Health Consultation

PRIVATE WELL WATERS FROM THE MILLS GAP ROAD AREA
SKYLAND, NORTH CAROLINA
EPA FACILITY ID: NCSFN0406988

Prepared by the
North Carolina Department of Public Health

JULY 8, 2010

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

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SUMMARY

INTRODUCTION

The N.C. Division of Public Health (DPH) understands the community’s concerns about contact with chemicals in private wells in the Mills Gap Road area in Skyland, NC. DPH’s top priority is to make sure the community near the site has the best science information available to safeguard its health.

The U.S. Environmental Protection Agency (EPA) and the N.C. Department of Environment and Natural Resources (DENR) have collected private well water samples in the area of Mills Gap Road in Skyland NC as part of the investigation of the Mills Gap Road/CTS site. Trichloroethylene (TCE) is the primary contaminant associated with the CTS site. The private wells are being sampled to determine if they have been contaminated by chemicals moving away from the former CTS site in the groundwater. DPH has evaluated the private well water analytical data for potential threats to the community’s health as part of its continuing review of the Mills Gap Road/CTS site. Private well water samples discussed in this Health Consultation were collected from June 2008 through January 2010. All detected substances were evaluated for potential health effects, regardless of possible source. The source or sources of the contaminants discussed in this Health Consultation have not been confirmed. There may be localized sources other than the CTS site.

While multiple samples were collected from some private wells, it is not known when contamination in any of the wells first appeared, and thus, when persons drinking water from those wells were first exposed to contamination. The concentration of contaminants over the full time period a well was contaminated is also not known. While this information is critical to provide the most accurate indication of potential health effects, DPH used health-protective methods to identify the possibility of adverse health effects associated with contact with the identified contaminants.

OVERVIEW

DPH has reached six important conclusions about the water from private wells.

CONCLUSION 1

DPH concludes that drinking or breathing the TCE in the well water over many years at the location discovered with 1200 micro-gram per liter (µg/L) in August 2009 could have harmed people’s health.

BASIS FOR DECISION

TCE contamination was discovered in the private well serving 2 homes in a sample collected in August 2009. The well was disconnected at that time. Residents indicated that family members had used the well for as long as 20 years. It is not known when the well was first contaminated or the TCE concentrations over the extent of the contamination period. An increased theoretical risk of cancer and non-cancer health effects is indicated if persons were using the well water over many years with a TCE concentration similar to that observed in the August 2009 sample. Drinking water contaminated with TCE and breathing TCE volatilized from the drinking water supply over many years in large amounts may
cause adverse health effects. These effects include increased risk of kidney or liver cancer; dizziness, lung irritation, impaired heart function; and nerve, kidney or liver damage. Reproductive effects such as impaired fetal growth or decreased fertility may also result. There may be an increased risk of birth defects or leukemia to children of women exposed during pregnancy.

NEXT STEPS

The DPH makes the following recommendations:

- Persons living in the homes served by this well should make their personal physicians aware of their exposure and monitor their health to detect effects related to TCE exposure. Health monitoring should include: CBC, liver and kidney function tests and urinalysis.

CONCLUSION 2

DPH cannot conclude if drinking TCE contaminated well water from 4 wells identified in the Oaks neighborhood could have harmed people’s health. It is not known how long the water was contaminated and at what concentrations over the entire time period of contamination. Harmful health effects are not indicated at the reported TCE concentrations.

BASIS FOR DECISION

Four wells were identified in 2007 and 2008 in the Oaks neighborhood with TCE contamination (TCE concentrations: 60 µg/L, 20 µg/L, 8.8 µg/L, 2.8 µg/L). All the wells were disconnected. It is not known when the wells were first contaminated or the TCE concentrations over time.

NEXT STEPS

The DPH makes the following recommendations:

- Inform persons that lived at the residences served by these wells before they were disconnected of the potential health effects and provide recommendations for follow-up with their personal physicians and continued health monitoring for effects associated with long-term TCE ingestion.

- Provide clean alternative drinking water sources to persons impacted by groundwater contamination of their private well at contaminant concentrations greater than regulatory values for public water systems or health guideline values. N.C. DPH recommends reducing exposures to known and suspected carcinogens to achieve a long-term cancer risk goal of less than 1 additional cancer in 1 million exposed persons.

- Continue to monitor groundwater contaminants moving away from the CTS site and identify points where the community may come into contact with the contaminants, such as private wells and surface waters. Determine the concentration of contaminants at these locations.

- Identify other possible sources of well water contamination in the area.

- Continue to identify and test private wells until the contamination sources and impacted areas are identified and controlled.

CONCLUSION 3

DPH cannot conclude if drinking water from a PAH (polycyclic aromatic hydrocarbons) contaminated well detected in September 2008 in the Oaks neighborhood (at 0.118 µg/L benzo(a)pyrene-
equivalent concentration) could have harmed resident’s health. It is not known how long the water was contaminated and at what concentrations over the entire time period of contamination. Harmful health effects are not indicated at the reported PAH concentrations.

**BASIS FOR DECISION**

Total PAHs at 0.118 µg/L benzo(a)pyrene-equivalent concentration were detected in a single sample collected from the well before it was closed. This concentration is less than the regulatory level for public water systems (0.2 µg/L MCL). It is not known when the well was first contaminated or the PAH concentrations over the extent of the contamination period.

**NEXT STEPS**

The DPH makes the following recommendations:

- Inform persons that lived at the residence served by this well before it was disconnected of the potential health effects and provide recommendations for follow-up with their personal physicians.

**CONCLUSION 4**

DPH cannot currently conclude if drinking well water from 2 wells with elevated copper concentrations could cause short-term health of children sensitive to low levels of copper.

**BASIS FOR DECISION**

The copper levels in these 2 wells do not exceed action levels for public water systems and long-term permanent health effects are not indicated. The health literature indicates possible temporary gastrointestinal effects at copper levels in the range observed in these 2 wells. DPH has observed these effects, but at copper levels 5 times higher. DPH is concerned that children sensitive to copper could suffer temporary effects by exposure to high levels of copper in these 2 wells if the water sits in the well delivery system for periods greater than several hours with no flushing.

**NEXT STEPS**

The DPH makes the following recommendations:

- Inform persons that lived at the residences served by these wells of the potential health effects, especially those to children, and provide recommendations for follow-up with their personal physicians.
- Identify the source of the copper. Determine if the copper contamination is in the groundwater or coming from the water lines.
- Flush the water lines for several minutes prior to collecting water to be used for drinking or cooking.
- Assist residents in identifying alternatives to reduce or eliminate the exposure.

**CONCLUSION 5**

DPH cannot currently conclude if drinking water from 3 wells for several years with elevated lead concentrations could harm children’s health.

**BASIS FOR DECISION**

Lead concentrations greater than the health guideline value referenced by the DPH for private well water supplies (15 µg/L) was detected in 3 wells. While adverse health effects to children were not indicated on the basis of the well water concentrations, only blood lead levels can confirm that children have not accumulated harmful concentration of lead from the well.
waters and other sources. The EPA is concerned that the intermittent elevated lead concentrations in these 3 wells may be coming from the water pipes and are not from the groundwater.

**NEXT STEPS**

The DPH makes the following recommendations:

- Inform persons that lived at the residences served by these wells of the potential health effects to children, provide a contact for blood lead testing, and provide recommendations for follow-up with their personal physicians.
- Identify the source of the lead. Determine if the lead contamination is in the groundwater or coming from the water lines.
- Flush the water lines for several minutes prior to collecting water to be used for drinking or cooking.
- Assist residents in identifying alternatives to reduce or eliminate the exposure.

**CONCLUSION**

DPH concludes that drinking waters from the other wells sampled in the area over many years is not expected to harm people’s health.

**BASIS FOR DECISION**

Other substances (carbon tetrachloride, cis-1,2-dichloroethene, vinyl chloride, arsenic, trihalomethanes, bis(2-chloroethyl)ether, antimony and n-nitroso-di-n-propylamine) detected in the private well water samples reviewed for this evaluation were not present at concentrations high enough to cause adverse health effects.

**NEXT STEPS**

DPH makes the following recommendations:

- Monitor vinyl chloride moving away from the CTS site in groundwater to identify potential exposure points including private wells and discharges to surface water.
- Evaluate the integrity of the septic and private well systems in the areas where the trihalomethane chemicals were detected (bromoform, bromodichloromethane and dibromochloromethane). Communicate the issues and implications to the residents.

**FOR MORE 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 N.C. Division of Public Health at (919) 707-5900, or send an e-mail to nchace@dhhs.nc.gov, and ask for information on the Mills Gap Road Area, Skyland NC Private Well Waters Health Consultation.
Background
The U.S. Environmental Protection Agency (EPA) and the N.C. Department of Environment and Natural Resources (DENR) are investigating contamination associated with the Mills Gap Road/CTS site in Skyland, NC (CTS). The N.C. Department of Public Health (DPH) issued the CTS/Mills Gap Road Public Health Assessment Initial/Public Release on January 19, 2010 [CTS PHA]. The Public Health Assessment (PHA) discusses the public health implications associated with the site and in the surrounding area. Trichloroethylene (TCE) is the primary contaminant associated with the CTS site. Sample collection of private wells in a radius around the CTS site was first performed by DENR in November 2007. Private well data collected from November 2007 through January 2008 was discussed in the CTS PHA. Subsequently, EPA has continued to identify and collect private well water samples in a radius near the Mills Gap Road/CTS site on a quarterly basis since January 2008. DPH evaluated the private well water data for potential negative health effects associated with drinking the water or breathing volatile substances escaping from the water that persons may be exposed to during activities such as showering or bathing. This document discusses the public health implications of approximately 520 private well water samples collected from June 2008 through January 2010. The health implications for private well water data evaluated in the prior PHA are not repeated in this document.

Discussion

Private Well Samples Included in the Health-Effect Evaluation
The DPH evaluated all private well water samples collected by EPA, DENR and their contractors near the Mills Gap Road/CTS site in Skyland, NC from November 2007 through January 2010 [MGRA 2010]. Discussion of samples collected prior to June 2008 is included in the CTS PHA. EPA and DENR are evaluating the well water data as it is finalized by the analytical laboratories and providing each residence with a letter that includes an explanation of the data, a summary of detected compounds, and in some instances the laboratory analytical report for the well water sample.

DPH evaluated data for more than 520 private well samples, plus the associated quality control samples. Many private well locations have been sampled multiple times. All wells were analyzed for a number of different chemicals using analytical methods that included “volatile organic chemicals” (VOCs), and many were also analyzed for metals, cyanide and “semi-volatile organic compounds” (SVOCs).

To protect individual’s privacy, well locations are not identified by address in this report. Well users will be contacted individually by N.C. DPH or Buncombe County Health Department to address potential health concerns identified in this evaluation.

The Health-Effect Evaluation Process
DPH examined the concentration of chemicals found in the well water samples for potential negative health effects related to ingesting (drinking) the well water. Breathing contaminants volatilizing out of the water during activities such as showering and bathing were also considered. It is not known when the detected substances 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. 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. Average concentrations were also assessed for some well detections. A 30-year exposure period was used to evaluate cancer risks. This time period approximates the maximum age of wells in the area and the time period identified as a typical maximum period a person is expected to live at one location. If the concentrations used in this evaluation for a particular contaminant are significantly higher than the actual exposure concentrations over the length of time the substances were present in a well, or the water was consumed for significantly less than 30 years, the health risks may be overestimated. To be protective of public health the process intentionally evaluates exposures to identify the maximum health risk.

A detailed description of the methods DPH uses to evaluate potential adverse health effects is provided in Appendix A. Much new information on the potential health effects of trichloroethylene (TCE) and exposure concentrations at which negative health impacts may occur is currently being evaluated by several organizations, including EPA and ATSDR. DPH used the most current health-protective values proposed by EPA and recommended by ATSDR for evaluation of TCE exposures. A discussion of the values used to evaluate TCE exposures and the most recent information on suspected TCE health effects is provided in Appendix B.

TCE has been identified as a contaminant of concern for the CTS Mills/Gap Road site. Historically, TCE has been used as a solvent for cleaning metal parts and as a solvent to make other chemicals. TCE can also be found in some household products, including paint removers, adhesives, and spot removers.

DPH’s evaluation of the well data is not intended to identify the source of the contamination, nor is it limited to contamination that is thought to be associated with the CTS/Mills Gap Road site. DPH evaluated all substances detected in the well water samples, regardless of possible source. Possible sources of some of the substances are discussed.

Evaluation of Private Well Water Data by Substance

Thirteen different substances were detected in 17 private wells at concentrations that exceed either a health-protective comparison value or a Maximum Contaminant Level (MCL) regulatory value applicable to public water systems. These substances are listed in Table 1. While the regulatory values are not applicable to the private wells, they were included in the evaluation for comparison to values that would apply to water supplied through a public water system. A discussion of the detections and health implications of these substances follows.

Detected concentrations, exposure dose estimates and health evaluation values are summarized in Table 2 for those substances that exceeded health guideline values. An estimate of the theoretical increased cancer risk for the substances known or suspected to be human carcinogens (chemicals that cause cancer) are provided in Table 3. This theoretical calculated cancer risk is not exact and tends to overestimate the actual risk associated with exposures that may have occurred [ATSDR 2005a]. The cancer risk is expressed as an estimate of the number of additional cancers over the number of cancers that occur in a typical population without these
Trichloroethylene (TCE)

Trichloroethylene (TCE) was detected in 6 wells that were in use as a residential water supply at the time the samples were collected. The TCE concentration at 5 of these locations exceeded either the federal regulatory value for public water systems (5 µg/L MCL) or EPA’s non-regulatory regional screening value for tap water (2.0 µg/L). EPA also lists a non-regulatory MCL goal (MCLG) of 0 µg/L for TCE in public drinking water supplies. N.C. DENR lists 3 µg/L as their Groundwater Standard for TCE [DENR 2010]. It is not known if TCE causes cancer in humans. ATSDR does not provide a comparison value for TCE but refers to the EPA’s MCL and proposed lower health screening values (Appendix B). TCE detections in the well waters ranged from 2.8 to 1,200 µg/L. Because it is not known when these wells were first contaminated, the available analytical data was used to evaluate the potential for health effects related to drinking the contaminated well water.

In addition to the detections noted above, TCE was detected in a groundwater well and in 2 spring wells directly east of the CTS site in 1999. These detections were discussed in the CTS/Mills Gap Road Public Health Assessment Initial/Public Release draft document released on January 12, 2010 [CTS PHA 2010]. A sample was collected at these drinking water source wells in 1999, high concentrations of TCE were detected (270 µg/L in the groundwater well and 21,000 and 15,000 µg/L in the spring wells), and the homes using these wells were provided alternative sources of drinking water. Health-effect evaluations for persons that may have
ingested (drank) waters from these wells over long periods indicated the potential for adverse health effects. It is not known when the contamination first appeared in these wells, or the concentrations of TCE in these waters over the length of time people were drinking from the wells. The health-effect evaluations were based on the only available data. DPH’s Public Health Physician has been in contact with members of the families that used the contaminated wells identified in 1999 to provide recommendations for medical follow-up to their TCE exposure.

Additional detections of TCE have been identified in the Oaks neighborhood which is approximately 4,000 feet northeast of the CTS site. EPA and DENR are currently investigating this area to determine if the TCE contamination found there is related to CTS, or may be due to another source of contamination. Private wells in the Oaks neighborhood were connected to the Asheville municipal water system in the fall of 2008. The first detection of TCE in the Oaks area was indentified in November 2007. TCE was detected in one well (at 57 µg/L) and the household was immediately supplied with bottled water. Additional samples collected from this well location (in June and September 2008), when it was no longer in use, found similar concentrations of TCE (60 µg/L average concentration). “Low” probability of additional cancers and no non-cancer adverse health effects were indicted for this exposure, as reported in the PHA [CTS 2010].

TCE concentrations greater than health-effect comparison values were identified in June 2008 in 5 additional wells in the Oaks neighborhood. There were 3 detections of TCE in one well (from January through September, 2008), with 2 samples having TCE concentrations greater than comparison values. The highest TCE concentration from this well (20 µg/L) was used for the health evaluation. Non-cancer adverse health effects are not indicated; while “very low” to “moderate” numbers of theoretical cancer risks (see Table 3) are indicated for persons drinking water contaminated with TCE at this concentration.

Two other wells in the Oaks neighborhood had single detections of TCE (8.8 and 2.8 µg/L) greater than comparison values. Non-cancer health effects are not indicated for either location. “Very low” to “low” numbers of theoretical increased cancers are estimated for the 8.8 µg/L detection. “No” increased cancers to “very low” numbers of increased cancers are indicated for the 2.8 µg/L TCE detection. The estimate of the number of increased cancers is listed in Table 3.

While non-cancer health risks are not indicated for the Oaks neighborhood samples and the estimates of increased theoretical cancer risks are within the acceptable cancer risk range (less than 100 additional cancers in 1 million exposed persons) N.C. DPH recommends a long-term target cancer risk of not more than 1 in a million exposed persons. Reducing or eliminating exposure to drinking water contaminants to achieve this level of theoretical risk should be the ultimate goal.

TCE at 1200 µg/L was found in a private well approximately 2500 feet northeast of the CTS site in August 2009. EPA immediately provided the residence with bottled water. Both non-cancer and theoretical cancer health effects are predicted if the 1200 µg/L detection is representative of the exposure concentration over a 30-year period. “Moderate” to “high” numbers of additional cancers are estimated (see Tables 2 and 3). In addition to the ingestion risks, there may have been additional risks due to inhalation (breathing) of TCE that escaped from the water to the air.
during activities such as showering or bathing. The potential for harmful health effects may be increased if the persons using the contaminated well water were exposed to both drinking and breathing high levels of TCE. A N.C. DPH Physician contacted the family residing at this location in August 2009 to provide medical recommendations. A family member indicated some family members had been using the well for as long as 20 years.

**Benzo(a)pyrene and Polycyclic Aromatic Hydrocarbons (PAHs)**

Polycyclic aromatic hydrocarbons (PAHs) are a group of structurally related organic chemicals that were evaluated together for potential health effects. PAHs are a group of compounds formed during the incomplete burning of coal, oil and gas, garbage, or other organic substances like tobacco or charbroiled meat. Some PAHs are manufactured. They are found in coal tar, crude oil, creosote, and roofing tar. Some are used in medicines or to make dyes, plastics, and pesticides. The potential health effects of PAHs were evaluated by converting individual detected PAH compounds to their benzo(a)pyrene equivalent concentration. Benzo(a)pyrene is considered a probable human carcinogen [ATSDR 2010 HG]. Additional information is provided in Appendix A.

None of the private well samples had PAH concentrations greater than the Federal regulatory level (0.2 µg/L MCL as benzo(a)pyrene) for public drinking water systems [EPA DW]. PAHs were found in 26 samples from 22 different wells. Three samples had benzo(a)pyrene-equivalent concentrations greater than the ATSDR cancer-effect comparison value (0.005 µg/L CREG). Two of these wells are near each other (approximately 9000 feet east of the CTS site) and the PAHs were detected (0.036 and 0.016 µg/L benzo(a)pyrene-equivalent) during the same sample collection event in January 2009. No PAHs were found in 2 subsequent samples collected at these 2 locations. “Very low” numbers of theoretical increased cancers are estimated for persons drinking the concentration of PAHs found in the single sample for each of these 2 wells (Table 3). The average PAH concentrations in these 2 wells do not indicate an increase in cancer risk. Adverse non-cancer or cancer health effects related to PAHs are not indicated for these 2 wells.

A third detection of PAHs (0.118 µg/L benzo(a)pyrene-equivalent) at a concentration greater than the cancer-effect comparison value was observed approximately 4000 feet northeast of the CTS site in September 2008 (in the Oaks neighborhood). The benzo(a)pyrene-equivalent concentration is less than the regulatory level for public water systems (0.2 µg/L MCL). A single sample was collected from this well before it was closed. “Low” numbers of increased cancers are estimated for the PAH concentration found in the single sample (Table 3).

Uncertainty exists regarding the potential health impacts related to PAHs in this well because of the lack of data available to determine if there was a long-term exposure and at what concentration. While non-cancer health risks associated with the PAH levels detected in this well and the estimates of theoretical increased cancer risks are within the acceptable cancer risk range N.C. DPH recommends a long-term target cancer risk of not more than 1 in a million exposed persons. Reducing or eliminating the exposure to the PAHs to achieve this level of risk is recommended.
Copper

Copper did not exceed the public water regulatory level (1300 µg/L MCL Action Level) in any well sample. One sample from each of 2 wells that were sampled multiple times did exceed the lower health-screening comparison value (100 and 400 µg/L for children and adults).

Copper at a maximum concentration of 160 µg/L was detected in a well sampled 7 times between May 2000 and October 2009. Copper was detected in each of the 7 samples (average = 80 µg/L). The exposure dose estimate for the highest detected copper concentration is equal to the health guideline value for children.

Copper was detected in all 4 samples collected from the second well, with the maximum concentration of 120 µg/L exceeding the lower health-screening comparison value. The average copper concentration for the 4 samples was 96 µg/L. Permanent adverse health effects are not indicated for these levels of copper.

The MCL action level is set at copper levels that result in permanent health effects. The most sensitive human exposure endpoint for copper ingestion is non-persistent gastrointestinal effects (nausea and vomiting) resulting from exposures as short as two months. More severe long-term effects to humans are associated with much higher exposure concentrations or exposure doses (5 to 10 times or more higher) than indicated by the data described above. The human health study values used for these evaluations are the highest at which no adverse effect was observed. Animal study data for long-term exposures do not indicate the copper concentrations observed in these samples would result in permanent adverse health effects [ATSDR 2004b]. This information would suggest the likelihood of adverse effects associated with ingestion of copper at the concentrations seen in these well samples is unlikely, but does not eliminate a concern for exposed children that may be sensitive to copper. N.C. DPH has not observed transient gastrointestinal effects at copper concentrations more 5 times the average levels seen in these 2 wells. If the elevated copper is coming from the components of the water supply system the residents could be exposed to much higher levels of copper when ingesting water that sits in the water lines for an extended period (such as overnight, or over the course of the day).

The 2 wells with the elevated copper concentrations are not located near each other. DPH recommends investigating whether the copper contribution may be from some component of the water lines. DPH also recommends person’s that may be exposed to these well waters be contacted to determine if they have experienced the type of temporary effects noted above and assisted with identifying alternatives to reduce or eliminate their exposure. Filter systems or reverse osmosis systems may be used to reduce or eliminate the concentration of copper in the well waters, thus reducing the exposure concentrations and the potential for adverse health effects. Flushing the water lines prior to collecting water for drinking or cooking may also reduce exposure concentrations.

Lead

There were 3 private wells with lead concentrations equal to or greater than the Federal regulatory action level for public water systems (15 µg/L) [EPA DW]. DPH uses the same value as a non-regulatory health guideline for private well water samples. All 3 wells had detections greater than the lead action level in samples collected in January 2009. For 2 of these wells,
none of the other samples collected have exceeded the lead action level. Because of the pattern of lead detections in these 3 wells, EPA became concerned that the elevated lead values detected in the January 2009 samples may have been due to inadequate purging of the water lines prior to sample collection. Regardless of the source of the lead (the pipes or the groundwater supply), the data indicates that persons using these water supplies could be exposed to lead concentrations greater than current health guideline values.

The first well was sampled 7 times between May 2000 and October 2009 and had 6 lead detections. Two detections were equal to or greater than the action level, including the January 2009 sample which was the highest concentration (16 µg/L lead). The October 2009 sample was reported at 15 µg/L lead, a concentration equivalent to the action level. EPA has stated they believe some of the elevated lead data at this location and the two locations discussed below may be related to lead leaching from plumbing fixtures and showing up in the well samples when the plumbing system is not purged adequately before sampling. Additional preliminary data reported for samples collected in February 2010 detected 39 µg/L lead in this well water. This result prompted EPA to perform follow-up sampling in April 2010. The objective of the April 2010 sampling is to determine if the elevated lead levels are being leached from the water pipes when the water sits in the pipes for an extended period. At the date of this report the April 2010 data was not available.

A second well had 5 detections of lead in 7 samples collected between September 2008 and February 2010. Only the January 2009 sample (22 µg/L) was greater than the action level. Duplicate samples collected from this well in March 2009 had two detections below the action level (11 and 14 µg/L). Low levels of lead were detected in one of two July 2009 duplicates (5.6 J µg/L) and the February 2010 sample (0.67 J µg/L) (“J” values indicate estimated concentrations).

The third well had 3 lead detections in 5 samples collected from January 2009 through February 2010, with only the January 2009 detection (29 µg/L) greater than the action level. The next higher lead detection from this well was 6.5 J µg/L in April 2009.

The EPA lists 0 µg/L as the goal for lead in drinking water. The Centers for Disease Control and Prevention (CDC) considers children to have an elevated blood lead level at 10 micro-grams lead per deci-liter of blood (µg/dL) or greater [ATSDR 2007e]. However, CDC identifies that blood lead levels in children less than 10 µg/dL may still result in decreased cognitive function, developmental delays, and behavior problems [CDC 2009]. There is no conclusive evidence that lead causes cancer in humans. Ingestion of large amounts of lead by laboratory animals has resulted in kidney cancer.

Blood lead levels were predicted using an ATSDR model and the maximum lead concentration for each well. None of the predicted blood lead levels exceed the 10 µg/dL action level for infants or children. While the blood lead level predictions for the well waters do not exceed the CDC definition of elevated blood lead, actually taking blood lead samples is the most reliable means to determine actual body burdens of lead.
DPH recommends collecting blood samples for lead determination and appropriate medical follow-up if children have been drinking from these 3 well waters. This will provide an indication of whether these infants and children are being exposed to lead from other sources in addition to well water and may have developed lead body burdens greater than that indicated by the well water only prediction. Blood lead tests would also help determine if children that may have drank from these wells were exposed to elevated lead by drinking water that sat in the well water delivery system and leached lead.

EPA has stated that off-site elevated lead concentrations in groundwater do not appear attributable to the CTS site (EPA 2010). Lead is often found in drinking water supplies as a result of contamination from components of the water supply lines (such as pipe solder), particularly in older homes. Determining if this is a likely source of the lead in the well waters is recommended. Filtration and reverse osmosis systems may be used to remove the lead and reduce exposures. Flushing the water lines prior to collecting water for drinking or cooking may also reduce exposure concentrations.

**Vinyl Chloride**
Vinyl chloride was detected in 1 well sample at a concentration greater than the cancer-effect comparison value in September 2008. The well with the detection (0.52 J µg/L) is approximately 1500 feet east of the CTS site and has not been used as a source of drinking water since 1999. (A “J” notation indicates the reported analytical concentration is an “estimated value.”) Since the well is no longer in use for drinking water, potential health effects were not evaluated. Vinyl chloride is a degradation product of TCE [UM BIOD] and a known human carcinogen [ATSDR HG]. Close monitoring of this chemical in groundwater down gradient of the site is recommended to prevent exposure to private well users. Groundwater may also discharge to surface water. Continued monitoring of surface waters down gradient of the site is also recommended to identify potential exposure points to TCE and its degradation products.

**Carbon Tetrachloride**
There was one detection of carbon tetrachloride (0.29 J µg/L, September 2008) at a concentration equal to the cancer-effect screening value. Carbon tetrachloride is considered a probable human carcinogen [ATSDR HG]. No additional cancer risk was indicated for this concentration.

**Bis-2-ethylhexylphthalate (BEHP)**
Bis-2-ethylhexylphthalate (BEHP) was detected in 3 wells in October 2009 at concentrations exceeding the health-effect comparison value. BEHP is commonly encountered in environmental samples as a contaminant occurring during the sampling or laboratory handling process. To investigate the BEHP detections, EPA resampled all 3 wells in January 2010, including one in duplicate. All follow-up samples were negative for BEHP. As a result, the prior BEHP detections are considered sample handling contamination and not indicative of contamination of the well waters. Potential health effects are not indicated.
Arsenic

The highest detection of the metal arsenic (0.65 µg/L, an estimated value, detected in September 2008) is less than the Federal regulatory value (10 µg/L MCL) for public water systems [EPA DW]. However, there were 11 detections of arsenic that exceeded ATSDR’s health-based comparison value for cancer-effects (0.02 µg/L CREG). Arsenic is a known human carcinogen [ATSDR HG]. The additional cancer risk evaluation for the highest detected concentration indicated no increased cancers. Adverse health effects are not indicated for the arsenic levels observed in the well waters.

Elevated arsenic concentrations were identified in sub-surface soil samples collected on and near the CTS site in December 2007. No elevated arsenic levels were noted in groundwater samples reviewed for the CTS PHA. Some areas of North Carolina have naturally-occurring arsenic in the groundwater. The observed arsenic detections may be naturally-occurring.

Total Trihalomethanes -
(Bromoform, Dibromochloromethane, Bromodichloromethane, Chloroform)

Bromodichloromethane (BDCM) was detected in 2 well samples at concentrations greater than the cancer-effect comparison value. One detection was in December 2007 in a well closed in 1999. The other BDCM detection was in January 2009 (2.9 µg/L). Bromoform was detected in one sample at a concentration exceeding the cancer-effect comparison value (11 µg/L, September 2008). Dibromochloromethane (DBCM) was detected twice (September 2008, October 2009) in the same well at concentrations greater than its cancer-effect comparison values (maximum concentration 2.1 µg/L). These three chemicals, along with chloroform, make up a group of chemicals collectively referred to as “trihalomethanes” (THMs). There were no detections of chloroform greater than comparison values. Both a bromoform and DBCM detection greater than comparison values were observed in the same well sample in September 2008. BDCM and bromoform are considered probable human carcinogens, and chloroform and DBCM are considered possible human carcinogens [ATSDR HG].

Exposure dose estimates did not exceed non-cancer health guideline values for any of the THM compounds. The exposure dose estimate for the sample with the highest combined concentration of all 4 compounds also did not exceed the non-cancer health guideline. Estimates of increased cancer risks for the individual compounds are all “very low”. The combined cancer risk of the bromoform and DBCM found in the same well sample is also “very low”. Based on the indications of very low additional cancer risk and the situation that the detections in all wells are infrequent, adverse health effects are not indicated for ingestion of the trihalomethane compounds.

There were approximately 26 THM detections in 9 different wells. The THM detections were scattered throughout the sampling area. The presence of THM chemicals in the well waters raises a question as to their source and could be related to residents’ activities, such as chlorinating their wells or adding chlorine-containing products to the laundry or toilets. If the latter is the source, it raises a concern with the effectiveness of the septic systems and the integrity of the wells in the areas where these chemicals were found. THMs are commonly seen in public water systems as a byproduct of the chlorination process. They are produced by the reaction of chlorine products with natural plant materials. The chlorine is added to prevent
exposure to disease-producing microorganisms. High volumes of chlorine-containing products going through a septic system may decrease the ability of the septic system to breakdown household and human waste. As a result, undesirable components of the waste may travel through the subsurface to where down-gradient well users may be exposed to either the chemicals or the disease-producing microorganisms. Well chlorination is an effective means to disinfect a well. Using more chlorine product than necessary for the size of the well could lead to exposure to the residual chlorinating components and by-products. DPH recommends following guidance provided by County or State agencies when chlorinating wells to optimize the disinfection process and prevent exposure to high concentrations of residual chemicals. DPH recommends investigating the situation leading to the observation of THMs in the well waters.

*Bis(2-chloroethyl)ether*

Bis(2-chloroethyl)ether (B2CEE) was detected in one well sample (approximately 9000 feet east of the CTS site) in September 2008 at an estimated concentration (2.7 J µg/L) greater than the cancer-effect comparison value. Four later samples collected at this location did not detect bis(2-chloroethyl)ether. EPA identifies B2CEE as a probable human carcinogen [ATSDR HG]. “Low” numbers of theoretical increased cancers are estimated for drinking water for many years with the detected concentration of bis(2-chloroethyl)ether. If the single B2CEE detected concentration is averaged for the number of samples collected (5) then no increased cancers are indicated. Adverse health effects are not indicated for this well.

Bis(2-chloroethyl)ether does not occur naturally. It is a component of pesticides, paints, varnishes, or rust inhibitors. It is also used as a solvent or cleaner, or to make other chemicals [ATSDR 2009].

*Antimony*

The metal antimony was detected in a single sample (22 µg/L J, an estimated concentration) in July 2009 at a concentration greater than the comparison value. Antimony was not detected in 2 prior and one later sample collected from the same well. Exposure dose estimates were greater than the non-cancer health guideline for both children and adults [ATSDR HG]. No human health study values were available for antimony. The dose estimates were compared to animal health study values [ATSDR 1992] following application of a 15% gastric absorption factor referenced by EPA [EPA RSL]. Comparison to animal health study data indicates adverse health effects would not be expected at the highest concentration. The intermittent detection of antimony would further indicate that adverse health effects are not expected, and may indicate that the antimony detection is due to contamination of the samples during handling.

*N-Nitrosodi-n-propylamine*

N-Nitrosodi-n-propylamine (NDPA) was detected in one of 2 duplicate well water samples collected from a well approximately ½-mile northeast of the CTS site in April 2009. The detection was an estimated value (0.82 J µg/L) less than the analytical minimum reporting level (MRL, 5.0 µg/L NPDA), but exceeded the ATSDR cancer-effect comparison value for drinking water (0.005 µg/L CREG) [ATSDR HG]. NDPA is identified as a likely human carcinogen by
EPA. NDPA was not detected in the duplicate well sample collected on the same day, nor 3 prior and 3 later samples collected from this well to date. “Low” numbers of increased cancers are estimated for drinking water for many years with NDPA at the concentration reported in the one April 2009 duplicate well sample. NDPA is not produced commercially in the U.S. It is most often seen as an impurity in some herbicides, a bi-product in foods treated with nitrite preservatives, some alcoholic beverages, in some textile and rubber industry processes, and in smoke from some tobacco products [ATSDR 1989b, IRIS]. No adverse health effects are indicated for this compound on the basis of the single detection in 8 sampling events from this well, the non-detect in the duplicate sample, and the single low reported concentration. Treatment of the well water (such as with carbon filtration) to remove NDPA from the well water and eliminate the drinking water exposure is recommended if NDPA is detected in future samples.

Substances Not Exceeding Health Guideline Values
A single detection of cis-1,2-dichloroethene was found in September 2008 in a well sample exceeding the MCL. The well has not been used as a drinking water source since 1999. Since there is not a drinking water exposure for this detection the chemical was not evaluated further.

Possible Health Effects Associated with Select Chemicals Detected in the Private Well Waters
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 for persons consuming water from the wells discussed in this report that contain these substances.

*Trichloroethylene (TCE)*
Trichloroethylene is a colorless liquid which is used as a solvent for cleaning metal parts and as a solvent to make other chemicals. Trichloroethylene can be found in some household products, including, paint removers, adhesives, and spot removers [ATSDR 2003].

While occupational studies have shown adverse health effects from breathing trichloroethylene (TCE), it is not known if drinking water contaminated with TCE causes non-cancer illness in humans. Childhood leukemia has been observed after maternal exposure to TCE contaminated drinking water during the prenatal period. Breathing small amounts may cause headaches, lung irritation, dizziness, poor coordination, and difficulty concentrating. Breathing large amounts of TCE may cause impaired heart function, unconsciousness, and death. Breathing it for long periods may cause nerve, kidney, and liver damage. Evidence from animal and epidemiological studies also suggest that exposure to TCE might be associated with congenital heart defects and poor intrauterine growth. Studies in rats and mice show that TCE can affect fertility, but the relevance to humans is not clear [NRC 2006]. Human epidemiological studies have been limited by difficulties in estimating exposure levels and by the presence of other solvents with similar toxic effects. In rats and mice, TCE begins affecting the liver, kidney, and developing fetus at doses as low as 1 mg/kg/day. These studies are limited, however, by inadequate characterization of exposure, inadequate quantification of results, or lack of endpoints suitable for deriving chronic endpoints. The current ATSDR cancer classifications listed for TCE are “under review” (EPA), “reasonably anticipated to be a carcinogen” (NTP),
and “probably carcinogenic to humans” [ATSDR 1997d, ATSDR 2003, EPA 2001, NJDHSS 2003]. In recent years evidence supporting TCE’s ability to cause cancer has been strengthened.

Copper

Copper is a reddish metal that occurs naturally in rock, soil, water, sediment, and, at low levels, in air. Copper also occurs naturally in all plants and animals. It is an essential element for all known living organisms including humans and other animals at low levels of intake. At much higher levels, toxic effects can occur. Drinking water that contains higher than normal levels of copper, may cause nausea, vomiting, stomach cramps, or diarrhea. Intentionally high intakes of copper can cause liver and kidney damage. It is not known if copper can cause cancer in humans. EPA does not classify copper as a human carcinogen because there are no adequate human or animal cancer studies.

Exposure to high levels of copper will result in the same types of effects in children and adults. It is not known if these effects occur at the same dose level in children and adults. Studies in animals suggest that children may have more severe effects than adults; 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 2004a].

Lead

Lead is a naturally occurring toxic metal. It may be found in its pure form or in combination with other minerals. Lead has no nutritional value, but is very valuable in manufacturing. In industry, lead is used in the production of batteries, solder, paints, ammunition, sheet metal, and other metal alloys. Lead was found in house paint sold before 1978. Since 1978, paint sold for residential use can contain no more than 600 parts per million lead. Most lead is now used to manufacture car batteries. Other lead sources include bullets, fishing weights, curtain weights, some glazed ceramics, and plumbing solders made before 1986.

Lead is a well known developmental neurotoxin (affects the nervous system), and also affects the kidneys, blood formation, reproduction, humoral immunity, and the peripheral nervous system. 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. Lead may also cause anemia. In pregnant women, high levels of exposure to lead may cause miscarriage. According to the ATSDR there is no conclusive proof that lead causes cancer, however both the U.S. Department of Health and Human Services and EPA have determined that lead is a probable human carcinogen. Children are more sensitive to the effects of lead than adults, and studies show that even low lead levels that do not affect adults can be detrimental to a child’s cognitive development [ATSDR 2007d].

Vinyl Chloride

Vinyl chloride is a colorless gas at room temperature. Vinyl chloride does not occur naturally. It can be formed in the environment when other manufactured substances, such as
trichloroethylene, trichloroethane, and tetrachloroethylene, are broken down by certain microorganisms. Vinyl chloride is used to make a plastic polymer called PVC, which is used to make a variety of plastic products. The effects of ingesting vinyl chloride are unknown. Several studies have suggested that breathing air or drinking water containing moderate levels (100 mg/L) of vinyl chloride might increase their risk for cancer. However, the levels used in these studies were much higher than levels found in the ambient air and/or most drinking water supplies. The U.S. Department of Health and Human Services has determined that vinyl chloride is a known carcinogen and EPA has determined that vinyl chloride is a human carcinogen. No studies are available that specifically address the effects of vinyl chloride in children [ATSDR 2006a].

**Polycyclic Aromatic Hydrocarbons (PAHs)**

PAHs are a group of chemicals that are formed during the incomplete burning of coal, oil, gas, wood, garbage, or other organic substances, such as tobacco and charbroiled meat. There are more than 100 different PAHs. PAHs usually occur naturally, generally as complex mixtures made-up of a number of different PAH compounds. A few PAHs are used in medicines and to make dyes, plastics, and pesticides. PAHs are found in asphalt used in road construction, substances such as crude oil, coal, coal tar pitch, creosote, and roofing tar. They are found throughout the environment in the air, water, and soil. PAHs enter the environment mostly as releases to air from volcanoes, forest fires, residential wood burning, and exhaust from automobiles and trucks. Some PAHs in soils can contaminant 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. But these effects have not been seen in people. Some PAHs may reasonably be 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 cancer. Some PAHs have caused cancer in laboratory animals when they breathed air containing PAHS (lung cancer), ingested PAHs in food (stomach cancer), or had PAHs applied to their skin (skin cancer) [ATSDR 1995, ATSDR 1996b].

**Total Trihalomethanes (TTHMs)**

“Total trihalomethanes” refers to bromoform, dibromochloromethane (DBCM), chloroform and bromodichloromethane (BDCM). These chemicals are most commonly seen as contaminants of drinking water that has been chlorinated to kill disease causing bacteria and viruses, forming when chlorine reacts with naturally occurring substances in water. In the past, bromoform was used by industry to dissolve dirt and grease and to make other chemicals. DBCM was used in the past to make other chemicals such as fire extinguisher fluids, spray can propellants, refrigerator fluid, and pesticides.
The main effect of swallowing or breathing large amounts of bromoform is a slowing of normal brain activities, resulting in sleepiness or sedation occurring quickly after the chemicals enter your body. In humans, these effects tend to disappear within a day. Some studies in animals indicate that exposure to high doses of bromoform or DBCM may also lead to liver and the kidney injury within a short period of time. Exposure to low levels of bromoform or DBCM does not appear to seriously affect the brain, liver, or kidneys. Other animal studies suggest that typical bromoform or DBCM exposures do not pose a high risk of affecting the chance of becoming pregnant or harming an unborn baby. However, studies in animals indicate that long-term intake of either bromoform or DBCM can cause liver and kidney cancer [ATSDR 2005b].

In humans, chloroform affects the central nervous system (brain), liver, and kidneys after a person breathes air or drinks liquids that contain large amounts of chloroform. In the past, chloroform was used as an anesthetic during surgery before its harmful effects on the liver and kidneys were recognized. Breathing air, eating food, or drinking water containing elevated levels of chloroform over a long period may result in liver and kidney damage. There is some evidence of an association with drinking chlorinated water and colon and bladder cancer. Chloroform is classified as a probable human carcinogen [ATSDR 1997a].

It is not known at what levels BDCM causes harmful health effects in people. In animals, the main effect of eating or drinking large amounts of BDCM is injury to the liver and kidneys. High levels can also cause effects on the brain, leading to incoordination and sleepiness. There is some evidence that BDCM can be toxic to developing fetuses. Studies in animals show that intake of BDCM for several years in food or water can lead to cancer of the liver, kidney and intestines. Although effects of BDCM have not been reported in humans, effects would probably occur if enough BDCM were taken into the body. It is not known at what levels BDCM causes harmful health effects in people [ATSDR 1989b].

**Arsenic**

Arsenic is a naturally occurring element that is widely distributed in the Earth’s crust. Arsenic is classified chemically as a metalloid, having both properties of a metal and a nonmetal; however, it is frequently referred to as a metal. In the past arsenic has been used as a wood preservative and in pesticides. Arsenic has also been used as an additive in animal feed. It may be combined with other metals to form alloys. The largest use is in lead-acid batteries for automobiles.

Inorganic arsenic has been recognized as a human poison since ancient times, and large oral doses (above 60,000 ppb in water which is 10,000 times higher than 80% of U.S. drinking water arsenic levels) can result in death. If you swallow lower levels of inorganic arsenic, ranging from about 300 to 30,000 ppb in water (100–10,000 times higher than most U.S. drinking water levels), you may experience irritation of your stomach and intestines, with symptoms such as stomach ache, nausea, vomiting, and diarrhea. Other effects you might experience from swallowing inorganic arsenic include decreased production of red and white blood cells, which may cause fatigue, abnormal heart rhythm, blood-vessel damage resulting in bruising, and
Table 2. Substances detected in private well samples collected around the Mills Gap Road area from June 2008 through January 2010. Data includes detections exceeding health comparison values or those with estimated exposure dose levels exceeding N.C. DPH health-effect guideline values. Exceedances are in bold. Cancer risk estimates are provided in Table 3. Table 2 continued on the next page.

<table>
<thead>
<tr>
<th>Detected Substance</th>
<th>Exposure Concentration (µg/L)</th>
<th>Health Effect Comparison values (^1) (µg/L)</th>
<th>Exposure Dose Estimate (mg/kg/d)</th>
<th>Health Guideline Exposure Dose (^1) (mg/kg/d)</th>
<th>Health Study Values (^2)</th>
</tr>
</thead>
<tbody>
<tr>
<td>TCE</td>
<td></td>
<td></td>
<td>0.0038 child, 0.0017 adult</td>
<td>Non-cancer</td>
<td>0.2</td>
</tr>
<tr>
<td></td>
<td>PW #1</td>
<td>60</td>
<td>2.0 (^3)</td>
<td>Cancer</td>
<td>0.0003 (^5)</td>
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<tr>
<td></td>
<td>PW #2</td>
<td>20</td>
<td>3 (^4)</td>
<td>0.02 to 0.4 (mg/kg/d)(^1)</td>
<td>1.0E-06 child, 4.6E-07 adult</td>
</tr>
<tr>
<td></td>
<td>PW #3</td>
<td>8.8</td>
<td>0.00055 child, 0.00025 adult</td>
<td>EPA CSF (^7)</td>
<td>1.0E-06 child, 4.6E-07 adult</td>
</tr>
<tr>
<td></td>
<td>PW #4</td>
<td>2.8</td>
<td>0.00018 child, 0.000080 adult</td>
<td>100 child, 400 adult</td>
<td>0.0075 child, 0.034 adult</td>
</tr>
<tr>
<td></td>
<td>PW #5</td>
<td>1200</td>
<td>5 MCL</td>
<td>0.010 child, 0.0046 adult</td>
<td>0.0044 child, 0.0020 adult</td>
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<tr>
<td></td>
<td></td>
<td></td>
<td>100 child, 400 adult, 1300 MCL AL</td>
<td>0.0075 child, 0.0034 adult</td>
<td>0.060 child, 0.0027 adult</td>
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<td>PAHs</td>
<td></td>
<td></td>
<td>2.2E-06 child, 1.0E-06 adult</td>
<td>0.01</td>
<td>0.04 RfD</td>
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<tr>
<td></td>
<td>PW #1</td>
<td>0.036</td>
<td>0.005 cancer</td>
<td>Non-cancer</td>
<td>0.042 human (^8)</td>
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<tr>
<td></td>
<td>PW #2</td>
<td>0.016</td>
<td>0.2 MCL</td>
<td>0.091 human (^9)</td>
<td>0.091 human (^9)</td>
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<tr>
<td></td>
<td>PW #3</td>
<td>0.118</td>
<td>1.0E-06 child, 4.6E-07 adult</td>
<td>0.17 human (^10)</td>
<td>0.17 human (^10)</td>
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<tr>
<td>Copper</td>
<td>PW #1</td>
<td>160 maximum 70 average</td>
<td>100 child, 400 adult, 1300 MCL AL</td>
<td>0.010 child, 0.0046 adult</td>
<td>0.0044 child, 0.0020 adult</td>
</tr>
<tr>
<td></td>
<td>PW #2</td>
<td>120 maximum 96 average</td>
<td>0.0075 child, 0.0034 adult</td>
<td>Maximum estimated blood lead level (µg/dL) –</td>
<td>0.0075 child, 0.0034 adult</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>15 AL</td>
<td>4.2 infants</td>
<td>0.0075 child, 0.0034 adult</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>5.7 infants</td>
<td>0.0075 child, 0.0034 adult</td>
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<td></td>
<td></td>
<td></td>
<td>7.5 infants</td>
<td>0.0075 child, 0.0034 adult</td>
</tr>
<tr>
<td>Lead</td>
<td>PW #1</td>
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<td>15 AL</td>
<td>10 µg/dL blood lead</td>
<td>-NA-</td>
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<tr>
<td></td>
<td>PW #2</td>
<td>22</td>
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<td>Maximum estimated blood lead level (µg/dL) –</td>
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<td>4.2 infants</td>
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<td>5.7 infants</td>
<td>-NA-</td>
</tr>
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<td></td>
<td></td>
<td></td>
<td></td>
<td>7.5 infants</td>
<td>-NA-</td>
</tr>
<tr>
<td>Carbon tetra-chloride</td>
<td>0.29 J</td>
<td>7 child, 20 adult 0.3 cancer</td>
<td>1.8E-05 child, 8.3E-06 adult</td>
<td>0.0007</td>
<td>-NA-</td>
</tr>
<tr>
<td>Arsenic</td>
<td>0.65 J</td>
<td>3 child, 10 adult 0.2 cancer</td>
<td>4.1E-05 child, 1.9E-05 adult</td>
<td>0.003</td>
<td>-NA-</td>
</tr>
</tbody>
</table>
Table 2, continued. Substances detected in private well samples collected around the Mills Gap Road area from June 2008 through January 2010. Data includes detections exceeding health comparison values or those with estimated exposure dose levels exceeding N.C. DPH health-effect guideline values. Exceedances are in bold. Cancer risk estimates are provided in Table 3.

<table>
<thead>
<tr>
<th>Detected Substance</th>
<th>Exposure Concentration (µg/L)</th>
<th>Health Effect Comparison values 1 (µg/L)</th>
<th>Exposure Dose Estimate (mg/kg/d)</th>
<th>Health Guideline Exposure Dose 1 (mg/kg/d)</th>
<th>Health Study Values (mg/kg/d) 2</th>
</tr>
</thead>
</table>
| THMs – as TTHMs    | 20.7                          | 80 MCL, 70 MCLG                        | 0.0013 child, 0.00059 adult     | 0.01 RfD 10                             | Non-cancer
|                    |                               | 200 child, 700 adult                   | 0.00018 child, 0.000083 adult   | 0.02                                     | -NA-                            |
|                    |                               | 80 MCL, 0 MCLG                         | 0.00069 child, 0.00031 adult    | 0.02                                     | -NA-                            |
| Bromoform          | 11                            | 200 child, 700 adult                   | 0.0011 child, 0.00049 adult     | 0.01 RfD 11                             | 0.96 human
|                    |                               | 80 MCL, 0 MCLG                         |                                |                                          | 0.96 human
| Chloroform         | 17                            | 100 child, 400 adult                   |                                |                                          |                                |
|                    |                               | 80 MCL, 70 MCLG                        |                                |                                          |                                |
| DBCM               | 2.1                           | 900 child, 3000 adult                  |                                |                                          |                                |
|                    |                               | 80 MCL, 60 MCLG                        |                                |                                          |                                |
| Bis(2-chloro-ethyl)ether | 2.7 J          | 0.03 cancer                            | 1.7E-04 child, 7.7E-05 adult    | 1.1 EPA 7                               | Non-cancer
|                    |                               |                                    |                                |                                          | 25 animal
|                     |                               |                                    |                                |                                          | Cancer
|                     |                               |                                    |                                |                                          | 41 animal
| Antimony           | 22 J                          | 4 child, 10 adult                      | 0.0014 child, 0.00063 adult     | 0.0004 RfD                             | 0.262 animal
|                    |                               | 6 MCL, MCLG                            |                                |                                          | 0.35 animal
| n-Nitroso-di-n-propylamine | 0.82 J     | 0.005 cancer                           | 2.1E-05 child, 2.3E-05 adult    | 0.1 7 CSF 7                             | No human cancer-effect values |

1. Value is an ATSDR non-cancer health effect comparison value unless otherwise noted. Comparison values are used to screen well water data and select detections for further study.
2. Values are ATSDR verified non-cancer health study values unless otherwise noted. These values used to screen exposure dose estimated for further evaluation. “Animal” denotes values derived from an animal study, “human” denotes values derived from human studies.
3. EPA Regional Screening Level for TCE
4. N.C. DENR Ground Water Standard
5. Proposed value (EPA 2001)
6. PAH data evaluated as benzo(a)pyrene-equivalent concentrations of all detected PAH compounds
7 EPA Cancer Slope Factor, unit of (mg/kg/d)^1
8 Human no adverse effect level for gastrointestinal effects
9 Human lowest adverse effect level for gastrointestinal effects
10 Human no adverse effect level for liver effects
11 The RfD for TTHMs and chloroform is considered protective for cancer effects

AL = Action Level
J = sample concentration is an estimated value
MCL = Maximum Contaminant Level, an EPA regulatory level for public drinking water systems
MCLG = Maximum Contaminant Level Goal, an EPA non-regulatory level for public drinking water systems
NA = not applicable
PW = private well
RfD = Reference dose, an EPA chronic oral exposure screening value
TTHM = total trihalomethanes (includes bromoform, chloroform, dibromochloromethane, bromodichloromethane)
THM = trihalomethanes (includes bromoform, chloroform, dibromochloromethane, bromodichloromethane)
TCE = trichloroethylene
Table 3. Estimates of the additional numbers of cancers for persons exposed at the indicated well water concentration for a 30 years period. Private well samples collected around the Mills Gap Road area from June 2008 through January 2010.

<table>
<thead>
<tr>
<th>Substance</th>
<th>Well Water Exposure Concentration (µg/L)</th>
<th>Number of Additional Cancers (^1) Predicted in 1 Million Exposed Persons</th>
<th>Number of Persons Exposed to Predict 1 Additional Cancer (^1)</th>
<th>Qualitative Additional Cancer Risk</th>
</tr>
</thead>
<tbody>
<tr>
<td>Arsenic</td>
<td>0.65 J</td>
<td>Less than 1</td>
<td>More than 2.7 million</td>
<td>No risk</td>
</tr>
<tr>
<td>Bis(2-chloro-ethyl)ether</td>
<td>2.7 J</td>
<td>36</td>
<td>28,000</td>
<td>Low risk</td>
</tr>
<tr>
<td>PAHs</td>
<td>0.036</td>
<td>3</td>
<td>310,000</td>
<td>Very low risk</td>
</tr>
<tr>
<td></td>
<td>0.016</td>
<td>1</td>
<td>690,000</td>
<td>Very low risk</td>
</tr>
<tr>
<td></td>
<td>0.118</td>
<td>10</td>
<td>94,000</td>
<td>Low risk</td>
</tr>
<tr>
<td>TCE</td>
<td>60</td>
<td>15 to 300</td>
<td>3300 to 67,000</td>
<td>Low to moderate risk</td>
</tr>
<tr>
<td></td>
<td>20</td>
<td>5 to 98</td>
<td>10,000 to 200,000</td>
<td>Very low to moderate risk</td>
</tr>
<tr>
<td></td>
<td>8.8</td>
<td>2 to 43</td>
<td>23,000 to 450,000</td>
<td>Very low to low risk</td>
</tr>
<tr>
<td></td>
<td>2.8</td>
<td>Less than 1 to 14</td>
<td>71,000 to 1.4 million</td>
<td>Very low to low risk</td>
</tr>
<tr>
<td></td>
<td>1200</td>
<td>290 to 5900</td>
<td>170 to 3400</td>
<td>Moderate to high risk</td>
</tr>
<tr>
<td>Carbon tetrachloride</td>
<td>0.29 J</td>
<td>Less than 1</td>
<td>More than 2.1 million</td>
<td>No risk</td>
</tr>
<tr>
<td>THMs –</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Bromoform</td>
<td>2.9</td>
<td>2</td>
<td>450,000</td>
<td>Very low risk</td>
</tr>
<tr>
<td>DBCM</td>
<td>11</td>
<td>1</td>
<td>900,000</td>
<td>Very low risk</td>
</tr>
<tr>
<td>n-Nitrosodi-n-propylamine</td>
<td>2.1</td>
<td>2</td>
<td>450,000</td>
<td>Very low risk</td>
</tr>
<tr>
<td></td>
<td>0.82 J</td>
<td>70</td>
<td>14,000</td>
<td>Low risk</td>
</tr>
</tbody>
</table>

\(^{1}\) “Additional cancers” = an estimate of the increased number of cancer cases over the background number of cases anticipated in a typical population.

J = sample concentration is an estimated value.
impaired nerve function causing a “pins and needles” sensation in your hands and feet. Long-
term oral exposure to inorganic arsenic may result in a pattern of skin changes. These include
patches of darkened skin and the appearance of small “corns” or “warts” on the palms, soles, and
torso, and are often associated with changes in the blood vessels of the skin. Skin cancer may
also develop. Swallowing arsenic has also been reported to increase the risk of cancer in the
liver, bladder, and lungs. 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 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. Thus, all health effects observed in adults are of potential concern in
children. 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.

There is some evidence that inhaled or ingested inorganic arsenic can injure pregnant women or
their unborn babies, although the studies are not definitive. Studies in animals show that large
doses of inorganic arsenic that cause illness in pregnant females can also cause low birth weight,
fetal malformations, and even fetal death. Arsenic can cross the placenta and has been found in
fetal tissues. Arsenic is found at low levels in breast milk [ATSDR 2007].

**Child Health Concerns**

In communities faced with water, air, or food contamination, the many physiological differences
between children and adults demand special emphasis. Children can be at greater risk than are
adults from certain kinds of exposure to hazardous substances. Children play outdoors and
sometimes engage in hand-to-mouth behaviors that increase their exposure potential. Children
are shorter than are adults; this means they are more likely to breathe dust, soil, and vapors close
to the ground. A child’s lower body weight and higher intake rate result in a greater dose of
hazardous substance per unit of body weight. If toxic exposures occur during critical growth
stages, the developing body systems of children can sustain permanent damage. Probably most
important, however, is that children depend on adults for risk identification and risk
management, housing, and access to medical care. Thus, adults should be aware of public health
risks in their community, so they can guide their children accordingly. Child-specific exposure
situations and health effects are taken into account in N.C. DPH health effect evaluations.

Children are more vulnerable than adults to lead poisoning if exposed to high levels of lead
within the environment [ATSDR Cu-Pb]. No safe blood lead level in children has been
determined. Lead affects children in different ways depending on the level of exposure. 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. Fetal exposure to
lead is associated with premature birth and low birth weight. Fetal and early childhood exposure to lead has also been linked to decreased cognitive development and reduced intelligence in early childhood, and evidence suggests that these effects may persist into adulthood. 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 in a child is to have their blood tested. A test to measure the amount of lead in the blood is called the Blood Lead Level test. The CDC recommends that a child’s blood lead level be no higher than 10 μg/dL (micrograms of lead per deciliter of blood). If a child’s blood lead level measures higher than 10 μg/dL precautions should be taken in order to reduce that child’s exposure to lead [ATSDR 2007d].

Community Health Concerns

The community living around the Mills Gap Road area has expressed concerns regarding the potential for adverse health effects, including cancer, to persons drinking contaminated well water, using contaminated well water for bathing or other household uses, and breathing air containing volatile contaminants associated with the site.

Conclusions

Private well samples have been collected in a radius around the CTS/Mills Gap Road Site since November 2007. Private well samples collected through January 2008 were previously discussed in the CTS/Mills Gap Road Public Health Assessment Initial/Public Release on January 19, 2010. In this Health Consultation N.C DPH reviewed environmental data for private well samples collected from June 2008 through January 2010 around the Mills Gap Road area and reached the following conclusions:

- Drinking or breathing the TCE in the well water over many years at the location discovered with 1200 micro-gram per liter (μg/L) in August 2009 could have harmed people’s health. TCE contamination was discovered in the private well serving 2 homes in a sample collected in August 2009. The well was disconnected at that time. Residents indicated that family members had used the well for as long as 20 years.
  - It is not known when the well was first contaminated or the TCE concentrations over the extent of the contamination period.
  - An increased theoretical risk of cancer and non-cancer health effects is indicated if persons were using the well water over many years with a TCE concentration similar to that observed in the August 2009 sample. Drinking water contaminated with TCE and breathing TCE volatilized from the drinking water supply over many years in large amounts may cause adverse health effects. These effects include a theoretical increased risk of kidney or liver cancer; dizziness, lung irritation, impaired heart function; and nerve, kidney or liver damage. Reproductive effects such as impaired fetal growth or decreased fertility may also result. There may be an increased risk of birth defects or leukemia to children of women exposed during pregnancy.

- DPH cannot conclude if drinking TCE contaminated well water from 4 wells identified in the Oaks neighborhood could have harmed people’s health. It is not known how long the water was contaminated and at what concentrations over the entire time period of
contamination. Harmful health effects are not indicated at the reported TCE concentrations.

- Four wells were identified in 2007 and 2008 in the Oaks neighborhood with TCE contamination (TCE concentrations: 60 µg/L, 20 µg/L, 8.8 µg/L, 2.8 µg/L). All the wells were disconnected. It is not known when the wells were first contaminated or the TCE concentrations over time.

- DPH cannot conclude if drinking water from a PAH (polycyclic aromatic hydrocarbons) contaminated well detected in September 2008 in the Oaks neighborhood (at 0.118 µg/L benzo(a)pyrene-equivalent concentration) could have harmed resident’s health. It is not known how long the water was contaminated and at what concentrations over the entire time period of contamination. Harmful health effects are not indicated at the reported TCE concentrations.

- Total PAHs at 0.118 µg/L benzo(a)pyrene-equivalent concentration were detected in a single sample collected from the well before it was closed. This concentration is less than the regulatory level for public water systems (0.2 µg/L MCL). It is not known when the well was first contaminated or the PAH concentrations over the extent of the contamination period.

- DPH cannot currently conclude if drinking well water from 2 wells with elevated copper concentrations could cause short-term health effects to children sensitive to low levels of copper.

- The copper levels in these 2 wells do not exceed action levels for public water systems and long-term permanent health effects are not indicated. While the health literature indicates possible temporary gastrointestinal effects at copper levels in the range observed in these 2 wells, DPH has observed these effects at levels more than 5 times higher. DPH is concerned that children sensitive to copper could suffer temporary effects by exposure to high levels of copper in these 2 wells if the water sits in the well delivery system for periods greater than several hours with no flushing.

- DPH can not currently conclude if drinking water from 3 wells for several years with elevated lead concentrations could harm children’s health.

- Lead concentrations greater than the health guideline value referenced by the DPH for private well water supplies (15 µg/L) was detected in 3 wells. While adverse health effects to children were not indicated on the basis of the well water concentrations, only blood lead levels can confirm that children have not accumulated harmful concentration of lead from the well waters and other sources. The EPA is concerned that the intermittent elevated lead concentrations in these 3 wells may be coming from the water pipes and are not from the groundwater.

- DPH concludes that drinking waters from the other wells sampled in the area over many years is not expected to harm people’s health.

- Other substances (carbon tetrachloride, cis-1,2-dichloroethene, vinyl chloride, arsenic, trihalomethanes, bis(2-chloroethyl)ether, antimony and n-nitroso-di-n-propylamine) detected in the private well water samples reviewed for this evaluation were not present at concentrations high enough to cause adverse health effects.
Recommendations

The N.C. DPH makes the following recommendations:

- Persons living in the homes served by the well where 1200 µg/L trichloroethylene (TCE) was detected in August 2009 should make their personal physicians aware of their exposure and monitor their health to detect effects related to TCE exposure. Health monitoring should include: complete blood count (CBC), liver and kidney function tests and urinalysis. N.C. DPH Public Health Physicians can provide additional guidance to medical personnel to identify negative health effects associated with the TCE exposure.

- Inform persons that were exposed in the Oak neighborhood before the wells were disconnected to TCE detections greater than 2.8 µg/L of the potential health effects. Provide recommendations for follow-up with their personal physicians and continued health monitoring for effects associated with long-term TCE ingestion. N.C. DPH Public Health Physicians can provide additional guidance to medical personnel to identify negative health effects associated with the TCE exposures.

- Continue to monitor groundwater contaminants moving away from the CTS site and identify points where the community may come into contact with the contaminants, such as private wells and surface waters.

- Identify other possible sources of well water contamination in the area.

- Continue to test private wells until the contamination sources and impacted areas are characterized and controlled.

- Provide clean alternative drinking water sources to persons impacted by groundwater contamination of their private well that exceed regulatory values used for public water systems or health guideline values. Reduce exposure to carcinogens to achieve a long-term cancer risk goal of less than 1 additional cancer in 1 million persons exposed.

- Inform residents exposed to the 0.118 µg/L benzo(a)pyrene-equivalent concentration of PAHs before the well was disconnected of the potential health effects and provide recommendations for follow-up with their personal physicians.

- 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.

- Inform persons that live at the residences where there were lead detections greater than the DPH 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. Provide assistance in determination if the lead is leaching from their water lines.

- Provide assistance to residents exposed to the elevated copper and lead to determine if the water pipes are a possible source. Provide alternatives for reducing their exposure if the lead and copper is a result of contamination from the well water lines. Flush the water lines for several minutes prior to collecting water to be used for drinking or cooking if other alternatives are not implemented.

- Monitor vinyl chloride moving away from the site in groundwater to identify potential exposure points including private wells and discharges to surface water.
Evaluate the integrity of the septic and private well systems in the areas where the trihalomethane chemicals were detected (bromoform, bromodichloromethane and dibromochloromethane). Communicate the issues and implications to the residents.

Public Health Action Plan

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

A. Public health actions completed:

- N.C. DPH has evaluated private well analytical data and health effects information to determine the potential for the health of the local community to be adversely impacted by substances identified in the private well waters.

B. Public health actions planned:

- The Health Consultation (HC) will be made available to the community, Buncombe County officials, N.C. DENR and EPA. A factsheet will also be prepared to summarize N.C. DPH's findings and recommendations specified in the Health Consultation. These documents and any accompanying information will be accessible from the N.C. DPH HACE web site. The Health Consultation will also be available from the ATSDR web site.
- N.C. DPH will work with Buncombe County Health Department to evaluate and respond to the concerns identified in the Health Consultation.
- N.C. DPH and Buncombe County staff will contact persons using the wells with potential adverse health impacts identified in the Health Consultation and provide assistance in eliminating or reducing their exposure and seeking appropriate medical follow-up.
- N.C. DPH will continue to review private well water data collected in association with the Mills Gap Road area.
- N.C. DPH staff will be available to discuss specifics regarding the private wells discussed in this Health Consultation.
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CERTIFICATION

This Public Health Consultation for the Private Well Waters from the Mills Gap Road Area of Skyland N.C. was prepared by the North Carolina Division of Public Health (N.C. DPH) under a cooperative agreement with the Federal Agency for Toxic Substances and Disease Registry (ATSDR). It is in accordance with approved methodology and procedures existing at the time the health consult and update was initiated. Editorial review was completed by the cooperative agreement partner.

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

[Signature]
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References:

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http://www.epa.gov/safewater/contaminants/index.html#mcls


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http://umbbd.msi.umn.edu/index.html
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Appendix A

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/kg [mg/kg = 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:

\( ED_w \) = exposure dose water (mg/kg/day)  
\( C \) = contaminant concentration (mg/kg)  
\( IR \) = 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 [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:

\( ED_s \) = exposure dose soil (mg/kg/day)  
\( C \) = contaminant concentration (mg/kg)  
\( IR \) = intake rate of contaminated medium (kilograms/day)  
\( EF \) = 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 (mg/m³) or parts per billion by volume (ppbv). The following equation is used to estimate the exposure doses resulting from inhalation of contaminated air.

\[
D = \frac{(C \times IR \times EF)}{BW}
\]

Where:
- \(D\) = exposure dose (mg/kg/day)
- \(C\) = contaminant concentration (mg/m³)
- \(IR\) = intake rate (m³/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).

\[
C_a = \frac{(C_w \times f \times F_w \times t)}{V_a}
\]

Where:
- \(C_a\) = bathroom air concentration (mg/m³)
- \(C_w\) = tap water concentration (mg/L)
- \(f\) = fractional volatilization rate (unitless)
- \(F_w\) = shower water flow rate (L/min)
- \(t\) = exposure time (min)
- \(V_a\) = bathroom volume (m³)

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 m³. 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.

**Polycyclic Aromatic Hydrocarbons (PAHs)**

ATSDR does not provide individual comparison values (CVs) for the group of structurally
related multi-carbon ring compounds known as polycyclic aromatic hydrocarbons or PAHs
(PAHs may also be called “polynuclear aromatic hydrocarbons” or “polyaromatic
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 calculated 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.

\[
PAH_{BaP-equ} = PAH_{conc} \times TEF
\]

\[
\text{Combined Cancer Risk}_{PAHs} = \sum PAH_{adj} \times CSF
\]
Where:

\[ \text{PAH}_{\text{BaP-eq}} = \text{Benzo(a)pyrene equivalent TEF adjusted PAH compound concentration, mg/kg} \]

\[ \text{PAH}_{\text{conc}} = \text{concentration of PAH compound, mg/kg} \]

\[ \text{TEF} = \text{Toxicity Equivalency Factor for PAH compound, unitless} \]

\[ \text{Combined Cancer Risk}_{\text{PAHs}} = \text{Summed cancer risk of all detected PAH compounds} \]

\[ \sum \text{PAH}_{\text{adj}} = \text{summed TEF-adjusted concentrations of all detected PAH compounds, mg/kg} \]

\[ \text{CSF} = \text{Cancer Slope Factor, mg/kg-d} \]

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

<table>
<thead>
<tr>
<th>PAH compounds</th>
<th>TEF value</th>
</tr>
</thead>
<tbody>
<tr>
<td>acenaphthene</td>
<td>0.001</td>
</tr>
<tr>
<td>acenaphthylene</td>
<td>0.001</td>
</tr>
<tr>
<td>anthracene</td>
<td>0.01</td>
</tr>
<tr>
<td>benzo(a)anthracene</td>
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</tr>
<tr>
<td>benzo(a)pyrene</td>
<td>1.00</td>
</tr>
<tr>
<td>benzo(b,k)fluoranthene</td>
<td>na</td>
</tr>
<tr>
<td>benzo(g,h,i)perylene</td>
<td>0.01</td>
</tr>
<tr>
<td>benzo(b)fluoranthene</td>
<td>0.1</td>
</tr>
<tr>
<td>benzo(k)fluoranthene</td>
<td>0.01</td>
</tr>
<tr>
<td>chrysene</td>
<td>0.001</td>
</tr>
<tr>
<td>dibenz(a,h)anthracene</td>
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<tr>
<td>fluoranthene</td>
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</tr>
<tr>
<td>fluorene</td>
<td>0.001</td>
</tr>
<tr>
<td>indeno(1,2,3-cd)pyrene</td>
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</tr>
<tr>
<td>2-methylnaphthalene</td>
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<td>naphthalene</td>
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<tr>
<td>phenanthrene</td>
<td>0.001</td>
</tr>
<tr>
<td>pyrene</td>
<td>0.001</td>
</tr>
</tbody>
</table>


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 N.C. Central Cancer Registry states:

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

<table>
<thead>
<tr>
<th>Estimated Number of Increased Cancers ¹</th>
<th>Qualitative Increased Risk Term</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;1/1,000,000</td>
<td>No Increase</td>
</tr>
<tr>
<td>&lt;1/100,000</td>
<td>Very Low</td>
</tr>
<tr>
<td>&lt;1/10,000</td>
<td>Low</td>
</tr>
<tr>
<td>&lt;1/1,000</td>
<td>Moderate</td>
</tr>
<tr>
<td>&lt;1/100</td>
<td>High</td>
</tr>
<tr>
<td>&gt;1/100</td>
<td>Very High</td>
</tr>
</tbody>
</table>

¹ 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.

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.

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:

\[ \text{HQ} = \frac{\text{estimated dose}}{\text{applicable health guideline}} \]

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:

\[
HI = HQ_1 + HQ_2 + HQ_3 + \ldots + HQ_n
\]

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 x 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.

**Reference:**
Appendix B

Current and Proposed Trichloroethene Environmental Screening and Health Effects Values
The current ATSDR drinking water CV for trichloroethene (TCE) is 5 µg/L MCL. Current
ASTDR CV cancer classifications are listed as “under review” (EPA), “reasonably anticipated to
be a carcinogen” (NTP), and “probably carcinogenic to humans” (IARC) (ATSDR 2008 HG).
The current ATSDR health guideline is an acute oral MRL (non-cancer effect) of 0.2 (mg/kg/d)⁻¹
(ATSDR 2008 HG). The ATSDR health guideline includes a reference to a EPA draft study that
proposes changing the TCE oral reference dose (RfD) to 0.0003 mg/kg/d and setting a cancer
slope factor (CSF) of 0.02 to 0.4 (mg/kg/d)⁻¹ (EPA 2001). Table 2 summarizes current and
proposed TCE screening values.

It is not known if drinking water contaminated with TCE causes non-cancer illness in humans.
Childhood leukemia has been observed after maternal exposure to TCE-contaminated drinking
water during the prenatal period. Evidence from animal and epidemiological studies also suggest
that exposure to TCE might be associated with congenital heart defects and poor intrauterine
growth. Studies in rats and mice show that trichloroethylene can affect fertility, but the
relevance to humans is not clear. Human epidemiological studies have been limited by
difficulties in estimating exposure levels and by the presence of other solvents with similar toxic
effects. In rats and mice, TCE begins affecting the liver, kidney, and developing fetus at doses
as low as 1 mg/kg/day. These studies are limited, however, by inadequate characterization of
exposure, inadequate quantification of results, or lack of endpoints suitable for deriving chronic
endpoints (EPA 2001).

The National Toxicology Program reviewed the carcinogenicity of TCE and concluded:

“Trichloroethylene (TCE) is reasonably anticipated to be a human carcinogen based on
limited evidence of carcinogenicity from studies in humans, sufficient evidence of
carcinogenicity from studies in experimental animals, which indicates there is an
increase incidence of malignant and/or a combination of malignant and benign tumors
at multiple tissue sites in multiple species of experimental animals and information
suggesting TCE acts through mechanisms that indicate it would likely cause cancer in
humans.” (NTP 2005)

In their 2001 draft assessment, EPA also reviewed the risk of cancer from exposure to TCE and
concluded:

“Epidemiological studies, considered as a whole, have associated TCE exposures with
excess risk of kidney cancer, liver cancer, lympho-hematopoietic cancer, cervical
cancer, and prostate cancer. TCE has been extensively tested in animals, with mice
developing liver tumors, lung tumors, and lymphomas, and rats developing kidney
tumors and testicular tumors. The epidemiologic evidence is strongest at sites where
the animals develop cancer, with site concordance for kidney cancer (in rats and
humans), liver cancer (in mice and humans), and lympho-hematopoietic cancer (in mice
and humans). TCE is also associated with cervical cancer and prostate cancer in
humans, sites for which there are no corresponding animal models.” (EPA 2001)
In 2006, the National Research Council (NRC) found that the evidence on carcinogenic risk and other health hazards from exposure to TCE has strengthened since 2001. The NRC found that enough credible human health information exists and recommended finalizing EPA’s 2001 draft risk assessment (NRC 2006).

In keeping with N.C. DPH’s and ATSDR’s conservative approach to public health assessments, the uncertainties of levels of TCE health effects, and the significant decrease in proposed TCE screening values, in this assessment N.C. DPH included evaluation of site TCE concentrations to the proposed lower screening values and applied the range of proposed cancer slope factors to calculate theoretical increased cancer risks.
Appendix C

N.C. DPH Fact Sheet -
Cancer and the Environment
Cancer and the Environment

Getting a diagnosis of cancer or learning that a loved one has been diagnosed with cancer can be devastating. It is usually at this moment that we become more aware of other people close to us who are also touched by this disease. It is natural to wonder why so many people around us seem to be suffering from cancer, and if there could be a connection to chemicals in the environment where you live and work.

Cancer is more common than most people realize.
In the U.S., cancer affects approximately 1 in 2 men and 1 in 3 women in their lifetime. An estimated 40% of North Carolinians will develop cancer in their lifetime.

Cancer is more likely to occur as people get older. Because people are living longer, more cases of cancer can be expected in the future. This increased life expectancy may create the impression that cancer is becoming much more common, even though an increase in the number of cases of cancer is related in large part to the growing number of elderly people in the population.

Cancer is not one disease.
Cancer is a group of more than 100 different types of conditions characterized by uncontrolled growth and spread of abnormal cells.

Cancer has many different causes.
Different types of cancer have different causes. What changes a breast cell into breast cancer is not the same as what changes a white blood cell...
There are many types of cancer-causing agents, or carcinogens, including some types of viruses and medicines, as well as chemicals and radiation. Cancer is likely to be caused by a combination of factors acting together over many years. These factors include:

- **Hereditary factors** (physical characteristics we inherit from our parents), and
- **Environmental factors**
  - how we live, also called *lifestyle factors*. This includes things we do such as exercise, and smoking, and the things we eat.
  - contact with cancer-causing agents (called "carcinogens").

Environmental factors make up an estimated 75% - 80% of cancer cases and deaths in the U.S. These factors include things we do, such as exercise and smoking, as well as contaminants in the environment.

However, contact with chemicals accounts for only a small percentage of cancer. The American Cancer Society estimates that contact with cancer-causing agents in workplaces causes about 4% of cancer, and contact with pollutants in non-workplace settings causes about 2%.

Different factors or combinations of factors can cause the same type of cancer. For example, one person’s breast cancer might be related to hereditary factors acting in combination with taking hormone pills prescribed by a doctor. Yet another person’s breast cancer might be caused by hereditary factors in combination with contact with chemicals during puberty.
Cancers today are usually related to events that happened many years ago. Cancer usually does not develop immediately after you have come in contact with a cancer-causing agent. Instead, it may take years, if not decades, between contact with a cancer-causing agent for you to be diagnosed with cancer. This delay between possible contact and the development of cancer often makes it difficult to determine which agent may have caused the cancer.

**How do I reduce my risk of cancer?**
- Avoid tobacco use
- Avoid excessive alcohol use
- Avoid excessive sun exposure
- Increase physical activity
- Maintain a recommended body weight
- Eat a healthy and nutritious diet
- Take advantage of cancer screenings

You can reduce your risk for developing cancer.

The American Cancer Society estimates that about 30% of cancer could be prevented by eliminating tobacco use, and another 35% could be prevented by reducing obesity, increasing physical activity, and eating a healthy diet.

**Cancer Clusters**

A *cancer cluster* is a greater-than-expected number of cancer cases that occur within a group of people in a geographic area over a period of time.

A cancer cluster usually involves:
- one type of cancer;
- a rare type of cancer;
- cancers diagnosed over a short period of time;
- groups of cases among children, or a type of cancer that is not usually found in a particular age group.

A greater than expected number of cancer cases could be the result of a variety of reasons, including chance. For example, it is like getting heads every time you flip a coin. You can get heads six times in a row although the probability of it happening is very small.
Even when scientist and/or the medical community identifies a true cancer cluster, there is usually no single external cause or cancer-causing agent that can be identified.

Trying to identify a cause for a cancer cluster has proven to be extremely difficult. Extensive follow-up investigations can be done, but these often take years to complete and require extensive research. In most instances, even when these activities are conducted, no cause is found.

Clusters of cancer cases have been identified in North Carolina. However, there have been no cases where a cluster of cancers was proven to occur as a result of an environmental exposure.

The NC Central Cancer Registry monitors cancer rates throughout the state. www.epi.state.nc.us/SCHS/CCR/

You can read more about how cancer is investigated in communities by visiting the CDC Web site at www.cdc.gov/nceh/clusters/faq.htm

You can report a suspected cancer cluster or obtain information on cancer statistics for your area. Contact your local health department at www.nchalhd.org/county.htm, or the N.C. Central Cancer Registry at (919) 715-4574. These agencies provide the first level of response and the most current data to answer your questions.

For more information
Centers for Disease Control and Prevention www.cdc.gov/nceh/clusters/faq.htm

National Cancer Institute www.cancer.gov/cancertopics/factsheet/Risk/clusters

Cancer Clusters, American Cancer Society www.cancer.org/docroot/PED/content/PED_1_3x_Cancer_Clusters.asp?sitearea=PED
Appendix D

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

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

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

Additive effect
A biologic response to exposure to multiple substances that equals the sum of responses of all the individual substances added together [compare with antagonistic effect and synergistic effect].

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

Aerobic
Requiring oxygen [compare with anaerobic].

Ambient
Surrounding (for example, ambient air).

Anaerobic
Requiring the absence of oxygen [compare with aerobic].

Analyte
A substance measured in the laboratory. A chemical for which a sample (such as water, air, or blood) is tested in a laboratory. For example, if the analyte is mercury, the laboratory test will determine the amount of mercury in the sample.

Analytic epidemiologic study
A study that evaluates the association between exposure to hazardous substances and disease by testing scientific hypotheses.

Antagonistic effect
A biologic response to exposure to multiple substances that is less than would be expected if the known effects of the individual substances were added together [compare with additive effect and synergistic effect].

Background level
An average or expected amount of a substance or radioactive material in a specific environment, or typical amounts of substances that occur naturally in an environment.
Biodegradation
Decomposition or breakdown of a substance through the action of microorganisms (such as bacteria or fungi) or other natural physical processes (such as sunlight).

Biologic indicators of exposure study
A study that uses (a) biomedical testing or (b) the measurement of a substance [an analyte], its metabolite, or another marker of exposure in human body fluids or tissues to confirm human exposure to a hazardous substance [also see exposure investigation].

Biologic monitoring
Measuring hazardous substances in biologic materials (such as blood, hair, urine, or breath) to determine whether exposure has occurred. A blood test for lead is an example of biologic monitoring.

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

Biomedical testing
Testing of persons to find out whether a change in a body function might have occurred because of exposure to a hazardous substance.

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

Body burden
The total amount of a substance in the body. Some substances build up in the body because they are stored in fat or bone or because they leave the body very slowly.

CAP
See Community Assistance Panel.

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

Cancer risk
A theoretical risk of for getting cancer if exposed to a substance every day for 70 years (a lifetime exposure). The true risk might be lower.

Carcinogen
A substance that causes cancer.

Case study
A medical or epidemiologic evaluation of one person or a small group of people to gather information about specific health conditions and past exposures.

Case-control study
A study that compares exposures of people who have a disease or condition (cases) with people who do not have the disease or condition (controls). Exposures that are more common among the cases may be considered as possible risk factors for the disease.
CAS registry number
A unique number assigned to a substance or mixture by the American Chemical Society Abstracts Service.

Central nervous system
The part of the nervous system that consists of the brain and the spinal cord.
CERCLA [see Comprehensive Environmental Response, Compensation, and Liability Act of 1980]

Chronic
Occurring over a long time (more than 1 year) [compare with acute].

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

Cluster investigation
A review of an unusual number, real or perceived, of health events (for example, reports of cancer) grouped together in time and location. Cluster investigations are designed to confirm case reports; determine whether they represent an unusual disease occurrence; and, if possible, explore possible causes and contributing environmental factors.

Community Assistance Panel (CAP)
A group of people, from a community and from health and environmental agencies, who work with ATSDR to resolve issues and problems related to hazardous substances in the community. CAP members work with ATSDR to gather and review community health concerns, provide information on how people might have been or might now be exposed to hazardous substances, and inform ATSDR on ways to involve the community in its activities.

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

Completed exposure pathway [see exposure pathway].

Comprehensive Environmental Response, Compensation, and Liability Act of 1980 (CERCLA)
CERCLA, also known as Superfund, is the federal law that concerns the removal or cleanup of hazardous substances in the environment and at hazardous waste sites. ATSDR, which was created by CERCLA, is responsible for assessing health issues and supporting public health activities related to hazardous waste sites or other environmental releases of hazardous substances.

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

Contaminant
A substance that is either present in an environment where it does not belong or is present at levels that might cause harmful (adverse) health effects.
Delayed health effect
A disease or injury that happens as a result of exposures that might have occurred in the past.

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

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

Descriptive epidemiology
The study of the amount and distribution of a disease in a specified population by person, place, and time.

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

Disease prevention
Measures used to prevent a disease or reduce its severity.

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

DOD
United States Department of Defense.

DOE
United States Department of Energy.

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

Dose (for radioactive chemicals)
The radiation dose is the amount of energy from radiation that is actually absorbed by the body. This is not the same as measurements of the amount of radiation in the environment.

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

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

Environmental media and transport mechanism
Environmental media include water, air, soil, and biota (plants and animals). Transport mechanisms move contaminants from the source to points where human exposure can occur.
The ongoing, systematic collection, analysis, and interpretation of health data. This activity also involves timely dissemination of the data and use for public health programs.

Epidemiology
The study of the distribution and determinants of disease or health status in a population; the study of the occurrence and causes of health effects in humans.

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

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

Exposure-dose reconstruction
A method of estimating the amount of people’s past exposure to hazardous substances. Computer and approximation methods are used when past information is limited, not available, or missing.

Exposure investigation
The collection and analysis of site-specific information and biologic tests (when appropriate) to determine whether people have been exposed to hazardous substances.

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

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

Feasibility study
A study by EPA to determine the best way to clean up environmental contamination. A number of factors are considered, including health risk, costs, and what methods will work well.

Geographic information system (GIS)
A mapping system that uses computers to collect, store, manipulate, analyze, and display data. For example, GIS can show the concentration of a contaminant within a community in relation to points of reference such as streets and homes.

Grand rounds
Training sessions for physicians and other health care providers about health topics.
Groundwater
Water beneath the earth’s surface in the spaces between soil particles and between rock surfaces [compare with surface water].

Half-life (t½)
The time it takes for half the original amount of a substance to disappear. In the environment, the half-life is the time it takes for half the original amount of a substance to disappear when it is changed to another chemical by bacteria, fungi, sunlight, or other chemical processes. In the human body, the half-life is the time it takes for half the original amount of the substance to disappear, either by being changed to another substance or by leaving the body. In the case of radioactive material, the half life is the amount of time necessary for one half the initial number of radioactive atoms to change or transform into another atom (that is normally not radioactive). After two half lives, 25% of the original number of radioactive atoms remains.

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

Hazardous Substance Release and Health Effects Database (HazDat)
The scientific and administrative database system developed by ATSDR to manage data collection, retrieval, and analysis of site-specific information on hazardous substances, community health concerns, and public health activities.

Hazardous waste
Potentially harmful substances that have been released or discarded into the environment.

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

Health education
Programs designed with a community to help it know about health risks and how to reduce these risks.

Health investigation
The collection and evaluation of information about the health of community residents. This information is used to describe or count the occurrence of a disease, symptom, or clinical measure and to estimate the possible association between the occurrence and exposure to hazardous substances.

Health promotion
The process of enabling people to increase control over, and to improve, their health.
**Health statistics review**
The analysis of existing health information (i.e., from death certificates, birth defects registries, and cancer registries) to determine if there is excess disease in a specific population, geographic area, and time period. A health statistics review is a descriptive epidemiologic study.

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

**Incidence**
The number of new cases of disease in a defined population over a specific time period [contrast with prevalence].

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

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

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

**In vitro**
In an artificial environment outside a living organism or body. For example, some toxicity testing is done on cell cultures or slices of tissue grown in the laboratory, rather than on a living animal [compare with in vivo].

**In vivo**
Within a living organism or body. For example, some toxicity testing is done on whole animals, such as rats or mice [compare with in vitro].

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

**Maximum Contaminant Level (MCL)**
The highest level of a contaminant that EPA allows in drinking water. MCLs ensure that drinking water does not pose either a short-term or long-term health risk. EPA sets MCLs at levels that are economically and technologically feasible. Some states set MCLs which are more strict than EPA's.
Medical monitoring
A set of medical tests and physical exams specifically designed to evaluate whether an individual’s exposure could negatively affect that person’s health.

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

Metabolite
Any product of metabolism.

mg/kg
Milligram per kilogram.

mg/cm²
Milligram per square centimeter (of a surface).

mg/m³
Milligram per cubic meter; a measure of the concentration of a chemical in a known volume (a cubic meter) of air, soil, or water.

Migration
Moving from one location to another.

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

Morbidity
State of being ill or diseased. Morbidity is the occurrence of a disease or condition that alters health and quality of life.

Mortality
Death. Usually the cause (a specific disease, condition, or injury) is stated.

Mutagen
A substance that causes mutations (genetic damage).

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

National Priorities List for Uncontrolled Hazardous Waste Sites (National Priorities List or NPL)
EPA’s list of the most serious uncontrolled or abandoned hazardous waste sites in the United States. The NPL is updated on a regular basis.
No apparent public health hazard
A category used in ATSDR’s public health assessments for sites where human exposure to contaminated media might be occurring, might have occurred in the past, or might occur in the future, but where the exposure is not expected to cause any harmful health effects.

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

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

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

Physiologically based pharmacokinetic model (PBPK model)
A computer model that describes what happens to a chemical in the body. This model describes how the chemical gets into the body, where it goes in the body, how it is changed by the body, and how it leaves the body.

Pica
A craving to eat nonfood items, such as dirt, paint chips, and clay. Some children exhibit pica-related behavior.

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

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

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

Potentially responsible party (PRP)
A company, government, or person legally responsible for cleaning up the pollution at a hazardous waste site under Superfund. There may be more than one PRP for a particular site.

ppb
Parts per billion.

ppm
Parts per million.
**Prevalence**
The number of existing disease cases in a defined population during a specific time period [contrast with incidence].

**Prevalence survey**
The measure of the current level of disease(s) or symptoms and exposures through a questionnaire that collects self-reported information from a defined population.

**Prevention**
Actions that reduce exposure or other risks, keep people from getting sick, or keep disease from getting worse.

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

**Public availability session**
An informal, drop-by meeting at which community members can meet one-on-one with ATSDR staff members to discuss health and site-related concerns.

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

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

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

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

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

**Public health statement**
The first chapter of an ATSDR toxicological profile. The public health statement is a summary written in words that are easy to understand. The public health statement explains how people might be exposed to a specific substance and describes the known health effects of that substance.
Public meeting
A public forum with community members for communication about a site.

Radioisotope
An unstable or radioactive isotope (form) of an element that can change into another element by giving off radiation.

Radionuclide
Any radioactive isotope (form) of any element.

RCRA [See Resource Conservation and Recovery Act (1976, 1984)]

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

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

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

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

This Act regulates management and disposal of hazardous wastes currently generated, treated, stored, disposed of, or distributed.

RFA
RCRA Facility Assessment. An assessment required by RCRA to identify potential and actual releases of hazardous chemicals.

RfD See reference dose

Risk
The probability that something will cause injury or harm.

Risk reduction
Actions that can decrease the likelihood that individuals, groups, or communities will experience disease or other health conditions.

Risk communication
The exchange of information to increase understanding of health risks.

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

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

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

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

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

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

Stakeholder
A person, group, or community who has an interest in activities at a hazardous waste site.

Statistics
A branch of mathematics that deals with collecting, reviewing, summarizing, and interpreting data or information. Statistics are used to determine whether differences between study groups are meaningful.

Substance
A chemical.

Substance-specific applied research
A program of research designed to fill important data needs for specific hazardous substances identified in ATSDR's toxicological profiles. Filling these data needs would allow more accurate assessment of human risks from specific substances contaminating the environment. This research might include human studies or laboratory experiments to determine health effects resulting from exposure to a given hazardous substance.

Superfund Amendments and Reauthorization Act (SARA)
In 1986, SARA amended CERCLA and expanded the health-related responsibilities of ATSDR. CERCLA and SARA direct ATSDR to look into the health effects from substance exposures at hazardous waste sites and to perform activities including health education, health studies, surveillance, health consultations, and toxicological profiles.

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

Surveillance [see epidemiologic surveillance]
Survey
A systematic collection of information or data. A survey can be conducted to collect information from a group of people or from the environment. Surveys of a group of people can be conducted by telephone, by mail, or in person. Some surveys are done by interviewing a group of people [see prevalence survey].

Synergistic effect
A biologic response to multiple substances where one substance worsens the effect of another substance. The combined effect of the substances acting together is greater than the sum of the effects of the substances acting by themselves [see additive effect and antagonistic effect].

Teratogen
A substance that causes defects in development between conception and birth. A teratogen is a substance that causes a structural or functional birth defect.

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

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

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

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

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

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

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