteristic data at all Census summary levels down to the Block Group level. This data was collected between January 1, 2006 and December 31, 2010. ACS replaces the decennial census sample data, and is not the 2010 Census population counts data. (<http://www.census.gov/acs/www/#fragment-3>)

**Margin of error (MOE):** The MOE provides a measure of the uncertainty in the estimate due to sampling error in the ACS survey. Applying the MOE value yields the confidence interval for the estimate. For example, an estimate value of 50 and +/- MOE of 5 means the true value is between 45 and 55 with a 90 percent certainty ([http://www.census.gov/acs/www/Downloads/data\\_documentation/Accuracy/MultiyearACSAccuracyofData2010.pdf](http://www.census.gov/acs/www/Downloads/data_documentation/Accuracy/MultiyearACSAccuracyofData2010.pdf)). Maximum MOE is shown for each value within study area.

**Source:** U.S. Census Bureau, American Community Survey (ACS) 2006 - 2010.



## Appendix C

### Tables

Table 1: Substances Detected in Surface Water

Substances Detected In Surface Water
Benzene
Carbon Tetrachloride
Chloroform
1,1-Dichloroethane
1,1-Dichloroethene
cis-1,2-Dichloroethene
trans-1,2-Dichloroethene
Trichloroethylene (TCE)
Tetrachloroethylene (PCE)
1,1,1-Trichloroethane
Vinyl chloride
Xylene

Table 2: Substances Detected in Drinking Water

Volatile organic compounds	Semi-volatile organic compounds	Inorganic Compounds
1,1,1-Trichloroethane	1,4-Dioxane	Aluminum
1,1-Dichloroethane	2-chlorophenol	Antimony
1,1,2-Trichloro-1,2,2-Trifluoroethane	2-Methylnaphthalene	Arsenic
1,2,4-Trichlorobenzene	4-chloro-3-methylphenol	Barium
Acetone	Acenaphthene	Beryllium
Bromodichloromethane	Acenaphthylene	Cadmium
Bromoform	Acetophenone	Calcium
Bromomethane	Antracene	Chromium
Carbon disulfide	Benzo(a)anthracene	Cobalt
Chloroform	Benzo(a)pyrene	Copper
Chloromethane	Benzo(b)fluoranthene	Cyanide
cis-1,2-Dichloroethene	Benzo(ghi)perylene	Iron
cis-1,3-Dichloropropene	Benzo(k)fluoranthene	Lead
Dibromochloromethane	Bis(2-ethylhexyl) phthalate	Magnesium
Dibromodifluoromethane (Freon 112)	Caprolactam	Manganese
Ethyl Benzene	Carbazole	Nickel
Methyl t-butyl ether (MTBE)	Chrysene	Potassium
Methylene Chloride	Dibenzo(ah)anthracene	Selenium
Tetrachloroethene (PCE)	Di-ethylhexyl phthalate	Silver
Toluene	Fluoranthene	Sodium
trans-1,2-Dichloroethene	Fluorene	Thallium
Trichloroethylene (TCE)	Indeno (1,2,3-cd) pyrene	Vanadium
Xylene	Naphthalene	Zinc
	Pentachlorophenol	
	Phenanthrene	
	Pyrene	

Table 3: Chemical/Metal Detected Above Comparison Value

Private Well	Feb 2010	June 2010	Sept 2010	Dec 2010	March 2011	June 2011	Sept 2011	Dec 2011
PW-001								
PW-002								
PW-003	X							X
PW-004								X
PW-005								
PW-006		X						
PW-007		X						X
PW-008								
PW-009								
PW-010	X			X				
PW-011								
PW-012								
PW-013								
PW-014		X	X	X	X	X		X
PW-015								
PW-016	X	X						
PW-017								X
PW-018								
PW-019								
PW-020								X
PW-021			X					
PW-022				X				
PW-023	X	X						
PW-024								X
PW-025								
PW-026								
PW-027					X			
PW-028		X						
PW-029		X						X
PW-030		X						
PW-031								
PW-032								X
PW-033					X			
PW-034								
PW-035			X					
PW-036								
PW-037								
PW-038								
PW-039								
PW-040		X						
PW-041		X						

Private Well	Feb 2010	June 2010	Sept 2010	Dec 2010	March 2011	June 2011	Sept 2011	Dec 2011
PW-042			X					
PW-043								
PW-044								
PW-045								
PW-046		X					X	
PW-047	X							
PW-048								
PW-049								
PW-050		X						
PW-051								
PW-052								
PW-053								
PW-054								
PW-055								
PW-056								
PW-057		X	X					
PW-058								
PW-059								X
PW-060		X						X
PW-061								X
PW-062								X
PW-063	X	X	X					
PW-064								
PW-065								
PW-066								X
PW-067		X						
PW-068								
PW-069			X		X			
PW-070		X						X
PW-071								
PW-072								
PW-073								
PW-074	X						X	
PW-075								
PW-076		X			X			X
PW-077								
PW-078		X						X
PW-079	X	X						X
PW-080	X	X	X	X				
PW-081				X				
PW-082		X						
PW-083								X
PW-084								

Private Well	Feb 2010	June 2010	Sept 2010	Dec 2010	March 2011	June 2011	Sept 2011	Dec 2011
PW-085				X				
PW-086								X
PW-087		X						X
PW-088		X						X
PW-089				X				X
PW-090								
PW-091								
PW-092								
PW-093								X
PW-094		X		X				
PW-095								X
PW-096								X
PW-097				X	X	X	X	
PW-098								
PW-099								X
PW-100								
PW-101	X	X	X		X	X		
PW-102				X				X
PW-103	X	X			X			
PW-104		X						

Note: ATSDR Comparison Values are lower than EPA screening levels for some compounds

Table 4: Data Summary for Drinking Water Contaminants of Concern

Contaminant	Highest Conc. µg/L	Samples > CV	Screening Value µg/L	CV Source
Polycyclic aromatic hydrocarbons (BAP eq)	1.71	14 6	0.005 0.2	CREG <sup>1</sup> MCL <sup>2</sup>
Pentachlorophenol	4.4J	27 0	0.09 10child/40 adult	CREG Chr. EMEG <sup>3</sup>
Di-ethylhexyl phthalate (DEHP)	75	24	2	CREG
1,4 Dioxane	0.36J	1 0	0.3 1000 child	CREG Chr. EMEG
Antimony	3.9	1	4 child/10 adult	RMEG <sup>4</sup>
Arsenic	17	24 2	0.02 3 child/10 adult	CREG EMEG
Copper	3300 <sup>5</sup>	15	100 child 400 adult	Int. EMEG <sup>6</sup>
Cyanide	32J	42	6 child/21 adult	RMEG
Lead	800 <sup>5</sup>	14	15	MCL
Manganese	540	1	300	LTHA <sup>7</sup>
Selenium	77	1	50 child/200 adult	Chr. EMEG

<sup>1</sup> Cancer Risk Evaluation Guide

<sup>2</sup> EPA Maximum Contaminant Level

<sup>3</sup> Chronic Environmental Media Evaluation Guide

<sup>4</sup> EPA Reference Dose Media Evaluation Guide

<sup>5</sup> First Draw Sample

<sup>6</sup> Intermediate Environmental Media Evaluation Guide

<sup>7</sup> Lifetime Health Advisory for Drinking Water

Table 5: Data Summary for Surface Water Contaminants of Concern

Contaminant	Highest Conc. µg/L	Samples > CV	Screening Value µg/L	CV Source
Benzene	29J	1 3	5 child/18 adult 0.64	Chr. EMEG <sup>1</sup> CREG <sup>2</sup>
1,1-Dichloroethane	8J	0 1	3,100 2.4	RSL <sup>3</sup> RSL cancer
Cis-1,2-Dichloroethene	2700	6	10	LTHA <sup>4</sup>
1,1,1-Trichloroethane	580	2	200	LTHA
Trichloroethylene	7,800	4 4	5 child/18 adult 0.76	Chr. EMEG CREG
Vinyl chloride	1.1	0 1	30 child/110 adult 0.025	Chr. EMEG CREG

<sup>1</sup> Chronic Environmental Media Evaluation Guide

<sup>2</sup> Cancer Risk Evaluation Guide

<sup>3</sup> Regional Screening Level

<sup>4</sup> Lifetime Health Advisory for Drinking Water

Table 6: Summary of Cancer Risk from Drinking Water

Contaminant	Highest Value mg/L	Slope Factor	Child		Adult	
			CTE <sup>1</sup> Cancers per people exposed	RME <sup>2</sup> Cancers per people exposed	CTE Cancers per people exposed	RME Cancers per people exposed
Polycyclic Aromatic Hydrocarbons (PAHs) <sup>3</sup>	0.00171	7.3	3/10,000	7/10,000	8/100,000	20/100,000
Pentachlorophenol	0.0044	0.4	9/1,000,000	22/1,000,000	9/1,000,000	27/1,000,000
Di-ethylhexyl phthalate (DEHP)	0.075	0.014	5/1,000,000	13/1,000,000	7/1,000,000	16/1,000,000
1,4-Dioxane	0.00036	0.1	<1/1,000,000	<1/1,000,000	<1/1,000,000	<1/1,000,000
Arsenic	0.017	1.5	1/10,000	3/10,000	2/10,000	4/10,000

<sup>1</sup> Central Tendency Exposure Water Intake Rates

<sup>2</sup> Reasonable Maximum Exposure (95<sup>th</sup> percentile) Water Intake Rates

<sup>3</sup> Cancer Risk with Age Dependant Adjustment Factors

Table 7: Summary of Cancer Risk from Incidental Ingestion of Springs

Contaminant	Highest Exposure mg/kg/day	Slope Factor	Estimated Risk Cancer per people exposed
Benzene	0.029	0.05-0.055	<1/1,000,000
1,1-Dichloroethane	8J <sup>1</sup>	0.0057	<1/1,000,000
Trichloroethylene <sup>2</sup> (TCE)	7,800	0.05	1/100,000
Vinyl chloride <sup>2</sup>	0.0011	1.4	<1/1,000,000

<sup>1</sup> Estimated Concentration

<sup>2</sup> Age Dependent Adjustment Factors Were Applied Because of Increased Early Life Susceptibility.

## Appendix D

### The ATSDR Health Effects Evaluation Process



## The ATSDR Health Effects Evaluation Process

The ATSDR health effects evaluation process consists of two steps: a screening analysis, and at some sites, based on the results of the screening analysis and community health concerns, a more in-depth analysis to determine possible public health implications of site-specific exposure estimates.

In evaluating data, ATSDR uses comparison values (CVs) to determine which chemicals to examine more closely. CVs are the contaminant concentrations found in a specific medium (soil, water, or air) and are used to select contaminants for further evaluation. CVs incorporate assumptions of daily exposure to the chemical and a standard amount of air, water and soil that someone may inhale or ingest each day.

The two step screening analysis process provides a consistent means to identify site contaminants that need to be evaluated more closely through the use of “comparison values” (CVs). The first step of the screening analysis is the “environmental guideline comparison” which involves comparing site contaminant concentrations to medium-specific comparison values derived by ATSDR from standard exposure default values. The second step is the “health guideline comparison” and involves looking more closely at site-specific exposure conditions, estimating exposure doses, and comparing them to dose-based health-effect comparison values.

As health-based thresholds, CVs are set at a concentration below which no known or anticipated adverse human health effects are expected to occur. CVs are not thresholds of toxicity and do not predict adverse health effects. CVs serve only as guidelines to provide an initial screen of human exposure to substances. Contaminant concentrations at or below the relevant CV may reasonably be considered safe, but it does not automatically follow that any environmental concentration that exceeds a CV would be expected to produce adverse health effects. Different CVs are developed for cancer and non-cancer health effects. Non-cancer levels are based on validated toxicological studies for a chemical, with appropriate safety factors included, and the assumption that small children (22 pounds) and adults are exposed every day. Cancer levels are the media concentrations at which there could be a one additional cancer in a one million person population (one in a million excess cancer risk for an adult) eating contaminated soil or drinking contaminated water every day for 70 years. For chemicals for which both cancer and non-cancer CVs exist, the lower level is used to be protective. Exceeding a CV does not mean that health effects will occur, just that more evaluation is needed.

After completing a screening analysis, site contaminants are divided into two categories. Those not exceeding CVs usually require no further analysis, and those exceeding CVs are selected for a more in-depth analysis to evaluate the likelihood of possible harmful effects.

The North Carolina Department of Public Health (N.C. DPH) uses the following screening values for public health assessments:

1. **Environmental Media Evaluation Guide (EMEG):** EMEGs are estimated contaminant concentrations in water, soil or air to which humans may be exposed over specified time periods and are not expected to result in adverse non-cancer health effects. EMEGs are based on ATSDR “minimum risk levels” (MRLs) and conservative (highly health protective) assumptions about exposure, such as intake rate, exposure frequency and duration, and body weight.

2. **Reference Dose Media Evaluation Guides (RMEGs):** RMEGs represent concentrations of substances in water and soil to which humans may be exposed over specified time periods without experiencing non-cancer adverse health effects. The RMEG is derived from the U.S. Environmental Protection Agency's (EPA's) oral reference dose (RfD).
3. **Cancer Risk Evaluation Guide (CREG):** CREGs are estimated media-specific contaminant concentrations that would be expected to cause no more than one additional excess cancer in one million persons exposed over a 70-year lifetime. CREGs are calculated from EPA's cancer slope factors (CSFs) or inhalation unit risk (IUR) values.
4. **Maximum Contaminant Levels (MCL):** A Federal Maximum Contaminant Level (MCL) is the regulatory limit set by EPA that establishes the maximum permissible level of a contaminant in water that is deliverable to the user of a public water system. MCLs are based on health data, also taking into account economic and technical feasibility to achieve that level. (ATSDR 2005a)
5. **EPA Regional Screening Levels (RSL):** "Regional Screening Levels for Chemical Contaminants at Superfund Sites" are tables of risk-based screening levels, calculated using the latest toxicity values, default exposure assumptions and physical and chemical properties. The Regional Screening table was developed with input from EPA Regions III, VI, and IX in an effort to improve consistency and incorporate updated guidance. ([http://www.epa.gov/reg3hwmd/risk/human/rb-concentration\\_table/index.htm](http://www.epa.gov/reg3hwmd/risk/human/rb-concentration_table/index.htm))

Contaminant concentrations exceeding the appropriate CVs are further evaluated against ATSDR health guidelines. N.C. DPH also retains for further assessment contaminants that are known or suspected to be cancer-causing agents. To determine exposure dose, N.C. DHHS uses standard assumptions about body weight, ingestion or inhalation rates, and duration of exposure. Important factors in determining the potential for adverse health effects also include the concentration of the chemical, the duration of exposure, the route of exposure, and the health status of those exposed. Site contaminant concentrations and site-specific exposure conditions are used to make conservative estimates of site-specific exposure doses for children and adults that are compared to ATSDR health guidelines (HGs), generally expressed as Minimal Risk Levels (MRLs). An exposure dose (generally 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 come into contact based on their actions and habits. Exposure dose calculations are based on the following assumptions as outlined by the ATSDR (ATSDR 2005a):

- Children between the ages of 1 and 6 ingest an average of 1 liter of water per day
- Children weigh an average of 15 kilograms
- Infants weigh an average of 10 kilograms
- Adults ingest an average of 2 liters of water per day
- Adults weigh an average of 70 kilograms

### **Ingestion of contaminants present in drinking water**

Exposure doses for ingestion of contaminants present in groundwater are calculated using the maximum and average detected concentrations of contaminants in milligrams per liter (mg/L = ppm). The

following equation is used to estimate the exposure doses resulting from ingestion of contaminated groundwater:

$$ED_w = \frac{C \times IR \times AF \times EF}{BW}$$

Where:

- ED<sub>w</sub> = exposure dose water (mg/kg/day)
- C = contaminant concentration (mg/L)
- IR = intake rate of contaminated medium (liters/day)
- AF = bioavailability factor (unitless)
- EF = exposure factor
- BW = body weight (kilograms)

### **Ingestion of contaminants present in soil**

Exposure doses for ingestion of contaminants present in soil are calculated using the maximum and average detected concentrations of contaminants in milligrams per kilogram (mg/kg = ppm). The following equation is used to estimate the exposure doses resulting from ingestion of contaminated soil:

$$ED_s = \frac{C \times IR \times AF \times EF}{BW}$$

Where:

- ED<sub>s</sub> = exposure dose soil (mg/kg/day)
- C = contaminant concentration (mg/kg)
- IR = intake rate of contaminated medium (kilograms/day)
- AF = bioavailability factor (unitless)
- EF = exposure factor (unitless)
- BW = body weight (kilograms)

The exposure factor is an expression of how often and how long a person may contact a substance in the environment. The exposure factor is calculated with the following general equation:

$$EF = \frac{F \times ED}{AT}$$

Where:

- F = frequency of exposure (days/year)
- ED = exposure duration (years)
- AT = averaging time (ED x 365 days/year)

### **Inhalation (breathing) of contaminants present in air**

Inhalation is an important pathway for human exposure to contaminants that exist as atmospheric gases or are adsorbed to airborne particles or fibers. Exposure doses for breathing contaminants in air were calculated using the maximum or average detected concentrations in milligrams per cubic meter ( $\text{mg}/\text{m}^3$ ) or parts per billion by volume (ppbv). The following equation is used to estimate the exposure doses resulting from inhalation of contaminated air.

$$D = (C \times IR \times EF) / BW$$

Where:

- D = exposure dose ( $\text{mg}/\text{kg}/\text{day}$ )
- C = contaminant concentration ( $\text{mg}/\text{m}^3$ )
- IR = intake rate ( $\text{m}^3/\text{day}$ )
- EF = exposure factor (unitless)
- BW = body weight (kg)

### **Calculations of Contaminant Exposures During Showering**

When showering in contaminated water a person may be exposed to the chemicals in the water by breathing a portion of the chemical that comes out of the water into the air (inhalation exposure), or by absorbing the chemical from the water through their skin (dermal exposure). Inhalation and dermal exposures to volatile organic compounds (VOCs) in the shower or bath may be equal to or greater than exposures from drinking the contaminated water. ATSDR uses conservative assumptions to estimate “worst case” exposures to VOCs during showering with contaminated water. The maximum concentration of VOC in the bathroom air is estimated with the following equation (Andelman 1990).

$$C_a = (C_w \times f \times F_w \times t) / V_a$$

Where:

- $C_a$  = bathroom air concentration ( $\text{mg}/\text{m}^3$ )
- $C_w$  = tap water concentration ( $\text{mg}/\text{L}$ )
- $f$  = fractional volatilization rate (unitless)
- $F_w$  = shower water flow rate ( $\text{L}/\text{min}$ )
- $t$  = exposure time (min)
- $V_a$  = bathroom volume ( $\text{m}^3$ )

Conservative calculation parameters are assumed, including a fractional volatilization of 0.9 for chlorinated VOCs, a flow rate of 8 L/min, and a small bathroom volume of 10  $\text{m}^3$ . Conservative calculations are also made by using the maximum concentration found for each VOC in the tap water. Calculated bathroom air concentrations of VOCs can then be compared to ATSDR inhalation comparison values. Inhalation exposure dose estimates can be made using ATSDR’s inhalation dose calculations.

Health guidelines represent daily human exposure to a substance that is likely to be without appreciable risk of adverse health effects during the specified exposure duration. The potential for adverse health effects exists under the representative exposure conditions if the estimated site-specific exposure doses exceed the health guidelines and they are retained for further evaluation. A MRL is an estimate of daily human exposure to a substance (in milligrams per kilogram per day [mg/kg/day] for oral exposures) that is likely to be without non-cancer health effects during a specified duration of exposure. Exposures are based on the assumption a person is exposed to the maximum concentration of the contaminant with a daily occurrence.

Generally, site-specific exposure doses that do not exceed screening values are dropped from further assessment. Exposure doses that exceed MRLs, or are known or suspected cancer-causing agents, are carried through to the health-effects evaluation. The health-effects evaluation includes an in-depth analysis examining and interpreting reliable substance-specific health effects data (toxicological, epidemiologic, medical, and health outcome data) related to dose-response relationships for the substance and pathways of interest. The magnitude of the public health issue may be estimated by comparing the estimated exposures to “no observed” (NOAELs) and “lowest observed” (LOAELs) adverse effect levels in animals and in humans, when available.

ATSDR’s toxicological profiles serve as the primary source of the health-effects data. Other sources of toxicological data include EPA’s Integrated Risk Information System (IRIS) database, International Agency for Research on Cancer (IARC) Monographs, and the National Toxicology Program (NTP). Standard toxicology textbooks and peer-reviewed scientific journals of environmental toxicology or environmental health can also be consulted.

### **Polynuclear Aromatic Hydrocarbons (PAHs)**

ATSDR does not provide individual comparison values (CVs) for the group of structurally related multi-carbon ring compounds known as polynuclear aromatic hydrocarbons or PAHs (PAHs may also be called “polycyclic aromatic hydrocarbons”). ATSDR does provide a CREG the PAH compound benzo(a)pyrene (BaP). BaP is the most studied of the individual chemicals of the PAH group, and is thought to be the most toxic. To evaluate potential adverse health effects associated with incidental ingestion of soil PAH concentrations, the concentrations of individual detected PAH compounds are converted to an equivalent BaP concentration and summed to provide a “BaP-equivalent” concentration for all detected PAHs. BaP-equivalent exposure dose are calculated by multiplying the concentration of individual detected PAH compounds by their “toxicity equivalency factor” (TEF), a value that relates the relative toxicity of the individual PAH compounds to the toxicity of BaP. Below is a table of TEF values used by N.C. DPH to calculate BaP-equivalent concentrations. An estimated soil ingestion BaP-equivalent exposure dose is calculated using soil exposure rates. Estimated numbers of increased cancers for the combined PAH exposure is calculated by multiplying the CREG value by the BaP-equivalent exposure dose.

$$PAH_{BaP-eq} = PAH_{conc} \times TEF$$

$$Combined\ Cancer\ Risk_{PAHs} = \sum PAH_{adj} \times CSF$$

Where:

$PAH_{BaP-eq}$  = Benzo(a)pyrene equivalent TEF adjusted PAH compound concentration, mg/kg

- $PAH_{conc}$  = concentration of PAH compound, mg/kg  
 $TEF$  = Toxicity Equivalency Factor for PAH compound, unitless  
 $Combined\ Cancer\ Risk_{PAHs}$  = Summed cancer risk of all detected PAH compounds  
 $\sum PAH_{adj}$  = summed TEF-adjusted concentrations of all detected PAH compounds, mg/kg  
 $CSF$  = Cancer Slope Factor, mg/kg-d

### PAH Toxicity Equivalency Factors (“TEFs”)

PAH compounds	TEF value
acenaphthene	0.001
acenaphthylene	0.001
anthracene	0.01
benzo(a)anthracene	0.1
benzo(a)pyrene	1.00
benzo(b,k)fluoranthene	na
benzo(g,h,i)perylene	0.01
benzo(b)fluoranthene	0.1
benzo(k)fluoranthene	0.01
chrysene	0.001
dibenzo(a,h)anthracene	1.00
fluoranthene	0.001
fluorene	0.001
indeno(1,2,3-cd)pyrene	0.1
2-methylnaphthalene	0.001
naphthalene	0.001
phenanthrene	0.001
pyrene	0.001

Source: *Toxicity equivalency factors for PAH and their applicability in shellfish pollution monitoring studies*. J Environ Monit, 2002, 4, 383-388  
 na = not available

### Cancer Health Effect Evaluations

Theoretical increased numbers of cancers are calculated for known or suspected cancer-causing contaminants using the estimated site-specific exposure dose and cancer slope factor (CSF) provided in ATSDR health guideline documents. This theoretical calculation is based on the assumption that there is no safe level of exposure to a chemical that causes cancer. However, the theoretical calculated risk is not exact and tends to overestimate the actual risk associated with exposures that may have occurred. This theoretical increased cancer risk estimate does not equal the increased number of cancer cases that will actually occur in the exposed population, but estimates a theoretical excess cancer risk expressed as the proportion of a population that may be affected by a carcinogen during a lifetime or

other selected period of exposure. For example, an estimated cancer risk of  $1 \times 10^{-4}$  predicts the probability of one additional cancer over the background number of cancers in a population of 10,000. Qualitative assessment of the predicted increased numbers of cancers is also used and represents terminology suggested by ATSDR and N.C. DPH.

The theoretical cancer risk calculation is:

$$\text{Theoretical Cancer Risk} = \text{Dose} \times \text{CSF}$$

or

$$\text{Theoretical Cancer Risk} = \text{Air Concentration} \times \text{IUR}$$

Where:

Theoretical Cancer Risk	= Expression of the cancer risk (unitless)
Dose	= Site-specific cancer dose (mg/kg/d)
Air Concentration	= Site-specific air concentration ( $\mu\text{g}/\text{m}^3$ )
CSF	= Cancer Slope Factor ( $[\text{mg}/\text{kg}/\text{d}]^{-1}$ )
IUR	= Inhalation Unit Risk ( $[\mu\text{g}/\text{m}^3]^{-1}$ )

### Age-Adjustment of Cancer Risk for Specific Chemicals

The U.S. EPA has identified a group of chemicals that cause cancer through mutagenic mechanisms and are believed to have higher levels of cancer causing potential to persons exposed early in life (before 16 years of age). Mutagenic mechanism of carcinogenicity act by causing mutations (changes) in the genetic material of cells (DNA) that lead to cancer development. EPA recommends applying age-specific adjustments (multipliers) when evaluating increased cancer risks to persons younger than 16 years for these chemicals. Chemical-specific adjustment factors are recommended when available and default adjustment factors are provided when not. The default adjustment factors are:

- a 10-fold adjustment for ages 0 - 2 years
- a 3-fold adjustment for ages 3 - <16 years
- no adjustment for 16 years and older

The current list of carcinogens that EPA has identified as acting through a mutagenic mechanism is available at: <http://www.epa.gov/oswer/riskassessment/sghandbook/chemicals.htm>

N.C. DPH will apply age-adjustment cancer risk factors for chemicals that EPA has identified as acting through a mutagenic mode of action. The age-adjusted cancer risk calculation is:

$$\begin{aligned} \text{Theoretical Cancer Risk} &= \text{Dose} \times \text{CSF} \times \text{ADAF} \\ \text{Theoretical Cancer Risk} &= \text{Air Concentration} \times \text{IUR} \times \text{ADAF} \end{aligned}$$

Where:

ADAF = age-dependent adjustment factor (cancer potency multiplier)

The N.C. Central Cancer Registry provides the following statement about cancer.

*“Although much has been learned about cancer over the past couple of decades, there is still much that is not known about the causes of cancer. What we do know is that cancer is not one disease, but a group of diseases that behave similarly. We know that different types of cancers are caused by different things. For example, cigarette smoking has been implicated in causing lung cancer, some chemical exposures are associated with leukemia, and prolonged exposure to sunlight causes some types of skin cancer. Genetic research has shown that defects in certain genes result in a much higher likelihood that a person will get cancer. What is not known is how genetic factors and exposures to cancer causing agents interact.*

*Many people do not realize how common cancers are. It is estimated that one out of every two men and one out of every three women will develop a cancer of some type during his or her lifetime. As a result, it is common to find what appear to be cancer cases clustering in neighborhoods over a period of years. This will occur in any neighborhood. As people age, their chance of getting cancer increases, and so as we look at a community, it is common to see increasing numbers of cancer cases as the people in the community age.*

*Cancers are diseases that develop over many years. As a result, it is difficult to know when any specific cancer began to develop, and consequently, what the specific factor was which caused the cancer. Because people in our society move several times during their lives, the evaluation of clusters of cancer cases is quite challenging. One can never be certain that a specific cancer was caused by something in the community in which the person currently resides. When we investigate clusters of cancer cases, we look for several things that are clues to likely associations with exposures in the community. These are:*

- 1. Groups of cases of all the same type of cancer (such as brain cancer or leukemia). Because different types of cancer are caused by different things, cases of many different types of cancer do not constitute a cluster of cases.*
- 2. Groups of cases among children, or ones with an unusual age distribution.*
- 3. Cases diagnosed during a relatively short time interval. Cases diagnosed over a span of years do not constitute a cluster of cases unless there is consistency in the type of cancer.*
- 4. Clusters of rare cancers. Because lung, breast, colon, and prostate cancers are so common, it is very difficult to find any association between them and exposures in a community.”*

N.C. DPH evaluates cancer health effects in terms of possible increased cancer risk. In North Carolina, approximately 30% of women and 50% of men (about 40% combined), will be diagnosed with cancer in their life-time from a variety of causes. This is referred to as the “background cancer risk”. The term “excess cancer risk” represents the risk on top of the background cancer risk. A “one-in-a-million” excess cancer risk (1/1,000,000 or  $10^{-6}$  cancer risk) means that if 1,000,000 people are exposed to the cancer-causing substance at a certain level every day of their life-time (considered 70 years), then one cancer above the background number of cancers may develop in those 1 million people. In numerical terms, the background number of cancers expected in 1 million people over their life-time is 400,000. If they are all exposed to the cancer-causing substance daily throughout their life-time, then 400,001 people may get cancer, instead of the expected 400,000. The expression of the estimated cancer risk is



not a prediction that cancer will occur, it represents the upper bound estimate of the probability of additional cancers, and merely suggests that there is a possibility. The actual risk may be much lower, or even no risk. For specific exposure situations N.C. DPH may use exposure periods of less than a lifetime to provide a more realistic estimation of the risks that are known or predicted to have occurred for a particular area. If information on the specifics of the exposure situations at a particular site is not known, then N.C. DPH will always use health protective values to estimate the maximum level of risk that we believe to be realistic.

**Estimates of Increased Number of Cancers Qualitative Assessment Categories Utilized by N.C. DPH**

Estimated Number of Increased Cancers <sup>a</sup>	Qualitative Increased Risk Term
< 1/1,000,000	No Increase
< 1/100,000	Very Low
< 1/10,000	Low
< 1/1,000	Moderate
< 1/100	High
> 1/100	Very High

<sup>a</sup> As number of increased cancers above typical background numbers of cancers in the stated population size. “<1/1,000,000” = less than one additional cancer in a population of 1 million persons.

**Assessment of Chemical Interactions**

To evaluate the risk for noncancerous effects in a mixture, ATSDR’s guidance manual (*Guidance Manual for the Assessment of Joint Toxic Action of Chemical Mixtures*, 2004) prescribes the calculation of a hazard quotient (HQ) for each chemical. The HQ is calculated using the following formula:

$$HQ = \text{estimated dose} \div \text{applicable health guideline}$$

Generally, whenever the HQ for a chemical exceeds 1, concern for the potential hazard of the chemical increases. Individual chemicals that have HQs less than 0.1 are considered unlikely to pose a health hazard from interactions and are eliminated from further evaluation. If all of the chemicals have HQs less than 0.1, harmful health effects are unlikely, and no further assessment of the mixture is necessary. If two or more chemicals have HQs greater than 0.1, then these chemicals are to be evaluated further as outlined below.

Since the HQ is greater than 1 for both adults and children the hazard index (HI) will be calculated. The HQ for each chemical then is used to determine the (HI) for the mixture of chemicals. An HI is the sum of the HQs and is calculated as follows:

$$HI = HQ1 + HQ2 + HQ3 + \dots HQ_n$$

The HI is used as a screening tool to indicate whether further evaluation is needed. If the HI is less than 1.0, significant additive or toxic interactions are highly unlikely, so no further evaluation is necessary. If the HI is greater than 1.0, then further evaluation is necessary, as described below.

For chemical mixtures with an HI greater than 1.0, the estimated doses of the individual chemicals are compared with their NOAELs or comparable values. If the dose of one or more of the individual chemicals is within one order of magnitude of its respective NOAEL ( $0.1 \times \text{NOAEL}$ ), then potential exists for additive or interactive effects. Under such circumstances, an in-depth mixtures evaluation should proceed as described in ATSDR's *Guidance Manual for the Assessment of Joint Action of Chemical Mixtures*.

If the estimated doses of the individual chemicals are less than 1/10 of their respective NOAELs, then significant additive or interactive effects are unlikely, and no further evaluation is necessary.

### **Limitations of the Health Evaluation Process**

Uncertainties are inherent in the public health assessment process. These uncertainties fall into the following categories: 1) the imprecision of the risk assessment process, 2) the incompleteness of the information collected and used in the assessment, and 3) the differences in opinion as to the implications of the information. These uncertainties are addressed in public health assessments by using worst-case assumptions when estimating or interpreting health risks. The health assessment calculations and screening values also incorporate safety margins. The assumptions, interpretations, and recommendations made throughout this public health assessment err in the direction of protecting public health.

### **Reference:**

(Andelman 1990). *Total Exposure of Volatile Organic Compounds in Potable Water*. In: Significance and Treatment of Volatile Organic Compounds in Water Supplies, Chapter 20. Lewis Publishers, Chelsea, MI.