



Public Health Assessment for

ABERDEEN CONTAMINATED GROUND WATER SITE
ABERDEEN, MOORE COUNTY, NORTH CAROLINA
EPA FACILITY ID: NCN000407447
MARCH 17, 2009

For Public Comment

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

Comment Period Ends:

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This document has previously been provided to EPA and the affected state in an initial release, as required by CERCLA section 104 (i) (6) (H) for their information and review. Where necessary, it has been revised in response to comments or additional relevant information provided by them to ATSDR. This revised document has now been released for a 30-day public comment period. Subsequent to the public comment period, ATSDR will address all public comments and revise or append the document as appropriate. The public health assessment will then be reissued. This will conclude the public health assessment process for this site, unless additional information is obtained by ATSDR which, in the agency’s opinion, indicates a need to revise or append the conclusions previously issued.

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PUBLIC HEALTH ASSESSMENT

ABERDEEN CONTAMINATED GROUND WATER SITE

ABERDEEN, MOORE COUNTY, NORTH CAROLINA

EPA FACILITY ID: NCN000407447

Prepared by:

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Division of Public Health
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Under a Cooperative Agreement with the
U.S. Department of Health and Human Services
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Acronyms

AEGL	Acute Exposure Guideline Level
AT	Averaging time
ATSDR	Agency for Toxic Substances and Disease Registry
CF	Conversion factor
cm	Centimeter
CREG	ATSDR Cancer Risk Evaluation Guide
CR	Contact rate
CV	Comparison Value
DAF	Dermal absorption efficiency
ED	Exposure duration
EF	Exposure frequency
EMEG	ATSDR Environmental Media Evaluation Guide
EPA	U.S. Environmental Protection Agency
EQRR	EQ Resource Recovery
HAZMAT	Hazardous Materials
IRi	Inhalation rate
IURF	Inhalation Unit Risk Factor
Kg	Kilogram
LOAEL	Lowest Observed Adverse Effect Level
MCLG	EPA Maximum Contaminant Level Goal
MCL	EPA Maximum Contaminant Level
M	Meter
mg	milligram
N.C. DENR	North Carolina Dept of Environment and Natural Resources
µg/dL	micro-gram per deci-liter
µg/L	micro-gram per liter
µg/m ³	micro-gram per cubic meter
µg	microgram
ng	nano-gram
NA	Not applicable
N.C. DHHS	North Carolina Dept. of Health and Human Services
NIOSH	National Institute for Occupational Safety and Health
NOAEL	No Observed Adverse Effect Level
PMCLG	EPA Proposed Maximum Contaminant Level Goal
ppm	Parts per million
ppb	Parts per billion
RfC	Reference Concentration
RfD	Reference Dose
SAd	Dermal surface area available for absorption
SAg	Dermal surface area available for ingestion
SVOC	Semi-volatile organic compound
VOC	Volatile organic compound

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

EXECUTIVE SUMMARY

The Aberdeen Contaminated Ground Water (CGW) site describes a general area east of Aberdeen, North Carolina in Moore County (Figure 1). The former Lee Paving site and the former Geigy Chemical Site are located in close proximity to the site. Previous investigations of the Lee Paving and Geigy Chemical sites found contamination related to these sites, but also found chlorinated compound contamination (primarily trichloroethene, "TCE") apparently originating from a third source. The former Powdered Metal Products (PMP) facility (now Diamond Exhaust Products) is thought to have used chlorinated solvents and is suspected as a potential source of the chlorinated compound contamination (USEPA 2008 HRS Ref. 12).

The Aberdeen CGW site is located southeast of the corporate limits of the Town of Aberdeen, North Carolina. The site area is a mix of industrial, commercial, and residential uses. Several of the industrialized areas have been investigated for environmental problems. Volatile organic compounds (VOCs), lead and pesticides have been detected in ground water monitoring wells, municipal drinking water supply wells, and private wells in the area of this site.

Trichloroethene (also known as trichloroethylene or "TCE", a VOC) contamination has been detected in a number of private wells and in the Town of Aberdeen municipal water supply wells (numbers 5 and 9) near the site. Most of the private well users in the area were connected to the municipal system from 1990 to 1995. Aberdeen's water supply is a blended system of 17 wells serving 4,655 people. The location of the municipal wells is identified in Figure 2. Each well supplies no more than 40% of the total make-up of 3 to 5 well blends (USEPA 2008 HRS Ref. 23). At least one other nearby municipal well appears to be in the path of the TCE contaminated ground water plume. The municipal system uses blending of waters from multiple wells to maintain TCE concentrations below the Federal regulatory Maximum Concentration Limit (MCL) in its supplied waters.

The North Carolina Division of Public Health (N.C. DPH) has performed a public health assessment for this site. The public health assessment evaluates ground water contamination in the area of this site to determine the potential for past and present adverse human health effects to persons that may be exposed to contaminated ground water. Past and present potential exposed populations include persons using private residential ground water sources, private commercial well users, and persons receiving water from the Town of Aberdeen municipal system. The ground water data evaluated includes samples collected from 1992 through October 2008 from multiple on-site and off-site investigations and supply well monitoring conducted by the Town of Aberdeen.

Based on a review of available ground water analytical data for the Aberdeen Contaminated Ground Water site, dose calculations, and review of health effects information, there is **no apparent health hazard** to persons that at one time used private wells, or to those currently using the Town of Aberdeen municipal system. The category of no apparent health hazard is contingent on continued containment of the TCE contaminant plume. The category is also contingent on the reviewed analytical data being representative of concentrations that exposed persons were actually ingesting, as well as the estimated time periods of exposure being representative of site conditions. Although evaluated ground water contaminant levels do not

indicate the potential for adverse impacts, NCDPH has several recommendations to prevent future exposures to TCE in contaminated ground water. Further monitoring of the TCE plume movement and contaminant concentrations is recommended. It is recommended that any private ground water wells in the area that may still be in service and affected by the contamination noted in this study be identified and tested. It is recommended that those private wells that are in the path of known contamination, or those that show evidence of contamination, be closed and the residence or facility connected to the Town of Aberdeen municipal supply. It is also recommended that further TCE impacts to the Town of Aberdeen municipal supply be controlled or eliminated as much as is practical.

An **indeterminate health hazard** exists for persons that may have ingested TCE contaminated ground waters supplied as drinking water at the PMP industrial facility. TCE concentrations in the PMP well water were at levels that may result in risk of increased numbers of cancers based on comparison to health study data. Actual risks associated with ingestion exposures at the PMP facility is likely much less than the calculated estimates due to the length of possible exposure, variability in exposure concentrations, and the likely reduced daily intake of drinking water.

An **indeterminate health hazard** exists for possible lead in ground water exposures detected in samples collected from 1991 to 1993 in rural residential areas within one mile south and east of the Geigy Chemical site. The maximum lead concentration detected in these samples may present a health hazard if persons were actually ingesting ground water with lead at these elevated levels. Whether persons were using ground water as a source of drinking water in this area, for what length of time, and at what actual concentrations is unknown. If regional lead ground water concentrations that persons may have ingested in this area are better represented by the lead geometric mean concentration, then no hazard would be anticipated.

PURPOSE AND HEALTH ISSUES

The Agency for Toxic Substances and Disease Registry (ATSDR) requested a public health assessment for the Aberdeen Contaminated Ground Water site when the site was placed on the National Priorities List (NPL) in March 2008. The State of North Carolina referred the site to the U.S. Environmental Protection Agency (USEPA) because of increasing levels of trichloroethene (TCE) contamination which are threatening the municipal water supply wells for the Town of Aberdeen, North Carolina. This health assessment evaluated private and municipal well water samples collected by USEPA, the North Carolina Department of the Environment and Natural Resources (N.C. DENR) and the Town of Aberdeen

BACKGROUND

Site Description and History

The Aberdeen Contaminated Ground Water site (USEPA ID NCN 000 407 447) is located southeast of the corporate limits of the Town of Aberdeen along Highway 211 approximately

1½ miles east of Highway 1 in Aberdeen, Moore County, NC (Figure 1). A trichloroethene (TCE) contaminated ground water plume is roughly bordered by Highway 211 to the north, Old Pee Dee Road to the west, Blues Bridge Road to the south and Blues Bridge Road and Crestline Lane to the east. The geographic coordinates for the site are 35.1224° north latitude and 79.4025° west longitude. The site reference point is the most highly contaminated well located on the former Powder Metal Products property (USEPA 2008 HRS Ref. 12). The study area incorporates several other areas that have had environmental investigations completed in the past to investigate unrelated ground water contamination problems (Geigy Chemical Corp., Route 211 Contaminated Wells, Lee Paving Company). TCE contamination has been detected in numerous private wells and in the Town of Aberdeen municipal water supply wells number 5 and 9 near the Aberdeen Contaminated Ground Water site study area. These TCE detections resulted in approximately 56 private well users being connected to municipal supplies between 1990 and 1995. One municipal well has a TCE concentration higher than the Federal Maximum Contaminant Level (MCL) allowed in drinking water (5 micrograms per liter, µg/L). At least one other municipal well lies in the presumed path of the TCE ground water plume. Aberdeen's water supply is a system of 17 wells in five regions, with each region having its own water tank. In 2007, it was estimated that the system served 4,655 people. The municipal water supply is a blended system with no one well supplying more than 40% of the regional blend (USEPA 2008 HRS Ref. 23).

The Former Powder Metal Products site (PMP) is currently the most likely source for the TCE contamination. The site is an approximately 1.5 acre property located along Lockey Drive and Crestline Lane just south of Highway 211 East in Aberdeen (Figure 1). PMP owned and operated a plant used to manufacture precision machine parts on this site from 1980 until 1995. A part of this process was a TCE dip bath. In 1995, the PMP Company was sold, and in 1998-99 PMP filed for Chapter 11 bankruptcy. The assets, not including the Aberdeen property, were purchased by Powder Metal Products of Indiana.

During the investigation of ground water contamination at the Geigy Chemical Corporation NPL site in 1990, which is located just on the other side of State Route 211, northwest of the PMP site, TCE, lead and pesticide contamination was detected in numerous private wells along Crestline Lane and Route 211. Investigations have identified contaminated soils near the former TCE dip-vat utilized by PMP as the source of TCE contamination in the ground water. The Geigy site operated from 1947 to 1989 in various forms of pesticide and farm chemical formulation and retail. In December 1988 the USEPA and Geigy site owners entered into an Administrative Order of Consent (AOC) after discovery of private and municipal well contamination with pesticides. Approximately 2000 tons of contaminated soils were removed from the Geigy Chemical site during clean-up activities in 1991.

In May 1990, due to lead and TCE contamination identified during work on the Route 211 Contaminated Well site, up to 10 private residences and businesses were connected to the municipal water system of Aberdeen. This work was done under a "Request for Removal Action" which was expanded in 1991 to as many as 40 residences and businesses (USEPA 2008 HRS Ref. 8). This site later became the Crestline Contaminated Well site.

In November 1995, a Site Inspection for the Crestline Contaminated Wells site (NCD 986 172 492) was performed by N.C. DENR. The study covered three industrial wells and two residential wells located near Crestline Drive, just east of the Geigy site. The Site Inspection evaluated risks associated with TCE and 1,1,1-Trichloroethane (1,1,1-TCA) contamination found in these wells during an investigation by the North Carolina Department of Transportation (N.C. DOT) on the Lee Paving site. The contaminated wells either were no longer used for potable sources, or had been abandoned, and the investigation resulted in a “No Further Remedial Action Planned” status for this site (USEPA 2008 HRS Ref. 9).

The former Lee Paving Company site is located approximately 500 feet south of the PMP site. As part of a Memorandum of Agreement (MOA) between the N.C. DOT and the N.C. DENR to address possible contamination at former asphalt testing sites, a Comprehensive Site Assessment was performed for the Lee Paving Company from 1995 to January 1997. Samples collected in 1994 and 1995 documented a co-mingled plume of TCE and 1,1,1-TCA originating in the southern portion of the Lee Paving site and migrating west in the surficial aquifer and the Black Creek Formation aquifer. TCE contamination on the Lee Paving site is believed to have migrated from an off-site source, most likely PMP. A Corrective Action Plan was submitted on January 6, 2000 indicates that TCE, 1,1,1-TCA and their degradation products detected in the surficial aquifer have not migrated off site and are not associated with the widespread TCE contamination.

In October 2000, the N.C. DENR Division of Water Quality (DWQ) installed and sampled four pairs of nested wells around the PMP property. These wells were screened in the surficial and the Upper Black Creek aquifers. All of the samples contained TCE, with higher TCE concentrations observed in the Upper Black Creek relative to the surficial aquifer.

In order to better document a source area for the TCE contamination apparently emanating from the PMP site, USEPA Region IV installed a nested pair of monitoring wells in 2004. These wells were approximately 100 feet northwest of the suspected location of the TCE vat used by PMP. The wells were screened in the surficial and Upper Black Creek Formation aquifers. High concentrations of TCE (1489 µg/L) and low concentrations of other volatile organics were detected. The well with the highest concentration of TCE was drilled close to the suspected location of the TCE dip-bath used by PMP. The high level of TCE indicates that the PMP site is the likely source of the TCE ground water contamination. The continued contamination of two disconnected private wells, as well as the detection of TCE in one of the municipal wells, indicates an ongoing threat to human health. The North Carolina Superfund Section has recommended the Aberdeen CGW site for an Expanded Site Inspection (ESI) under CERCLA (“Superfund”).

Hydrogeologic Setting

The hydrogeology of the local Aberdeen area consists of five distinct hydrogeologic units. These include an unconfined surficial aquifer, the upper and lower Black Creek aquifers, the upper Cape Fear Formation, and the saprolite-bedrock, or basement, formation. Clay units at the top of the lower Black Creek aquifer, upper Cape Fear Formation, and saprolite-bedrock aquifer act as confining layers above these units. A discontinuous clay unit at the top of the upper Black Creek

aquifer creates locally perched water table conditions in the overlying surficial aquifer. The surficial aquifer contains no confining units. The major water supply aquifer is the lower Black Creek aquifer. The three aquifers in the Site vicinity are interconnected. Deposits overlying the Cape Fear Formation are considered to comprise a single aquifer system composed of hydrogeologic units of varying permeability and areal extent, all more or less hydraulically connected. The two confining layers separating the three aquifers have been documented to be absent in some locations in the immediate vicinity of the site. The Surficial aquifer is unconfined and consists mainly of lenses of perched ground water underlain by clay beds. The clay bed base overlies the Black Creek aquifer. The Surficial and the Upper Black Creek aquifers locally are vertically connected. The Upper Black Creek and Lower Black Creek aquifer units are separated by a semi-continuous confining bed. The absence of the confining layer has been located within a maximum 700 feet (0.13 mile) from the source contaminant plume. The approximate center of the plume is 1200 feet (0.23 mile) from the nearest documented area of aquifer connectivity. The Upper and Lower Black Creek aquifers are in hydraulic communication where this discontinuity exists and are considered as one aquifer in this area. The surficial aquifer is widely used throughout the state for individual home wells. The shallowest surficial aquifer is most susceptible to contamination from near surface sources. The surficial aquifer is also very sensitive to variations in rainfall amounts. The Black Creek Aquifer is recognized as a regional aquifer throughout the North Carolina Coastal Plain and is the primary source of water in the Aberdeen area. Ground water flows westward from the PMP property and discharges into Aberdeen Creek and its tributaries. The water table in the surficial aquifer has historically ranged from 42 to 500 feet below land surface and 72 to 82 feet below land surface in the Black Creek aquifer. The Surficial Aquifer occurs in sand and clayey sands of the Middendorf Formation and contains a perched saturated zone above the clay unit, which forms its base. The clay unit, 5 to 15 feet thick, overlies the Black Creek aquifer and is encountered at most Lee Paving Company site wells at a depth of 60 to 70 feet. It is absent or discontinuous on some areas of the Lee Paving property. The Black Creek aquifer, comprised of sands and clayey sands of the Middendorf Formation, occurs at depth ranging from 70 to 80 feet at the Lee Paving Company site. The upper unit of the Black Creek aquifer is an unconfined recharge to the aquifer and occurs indirectly by leakage through the overlying clay bed and by direct infiltration in areas where the clay unit is not present (USEPA 2008 HRS DRR).

DEMOGRAPHICS

Based on Census 2000 demographic data, 3,400 persons live in the Town of Aberdeen, North Carolina. It is estimated that 862 individuals live within a 1-mile radius of the Aberdeen CGW site. Twenty-three percent (23%) of the population is African-American in comparison to the state average (21.5%) and the national average (12%). The percentage of people with a high school diploma or higher is higher than the national average (86%), and considerably higher than the rest of the state (76%). The percentage of owner occupied housing units is also considerably higher (79%) than the national average (66%). The poverty level is slightly lower (10%) than the national average (12%). If we look at the population living within a two-mile radius of the site, the differences noted above stay consistent.

Demographic figures change dramatically for the census track south of NC 211 and in the area covering the TCE plume. The population in that area is 66% minority. The poverty level is

26%, more than two times the national average. Fifty-three percent (53%) of the population has a high school diploma or higher and 35% of the population in that area does not own the housing they live in.

COMMUNITY HEALTH CONCERNS

The USEPA organized a community meeting on January 8, 2009 at the Aberdeen Fire Department, 800 Holly Street in Aberdeen, North Carolina. The purpose of the meeting was to discuss the site status and proposed future site investigations, and hear input from local residents. USEPA mailed approximately 100 announcements of the meeting to area residents, local media and local government officials. Representatives of USEPA, N.C. DENR, Department of Waste Management, and N.C. DHHS, Division of Public Health attended the meeting. N.C. DHHS, Division of Public Health staff attended the meeting to meet directly with local residents and hear their concerns. There were no public attendees at the meeting.

DISCUSSION

Exposure Pathway Analysis

Chemical contaminants in the environment can harm people's health, but only if people have contact with those contaminants at a high enough concentration (dose) to cause a health effect. Knowing or estimating the frequency with which people have contact with hazardous substances is essential to assessing the public health importance of these contaminants. The human exposure pathway is evaluated to determine if people can come into contact with site contaminants.

According to the ATSDR, a completed exposure pathway is one that contains the following elements:

- a source of contamination, such as a hazardous waste site or contaminated industrial site,
- travel of the contaminant through a medium such as air, water, or soil,
- a point where people come in contact with a contaminated medium, such as drinking water, soil in a garden, or in the air,
- an exposure route, such as drinking contaminated well water or eating contaminated soil on homegrown vegetables, or inhaling contaminated air, and
- a population that can come into contact with the contaminants (be exposed)

A completed pathway is one in which all five pathway components exist and exposure to a contaminant has occurred, is occurring, or will occur. If one of the five elements is not present, but could be at some point, the exposure is considered a potential pathway. An exposure pathway is eliminated from further assessment if one of the five parts is missing and will not occur in the future. The length of the exposure period, the concentration of the contaminants at the time of exposure, and the route of exposure (skin contact, ingestion, and inhalation) are all critical elements considered in defining a particular exposure event.

A. Completed Exposure Pathway

The exposed population for the Aberdeen Contaminated Ground water (CGW) site is persons that ingest water from contaminated ground water wells. The completed pathways for this site are ingestion (drinking the contaminated ground water) and dermal (contact with contaminated drinking water). The completed exposure pathway for this site is illustrated in Table 1.

Table 1. Completed Exposure Pathway for the Aberdeen Contaminated Ground Water Site

Source	Medium	Exposure Point	Route of Exposure	Exposed Population
Contaminated ground water	Ground water	Well water	Ingestion, dermal (contact)	Persons in the past and present with contaminated well water

The Aberdeen CGW site was initiated when TCE was identified in private wells that were sampled during investigations of the Geigy Chemical NPL site. Subsequent investigations identified TCE and other contaminants, likely from multiple sites, in a number of private wells as well as the Town of Aberdeen municipal drinking water system's ground water wells. Initially the source of the TCE was not known, but was eventually identified as likely emanating from the former Powder Metal Products (PMP) industrial facility site. PMP began operations on the site in 1980. TCE was initially observed in ground water in the area in 1987. The exact date of initial contamination of any of the wells is not known.

B. Potential Exposure Pathway

It has not been confirmed that all private wells have been disconnected within the outlying areas of ground water contamination associated with the Aberdeen GCW site. Any existing private wells still being used within the areas of known TCE contamination present a potential exposure pathway if TCE concentrations exceed health guidelines and extended exposures periods exist.

Prior investigations on the site by USEPA and N.C. DENR have identified that other exposure pathways, such as contaminated soils or surface water, are not of concern for this site. The site includes only a contaminated ground water plume.

Contaminated ground water being supplied to a household or commercial facility may provide a potential exposure pathway through ingestion (by drinking the water), inhalation (from volatilization during a shower), and dermal contact (when taking a shower or bath). Many variables influence the levels of volatile chemicals entering a home from a water supply, including the chemical's physical and chemical properties, seasonal variations, and building construction. Confounding factors to consider when evaluating indoor exposures to volatile chemicals in supplied waters is that some of these chemicals are also common components of materials routinely used or present in the home or commercial and industrial operations, such as cleaning chemicals, textiles, or building materials.

Potential exposure pathways include inhalation and dermal contact for past residential and industrial users of contaminated private well water supplies. During investigations of the Aberdeen CGW site and the other nearby contaminated ground water sites private drinking water wells were disconnected. Users of the contaminated private drinking water wells were provided access to the Town of Aberdeen municipal drinking water supply when contamination was observed. This eliminated current inhalation and dermal pathways for exposure to contaminated ground water. Potential future exposure pathways exist for persons that may use contaminated private well water supplies or contaminated municipal supplies in the future. Control of the transport of the TCE ground water plume and/or remediation of the TCE plume would eliminate this potential exposure pathway.

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.

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.

ATSDR comparison values are set well below levels known or anticipated to result in adverse health effects. 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.

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.

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.

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

1. **Cancer Risk Evaluation Guide (CREG):** A CREG is the contaminant concentration estimated to result in no more than one excess cancer per 1 million persons exposed during a lifetime (i.e., 70 years). ATSDR calculates CREGs from EPA-established cancer slope factors.
2. **Environmental Media Evaluation Guide (EMEG):** EMEGs are estimated contaminant concentrations that are not expected to result in adverse non-cancer health effects. EMEGs are based on ATSDR “minimum risk levels” and conservative assumptions about exposure, such as intake rate, exposure frequency and duration, and body weight.
3. **Reference Dose Media Evaluation Guides (RMEGs):** ATSDR derives RMEGs from USEPA’s oral reference doses (RfD). RMEGs represent the concentration in water or soil at which daily human exposure is unlikely to result in adverse non-cancer effects.
4. **Maximum Contaminant Levels (MCL):** A Federal Maximum Contaminant Level (MCL) is the regulatory limit set by USEPA 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)

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, 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

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. 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 USEPA’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.

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 1×10^{-4} predicts the probability of one additional cancer over the background number of cancers in a population of 10,000. (The expected numbers of cancers in a population of 10,000 is approximately 3,300.) Qualitative assessment of the predicted increased numbers of cancers is also used and represents terminology suggested by ATSDR and N.C. DPH.

Thirty-year exposures were used for most of the increased cancer risk calculations in this study. This number is based on the maximum expected exposure period for TCE (PMP began operations in 1980) and is the risk assessment parameter that references a typical 30-year period for residence at a single location. A 70-year exposure period was used for pesticide cancer risk calculations based on operations initiated at the Geigy location in 1947.

Ground Water Data Sets Evaluated for Potential Health Effects

Multiple ground water data sets collected for the Aberdeen CGW site and other nearby contaminated sites were evaluated in this public health assessment. Each data set is discussed below. Contaminant concentrations and environmental drinking water screening values are summarized in separate tables for each sample set. A second table for each sample set summarizes child and adult estimated site-specific oral exposure doses and compares dose estimates to ATSDR non-cancer health guidelines (MRLs). Table 15 combines contaminants for

all sample sets that were retained for cancer-effect evaluation, listing calculated numbers of increased theoretical cancer cases for a population of 10,000. This population figure was selected as it represents a standard level of increased cancer risk estimation (10^{-4}) that is closest to the number of persons supplied drinking water through the Town of Aberdeen municipal system. Table 17 summarizes the qualitative levels of increased cancer risk used in this report. Table 16 summarizes oral exposure health effects data taken from ATSDR toxicological profiles and used for final evaluation of site-specific estimated exposure doses.

Ground Water Samples Collected by USEPA, 1991 to 1993: During 1991 to 1993 USEPA collected 31 ground water samples at 25 different locations in a rural mainly residential neighborhood immediately south of Highway 211 and less than one mile south and east of the Geigy Chemical site (Figure 3). This investigation was part of the Route 211 Contaminated Well site (which was later called the “Crestline Contaminated Well” site, USEPA 2008 HRS Ref. 8). The Geigy site is less than ½ mile northwest of the Aberdeen CGW site. Locations included 20 private residential and three commercial/industrial wells, and two municipal system wells. Some locations were sampled more than once. Some private well users in the area had previously been told to use bottled water. TCE, lead and several forms (isomers) of the pesticide hexachlorocyclohexane were detected (HCH, also known as benzene hexachloride or “BHC”, the gamma-BHC isomer is also commonly called “Lindane”).

TCE was detected in three samples collected at two locations. The highest TCE concentration observed on the PMP site was 730 µg/L (ppb). Site documentation indicates that the PMP well had been removed as a drinking water source in 1990. TCE was observed in a single residential well west of the PMP site at 34 µg/L. All detections exceeded the ATSDR CV for TCE (5 µg/L MCL) (Table 3). Estimated exposure doses for children and adults (Table 4) calculated using the 34 µg/L residential well TCE concentration are less than the current non-cancer effect health guideline (MRL) and 23,000 and 51,00 times less, respectively, than the lowest dose for non-cancer effects used to develop the ATSDR’s MRL value (Table 16). The estimated exposure dose for adults for the 730 µg/L detection from the PMP well is 2,400 times lower than the low non-cancer effect level. The critical study used for MRL development was a lowest observed effect level for an acute mouse study using an oral gavage exposure route. An alternative study for comparison is a chronic rat study, also using an oral gavage exposure route, had a no-effect level equal to the critical MRL study. No human health effect studies for TCE are available in the ATSDR toxicological profile.

A current ATSDR cancer-effect health guideline is not listed. USEPA proposed ranges of cancer slope factors (CSFs) (0.02 to 0.4 [$\text{mg}/\text{kg}/\text{day}$] $^{-1}$) were used to calculate theoretical increases in cancer risk. Adults ingesting ground water from the most contaminated well (730 µg/L TCE) for 30 years have a “moderate” to “high” risk (2 to 40 additional cases per 10,000 population) of theoretical increased numbers of cancers over the number of expected cancers for this population (Table 4). Site-specific estimates of increased cancers is likely less than these calculations since PMP did not begin operation until 1980 and documents indicate the PMP well was closed in 1990. Adults ingesting ground water from the residential well for 30 years have a “low” to “moderate” risk (less than 1 to 1 additional cases per 10,000) of increased cancer risk. Diabetes or chronic alcohol consumption may further increase the TCE cancer risk (EPA 2001). The estimated exposure dose calculated from the highest TCE ground water concentration is 800

times lower than lowest cancer-effect level animal study data. The residential well concentration is 18,000 times lower than the lowest cancer-effect level animal study data (Table 16). Uncertainty exists in the information regarding the number and location of private wells in use in the area and when they were removed as drinking water sources.

This public health assessment makes the health-protective conservative assumption that persons drank ground water daily with the highest concentration of trichloroethene found in a well on the PMP property (730 µg/L) and in the residential well (34 µg/L). If they drank contaminated ground water, the trichloroethene concentrations persons were actually exposed to in the past likely varied over time. The concentration of trichloroethene in the ground water in the past may have been lower or higher than 730 µg/L. In addition, the exposure may have been for a shorter time period since operations on the PMP site, believed to be the source of the TCE, did not start until 1980. Also, the quantity of water consumed by persons at the PMP facility may have been less than the default volume used in the exposure calculations. Most of the private wells in the area were disconnected between 1990 and 1995. Proportionate drops in exposure and theoretical cancer risk would be seen with reduced exposures periods.

Lead was detected above reporting limits in 27 of the 31 ground water samples collected in 1991 to 1993 (Table 3). The geometric mean concentration for the 27 detections was 28 µg/L. The highest concentration was 900 µg/L in a disconnected residential well. A source of the lead was not indicated in the site documents. ATSDR's lead CV is the MCL action level of 15 µg/L, with the notation that action is to be taken if more than 10% of tap water samples exceed this level. Under the Lead Copper Rule [LCR], USEPA requires testing of public water systems. If more than 10% of the samples at residences contain lead levels over 15 µg/L, actions must be taken to lower these levels. Eighteen of the ground water wells had lead levels exceeding the CV.

ATSDR does not publish health guidelines for lead, but provides a guidance framework to estimate blood lead dose levels in the lead toxicological profile. It provides blood lead slope values used to calculate estimated blood lead levels (BPb) from various exposure routes, including water. Table 5 lists ATSDR water matrix slope factors and estimated BPb for this set of ground water data, using the highest and geometric mean lead concentrations. The Centers for Disease Control and Prevention (CDC) considers children to have an elevated level of blood lead if the level is equal to or above 10 µg/dL (ATSDR 2007b).

For this data set, blood lead level estimates using the maximum lead concentration (900 µg/L) exceeded the action level for children and infants, but were approximately one-tenth the action level using the geometric mean lead concentration. Table 6 lists selected health effects for corresponding BPb levels representing sensitive endpoints (ATSDR 2007). The maximum lead concentration was observed in a residential well approximately ¼ mile northwest of the PMP site. Study documentation (USEPA 2008 HRS Ref. 8) indicates this well was closed at the time of sampling. The blood lead levels calculated with the maximum lead ground water concentration exceed multiple values associated with adverse health effects in infants and children. The estimated maximum blood lead levels for adult males and females also exceed multiple sensitive endpoints. Estimated blood lead levels calculated with the geometric mean concentration were less than health effect levels for all population sectors. The calculated blood lead level estimates assume extended periods of ingestion of the contaminated water at the

maximum and geometric mean concentrations. They also assume that all ingested water comes from the single contaminated source. Testing blood lead levels of the past and present occupants of the residence where the maximum lead concentrations were detected is advised if they used the well water as a drinking water source. Testing occupants of other residences served by wells that had waters exceeding the 15 µg/L Maximum Contaminant Level Action Level is also recommended.

The pesticide Lindane (hexachlorocyclohexane or gamma-HCH) and its related compounds alpha, beta, and delta-HCH were detected above reporting limits in 10 samples collected at 8 locations that included residential, commercial/industrial and the Town of Aberdeen supply wells. Lindane was detected in 10 wells at a maximum concentration of 1.5 µg/L and a geometric mean concentration of 0.38 µg/L. Both values exceed the child comparison value and the maximum concentration exceeds the adult comparison value (Table 3). Both the maximum and geometric mean estimated child and adult exposure doses exceed the non-cancer health guideline (Table 4). No human health studies are available for Lindane. The non-cancer lowest effect level from the intermediate exposure period study used to develop the health guideline MRL value (Table 16) is 80 times lower than the child, and 280 times lower than the adult estimated doses using the maximum Lindane concentration. ATSDR classifies Lindane as a “possible” human carcinogen, with no human studies documenting cancer development and limited animal studies indicating cancer development. ATSDR does not list a cancer screening value for Lindane, although cancer-effect animal health studies are provided in the toxicological profile. The Lindane maximum exposure dose is 300,000 times lower than the lowest cancer-effect level (Table 16).

Alpha, beta and delta-HCH were each detected in the two municipal wells. Alpha, beta, and total HCH (total HCH equals the summed concentrations of Lindane, alpha, beta, and gamma-HCH) maximum and geometric mean concentrations exceed their cancer-effect screening levels (Table 3). There are no screening values for delta-HCH. Concentrations of all detected HCH isomers were totaled and carried through the health effects evaluation, comparing concentrations and doses to technical grade HCH comparison values. The maximum total HCH concentration was 4.56 µg/L, and the geometric mean 1.5 µg/L. Both values exceeded the cancer-effect screening values. The estimated child and adult exposure doses using the maximum concentrations for alpha and beta-HCH were all at least 10 times less than their respective non-cancer health guideline values (Table 4). There were no non-cancer health-effect guideline values for delta and total-HCH.

ATSDR identifies alpha-HCH as a “probable” human carcinogen. Studies indicate its ability to cause cancer in animals, but inadequate human studies are available to prove its human-carcinogenic potential. ATSDR classifies beta-HCH as a “possible” human carcinogen, with no human studies documenting cancer development and limited animal studies indicating cancer development. Alpha, beta and total-HCH theoretical increased cancer risk calculations indicate, for a 70-year exposure using maximum and geometric mean concentrations, “moderate” levels of increased cancer risk for alpha-HCH, “low” increased cases for beta-HCH and “high” increased cases for total HCH (Table 15). Estimated exposure doses for alpha, beta and total-HCH were compared to cancer health-effects study data. The alpha-HCH estimated exposure dose at the maximum ground water concentration is 54,000 times lower, the beta-HCH estimated exposure

dose is 200,000 times lower, and the total HCH estimated exposure dose is 1,100 times lower, than the lowest cancer-effect level for HCH isomers (Table 16).

Based on comparison of estimated site-specific exposure doses to health effects study levels, no impacts related to ingestion of the waters contaminated with HCH pesticides at the concentrations observed in this data set would be anticipated for persons ingesting the water over a 70-year period. These estimates do not take into consideration that the Town of Aberdeen municipal water supply blends waters from multiple wells for supplying to end users and no one well makes up more than 40% of the contribution. A maximum 40% contribution would proportionally decrease the above estimated exposure concentrations, doses and increased cancer risk.

1995 N.C. DENR Crestline Site Investigation: In July and August 1995 N.C. DENR collected ground water samples for a Site Inspection at the Crestline Contaminated Wells site (NCD # 986 172 492). Water was collected from two residential and three industrial private wells. All were identified as inactive at the time. The industrial wells were located at the Sandhills Recycling Center, Lee Paving Company, and on the PMP property. TCE contamination had been found in these wells during earlier investigations associated with the Geigy Chemical site (USEPA 2008 HRS Ref 9).

TCE, 1,1,1-trichloroethane (1,1,1-TCA), and 1,1-dichloroethene (1,1-DCE) were found in ground water samples (Table 7). 1,1,1-TCA was found in two wells at a maximum concentration of 147 µg/L and did not exceed ATSDR screening values.

1,1-DCE was also found in two wells, at a maximum concentration of 26.7 µg/L and a geometric mean concentration of 12.7 µg/L. The 1,1-DCE concentrations do not exceed the ATSDR child or adult CV, but does exceed the MCL (7 µg/L). Estimated exposure doses are less than a factor of 10 below the ATSDR non-cancer health guideline (Table 8). Estimated exposure doses (Table 8) calculated with the maximum 1,1-DCE concentration are more than 11,000 times lower than the lowest non-cancer effect level from the critical animal study used to develop the non-cancer health guideline value. There are no ATSDR-referenced human studies for 1,1-DCE health effects.

ATSDR lists no cancer-effect CVs for 1,1-DCE, but ATSDR notes there is “suggestive evidence of carcinogenic potential”. ATSDR references a USEPA IRIS cancer slope factor (ATSDR 1994), which was used for calculation of theoretical increased cancer risks. A 30-year exposure is used for 1,1-DCE to coincide with that used for TCE. Estimates of theoretical increased cancer risk for 30-year exposures using the maximum concentration indicate a “moderate” risk of two increased cancers per 10,000 population, and a low increased risk (<1) for the geometric mean concentration (Table 15). The maximum estimated exposure dose is more than 6,500 times lower than the lowest cancer-effect level noted in the IRIS data (Table 16). Based on comparison of site-specific exposure doses to health effects data, no adverse health effects would be anticipated from ingestion of waters with 1,1-DCE concentrations representative of those observed in this sample set.

TCE was found in all 5 wells sampled in 1995. The maximum and geometric mean TCE concentrations are 730 µg/L and 73.1 µg/L, respectively. Both values exceed the 5 µg/L screening value (Table 7). Estimated dose calculations for neither concentration exceed the current health guideline, but do exceed the USEPA proposed reference dose (Table 8). Using the maximum TCE concentration, the USEPA proposed range of cancer slope factors, and a 30-year exposure period, “moderate” to “high” numbers of theoretical increased cancers are calculated (2 to 40 per 10,000 population). “Low” to “moderate” numbers of increased cancers (<1 and 6 per 10,000) are predicted using the geometric mean TCE concentration (Table 15). Comparison to health effects data indicates the estimated maximum exposure dose is 2,400 times lower than the chronic animal study used for development of the ATSDR non-cancer health guideline and 800 times lower than the lowest cancer-effect study. Based on this evaluation, no adverse health effects would be anticipated due to drinking the waters from these wells, assuming the maximum concentration used for site-specific exposure doses is representative of the actual well water concentrations of TCE.

Town of Aberdeen Drinking Water Supply Monitoring Data Collected from 1992 through 2008: The Town of Aberdeen (TOA) wells #5 and #9 are approximately 1 mile southwest of the PMP site, down gradient of the suspected source of the ground water TCE contamination. Volatile organic compound (VOC) analyses collected by the Town of Aberdeen to monitor municipal drinking water supply wells # 5 and #9 from March 1992 through October 2008 were evaluated (USEPA 2008 HRS Ref. 17; NCDENR PWS). TCE was the only VOC exceeding drinking water regulatory levels. The highest TCE concentration was 8.8 and 3.5 µg/L, respectively, for well #5 and #9. There were 12 TCE concentrations greater than the MCL in well #5 and none in well #9. The geometric mean TCE concentration in well #5 is 2.9 µg/L (Table 9). Estimated maximum exposure doses were calculated for children and adults using the maximum and geometric mean concentrations observed in well #5. All estimated exposure doses were less than the ATSDR non-cancer health guideline. Only the adult dose calculated with the well #5 geometric mean concentration was more than 10 times lower than the proposed non-cancer health guideline (Table 10). The maximum estimated exposure dose is one-million times lower than the lowest non-cancer animal study effect level used by ATSDR to develop the MRL. A “low” (<1 in 10,000 population) theoretical increased cancer risk is estimated for a 30-year exposure at the maximum estimated dose (Table 15). The maximum estimated exposure dose is more than 400,000 times lower than the lowest cancer-effect health study level. No adverse health effects would be expected for persons consuming water from the Town of Aberdeen wells #5 or #9. In addition, the Town of Aberdeen blends waters from multiple wells for distribution, with no one well making up more than 40% of the total supplied volume. Applying the 40% contribution maximum to the above calculations results in proportional decreases in estimated doses and increased cancer risks, further lowering the potential for adverse effects.

The highest TCE concentration in well #5 was observed in October 2008, the highest in well #9 in July 2003. There appears to be a pattern of increasing frequency of TCE detections in both wells. The TCE concentrations in well #5 appear to show a slight upward trend with time. The health effects evaluation for the last year of data for both wells does not differ from that of the 1992 through 2008 time period.

2000 and 2004 N.C. DENR Ground water Collections Near the PMP Site: In 2000 and 2004, N.C. DENR collected ground water samples from five monitoring wells (MWs), two Town of Aberdeen supply wells, and two inactive private residential wells (N.C. DENR PASI). The five MWs encircled the PMP property site, all within approximately 700 feet of the site boundary. MW collections included samples from both the surficial aquifer and the underlying Upper Black Creek Formation (UBCF) aquifer, both of which are used for local drinking water sources. The Town of Aberdeen municipal wells were #8 and #9, both down gradient of ground water flow from the PMP site. Municipal well #9 is approximately 4,000 feet southwest of the PMP site and has shown TCE contamination since 1996. Municipal well #8 is an additional 1,800 feet southwest of municipal well #9 and has not shown TCE contamination. One of the disconnected private residence wells is approximately 1200 feet northwest of the PMP site and believed to be the closest private residential well. The other residential well is approximately 1200 feet directly west of the PMP site. Both residential wells had shown TCE contamination in past collections. All samples were analyzed for VOCs. Detected VOCs included TCE, 1,1-DCE, cis-1,2-dichloroethene (c-1,2-DCE), and chloroform. The 2004 concentrations were generally higher than the 2000, consequently the 2004 data is used for the health evaluation.

Chloroform was detected in a single sample from the MW west of PMP in the lower screened aquifer (UBCF). The concentration (0.39 µg/L) was less than the non-cancer screening value (Table 11). ATSDR does not provide a cancer screening value for chloroform, but identifies chloroform as a “likely” or “probable” carcinogen. The USEPA has developed a reference dose (RfD) for chloroform which they identify as protective for cancer effects. Estimated exposure doses calculated with the highest 2004 detection are more than 900 times lower than the RfD (Table 12). No adverse health effects are expected with exposure to chloroform at concentrations represented by this data.

cis-1,2-DCE was detected in two MWs and one residential well, all at concentrations less than the non-cancer CVs. Based on no carcinogenicity data in humans or animals and non-positive results in laboratory mutagenicity assays (IRIS) c-1,2-DCE is not classified as to carcinogenic potential. No further evaluation of c-1,2-DCE was undertaken. No adverse health effects are expected with exposure to c-1,2-DCE at these concentrations.

1,1-DCE was also found in two monitoring wells and the two disconnected residential wells in 2004. All 1,1-DCE detections are less than ATSDR comparison values. Both the highest 1,1-DCE detection (15.8 µg/L) and the geometric mean concentration (3.9 µg/L) exceed the MCL (Table 11). The highest 1,1-DCE concentration was observed in the lower aquifer sample taken from the MW closest to the west side of the PMP property. Estimated exposure doses for the maximum and geometric mean concentrations were more than 10 times lower than the non-cancer health guideline except for children at the maximum concentration (Table 12). The maximum estimated child site-specific exposure does is more than 5,000 times lower than the lowest non-cancer health effects study (Table 16).

The USEPA states there is “suggestive evidence” for cancer-effects for 1,1-DCE. Theoretical estimates of increased cancer risk for 30-year exposures indicate “moderate” to “low” increased cancers (3 and <1 per 10,000 population, respectively) using the highest and geometric mean detections in 2004 (Table 15). The estimated maximum exposure dose for 1,1-DCE is 11,000

times lower than lowest cancer effect level in the animal study used by the USEPA to develop their cancer slope factor (Table 16). No health effects would be anticipated for persons ingesting water contaminated with 1,1-DCE at the levels detected in the 2000 and 2004 concentrations.

In 2000, TCE was detected in four of five MWs from both aquifers (highest concentration 640 µg/L, geometric mean 5.5 µg/L). No TCE was detected in the MW nearest the west side of the PMP property, nor in either of the municipal supply wells or the residential well. In 2004, TCE was found in three MWs, one municipal well, and both disconnected residential wells. The highest TCE concentration was observed in 2004 (1,489 µg/L) from the MW nearest the west side of the PMP property in the underlying aquifer (UBCF), where no TCE had been seen on 2000. The highest TCE concentration and the geometric mean TCE concentration (34.5 µg/L) for the 2004 data (Table 11) both exceed the MCL screening value.

Site-specific estimated exposure doses were calculated for TCE maximum and geometric mean concentrations for the 2004 samples. Both child and adult maximum exposure doses were less than 10 times lower than the current ATSDR non-cancer health guideline. All but the adult geometric mean exposure dose was greater than the USEPA proposed health guideline (Table 12). The estimated maximum child exposure dose for the 2004 data more than 300 times lower than the lowest acute exposure effect used by ATSDR to develop the non-cancer health guideline, as well as the lowest no-effect study for chronic exposures.

Calculations of theoretical increased risk of cancer for 30-year exposures at the highest 2004 TCE detection predict “moderate” to “high” numbers of increased cancers (2 and 40 per 10,000 population, respectively). “Low” to “moderate” increased cancers (<1 and 4 per 10,000 population, respectively) are indicated at the 2004 geometric mean concentration (Table 15). Comparing estimated adult exposure doses to cancer health effects study data indicates the maximum dose is more than 400 times less than the lowest cancer-effect level and the geometric mean dose more than 18,000 times lower.

Person ingesting water at concentrations represented by the highest concentrations of TCE in the 2004 sample set would not be expected to be at risk of adverse health effects with long-term exposure (up to 30-years).

April 2008 USEPA Ground water Collections: In April 2008 USEPA collected ground water samples from two MWs located approximately 4,500 feet south east and 9,000 feet northwest of the PMP property (USEPA 2008 GW) to document the spread of contaminants outward from the PMP site. Both MWs are outside of the radius of the Town of Aberdeen municipal system of ground water supply wells. These two wells are within a 2-mile radius of the PMP site and near locations of known or suspected private drinking water wells identified in the September 2007 well survey (Figure 4). The pesticides p,p'-DDD, p,p'-DDT, endosulfan sulfate, endrin aldehyde, and endrin ketone, and the VOCs chloroform and methyl-tert-butyl ether (MTBE) were detected in the northwest well, all at estimated concentrations less than the laboratory minimum reporting limit, and less than screening values. No TCE was detected in these samples (Figure 5, Table 13).

PUBLIC HEALTH IMPLICATIONS

This section discusses the health effects that could plausibly result from exposures to specific contaminants identified at the Aberdeen CGW site. For a public health hazard to exist, people must contact contamination at levels high enough and for a long enough time to adversely affect their health. Evaluation of potential public health hazards are based on ATSDR assessment procedures. The environmental data and conditions at the site revealed one major completed exposure pathway - use of private wells for potable purposes.

ATSDR prefers to use site-specific conditions whenever possible to evaluate whether people are being exposed to contaminants at levels of health concern. However, two important site-specific determinants are not known for this site: 1) when the contaminants first reached private and municipal drinking water wells; and, 2) what levels of contamination residents might have been exposed to over time (the levels could have been higher or lower than those detailed in this study). Because of these unknowns, ATSDR must rely on reasonable assumptions rather than site-specific information in this instance.

Some of the contaminants identified in this study are identified as “volatile” and could volatilize (out-gas) from waters during activities such as showering, bathing or car washing. Concentrations in ground water may not be high enough to cause adverse health effects in an outdoor environment. No air sampling data is available to evaluate indoor exposures, although again, water concentrations are likely not high enough to be anticipated as a problem.

Contaminants of concern specific to each sampling event are discussed in the previous section. The following text provides discussion of the potential health effects of the contaminants identified for this site that may present a health concern. Review of analytical data, calculation of estimated site-specific exposure doses, and review of health effects study information indicated that only lead contamination in the ground waters collected in 1991 to 1993 may pose potential health hazards. All other identified and evaluated site contaminants, including TCE, present no apparent health hazard. Potential exposures to lead associated with this site are uncertain as indicated in the discussion of the 1991 to 1993 ground water sampling event. The primary concern for lead exposure, if it did take place, is with children that may have been exposed by drinking the ground water for extended periods of time at concentrations near the maximum detected concentration. Breast-fed infants of lead-exposed mothers also represent a group of increased susceptibility since lead may be excreted in breast milk. Exposures to children at concentrations represented by the mean concentration found in this sampling event would not be considered a health hazard. Determination of potential health effects for ingestion of ground water containing lead were based on estimated blood lead levels calculated with ATSDR blood lead slope values, as discussed above. The developing nervous system, the hematological (blood) and cardiovascular systems, and the kidney are the most sensitive targets to elevated lead exposure, although any system or organ in the body may ultimately be affected. Children less than 5 years old appear to absorb lead through ingestion more efficiently than adults. Nutritional deficiencies in children may heighten the toxic effects. Toxic effects associated with lead exposure in children, including hematological and nervous system effects,

have been documented at lower blood lead levels, with resulting effects also more severe (ATSDR 2007b). Recommendations are made for follow-up of the potential lead exposures.

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” (USEPA), “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 USEPA 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 (USEPA 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. Studies of women exposed to mixtures of chlorinated solvents (including TCE) in drinking water during pregnancy also suggest that TCE may increase the risk of birth defects (e.g., neural tube defects, oral cleft defects, and congenital heart defects) and/or childhood leukemia (ATSDR, 1997c). In each of the drinking water studies, however, there are uncertainties about how much contaminated water the women drank during pregnancy and about how much TCE was in the water the women drank while pregnant. 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 (NRC 2006). 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 (USEPA 2001). It is not known if the health effects observed in the studies of human exposure to TCE in workplace air and in drinking water are due to TCE or other factors, including exposure to other chemicals, smoking, alcohol consumption, and lifestyle choices. Since these potential confounding factors were not well controlled, and because there were uncertainties about actual exposures, the studies in humans suggest, but do not prove, that exposure to TCE can cause cancer, developmental effects and reproductive effects in humans. In humans, long-term exposure in the workplace to high levels of TCE in air is linked to effects on the central nervous system and irritation of the mucous membranes. Some studies of people exposed to high levels of TCE in workplace air or in drinking water show an association between exposure to TCE and increased risks for certain types of cancer, including cancers of the kidney, liver, esophagus, and non-Hodgkin’s lymphoma. Other studies suggest an association between workplace TCE exposure and reproductive effects (alterations in sperm counts) in men.

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, USEPA 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.” (USEPA 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 USEPA’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.

Health Effects Information for Other Contaminants of Concern Discussed in this Public Health Assessment

Lead: Lead is a naturally occurring metal that is dispersed throughout the environment. The most likely source of exposure to lead is the ingestion of contaminated food and drinking water, or the unintended ingestion of contaminated soil, dust or lead-based paint. Lead levels in surface waters and ground waters in the U.S. range between 5 and 30 µg/L. The rate of uptake of ingested lead is influenced by the health status of the exposed individual and the form of lead. Children tend to take up lead at a higher proportion than adults. Normal blood lead levels are 1.9 µg/dL for children 1 to 5 years of age, and 1.5 µg/dL for adults 20 to 59 years of age. Children are more vulnerable to the effects of lead than adults. The most common source of exposure to lead for children is lead-based paint. Lead exposure during infancy or childhood may result in anemia, neurological impairment, renal alterations, colic, and impaired Vitamin D metabolism,

IQ deficits, low birth weight, growth retardation, and delayed sexual maturation in girls (ATSDR 2007b).

Isomers of Hexachlorocyclohexane: Hexachlorocyclohexane (“HCH”) is a manufactured chemical that exists in eight chemical forms called isomers. One of these forms, gamma-HCH (commonly called Lindane) is used as an insecticide on fruit, vegetables, and forest crops. It is also available as a prescription (lotion, cream, or shampoo) to treat lice and scabies. Lindane has not been produced in the United States since 1976, but is imported for insecticide use. Technical-grade HCH was used as an insecticide in the United States and typically contains a mixture of gamma, alpha, beta, delta and other forms of HCH. Technical-grade HCH has not been produced or used in the United States in over 20 years. Some people who breathed contaminated workplace air during manufacturing of pesticides, including gamma-HCH, had blood disorders, dizziness, headaches, and changes in the levels of sex hormones. Some people who swallowed large amounts had seizures and sometimes died. Animals fed gamma and alpha-HCH have had convulsions, and animals fed beta-HCH have become comatose. All isomers can produce liver and kidney effects. Reduced ability to fight infection was reported in animals fed gamma-HCH, and injury to the ovaries and testes was reported in animals given gamma-HCH or beta-HCH. Long-term oral administration of alpha-HCH, beta-HCH, gamma-HCH, or technical-grade HCH to laboratory rodents produced liver cancer. The U.S. Department of Health and Human Services (DHHS) has determined that HCH (all isomers) may reasonably be anticipated to cause cancer in humans. The International Agency for Research on Cancer (IARC) has classified HCH (all isomers) as possibly carcinogenic to humans. The USEPA has determined that there is suggestive evidence that gamma-HCH (Lindane) is carcinogenic, but the evidence is not sufficient to assess its human carcinogenic potential. The USEPA has additionally classified technical HCH and alpha-HCH as probable human carcinogens, beta-HCH as a possible human carcinogen, and gamma-HCH as not classifiable as to human carcinogenicity (ATSDR 2005c).

1,1,1-Trichloroethane: 1,1,1-Trichloroethane (1,1,1-TCA) is a synthetic chemical that does not occur naturally in the environment. It also is known as methylchloroform or trichloromethylmethane. 1,1,1-TCA is a colorless, volatile, nonflammable liquid with a sweet, chloroform-like odor. 1,1,1-Trichloroethane had many industrial and household uses, including use as a solvent to dissolve other substances, such as glues and paints; to remove oil or grease from manufactured metal parts; and as an ingredient of household products such as spot cleaners, glues, and aerosol sprays. 1,1,1-TCA is rapidly absorbed if inhaled or ingested. Dermal absorption is slow and does not contribute significantly to systemic toxicity. 1,1,1-TCA crosses the placenta and is excreted in breast milk. Breathing air containing high levels of 1,1,1-TCA for a short time may result in dizziness and lightheadedness and possibly loss of coordination. These effects rapidly disappear after breathing contaminated air is stopped. Breathing in much higher levels may result in unconsciousness, blood pressure may decrease, and the heart may stop beating. The health effects of breathing low levels of 1,1,1-TCA for a long time are not known. Studies in animals show that breathing air that contains very high levels of 1,1,1-TCA damages the breathing passages and causes mild effects in the liver and the nervous system. There are no studies in humans that determine whether eating food or drinking water contaminated with 1,1,1-TCA could harm health. Placing large amounts of 1,1,1-TCA in the stomachs of animals has caused effects on the nervous system, mild liver damage, unconsciousness, and even death. Contact of the skin with 1,1,1-TCA may result in skin irritation. Studies in animals suggest that

repeated exposure of the skin might affect the liver and that very large amounts may cause death. These effects occurred only when evaporation was prevented. Children exposed to large amounts of 1,1,1-TCA probably would be affected in the same manner as adults. Available information does not indicate that 1,1,1-TCA causes cancer (ATSDR 2006b, ATSDR TCA MMG).

1,1-Dichloroethene: 1,1-Dichloroethene (1,1-DCA) is an industrial chemical that is not found naturally in the environment. It is a colorless liquid with a mild, sweet smell. It is also called vinylidene chloride. 1,1-DCA is used to make certain plastics, such as flexible films like food wrap, and in packaging materials. It is also used to make flame retardant coatings for fiber and carpet backings, and in piping, coating for steel pipes, and in adhesive applications. Breathing lower levels of 1,1-DCA in air for a long time may damage your nervous system, liver, and lungs. Workers exposed to 1,1-DCA have reported a loss in liver function, but other chemicals were present. Animals that breathed high levels of 1,1-DCA had damaged livers, kidneys, and lungs. The offspring of some of the animals had a higher number of birth defects. We do not know if birth defects occur when people are exposed to 1,1-DCA. Animals that ingested high levels of 1,1-DCA had damaged livers, kidneys, and lungs. There were no birth defects in animals that ingested the chemical. Spilling 1,1-DCA on your skin or in your eyes can cause irritation. The USEPA has determined that 1,1-DCA is a possible human carcinogen. Studies on workers who breathed 1,1-DCA have not shown an increase in cancer. These studies, however, are not conclusive because of the small numbers of workers and the short time studied. Animal studies have shown mixed results. Several studies reported an increase in tumors in rats and mice, and other studies reported no such effects (ATSDR 1995).

HEALTH OUTCOME DATA

In addition to studying exposure and chemical-specific toxicity data as part of the public health assessment process, N.C. DPH also considers health outcome data, such as mortality and morbidity data. The following criteria are evaluated when determining if a study of health outcome data is reasonable: (1) presence of a completed human exposure pathway, (2) high enough concentrations of contaminant to result in measureable adverse health effects, (3) sufficient numbers of exposed people in the pathway for effects to be measured, and (4) a health outcome database where disease rates for the population of concern can be identified.

N.C. DPH identified a completed ground water ingestion exposure pathway for the Aberdeen CGW site. The limited number of potentially exposed persons, the short length of the potential exposure period, the concentrations of contaminants of potential exposure, and the potential long-term health effects associated with the site contaminants make the study of health outcome data related to this site impractical.

CHILD HEALTH CONSIDERATIONS

The ATSDR recognizes there are unique exposure risks concerning children that do not apply to adults. Children engage in increased outdoor activities and hand to mouth actions. Children have lower body weights and higher intake rate than adults, which result in a greater dose of

hazardous substance per unit of body weight. Other variables that can affect a child's exposure response include genetic makeup, age, health, nutritional status, and exposure to other environmental substances. If toxic exposure levels are high enough during critical growth stages, the developing body systems of children can sustain permanent damage (ATSDR, 1999). Because adults are in charge of the housing, medical care, and risk identification of children they should have as much information as possible about environmental contaminants in order to make informed decisions, which can affect the child's health.

Children are particularly sensitive to lead as compared to adults. There is the potential that children were exposed to ground water drinking water sources containing lead at levels that may have possible health effects. Calculated blood lead levels for children using the maximum concentration for the ground water samples collected in 1991 to 1993, which included residential wells, indicated estimated blood lead levels exceeding the ATSDR health effects action level. The number of children possibly exposed, if any, the length of the exposure, and actual exposure concentrations over time are unknown. Data evaluations do not indicate any increased risks to children due to the other chemicals evaluated in this study.

CONCLUSIONS

An **Indeterminate Health Hazard** was identified for persons that may have been exposed to TCE in the drinking water supply at the PMP industrial facility. TCE levels detected from 1991 to 1993 were at levels that may result in increased cancer risk over the expected background numbers of cancers. Uncertainty exists on the length of exposure for this source, with the maximum exposure period of approximately 10 years. The actual length of exposure may have much less since it is not known when the contamination was first present in the ground water on the PMP site.

An **Indeterminate Health Hazard** was identified for potential ground water lead exposures associated with samples collected from 1991 to 1993 in rural residential areas within one mile south and east of the Geigy Chemical site. This conclusion is based on a lack of knowledge of whether these waters were actually used as a source of drinking water and if so, for how long. Children or infants exposed at lead levels represented by the highest concentration found in the 1991 to 1993 sample set would have the potential for adverse health effects. No health effects would be expected for those exposed at lead concentrations represented by the geometric mean lead concentration. Site documents noted that the private well where the highest lead concentration was collected was disconnected at the time of collection.

Based on sampling data, estimates of site-specific exposure doses, cancer risk estimates, and comparisons of those dose estimates to health-effect study data, this site is considered **No Apparent Public Health Hazard** for past and present exposures. This conclusion is based on the assumption that the ground water concentrations represented by the sampling data provided in the referenced sample sets collected from 1991 through 2008 are representative of actual exposures to persons ingesting ground water in the area of the Aberdeen Contaminated Ground water site.

RECOMMENDATIONS

To prevent future exposures to contaminated ground water, NCDPH have the following recommendations:

- Efforts to identify private well users in the area should be continued. The most recent survey of area residences in the areas near where contaminated ground waters have been found may be using private ground water wells for drinking and other uses.
- Private wells in the area should be tested for volatile organic compounds, pesticides, and metals (lead). Wells with contaminant concentrations exceeding regulatory levels or health guideline levels should be closed and the user should be connected to the Town of Aberdeen municipal water supply system.
- The Town of Aberdeen municipal supply wells in the path of documented contaminant plumes should continue to be closely monitored, including wells #5, #9 and #8. Wells #5 and #9 may be indicating a pattern of increasing frequency and concentrations of TCE contamination, with the concentrations in well #5 showing the higher concentrations. Blending or other means should continue to be used to insure that finished waters supplied meet regulatory criteria and health guideline recommendations.
- Efforts should be made to control further migration of the TCE plume to prevent further impact to the Town of Aberdeen municipal water supply.
- Other known or suspected contaminant plumes that may impact the Town of Aberdeen municipal water wells should be adequately characterized and controlled.
- Testing blood lead levels of the past and present occupants of the residence where the maximum lead concentrations were detected in the 1991 to 1993 sampling event is advised if the well water was a drinking water source. Testing occupants of other nearby residences with long-term exposure from wells that had waters exceeding the 15 µg/L MCL Action Level is also recommended.

PUBLIC HEALTH ACTION PLAN

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

Public Health Actions Completed

- N.C. DENR and USEPA have conducted ground water characterization studies identified in this report.
- The Town of Aberdeen contracts for quarterly analytical quality testing of the municipal water supply well system.
- USEPA and N.C. DENR held a public availability session on January 8, 2009 to gather health concerns from the community and present details of the planned Remedial Investigation/Feasibility Study; however, no community members participated.

Public Health Actions Planned

- Educational outreach materials will be distributed through the Moore County Health Department for residents that live near the site. The educational materials will detail information about the contaminants of concern in the form of fact sheets or other written materials designed to educate the public. N.C. DHHS will provide the educational materials to Moore County Health Department. Accurate alternate sources of information can be obtained from the Moore County Health Department if desired. The alternate sources will include printed material obtained from internet resources provided by organizations such as the USEPA or ATSDR.
- N.C. DHHS will continue to provide health assessment and other services to support N.C. DENR and USEPA efforts on the Aberdeen Contaminated Ground water site, including future public health assessments of additional ground water data as it is gathered.
- N.C. DHHS will undertake efforts to inform and engage persons living and working in the area about activities and issues related to the Aberdeen Contaminated Ground water site.

If any citizen has questions or concerns about this report, please contact the NCDHHS Occupational and Environmental Epidemiology Branch at (919) 707-5900.

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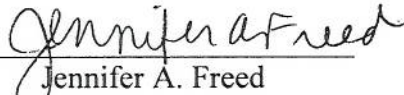
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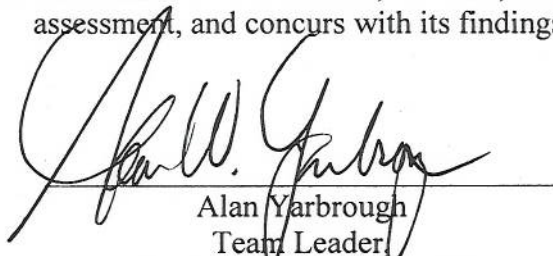
CERTIFICATION

This Public Health Assessment for the Aberdeen Contaminated Ground Water Site was prepared by the North Carolina Division of Public Health (NC DHHS) 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 public health assessment, and concurs with its findings.



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Team Leader
CAT, CAPEB, DHAC, ATSDR

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Appendix A

Figures

Figure 1. Aberdeen Contaminated Ground Water NPL Site and Other Nearby Known Contaminated Sites

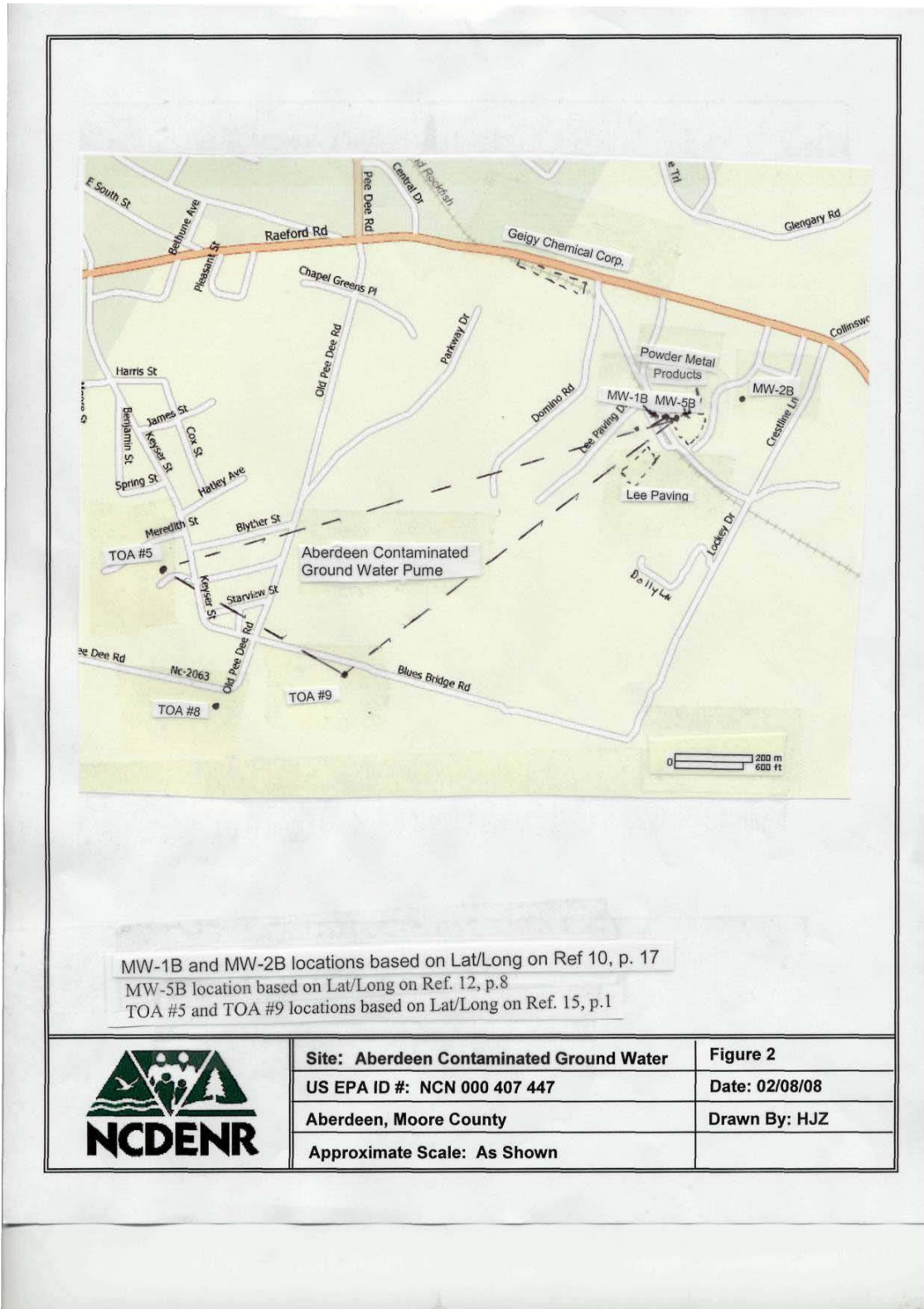


Figure 2. Location of Town of Aberdeen Municipal Drinking Water System Supply Wells

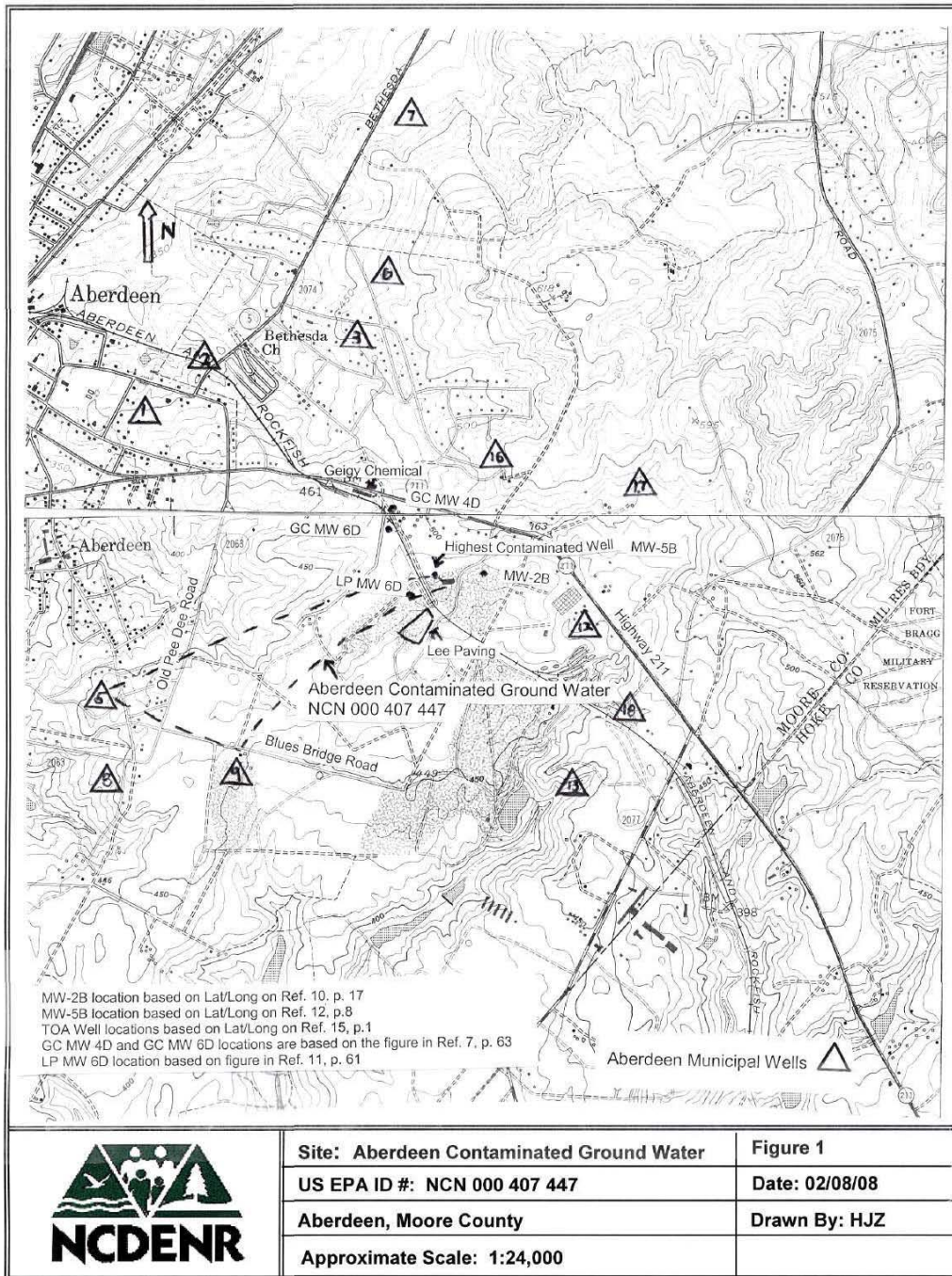


Figure 3. Ground Water Sample Locations Collected by the USEPA in 1991 to 1993

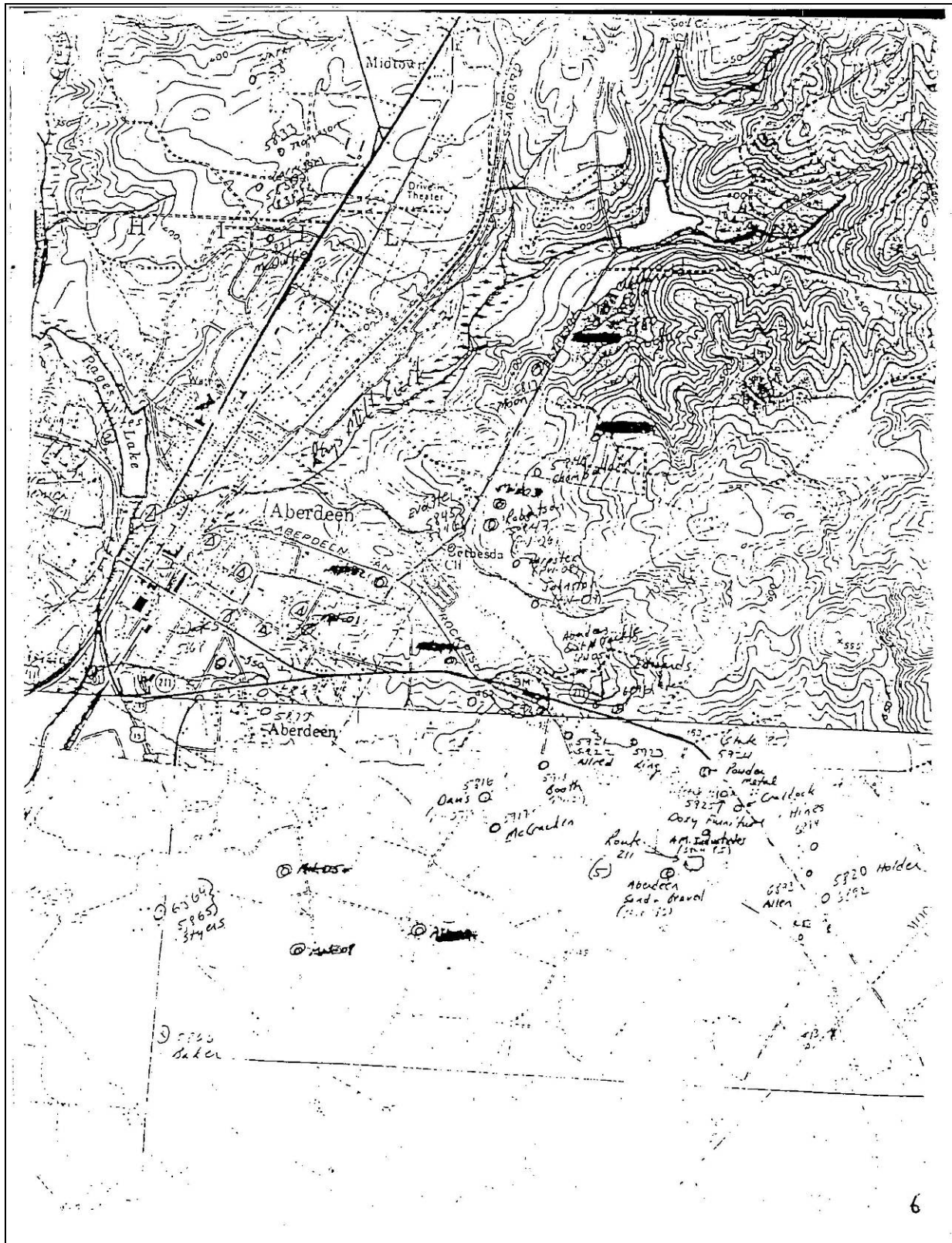


Figure 4. 2007 Private Wells Survey in 2-Mile Radius around Aberdeen Contaminated Ground Water NPL Site

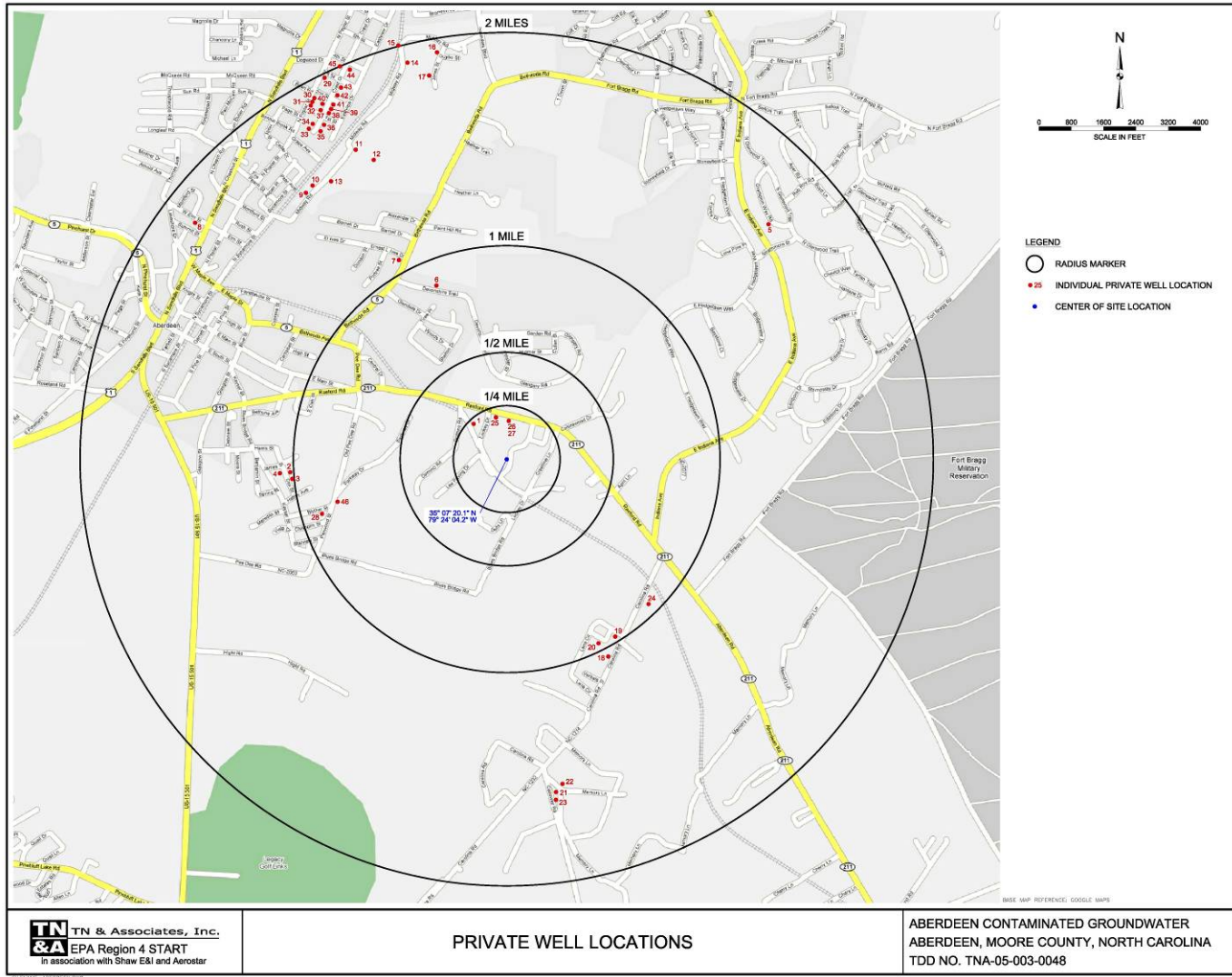
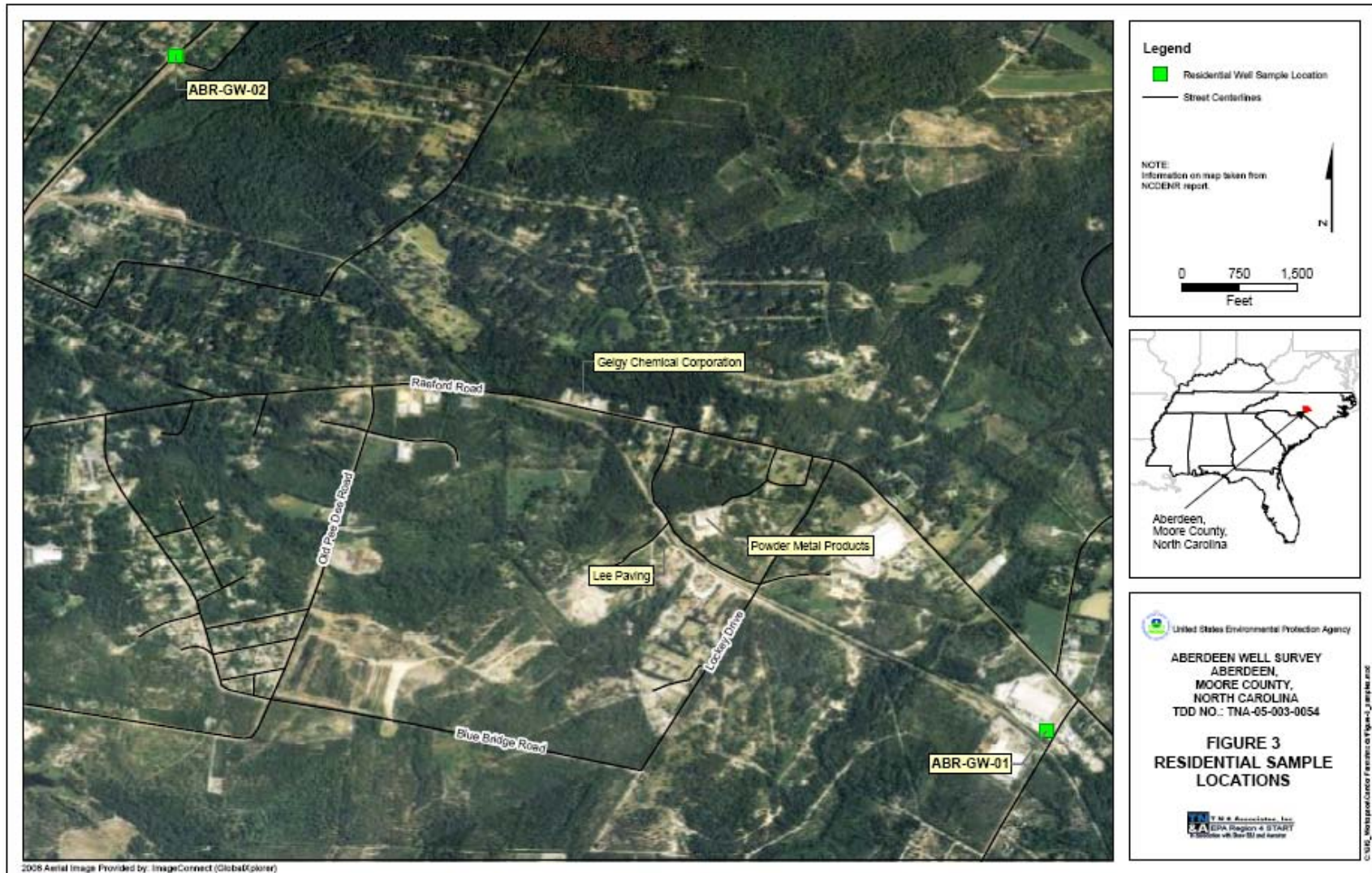


Figure 5. April 2008 USEPA Ground Water Sampling Locations



Appendix B

Tables

Table 2. Current and Proposed Drinking Water Screening Values for Trichloroethene (TCE).

Current ATSDR SV, Type	Current ATSDR Oral MRL, Exposure Scenario	Current ATSDR Cancer Classification(s)	Proposed USEPA SV	Proposed Cancer Slope Factor
5 µg/L MCL	0.2 mg/kg/d Acute	“Under Review” (USEPA); “Reasonably Anticipated to be a Carcinogen: (NTP); “Probably carcinogenic to humans (limited human evidence; sufficient evidence in animals)” (IARC)	0.0003 mg/kg/d RfD (chronic)	0.02 to 0.4 (mg/kg/d) ⁻¹

Notes: SV = screening value
MRL = minimum risk level
MCL = Minimum Concentration Limit, USEPA Federal Drinking Water Standard
RfD = Reference Dose

Table 3. Data Summary and Screening Value Analysis for Private Well Samples Collected by USEPA from 1991 to 1993 for the Aberdeen Contaminated Ground Water site.

Contaminant	Frequency of Detection	Range of Concentrations (µg/L)	Number of Detections Greater Than CV	Geometric Mean Concentration (µg/L)	Comparison Value (CV) (µg/L)	Type of CV
TCE	3/31	34 – 730	3	202	5	MCL
Lead	27/31	15 - 900	18	28	15	MCL AL
gamma-HCH (Lindane)	10/31	0.08 – 1.5	9	0.38	0.1 child 0.4 adult	RMEG
alpha-HCH	2/31	0.22 – 1.30	2	0.53	80 child 300 adult	Chronic EMEG
					0.006	CREG
beta-HCH	2/31	0.03 – 0.36	2	0.10	6 child 20 adult	Intermediate EMEG
					0.02	CREG
delta-HCH	2/31	0.06 – 1.40	2	0.29	Not Available	-----
Total HCH	2/31	0.51 – 4.56	2	1.5	0.02 (as “technical grade”)	CREG

Notes: CV = Comparison value (ATSDR established screening values)

TCE = Trichloroethene

AL = Action Level

HCH = Hexachlorocyclohexane

RMEG = Reference Dose Media Evaluation Guide

EMEG = Environmental Media Evaluation Guide

CREG = Cancer Risk Evaluation Guide

µg/L = micrograms per liter, parts per billion (ppb)

Table 4. Site-Specific Exposure Dose Estimates and Health Guideline Comparison for Private Well Waters Collected by USEPA from 1991 to 1993 for the Aberdeen Contaminated Ground Water sites.

Contaminant	Calculated Maximum Exposure Dose (mg/kg/d) ⁽¹⁾	Calculated Geometric Mean Exposure Dose (mg/kg/d) ⁽¹⁾	ATSDR MRL (non-cancer) (mg/kg/d)	Does Calculated Maximum Exposure Dose Exceed non-CA HG (child/adult)	Does Calculated Geometric Mean Exposure Dose Exceed non-CA HG (child/adult)
TCE (at 730 µg/L in PMP well)	Child 0.073 Adult 0.021	Child 0.020 Adult 0.0058	0.2 acute oral; Proposed changes: RfD = 0.0003 mg/kg/d	<u>Current HG:</u> Child NO Adult NO <u>Proposed HG:</u> Child YES Adult YES	<u>Current HG:</u> Child NO Adult NO <u>Proposed HG:</u> Child YES Adult YES
TCE (at 34 µg/L in residential well)	Child 0.0021 Adult 0.00097	----	0.2 acute oral; Proposed changes: RfD = 0.0003 mg/kg/d	<u>Current HG:</u> Child NO Adult NO <u>Proposed HG:</u> Child YES Adult YES	----
Lead	Child 0.090 Adult 0.026	Child 0.0029 Adult 0.00080	Not Available	----	----
gamma-HCH	Child 0.00015 Adult 0.000043	Child 0.000038 Adult 0.000011	0.00001	Child YES Adult YES	Child YES Adult YES
alpha-HCH	Child 0.00013 Adult 0.000037	Child 0.000053 Adult 0.000015	0.008	Child NO Adult NO	Child NO Adult NO
beta-HCH	Child 0.000036 Adult 0.000010	Child 0.000010 Adult 0.000029	0.0006	Child NO Adult NO	Child NO Adult NO
delta-HCH	Child 0.000014 Adult 0.00004	Child 0.000029 Adult 0.000083	Not Available	----	----
Total HCH	Child 0.00046 Adult 0.0013	Child 0.00015 Adult 0.0030	Not Available	----	----

Notes: Non-CA = non-cancer

Table 5. ATSDR Blood Lead Slope Factors for Water Exposures and Estimated Blood Lead Levels for Ground Waters Collected in 1991 to 1993 for the Route 211 Contaminated Well Site (Later re-named Aberdeen Contaminated Ground Water Site).

Population	Slope Factor⁽¹⁾	µg/dL Blood Lead at Max. Water Concentration⁽²⁾	µg/dL Blood Lead at Geo-Mean Water Concentration⁽³⁾
Infants	0.04	36	1.1
Children	0.03	27	0.84
Adult Males	0.06	54	1.7
Adult Females	0.03	27	0.84

Notes: Reference: *Toxicological Profile for Lead*. 2007. ATSDR. Appendix D Table 1.
 Slope factors for >15 µg/L water concentrations used to calculate blood lead levels
⁽¹⁾ Slope factor as µg/dL blood lead per µg lead/L water lead
⁽²⁾ Maximum ground water lead concentration for sample set = 900 µg/L
⁽³⁾ Geometric mean ground water lead concentration for sample set = 28 µg/L

Table 6. ATSDR Blood lead Concentrations Corresponding to Adverse Health Effects

Age	Effect	Blood Lead Associated with Effect, µg/dL
Children	Depressed aminolevulinic acid dehydratase	<5
Children	Neurodevelopmental effects	<10
Children	Sexual maturation	<10
Children	Depressed vitamin D	>15
Children	Elevated erythrocyte protoporphyrin	>15
Children	Depressed nerve conduction velocity	>30
Children	Depressed hemoglobin	>40
Children	Colic	>60
Adults (elderly)	Neurobehavioral effects	>4
Adults	Depressed aminolevulinic acid dehydratase	<5
Adults	Depressed glomerular filtration rate	<10
Adults	Elevated blood pressure	<10
Adults	Elevated erythrocyte protoporphyrin (females)	>20
Adults	Enzymuria/proteinuria	>30
Adults	Peripheral neuropathy	>40
Adults	Neurobehavioral effects	>40
Adults	Altered thyroid hormone	>40
Adults	Reduced fertility	>40
Adults	Depressed hemoglobin	>50

Reference: *Toxicological Profile for Lead*. 2007. ATSDR. Table 2-1.

Table 7. Data Summary and Screening Value Analysis for Private Well Samples Collected In July to August 1995 for Crestline Area Investigation.

Contaminant	Frequency of Detection	Range of Concentrations (µg/L)	Number of Detections Greater Than CV	Geometric Mean Concentration (µg/L)	ATSDR Health-Based CV (µg/L)	Type of CV
TCE	5/5	34 – 730	5	73.1	5	MCL
1,1,1-TCA	2/5	22 – 147.1	0	56.9	Child 20,000 Adult 70,000	RMEG
					200	MCL, LTHA
1,1-DCE	2/5	6.0 – 26.7	1	12.7	Child 90 Adult 300	Chronic EMEG
					7	MCL

Notes: 1,1,1-TCA = 1,1,1-Trichloroethane
1,1-DCE = 1,1-Dichloroethene

Table 8. Site-Specific Exposure Dose Estimates and Health Guideline Comparison for Private Well Samples Collected in July to August 1995 for Crestline Area Investigation.

Contaminant	Calculated Maximum Exposure Dose (mg/kg/d) ⁽¹⁾	Calculated Geometric Mean Exposure Dose (mg/kg/d) ⁽¹⁾	ATSDR MRL (non-cancer) (mg/kg/d)	Does Calculated Maximum Exposure Dose Exceed non-CA HG (child/adult)	Does Calculated Geometric Mean Exposure Dose Exceed non-CA HG (child/adult)
TCE	Child 0.074 Adult 0.021	Child 0.0073 Adult 0.0037	0.2 acute oral; Proposed changes: RfD = 0.0003 mg/kg/d	<u>Current HG:</u> Child NO Adult NO <u>Proposed HG:</u> Child YES Adult YES	<u>Current HG:</u> Child NO Adult NO <u>Proposed HG:</u> Child YES Adult YES
1,1-DCE	Child 0.0027 Adult 0.00076	Child 0.0013 Adult 0.00036	0.009	Child NO Adult NO	Child NO Adult NO

Table 9. Town Of Aberdeen Municipal Drinking Water Well Data Collected From March 1992 Through October 2008 Summary TCE Analytical Data and ATSDR Comparison Values.

Location	Contaminant	Frequency of Detection	Range of Concentrations Greater Than the CV (µg/L)	Number of Detections Greater Than CV	Geometric Mean Concentration (µg/L)	Health-Based Comparison Value (CV) (µg/L)	Type of CV
TOA well #5	TCE	37/59	5.1 – 8.8	12	2.9	5	MCL
TOA well #9	TCE	20/34	NA	0	2.0	5	MCL

Table 10. Town Of Aberdeen Municipal Drinking Water Well Data Collected From March 1992 Through October 2008 Exposure Dose Estimate Data and Health Guideline Comparison Data.

Contaminant	Calculated Maximum Exposure Dose (mg/kg/d) ⁽¹⁾	Calculated Geometric Mean Exposure Dose (mg/kg/d) ⁽¹⁾	ATSDR MRL (non-cancer) (mg/kg/d)	Does Calculated Maximum Exposure Dose Exceeds non-CA HG (child/adult)	Does Calculated Geometric Mean Exposure Dose Exceeds non-CA HG (child/adult)
TCE well #5	Child 0.00035 Adult 0.00010	Child 0.00012 Adult 0.000083	0.2 acute oral; Proposed changes: RfD = 0.0003 mg/kg/d	<u>Current HG:</u> Child NO Adult NO <u>Proposed HG:</u> Child YES Adult NO	<u>Current HG:</u> Child NO Adult NO <u>Proposed HG:</u> Child NO Adult NO

Notes: ⁽¹⁾ Assumes 40% maximum well contribution for blended water supply
HG = Health Guideline

Table 11. Data Summary and Screening Values Analysis for Ground Water Samples Collected by NCDENR in 2004 for Powder Metal Products Investigation.

Contaminant	Frequency of Detection	Range of Concentrations (µg/L)	Number of Detections Greater Than CV	Geometric Mean Concentration (µg/L)	ATSDR Health-Based CV (µg/L)	Type of CV
TCE	7/14	0.6 to 1489	5	34.5	5	MCL
1,1-DCE	4/14	0.9 to 15.8	2	3.9	90 Child 300 Adult	Chronic EMEG
					7	MCL
cis-1,2-DCE	4/14	1 to 8.9	0	2.5	3,000 Child 10,000 Adult	Intermediate EMEG
					70	MCL
Chloroform	1/14	0.39	1	Not Applicable	100 Child 400 Adult	Chronic EMEG
					80 (as Trihalomethanes)	MCL

Table 12. Site-Specific Exposure Dose Estimates and Health Guideline Comparison for Ground Water Samples Collected by NCDENR in 2004 for Powder Metal Products Investigation.

Contaminant	Calculated Maximum Exposure Dose (mg/kg/d) ⁽¹⁾	Calculated Geometric Mean Exposure Dose (mg/kg/d) ⁽¹⁾	ATSDR MRL (non-cancer) (mg/kg/d)	Does Calculated Maximum Exposure Dose Exceed non-CA HG (child/adult)	Does Calculated Geometric Mean Exposure Dose Exceed non-CA HG (child/adult)
TCE	Child 0.15 Adult 0.042	Child 0.0034 Adult 0.00099	0.2 acute oral; Proposed changes: RfD = 0.0003 mg/kg/d	<u>Current HG:</u> Child NO Adult NO <u>Proposed HG:</u> Child YES Adult YES	<u>Current HG:</u> Child NO Adult NO <u>Proposed HG:</u> Child YES Adult NO
1,1-DCE	Child 0.0016 Adult 0.00045	Child 0.00039 Adult 0.00011	0.009 Chronic	Child NO Adult NO	Child NO Adult NO
Chloroform	Child 0.000039 Adult 0.000011	-----	0.01 (EPA RfD _{oral} 0.01)	Child NO Adult NO	----

Table 13. Data Summary and Screening Value Analysis for Ground Water Samples Collected by NCDENR for April 2008 Aberdeen Well Survey.

Contaminant	Frequency of Detection	Range of Concentrations (µg/L)	Number of Detections Greater Than CV	Geometric Mean Concentration (µg/L)	ATSDR Health-Based CV (µg/L)	Type of CV
p,p-DDD	1/2	0.012 J	0	----	5 Child 20 Adult	Intermediate EMEG
					0.1	CREG
p,p-DDT	1/2	0.011 J	0	----	5 Child 20 Adult	Intermediate EMEG
					0.1	CREG
Endosulfan Sulfate	1/2	0.014 J	0	----	20 Child 70 Adult ⁽¹⁾	Intermediate EMEG
Endrin Aldehyde	1/2	0.022 J	0	----	3 Child 10 Adult ⁽²⁾	Chronic EMEG
Endrin Ketone	1/2	0.017 J	0	----	3 Child 10 Adult ⁽²⁾	Chronic EMEG
Chloroform	1/2	0.011 J	0	----	100 Child 400 Adult	Chronic EMEG
					80 ⁽³⁾	MCL
MTBE	1/2	0.12 J	0	----	3000 Child 10,000 Adult	Intermediate EM

Notes: ⁽¹⁾ ATSDR comparison values not given for Endosulfan Sulfate. Listed CVs are for Endosulfan.

⁽²⁾ ATSDR comparison values not given for Endrin Aldehyde or Endrin Ketone, Listed CVs are for Endrin.

⁽³⁾ MCL listed is for Trihalomethanes. Chloroform identified as “likely” carcinogen by USEPA and is “reasonably anticipated to be a carcinogen” by National Toxicology Program. ATSDR identifies USEPA RfD_{oral} (0.01 mg/kg/d) to be protective against cancer risk, so no ATSDR CREG has been derived.

Table 14. Site-Specific Exposure Dose Estimates and Health Guideline Comparison for Ground Water Samples Collected by NCDENR for April 2008 Aberdeen Well Survey.

Contaminant	Calculated Maximum Exposure Dose (mg/kg/d) ⁽¹⁾	Calculated Geometric Mean Exposure Dose (mg/kg/d) ⁽¹⁾	ATSDR MRL (non-cancer), Type (mg/kg/d)	Does Calculated Maximum Exposure Dose Exceed non-CA HG (child/adult)	Does Calculated Geometric Mean Exposure Dose Exceed non-CA HG (child/adult)
p,p-DDD	Child 0.0000012 Adult 0.0000034	----	-----	----	----
p,p-DDT	Child 0.0000011 Adult 0.0000031	----	0.0005 Intermediate	Child NO Adult NO	----
Endosulfan Sulfate	Child 0.0000014 Adult 0.0000040	-----	0.002 Chronic ⁽¹⁾	Child NO Adult NO	----
Endrin Aldehyde	Child 0.0000022 Adult 0.0000063	----	0.0003 Chronic ⁽²⁾	Child NO Adult NO	----
Endrin Ketone	Child 0.0000017 Adult 0.0000049	----	0.0003 Chronic ⁽²⁾	Child NO Adult NO	----
Chloroform	Child 0.000011 Adult 0.0000031	----	0.01 Chronic ⁽³⁾	Child NO Adult NO	----
MTBE	Child 0.000012 Adult 0.0000034	----	0.3 Intermediate	Child NO Adult NO	----

Notes: ⁽¹⁾ ATSDR Health Guidelines not given for Endosulfan Sulfate. Listed CVs are for Endosulfan.
⁽²⁾ Health Guidelines not given for Endrin Aldehyde or Endrin Ketone, Listed CVs are for Endrin.
⁽³⁾ Chloroform identified as “likely” carcinogen by USEPA and is “reasonably anticipated to be a carcinogen” by National Toxicology Program. ATSDR identifies USEPA RfD_{oral} (0.01 mg/kg/d) to be protective against cancer risk, so no ATSDR CREG has been derived.

Table 15. Calculated Theoretical Increased Cancer Risk for a Population of 10,000. Risk Estimates Assume Exposure to the Highest Detected Contaminant Concentration for Each Sampling Event.

Sampling Event	Contaminant Of Concern	Cancer Slope Factor (CSF) (mg/kg/d) ⁻¹	30-Year Exposure at Maximum Concentration	30-Year Exposure at Geometric Mean Concentration	70-year Exposure at Maximum Concentration	70-year Exposure at Geometric Mean Concentration
1991-1993 Private Well Waters for Route 211 Site	TCE	No current CSF; Proposed changes: CSF range from 0.02 to 0.4 (mg/kg/d) ⁻¹	2 to 40	<1 to 10	----	----
	Alpha-HCH	6.3	----	----	2	1
	Beta-HCH	1.8	----	----	<1	<1
	Total HCH	1.8	----	----	20	50
Jul – Aug 1995 Crestline Investigation of Private Wells	TCE	(see above)	2 to 40	<1 to 6	----	----
	1,1-DCE	0.6 (IRIS)	2	<1	----	----
1992 – 2008 TOA Well #5 ⁽¹⁾	TCE	(see above)	<1	<1	----	----
2004 NCDENR PMP Ground Water	TCE	(see above)	4 to 70	<1 to 4	----	----
	Chloroform	0.01 (USEPA RfD _{oral})	Estimated Exposure Dose < RfD	----	----	----
	1,1-DCE	0.6 (IRIS)	3	<1	----	----
2008 NCDENR Aberdeen Well Survey	p,p-DDD	0.24	----	----	<1	----
	p,p-DDT	0.34	----	----	<1	----
	Chloroform	0.01 (USEPA RfD _{oral})	----	----	Estimated Exposure Dose < RfD	----

Table 16. Oral Exposure Route Health Effects Data Selected for Site-Specific Exposure Dose Comparisons

Contaminant Of Concern	Non-Cancer Effects Study Data	Cancer Effects Study Data
TCE	Lowest NOAEL chronic exposure: Rat, gavage in oil, renal effects at 50 mg/kg/d; Lowest LOAEL chronic exposure: Rat, gavage in oil, multiple systemic effects at 250 mg/kg/d; Acute LOAEL - Critical study for MRL: Mouse pups, gavage in 20% oil, behavioral & developmental effects at 50 mg/kg/d (UCF = 300); ATSDR: Lack of adequate studies with suitable endpoints to develop intermediate or chronic MRL; [No human study data available]	LOAEL intermediate exposure: Male mouse, gastrointestinal effects at 18 mg/kg/d [No human study data available]
1,1-DCE	LOAEL: critical study - Rat, chronic exposure in drinking water, Hepatic effects at 9 mg/kg/d (UCF = 1000); Lowest chronic NOAEL: Rat, exposure in water, hepatic effects at 10 mg/kg/d; Lowest intermediate NOAEL: Dog, in drinking water, hematological/hepatic/renal effects at 25 mg/kg/d [No human study data available]	USEPA IRIS (1992): Rat, chronic exposure by gavage, lowest CEL 5 mg/kg/d, pheochromocytomas; CSF 0.6 (mg/kg/d) ⁻¹
gamma-HCH (Lindane)	LOAEL intermediate exposure, Critical study - Female mouse, food exposure, immunological effects at 0.012 mg/kg/d; Lowest chronic NOAEL: Female rat, food exposure, hepatic/renal effects at 4 mg/kg/d; Lowest chronic LOAEL: Female rat, food exposure, hepatic/renal effects at 7 mg/kg/d; [No human study data available]	Male mouse, food exposure, hepatocellular carcinoma at 13.6 mg/kg/d [No human study data available]
Other isomers of HCH	Chronic Critical study NOAEL: Female rat, food exposure, hepatic effects at 0.8 mg/kg/d; Lowest chronic LOAEL: Female rat, food exposure, hepatic effects at 0.8 mg/kg/d; males at 56 mg/kg/d; Lowest intermediate NOAEL: Male rat, food exposure, neurological effects at 0.04 mg/kg/d and Female, reproductive effects at 0.2 mg/kg/d [No human study data available]	Lowest chronic exposure CEL: Mouse, food exposure, hepatocellular carcinoma at 10 & 17 mg/kg/d; Lowest intermediate exposure CEL: Female rat, food exposure, hepatic effects at 2 & 3 mg/kg/d [No human study data available]

Notes: Source ATSDR Toxicological Profiles (see References)
 Selected references are lowest NOAEL or LOAEL study or study for non-cancer or cancer effects, or "Critical Study" utilized for development of MRL
 NOAEL = no observed adverse affect level
 LOAEL = lowest observed adverse affect level
 UCF = Uncertainty Factor
 CEL = cancer effect level

Table 17. Theoretical Increased Number of Cancers Qualitative Assessment Categories Utilized by NCDHHS for Aberdeen Contaminated Ground Water Study

Per population of	No Increased Risk	No Apparent Increased Risk	“Low”	“Moderate”	“High”	“Very High”
10,000	---	---	<1	1 to 9	10 to 100	>100
100,000	---	<1	1 to 9	10 to 99	100 to 1,000	>1,000
1,000,000	<1	10 to 99	<100	101 to 999	1,000 to 10,000	>10,000

Notes: “Low” theoretical increased number of cancers = 0.01%, and “Very High” = 1% increase over expected number of cancer cases in a typical population (approximately 33%)

Appendix C
ATSDR Evaluation Process

Comparison Values and the Screening Process

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 or water) 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.

As health-based thresholds, CVs are set at a concentration below which no known or anticipated adverse human health effects are expected to occur. Different CVs are developed for cancer and non-cancer health effects. Non-cancer levels are based on validated toxicologic 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.

CVs used to select contaminants for further evaluation:

Environmental Media Evaluation Guides (EMEGs) represent concentrations of substances in water, soil, and air to which humans may be exposed over specified time periods without experiencing non-cancer adverse health effects. The EMEG is derived from the Agency for Toxic Substances and Disease Registry's (ATSDR) minimal risk level (MRL).

Reference Dose Media Evaluation Guides (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 Environmental Protection Agency's (EPA's) oral reference dose (RfD).

Cancer Risk Evaluation Guides (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 lifetime. CREGs are calculated from EPA's cancer slope factors (CSFs) or inhalation unit risk (IUR) values.

Risk-Based Concentrations (RBCs) are the estimated contaminant concentrations in media where non-carcinogenic health effects are unlikely. The RBCs used in this PHA were derived by EPA's Region 3 toxicologists.

EPA Soil Screening Levels (SSLs) are estimated contaminant concentrations in soil at which additional evaluation is needed to determine if action is required to eliminate or reduce exposure.

Estimation of Exposure Dose

The next step is to consider those contaminants that are present at levels above the CVs and further identify which chemicals and exposure situations are likely to be a health hazard. Child and adult exposure doses are calculated for the site-specific exposure scenario, using our assumptions of who goes on the site and how often they contact the site contaminants. The exposure dose is the estimated amount of a contaminant that gets into a person's body.

Non-Cancer Health Effects

The doses calculated for exposure to each individual chemical are then compared to an established health guideline, such as a MRL or RfD, in order to assess whether adverse health impacts from exposure are expected. These health guidelines, developed by ATSDR and EPA, are chemical-specific values that are based on the available scientific literature and are considered protective of human health. Non-carcinogenic effects, unlike carcinogenic effects, are believed to have a threshold, that is, a dose below which adverse health effects will not occur. As a result, the current practice for deriving health guidelines is to identify, usually from animal toxicology experiments, a No Observed Adverse Effect Level (or NOAEL), which indicates that no effects are observed at a particular exposure level. This is the experimental exposure level in animals (and sometimes humans) at which no adverse toxic effect is observed. The NOAEL is then modified with an uncertainty (or safety) factor, which reflects the degree of uncertainty that exists when experimental animal data are extrapolated to the general human population. The magnitude of the uncertainty factor considers various factors such as sensitive subpopulations (for example; children, pregnant women, and the elderly), extrapolation from animals to humans, and the completeness of available data. Thus, exposure doses at or below the established health guideline are not expected to result in adverse health effects because these values are much lower (and more human health protective) than doses that do not cause adverse health effects in laboratory animal studies. For non-cancer health effects, the following health guidelines are described below in more detail. It is important to consider that the methodology used to develop these health guidelines does not provide any information on the presence, absence, or level of cancer risk. Therefore, a separate cancer evaluation is necessary for potentially cancer-causing chemicals detected in samples at this site. A more detailed discussion of the evaluation of cancer risks is presented in the following section.

Minimal Risk Levels (MRLs) – developed by ATSDR

ATSDR has developed MRLs for contaminants commonly found at hazardous waste sites. The MRL is an estimate of daily exposure to a contaminant below which non-cancer, adverse health effects are unlikely to occur. MRLs are developed for different routes of exposure, such as inhalation and ingestion, and for lengths of exposure, such as acute (less than 14 days), intermediate (15-364 days), and chronic (365 days or greater). At this time, ATSDR has not developed MRLs for dermal exposure. A complete list of the available MRLs can be found at <http://www.atsdr.cdc.gov/mrls.html>.

References Doses (RfDs) – developed by EPA

The RfDs are an estimate of the daily, lifetime exposure of human populations to a possible hazard that is not likely to cause non-cancerous health effects. RfDs consider exposures to sensitive sub-populations, such as the elderly, children, and the developing fetus. USEPA RfDs

have been developed using information from the available scientific literature and have been calculated for oral and inhalation exposures. A complete list of the available RfDs can be found at <http://www.epa.gov/iris>.

If the estimated exposure dose for a chemical is less than the health guideline value, the exposure is unlikely to result in non-cancer health effects. If the calculated exposure dose is greater than the health guideline, the exposure dose is compared to known toxicological values for the particular chemical and is discussed in more detail in the text of the assessment. The known toxicological values are doses derived from human and animal studies that are presented in the ATSDR Toxicological Profiles and EPA's Integrated Risk Information System (IRIS). A direct comparison of site-specific exposure doses to study-derived exposures and doses found to cause adverse health effects is the basis for deciding whether health effects are likely to occur. This in-depth evaluation is performed by comparing calculated exposure doses with known toxicological values, such as the no-observed adverse-effect-level (NOAEL) and the lowest-observed-adverse-effect-level (LOAEL) from studies used to derive the MRL or RfD for a chemical.

Cancer Risks

Exposure to a cancer-causing compound, even at low concentrations, is assumed associated with some increased risk for evaluation purposes. The estimated excess risk of developing cancer from exposure to contaminants associated with the site was calculated by multiplying the site-specific adult exposure doses, with a slight modification, by EPA's chemical-specific cancer slope factors (CSFs or cancer potency estimates), which are available at <http://www.epa.gov/iris>. Calculated dermal doses were compared with the oral CSFs.

Because of the uncertainties involved with estimating carcinogenic risk, ATSDR employs a weight-of-evidence approach in evaluating all relevant data. Therefore, the carcinogenic risk is also described in words (qualitatively) rather than giving a numerical risk estimate only.

Exposure Dose Calculations and Results for the Aberdeen Contaminated Ground Water Site

When contaminant concentrations at the site exceed established CVs, the chemical needs additional evaluation. To evaluate the potential for human exposure to contaminants present at the site and potential health effects from site-specific activities, ATSDR estimates human exposure to the site contaminant from different environmental media by calculating exposure doses. A brief discussion of the calculations and assumptions is presented below.

Well Water Pathway (Ingestion, Inhalation, Dermal Contact)

The ATSDR exposure dose formula used for the well water pathway is:

$$ED = C \times IR \times EF / 1000 \times BW$$

where:

ED = exposure dose in milligrams per kilogram per day (mg/kg/day)

C = concentration of contaminant in water in parts per billion (ppb or µg/L)

IR = ingestion rate in liters per day (L/day)

EF = exposure factor, days of exposure divided by 365 (unitless) 1000 = conversion factor in micrograms per milligram (µg/mg)

BW = body weight in kilogram (kg) Assumptions used were based on default values and/or professional judgment.

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:

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

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

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:

$$\text{HI} = \text{HQ}_1 + \dots + \text{HQ}_n$$

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 Toxic Action of Chemical Mixtures* (ATSDR 2004).

Appendix D
ATSDR ToxFAQs Sheets

This fact sheet answers the most frequently asked health questions (FAQs) about trichloroethylene. For more information, call the ATSDR Information Center at 1-888-422-8737. This fact sheet is one in a series of summaries about hazardous substances and their health effects. This information is important because this substance may harm you. The effects of exposure to any hazardous substance depend on the dose, the duration, how you are exposed, personal traits and habits, and whether other chemicals are present.

HIGHLIGHTS: Trichloroethylene is a colorless liquid which is used as a solvent for cleaning metal parts. Drinking or breathing high levels of trichloroethylene may cause nervous system effects, liver and lung damage, abnormal heartbeat, coma, and possibly death. Trichloroethylene has been found in at least 852 of the 1,430 National Priorities List sites identified by the Environmental Protection Agency (EPA).

What is trichloroethylene?

Trichloroethylene (TCE) is a nonflammable, colorless liquid with a somewhat sweet odor and a sweet, burning taste. It is used mainly as a solvent to remove grease from metal parts, but it is also an ingredient in adhesives, paint removers, typewriter correction fluids, and spot removers.

Trichloroethylene is not thought to occur naturally in the environment. However, it has been found in underground water sources and many surface waters as a result of the manufacture, use, and disposal of the chemical.

What happens to trichloroethylene when it enters the environment?

- ❑ Trichloroethylene dissolves a little in water, but it can remain in ground water for a long time.
- ❑ Trichloroethylene quickly evaporates from surface water, so it is commonly found as a vapor in the air.
- ❑ Trichloroethylene evaporates less easily from the soil than from surface water. It may stick to particles and remain for a long time.
- ❑ Trichloroethylene may stick to particles in water, which will cause it to eventually settle to the bottom sediment.
- ❑ Trichloroethylene does not build up significantly in

plants and animals.

How might I be exposed to trichloroethylene?

- ❑ Breathing air in and around the home which has been contaminated with trichloroethylene vapors from shower water or household products such as spot removers and typewriter correction fluid.
- ❑ Drinking, swimming, or showering in water that has been contaminated with trichloroethylene.
- ❑ Contact with soil contaminated with trichloroethylene, such as near a hazardous waste site.
- ❑ Contact with the skin or breathing contaminated air while manufacturing trichloroethylene or using it at work to wash paint or grease from skin or equipment.

How can trichloroethylene affect my health?

Breathing small amounts may cause headaches, lung irritation, dizziness, poor coordination, and difficulty concentrating.

Breathing large amounts of trichloroethylene may cause impaired heart function, unconsciousness, and death. Breathing it for long periods may cause nerve, kidney, and liver damage.

ToxFAQs™ Internet address is <http://www.atsdr.cdc.gov/toxfaq.html>

Drinking large amounts of trichloroethylene may cause nausea, liver damage, unconsciousness, impaired heart function, or death.

Drinking small amounts of trichloroethylene for long periods may cause liver and kidney damage, impaired immune system function, and impaired fetal development in pregnant women, although the extent of some of these effects is not yet clear.

Skin contact with trichloroethylene for short periods may cause skin rashes.

How likely is trichloroethylene to cause cancer?

Some studies with mice and rats have suggested that high levels of trichloroethylene may cause liver, kidney, or lung cancer. Some studies of people exposed over long periods to high levels of trichloroethylene in drinking water or in workplace air have found evidence of increased cancer. Although, there are some concerns about the studies of people who were exposed to trichloroethylene, some of the effects found in people were similar to effects in animals.

In its 9th Report on Carcinogens, the National Toxicology Program (NTP) determined that trichloroethylene is "reasonably anticipated to be a human carcinogen." The International Agency for Research on Cancer (IARC) has determined that trichloroethylene is "probably carcinogenic to humans."

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

If you have recently been exposed to trichloroethylene, it can be detected in your breath, blood, or urine. The breath test, if it is performed soon after exposure, can tell if you have been exposed to even a small amount of trichloroethylene.

Exposure to larger amounts is assessed by blood

and urine tests, which can detect trichloroethylene and many of its breakdown products for up to a week after exposure. However, exposure to other similar chemicals can produce the same breakdown products, so their detection is not absolute proof of exposure to trichloroethylene. This test isn't available at most doctors' offices, but can be done at special laboratories that have the right equipment.

Has the federal government made recommendations to protect human health?

The EPA has set a maximum contaminant level for trichloroethylene in drinking water at 0.005 milligrams per liter (0.005 mg/L) or 5 parts of TCE per billion parts water.

The EPA has also developed regulations for the handling and disposal of trichloroethylene.

The Occupational Safety and Health Administration (OSHA) has set an exposure limit of 100 parts of trichloroethylene per million parts of air (100 ppm) for an 8-hour workday, 40-hour workweek.

Glossary

Carcinogenicity: The ability of a substance to cause cancer.

CAS: Chemical Abstracts Service.

Evaporate: To change into a vapor or gas.

Milligram (mg): One thousandth of a gram.

Nonflammable: Will not burn.

ppm: Parts per million.

Sediment: Mud and debris that have settled to the bottom of a body of water.

Solvent: A chemical that dissolves other substances.

References

This ToxFAQs information is taken from the 1997 Toxicological Profile for Trichloroethylene (update) produced by the Agency for Toxic Substances and Disease Registry, Public Health Service, U.S. Department of Health and Human Services, Public Health Service in Atlanta, GA.

Where can I get more information? For more information, contact the Agency for Toxic Substances and Disease Registry, Division of Toxicology, 1600 Clifton Road NE, Mailstop F-32, Atlanta, GA 30333. Phone: 1-888-422-8737, FAX: 770-488-4178. ToxFAQs™ Internet address is <http://www.atsdr.cdc.gov/toxfaq.html>. ATSDR can tell you where to find occupational and environmental health clinics. Their specialists can recognize, evaluate, and treat illnesses resulting from exposure to hazardous substances. You can also contact your community or state health or environmental quality department if you have any more questions or concerns.

The ToxGuide™ is developed to be used as a pocket guide. Tear off at perforation and fold along lines.

Sources of Exposure	Toxicokinetics and Normal Human Levels	Biomarkers/Environmental Levels	ToxGuide™ for Lead Pb CAS# 7439-92-1 October 2007
<p>General Populations</p> <ul style="list-style-type: none"> The most likely source of exposure is ingestion of contaminated food and drinking water. Exposure can also occur via inadvertent ingestion of contaminated soil/dust or lead-based paint. Lead can leach into drinking water from lead-soldered joints or leaded pipes in water distribution systems or individual houses. Lead may also enter foods if they are put into improperly glazed pottery or ceramic dishes. Some non-Western folk remedies may contain substantial amounts of lead. Some types of hair dyes and cosmetics may contain lead compounds. Other potential sources of exposure are hobbies that use lead: casting ammunition and m fishing weights; soldering with lead solder; making stained glass; using firing ranges. Leaded gasoline is still used in some race cars, airplanes, and off-road vehicles. <p>Occupational Populations</p> <ul style="list-style-type: none"> Potentially high levels of lead may occur in the following industries: lead smelting and refining industries, battery manufacturing plants, steel welding or cutting operations, construction, rubber products and plastics industries, printing industries, firing ranges, radiator repair shops and other industries requiring flame soldering of lead solder. 	<p>Toxicokinetics</p> <ul style="list-style-type: none"> Approximately 95% of deposited inorganic lead that is inhaled is absorbed. The extent and rate of gastrointestinal absorption of inorganic lead are influenced by the physiological state of the exposed individual and the species of the lead compound. Gastrointestinal absorption of lead is higher in children (40–50%) than in adults (3–10%). The presence of food in the gastrointestinal tract decreases absorption. Absorption of lead from soil is less than that of dissolved lead, but is similarly depressed by meals (26% fasted; 2.5% when ingested with a meal). In adults, about 94% of the total amount of lead in the body is contained in the bones and teeth versus about 73% in children. The elimination half-lives for inorganic lead in blood and bone are approximately 30 days and 27 years, respectively. Independent of the route of exposure, absorbed lead is excreted primarily in urine and feces. <p>Normal Human Levels</p> <ul style="list-style-type: none"> Lead levels in blood (geometric mean, 1999-2002): <ul style="list-style-type: none"> 1.9 µg/dL for children 1-5 years 1.5 µg/dL for adults 20–59 years Lead levels in urine (geometric mean, 2001-2002): <ul style="list-style-type: none"> 0.677 µg/L for ≥6 years of age 	<p>Biomarkers</p> <ul style="list-style-type: none"> Analysis of lead in whole blood is the most common and accurate method of assessing lead exposure. Erythrocyte protoporphyrin (EP) tests can also be used, but are not as sensitive at low blood lead levels (<20 µg/dL). Lead in blood reflects recent exposure. Bone lead measurements are an indicator of cumulative exposure. Measurements of urinary lead levels and hair have been used to assess lead exposure; however, they are not as reliable. <p>Environmental Levels</p> <p><i>Air</i></p> <ul style="list-style-type: none"> The concentration of lead in air samples (2002) is <0.05 µg/m³. <p><i>Sediment and Soil</i></p> <ul style="list-style-type: none"> The natural lead content of soil typically ranges from <10 to 30 µg/g. However, lead levels in the top layers of soil vary widely due to deposition and accumulation of atmospheric particulates from anthropogenic sources. <p><i>Water</i></p> <ul style="list-style-type: none"> Levels of lead in surface water and groundwater in the U.S. range between 5 and 30 µg/L. <p>Reference</p> <p>Agency for Toxic Substances and Disease Registry (ATSDR). 2007. Toxicological Profile for Lead. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Services.</p>	

U.S. Department of Health and Human Services
 Public Health Service
 Agency for Toxic Substances and Disease Registry
www.atsdr.cdc.gov

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 Applied Toxicology Branch

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www.atsdr.cdc.gov/toxpro2.html



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Chemical and Physical Information	Routes of Exposure	Relevance to Public Health (Health Effects)
<p>Lead is a metal</p> <ul style="list-style-type: none"> Lead is a naturally-occurring bluish gray metal that is rarely found in its elemental form, but occurs in the Earth's crust primarily as the mineral galena (PbS), and to a lesser extent as anglesite (PbSO₄) and cerussite (PbCO₃). Lead is not a particularly abundant element, but its ore deposits are readily accessible and widely distributed throughout the world. Its properties, such as corrosion resistance, density, and low melting point, make it a familiar metal in pipes, solder, weights, and storage batteries. Natural lead is a mixture of four stable isotopes, ²⁰⁸Pb (51–53%), ²⁰⁶Pb (23.5–27%), ²⁰⁷Pb (20.5–23%), and ²⁰⁴Pb (1.35–1.5%). Lead isotopes are the stable decay product of three naturally radioactive elements: ²⁰³Pb from uranium, ²⁰⁷Pb from actinium, and ²⁰⁸Pb from thorium. 	<ul style="list-style-type: none"> Inhalation – Primary route for occupational exposure. Larger particles (>2.5 μm) that are deposited in the ciliated airways (nasopharyngeal and tracheobronchial regions) can be transferred by mucociliary transport into the esophagus and swallowed. Oral – Primary route of exposure for the general population. Dermal – Studies in animals have shown that organic lead is well absorbed through the skin. <p>Lead in the Environment</p> <ul style="list-style-type: none"> Lead is dispersed throughout the environment primarily as the result of anthropogenic activities. In the air, lead is in the form of particles and is removed by rain or gravitational settling. The fate of lead in soil is affected by the adsorption at mineral interfaces, which are dependent upon physical and chemical characteristics of the soil (e.g., pH, soil type, particle size, organic matter content). Sources of lead in dust and soil can include lead from weathering and chipping of lead-based paint from buildings, bridges, and other structures. The solubility of lead compounds in water is a function of pH, hardness, salinity, and the presence of humic material. Solubility is highest in soft, acidic water. 	<p>Health effects are determined by the dose (how much), the duration (how long), and the route of exposure.</p> <p>Minimal Risk Levels (MRLs)</p> <ul style="list-style-type: none"> MRLs were not derived for lead because a clear threshold for some of the more sensitive effects in humans has not been identified. In lieu of MRLs, ATSDR has developed a framework to guide decisions at lead sites. This approach utilizes site-specific exposure data to estimate internal doses as measured by blood lead levels (PbBs) (see Appendix D in the Toxicological Profile). <p>Health Effects</p> <p><i>Hematological</i></p> <ul style="list-style-type: none"> Decreased activity of several heme biosynthesis enzymes at PbB <10 μg/dL. <p><i>Gastrointestinal</i></p> <ul style="list-style-type: none"> Colic in children – PbB 60–100 μg/dL. <p><i>Cardiovascular</i></p> <ul style="list-style-type: none"> Elevated blood pressure – PbB <10 μg/dL. <p><i>Renal</i></p> <ul style="list-style-type: none"> Decreased glomerular filtration rate at mean PbB <20 μg/dL. <p><i>Neurological</i></p> <ul style="list-style-type: none"> Encephalopathy – PbB 100–120 μg/dL (adults) 70–100 μg/dL (children). Peripheral neuropathy – PbB 40 μg/dL. Neurobehavioral and neuropsychological effects in adults – PbB 40–80 μg/dL. Cognitive and neurobehavioral effects in children at PbB <10 μg/dL. <p><i>Reproductive</i></p> <ul style="list-style-type: none"> Reduced fertility – PbB >40 μg/dL. <p>Children's Health</p> <ul style="list-style-type: none"> Children are more vulnerable to the effects of lead than adults. The most common source of lead exposure for children is lead-based paint. Lead exposures during infancy or childhood may result in anemia, neurological impairment, renal alterations, colic, and impaired metabolism of vitamin D. Lead exposures either <i>in utero</i>, during infancy, or during childhood may result in delays or impairment of neurological development, neurobehavioral deficits including IQ deficits, low birth weight, and low gestational age, growth retardation, and delayed sexual maturation in girls. Ensuring a diet that is nutritionally adequate in calcium and iron may decrease the absorbed dose of lead.

This fact sheet answers the most frequently asked health questions (FAQs) about 1,1-dichloroethene. For more information, call the ATSDR Information Center at 1-888-422-8737. This fact sheet is one in a series of summaries about hazardous substances and their health effects. It's important you understand this information because this substance may harm you. The effects of exposure to any hazardous substance depend on the dose, the duration, how you are exposed, personal traits and habits, and whether other chemicals are present.

SUMMARY: Exposure to 1,1-dichloroethene occurs mainly in the workplace. Breathing high levels of 1,1-dichloroethene can affect the liver, kidney, and central nervous system. This chemical has been found in at least 515 of 1,416 National Priorities List sites identified by the Environmental Protection Agency.

What is 1,1-dichloroethene?

(Pronounced 1,1-dī'klōr'ō ēth'ēn)

1,1-Dichloroethene is an industrial chemical that is not found naturally in the environment. It is a colorless liquid with a mild, sweet smell. It is also called vinylidene chloride.

1,1-Dichloroethene is used to make certain plastics, such as flexible films like food wrap, and in packaging materials. It is also used to make flame retardant coatings for fiber and carpet backings, and in piping, coating for steel pipes, and in adhesive applications.

What happens to 1,1-dichloroethene when it enters the environment?

- 1,1-Dichloroethene enters the environment from industries that make or use it.
- 1,1-Dichloroethene evaporates very quickly from water and soil to the air.
- In the air, it takes about 4 days for it to break down.
- 1,1-Dichloroethene breaks down very slowly in water.
- It does not accumulate very much in fish or birds.
- In soil, 1,1-dichloroethene is slowly transformed to other less harmful chemicals.

How might I be exposed to 1,1-dichloroethene?

- Workers may be exposed in industries that make or use 1,1-dichloroethene (these industries are mainly in Texas and Louisiana).
- Food that is wrapped in plastic wrap may contain very low levels of 1,1-dichloroethene. The government controls these levels to prevent harm to your health.
- A small percentage (3%) of the drinking water supplies may contain very low levels of 1,1-dichloroethene.
- Air near factories that make or use 1,1-dichloroethene and air near hazardous waste sites may contain low levels of it.

How can 1,1-dichloroethene affect my health?

The main effect from breathing high levels of 1,1-dichloroethene is on the central nervous system. Some people lost their breath and fainted after breathing high levels of the chemical.

Breathing lower levels of 1,1-dichloroethene in air for a long time may damage your nervous system, liver, and lungs. Workers exposed to 1,1-dichloroethene have reported a loss in liver function, but other chemicals were present.

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Animals that breathed high levels of 1,1-dichloroethene had damaged livers, kidneys, and lungs. The offspring of some of the animals had a higher number of birth defects. We do not know if birth defects occur when people are exposed to 1,1-dichloroethene.

Animals that ingested high levels of 1,1-dichloroethene had damaged livers, kidneys, and lungs. There were no birth defects in animals that ingested the chemical.

Spilling 1,1-dichloroethene on your skin or in your eyes can cause irritation.

How likely is 1,1-dichloroethene to cause cancer?

The Environmental Protection Agency (EPA) has determined that 1,1-dichloroethene is a possible human carcinogen.

Studies on workers who breathed 1,1-dichloroethene have not shown an increase in cancer. These studies, however, are not conclusive because of the small numbers of workers and the short time studied.

Animal studies have shown mixed results. Several studies reported an increase in tumors in rats and mice, and other studies reported no such effects.

Is there a medical test to show whether I've been exposed to 1,1-dichloroethene?

Tests are available to measure levels of 1,1-dichloroethene in breath, urine, and body tissues. These tests are not usually available in your doctor's office. However, a sample taken in your doctor's office can be sent to a special laboratory if necessary.

Because 1,1-dichloroethene leaves the body fairly quickly, these methods are useful only for finding exposures that have occurred within the last few days. These tests can't tell you if adverse health effects will occur from exposure to 1,1-dichloroethene.

Has the federal government made recommendations to protect human health?

The EPA has set a limit in drinking water of 0.007 parts of 1,1-dichloroethene per million parts of drinking water (0.007 ppm). EPA requires that discharges or spills into the environment of 5,000 pounds or more of 1,1-dichloroethene be reported.

The Occupational Safety and Health Administration (OSHA) has set an occupational exposure limit of 1 ppm of 1,1-dichloroethene in workplace air for an 8-hour workday, 40-hour workweek.

The National Institute for Occupational Safety and Health (NIOSH) currently recommends that workers breathe as little 1,1-dichloroethene as possible.

Glossary

Carcinogen: A substance that can cause cancer.

CAS: Chemical Abstracts Service.

Ingesting: Taking food or drink into your body.

ppm: Parts per million.

Tumor: An abnormal mass of tissue.

References

Agency for Toxic Substances and Disease Registry (ATSDR). 1994. Toxicological profile for 1,1-dichloroethene. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service.

Where can I get more information? For more information, contact the Agency for Toxic Substances and Disease Registry, Division of Toxicology, 1600 Clifton Road NE, Mailstop E-29, Atlanta, GA 30333. Phone: 1-888-422-8737, FAX: 404-498-0093. ToxFAQs Internet address via WWW is <http://www.atsdr.cdc.gov/toxfaq.html>. ATSDR can tell you where to find occupational and environmental health clinics. Their specialists can recognize, evaluate, and treat illnesses resulting from exposure to hazardous substances. You can also contact your community or state health or environmental quality department if you have any more questions or concerns. [\(Link to Public Health Statement\)](#)



This fact sheet answers the most frequently asked health questions (FAQs) about hexachlorocyclohexane. For more information, call the ATSDR Information Center at 1-888-422-8737. This fact sheet is one in a series of summaries about hazardous substances and their health effects. It is important you understand this information because this substance may harm you. The effects of exposure to any hazardous substance depend on the dose, the duration, how you are exposed, personal traits and habits, and whether other chemicals are present.

HIGHLIGHTS: Exposure to hexachlorocyclohexane (HCH) happens mostly from eating contaminated food or by breathing contaminated air in the workplace. Exposure to high levels of HCH can cause blood disorders, dizziness, headaches, seizures, and changes in the levels of sex hormones. HCH has caused cancer in animals. α -, β -, γ -, and δ -HCH have been found in at least 146, 159, 189, and 126, respectively, of the 1,662 National Priority List sites identified by the Environmental Protection Agency (EPA).

What is hexachlorocyclohexane?

Hexachlorocyclohexane (HCH) is a manufactured chemical that exists in eight chemical forms called isomers. One of these forms, gamma-HCH (or γ -HCH, commonly called lindane) is produced and used as an insecticide on fruit, vegetables, and forest crops. It is a white solid that may evaporate into the air as a colorless vapor with a slightly musty odor. It is also available as a prescription (lotion, cream, or shampoo) to treat head and body lice, and scabies. Lindane has not been produced in the United States since 1976, but is imported for insecticide use.

Technical-grade HCH was used as an insecticide in the United States and typically contained 10-15% γ -HCH as well as the alpha (α), beta (β), delta (δ), and epsilon (ϵ) forms of HCH. Virtually all the insecticidal properties resided in γ -HCH. Technical-grade HCH has not been produced or used in the United States in over 20 years.

What happens to hexachlorocyclohexane when it enters the environment?

- The components of technical-grade HCH have been found in soil and surface waters near hazardous waste sites.
- In the air, the different forms of HCH can exist as a vapor or attached to small particles such as soil and dust. The particles may be removed from the air by rain or degraded by other compounds in the atmosphere.
- HCH can remain in the air for long periods of time and travel great distances.

- In soil, sediments, and water, HCH is broken down to less toxic substances by algae, fungi, and bacteria, but this process can take a long time.

- HCH can accumulate in the fatty tissue of fish.

How might I be exposed to hexachlorocyclohexane?

- Eating food or drinking water contaminated with HCH.
- Breathing air contaminated with HCH in or near factories where products using γ -HCH are made.
- Through the skin when applied as a lotion or shampoo to treat lice or scabies.
- Workers involved in the formulation or application of products containing γ -HCH may be exposed to higher concentrations.

How can hexachlorocyclohexane affect my health?

Some people who breathed contaminated workplace air during manufacturing of pesticides, including γ -HCH, had blood disorders, dizziness, headaches, and changes in the levels of sex hormones. Some people who swallowed large amounts had seizures and sometimes died.

Animals fed γ - and α -HCH have had convulsions, and animals fed β -HCH have become comatose. All isomers can produce liver and kidney effects. Reduced ability to fight infection was reported in animals fed γ -HCH, and injury to the ovaries and testes was reported in animals given γ -HCH or β -HCH.

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How likely is hexachlorocyclohexane to cause cancer?

Long-term oral administration of α -HCH, β -HCH, γ -HCH, or technical-grade HCH to laboratory rodents produced liver cancer. The Department of Health and Human Services (DHHS) has determined that HCH (all isomers) may reasonably be anticipated to cause cancer in humans. The International Agency for Research on Cancer (IARC) has classified HCH (all isomers) as possibly carcinogenic to humans. The EPA has determined that there is suggestive evidence that lindane (γ -HCH) is carcinogenic, but the evidence is not sufficient to assess its human carcinogenic potential. The EPA has additionally classified technical HCH and α -HCH as probable human carcinogens, β -HCH as a possible human carcinogen, and δ - and ϵ -HCH as not classifiable as to human carcinogenicity.

How can hexachlorocyclohexane affect children?

Health effects observed in adults should also be of potential concern in children. Children can experience convulsions from exposure to γ -HCH. Accidentally eating high amounts of γ -HCH can kill a child. We do not know whether children are more susceptible than adults to health effects from exposure to γ -HCH. However, a study performed on rabbits showed that young animals had higher death rates and greater sensitivity than adults when γ -HCH was applied to the skin. We do not know whether HCH causes birth defects in humans. Technical grade and γ -HCH do not cause serious birth defects in animals. HCH has been shown to cross the placenta in pregnant women. HCH has been detected in human breast milk, suggesting that it can be transferred to infants from women who nurse.

How can families reduce the risks of exposure to hexachlorocyclohexane?

- γ -HCH is a restricted use pesticide, it can be applied only by persons licensed and certified.
- If you work with HCH, take the necessary precautions to avoid bringing the dust home in your clothing or tools.
- If you use products containing γ -HCH, such as shampoos or lotions to treat lice, follow the directions for

use carefully. There are alternatives that do not involve γ -HCH.

- Make sure you keep products containing γ -HCH in tightly covered containers stored in the original container, and out of the reach of children.

Is there a medical test to determine whether I've been exposed to hexachlorocyclohexane?

HCH isomers can be measured in blood, urine, body fat, breast milk, and semen of exposed persons. Samples of blood, urine, and semen can be collected at the doctor's office and sent to a laboratory that has the special equipment needed to measure the levels of HCH. These tests cannot tell the levels you were exposed to or whether you will experience adverse health effects. The urine of potentially exposed persons can be tested for breakdown products of HCH formed in the body to determine whether a person was exposed to HCH or to a group of chemicals similar to HCH.

Has the federal government made recommendations to protect human health?

The EPA recommends that drinking water that children consume for up to 10 days should not contain more than 1.2 mg of γ -HCH per liter of water (1 mg/L) and that drinking water for children consumed for lifetime should not contain more than 0.0002 mg/L.

The Occupational Safety and Health Administration (OSHA) has set a limit for γ -HCH in the workplace air of 0.5 mg per cubic meter of air (0.5 mg/m³) during an 8-hour workday, 40-hour workweek.

References

Agency for Toxic Substances and Disease Registry (ATSDR). 2005. Toxicological Profile for Alpha-, Beta-, Gamma-, and Delta-Hexachlorocyclohexane. (Update). Atlanta, GA: U.S. Department of Public Health and Human Services, Public Health Service.

Where can I get more information? For more information, contact the Agency for Toxic Substances and Disease Registry, Division of Toxicology, 1600 Clifton Road NE, Mailstop F-32, Atlanta, GA 30333. Phone: 1-888-422-8737, FAX: 770-488-4178. ToxFAQs Internet address via WWW is <http://www.atsdr.cdc.gov/toxfaq.html>. ATSDR can tell you where to find occupational and environmental health clinics. Their specialists can recognize, evaluate, and treat illnesses resulting from exposure to hazardous substances. You can also contact your community or state health or environmental quality department if you have any more questions or concerns.



This fact sheet answers the most frequently asked health questions (FAQs) about 1,1,1-trichloroethane. For more information, call the ATSDR Information Center at 1-888-422-8737. This fact sheet is one in a series of summaries about hazardous substances and their health effects. It is important you understand this information because this substance may harm you. The effects of exposure to any hazardous substance depend on the dose, the duration, how you are exposed, personal traits and habits, and whether other chemicals are present.

HIGHLIGHTS: Exposure to 1,1,1-trichloroethane usually occurs by breathing contaminated air. It is found in building materials, cleaning products, paints, and metal degreasing agents. You are not likely to be exposed to large enough amounts to cause adverse health effects. Inhaling high levels of 1,1,1-trichloroethane can cause you to become dizzy and lightheaded. Exposure to much higher levels can cause unconsciousness and other effects. This substance has been found in at least 823 of the 1,662 National Priorities List sites identified by the Environmental Protection Agency (EPA).

What is 1,1,1-trichloroethane?

1,1,1-Trichloroethane is a synthetic chemical that does not occur naturally in the environment. It also is known as methylchloroform, methyltrichloromethane, trichloromethylmethane, and α -trichloromethane. Its registered trade names are chloroethene NU® and Aerothene TT®.

No 1,1,1-trichloroethane is supposed to be manufactured for domestic use in the United States after January 1, 2002 because it affects the ozone layer. 1,1,1-Trichloroethane had many industrial and household uses, including use as a solvent to dissolve other substances, such as glues and paints; to remove oil or grease from manufactured metal parts; and as an ingredient of household products such as spot cleaners, glues, and aerosol sprays.

What happens to 1,1,1-trichloroethane when it enters the environment?

- Most of the 1,1,1-trichloroethane released into the environment enters the air, where it lasts for about 6 years.
- Once in the air, it can travel to the ozone layer where sunlight can break it down into chemicals that may reduce the ozone layer.
- Contaminated water from landfills and hazardous waste sites can contaminate surrounding soil and nearby surface water or groundwater.
- From lakes and rivers, most of the 1,1,1-trichloroethane evaporates quickly into the air.

Water can carry 1,1,1-trichloroethane through the soil and into the groundwater where it can evaporate and pass through the soil as a gas, then be released to the air.

Organisms living in soil or water may also break down 1,1,1-trichloroethane.

It will not build up in plants or animals.

How might I be exposed to 1,1,1-trichloroethane?

Breathing 1,1,1-trichloroethane in contaminated outdoor and indoor air. Because 1,1,1-trichloroethane was used so frequently in home and office products, you are likely to be exposed to higher levels indoors than outdoors or near hazardous waste sites. However, since 2002, 1,1,1-trichloroethane is not expected to be commonly used, and therefore, the likelihood of being exposed to it is remote.

In the workplace, you could have been exposed to 1,1,1-trichloroethane while using some metal degreasing agents, paints, glues, and cleaning products.

Ingesting contaminated drinking water and food.

How can 1,1,1-trichloroethane affect my health?

If you breathe air containing high levels of 1,1,1-trichloroethane for a short time, you may become dizzy and lightheaded and possibly lose your coordination. These effects rapidly disappear after you stop breathing contaminated air. If you breathe in much higher levels, you may become unconscious, your blood pressure may decrease, and your heart may stop beating. Whether breathing low levels of 1,1,1-trichloroethane for a long

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time causes harmful effects is not known. Studies in animals show that breathing air that contains very high levels of 1,1,1-trichloroethane damages the breathing passages and causes mild effects in the liver, in addition to affecting the nervous system. There are no studies in humans that determine whether eating food or drinking water contaminated with 1,1,1-trichloroethane could harm health. Placing large amounts of 1,1,1-trichloroethane in the stomachs of animals has caused effects on the nervous system, mild liver damage, unconsciousness, and even death. If your skin contacts 1,1,1-trichloroethane, you might feel some irritation. Studies in animals suggest that repeated exposure of the skin might affect the liver and that very large amounts may cause death. These effects occurred only when evaporation was prevented.

How likely is 1,1,1-trichloroethane to cause cancer?

Available information does not indicate that 1,1,1-trichloroethane causes cancer. The International Agency for Research on Cancer (IARC) and the EPA have determined that 1,1,1-trichloroethane is not classifiable as to its carcinogenicity in humans.

How can 1,1,1-trichloroethane affect children?

Children exposed to large amounts of 1,1,1-trichloroethane probably would be affected in the same manner as adults. In animals, it has been shown that 1,1,1-trichloroethane can pass from the mother's blood into a fetus. When pregnant mice were exposed to high levels of 1,1,1-trichloroethane in air, their babies developed more slowly than normal and had some behavioral problems. However, whether similar effects occur in humans has not been demonstrated.

How can families reduce the risk of exposure to 1,1,1-trichloroethane?

Children can be exposed to 1,1,1-trichloroethane in household products, such as adhesives and cleaners. Parents should store household chemicals out of reach of young children to prevent accidental poisonings or skin irritation. Always store household chemicals in their original labeled containers. Never store household chemicals in containers that children would find attractive to eat or drink from, such as old soda bottles. Keep your Poison Control Center's number near the phone.

Sometimes older children sniff household chemicals in an attempt to get high. Your children may be exposed to 1,1,1-trichloroethane by inhaling products containing it. Talk with your children about the dangers of sniffing chemicals.

Is there a medical test to show whether I've been exposed to 1,1,1-trichloroethane?

Samples of your breath, blood, and urine can be tested to determine if you have recently been exposed to 1,1,1-trichloroethane. In some cases, these tests can estimate how much 1,1,1-trichloroethane has entered your body. To be of any value, samples of your breath or blood have to be taken within hours after exposure, and samples of urine have to be taken within 2 days after exposure. However, these tests will not tell you whether your health will be affected by exposure to 1,1,1-trichloroethane. The exposure tests are not routinely available in hospitals and clinics because they require special analytical equipment.

Has the federal government made recommendations to protect human health?

EPA regulates the levels of 1,1,1-trichloroethane that are allowable in drinking water. The highest level of 1,1,1-trichloroethane allowed in drinking water is 0.2 parts 1,1,1-trichloroethane per 1 million parts of water (0.2 ppm).

The Occupational Safety and Health Administration (OSHA) has set a limit of 350 parts 1,1,1-trichloroethane per 1 million parts of air (350 ppm) in the workplace.

Reference

Agency for Toxic Substances and Disease Registry (ATSDR). 2006. Toxicological Profile for 1,1,1-Trichloroethane (Update). Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service.

Where can I get more information? For more information, contact the Agency for Toxic Substances and Disease Registry, Division of Toxicology and Environmental Medicine, 1600 Clifton Road NE, Mailstop F-32, Atlanta, GA 30333. Phone: 1-888-422-8737, FAX: 770-488-4178. ToxFAQs Internet address via WWW is <http://www.atsdr.cdc.gov/toxfaq.html>. ATSDR can tell you where to find occupational and environmental health clinics. Their specialists can recognize, evaluate, and treat illnesses resulting from exposure to hazardous substances. You can also contact your community or state health or environmental quality department if you have any more questions or concerns.



This fact sheet answers the most frequently asked health questions (FAQs) about DDT, DDE, and DDD. For more information, call the ATSDR Information Center at 1-888-422-8737. This fact sheet is one in a series of summaries about hazardous substances and their health effects. It is important you understand this information because this substance may harm you. The effects of exposure to any hazardous substance depend on the dose, the duration, how you are exposed, personal traits and habits, and whether other chemicals are present.

HIGHLIGHTS: Exposure to DDT, DDE, and DDD occurs mostly from eating foods containing small amounts of these compounds, particularly meat, fish and poultry. High levels of DDT can affect the nervous system causing excitability, tremors and seizures. In women, DDE can cause a reduction in the duration of lactation and an increased chance of having a premature baby. DDT, DDE, and DDD have been found in at least 441 of the 1,613 National Priorities List sites identified by the Environmental Protection Agency (EPA).

What are DDT, DDE, and DDD?

DDT (dichlorodiphenyltrichloroethane) is a pesticide once widely used to control insects in agriculture and insects that carry diseases such as malaria. DDT is a white, crystalline solid with no odor or taste. Its use in the U.S. was banned in 1972 because of damage to wildlife, but is still used in some countries.

DDE (dichlorodiphenyldichloroethylene) and DDD (dichlorodiphenyldichloroethane) are chemicals similar to DDT that contaminate commercial DDT preparations. DDE has no commercial use. DDD was also used to kill pests, but its use has also been banned. One form of DDD has been used medically to treat cancer of the adrenal gland.

What happens to DDT, DDE, and DDD when they enter the environment?

- ❑ DDT entered the environment when it was used as a pesticide; it still enters the environment due to current use in other countries.
- ❑ DDE enters the environment as contaminant or breakdown product of DDT; DDD also enters the environment as a breakdown product of DDT.
- ❑ DDT, DDE, and DDD in air are rapidly broken down by sunlight. Half of what's in air breaks down within 2 days.
- ❑ They stick strongly to soil; most DDT in soil is broken down slowly to DDE and DDD by microorganisms; half the DDT in soil will break down in 2-15 years, depending on the type of soil.

- ❑ Only a small amount will go through the soil into groundwater; they do not dissolve easily in water.
- ❑ DDT, and especially DDE, build up in plants and in fatty tissues of fish, birds, and other animals.

How might I be exposed to DDT, DDE, and DDD?

- ❑ Eating contaminated foods, such as root and leafy vegetables, fatty meat, fish, and poultry, but levels are very low.
- ❑ Eating contaminated imported foods from countries that still allow the use of DDT to control pests.
- ❑ Breathing contaminated air or drinking contaminated water near waste sites and landfills that may contain higher levels of these chemicals.
- ❑ Infants fed on breast milk from mothers who have been exposed.
- ❑ Breathing or swallowing soil particles near waste sites or landfills that contain these chemicals.

How can DDT, DDE, and DDD affect my health?

DDT affects the nervous system. People who accidentally swallowed large amounts of DDT became excitable and had tremors and seizures. These effects went away after the exposure stopped. No effects were seen in people who took small daily doses of DDT by capsule for 18 months. A study in humans showed that women who had high amounts of a form of DDE in their breast milk were unable to

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breast feed their babies for as long as women who had little DDE in the breast milk. Another study in humans showed that women who had high amounts of DDE in breast milk had an increased chance of having premature babies. In animals, short-term exposure to large amounts of DDT in food affected the nervous system, while long-term exposure to smaller amounts affected the liver. Also in animals, short-term oral exposure to small amounts of DDT or its breakdown products may also have harmful effects on reproduction.

How likely are DDT, DDE, and DDD to cause cancer?

Studies in DDT-exposed workers did not show increases in cancer. Studies in animals given DDT with the food have shown that DDT can cause liver cancer. The Department of Health and Human Services (DHHS) determined that DDT may reasonable be anticipated to be a human carcinogen. The International Agency for Research on Cancer (IARC) determined that DDT may possibly cause cancer in humans. The EPA determined that DDT, DDE, and DDD are probable human carcinogens.

How can DDT, DDE, and DDD affect children?

There are no studies on the health effects of children exposed to DDT, DDE, or DDD. We can assume that children exposed to large amounts of DDT will have health effects similar to the effects seen in adults. However, we do not know whether children differ from adults in their susceptibility to these substances. There is no evidence that DDT, DDE, or DDD cause birth defects in people. A study showed that teenage boys whose mothers had higher DDE amounts in the blood when they were pregnant were taller than those whose mothers had lower DDE levels. However, a different study found the opposite in preteen girls. The reason for the discrepancy between these studies is unknown. Studies in rats have shown that DDT and DDE can mimic the action of natural hormones and in this way affect the development of the reproductive and nervous systems. Puberty was delayed in male rats given high amounts of DDE as juveniles. This could possibly happen in humans.

A study in mice showed that exposure to DDT during the first weeks of life may cause neurobehavioral problems later in life.

How can families reduce the risk of exposure to DDT, DDE, and DDE?

- Most families will be exposed to DDT by eating food or drinking liquids contaminated with small amounts of DDT.
- Cooking will reduce the amount of DDT in fish.
- Washing fruit and vegetables will remove most DDT from their surface.
- Follow health advisories that tell you about consumption of fish and wildlife caught in contaminated areas.

Is there a medical test to show whether I've been exposed to DDT, DDE, and DDD?

Laboratory tests can detect DDT, DDE, and DDD in fat, blood, urine, semen, and breast milk. These tests may show low, moderate, or excessive exposure to these compounds, but cannot tell the exact amount you were exposed to, or whether you will experience adverse effects. These tests are not routinely available at the doctor's office because they require special equipment.

Has the federal government made recommendations to protect human health?

The Occupational Safety and Health Administration (OSHA) sets a limit of 1 milligram of DDT per cubic meter of air (1 mg/m³) in the workplace for an 8-hour shift, 40-hour workweek. The Food and Drug Administration (FDA) has set limits for DDT, DDE, and DDD in foodstuff at or above which the agency will take legal action to remove the products from the market.

References

Agency for Toxic Substances and Disease Registry (ATSDR). 2002. Toxicological Profile for DDT/DDE/DDD (Update). Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service.

Where can I get more information? For more information, contact the Agency for Toxic Substances and Disease Registry, Division of Toxicology, 1600 Clifton Road NE, Mailstop F-32, Atlanta, GA 30333. Phone: 1-888-422-8737, FAX: 770-488-4178. ToxFAQs Internet address via WWW is <http://www.atsdr.cdc.gov/toxfaq.html>. ATSDR can tell you where to find occupational and environmental health clinics. Their specialists can recognize, evaluate, and treat illnesses resulting from exposure to hazardous substances. You can also contact your community or state health or environmental quality department if you have any more questions or concerns.



This fact sheet answers the most frequently asked health questions (FAQs) about chloroform. For more information, call the ATSDR Information Center at 1-888-422-8737. This fact sheet is one in a series of summaries about hazardous substances and their health effects. It's important you understand this information because this substance may harm you. The effects of exposure to any hazardous substance depend on the dose, the duration, how you are exposed, personal traits and habits, and whether other chemicals are present.

HIGHLIGHTS: Exposure to chloroform can occur when breathing contaminated air or when drinking or touching the substance or water containing it. Breathing chloroform can cause dizziness, fatigue, and headaches. Breathing chloroform or ingesting chloroform over long periods of time may damage your liver and kidneys. It can cause sores if large amounts touch your skin. This substance has been found in at least 717 of the 1,430 National Priorities List sites identified by the Environmental Protection Agency (EPA).

What is chloroform?

(Pronounced klôr'ə-fôrm')

Chloroform is a colorless liquid with a pleasant, nonirritating odor and a slightly sweet taste. It will burn only when it reaches very high temperatures.

In the past, chloroform was used as an inhaled anesthetic during surgery, but it isn't used that way today. Today, chloroform is used to make other chemicals and can also be formed in small amounts when chlorine is added to water.

Other names for chloroform are trichloromethane and methyl trichloride.

What happens to chloroform when it enters the environment?

- Chloroform evaporates easily into the air.
- Most of the chloroform in air breaks down eventually, but it is a slow process.
- The breakdown products in air include phosgene and hydrogen chloride, which are both toxic.
- It doesn't stick to soil very well and can travel through soil to groundwater.

- Chloroform dissolves easily in water and some of it may break down to other chemicals.
- Chloroform lasts a long time in groundwater.
- Chloroform doesn't appear to build up in great amounts in plants and animals.

How might I be exposed to chloroform?

- Drinking water or beverages made using water containing chloroform.
- Breathing indoor or outdoor air containing it, especially in the workplace.
- Eating food that contains it.
- Skin contact with chloroform or water that contains it, such as in swimming pools.

How can chloroform affect my health?

Breathing about 900 parts of chloroform per million parts air (900 ppm) for a short time can cause dizziness, fatigue, and headache. Breathing air, eating food, or drinking water containing high levels of chloroform for long periods of time may damage your liver and kidneys. Large amounts of chloroform can cause sores when chloroform touches your skin.

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It isn't known whether chloroform causes reproductive effects or birth defects in people.

Animal studies have shown that miscarriages occurred in rats and mice that breathed air containing 30 to 300 ppm chloroform during pregnancy and also in rats that ate chloroform during pregnancy. Offspring of rats and mice that breathed chloroform during pregnancy had birth defects. Abnormal sperm were found in mice that breathed air containing 400 ppm chloroform for a few days.

How likely is chloroform to cause cancer?

The Department of Health and Human Services (DHHS) has determined that chloroform may reasonably be anticipated to be a carcinogen.

Rats and mice that ate food or drank water with chloroform developed cancer of the liver and kidneys.

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

Although the amounts of chloroform in the air that you exhale and in blood, urine, and body tissues can be measured, there is no reliable test to determine how much chloroform you have been exposed to or whether you will experience any harmful effects.

The measurement of chloroform in body fluids and tissues may help to determine if you have come into contact with large amounts of chloroform, but these tests are useful for only a short time after you are exposed. Chloroform in your body might also indicate that you have come into contact with other chemicals.

Has the federal government made recommendations to protect human health?

The EPA drinking water limit for total trihalomethanes, a class of chemicals that includes chloroform, is 100 micrograms per liter of water (100 µg/L).

The EPA requires that spills or accidental releases of 10 pounds or more of chloroform into the environment be reported to the EPA.

The Occupational Safety and Health Administration (OSHA) has set the maximum allowable concentration of chloroform in workroom air during an 8-hour workday in a 40-hour workweek at 50 ppm.

Glossary

Carcinogenicity: A substance with the ability to cause cancer.

CAS: Chemical Abstracts Service.

Ingesting: Taking food or drink into your body.

Microgram (µg): One millionth of a gram.

Miscarriage: Pregnancy loss.

ppm: Parts per million.

References

This ToxFAQs information is taken from the 1997 Toxicological Profile for Chloroform (update) produced by the Agency for Toxic Substances and Disease Registry, Public Health Service, U.S. Department of Health and Human Services, Public Health Service in Atlanta, GA.

Where can I get more information? For more information, contact the Agency for Toxic Substances and Disease Registry, Division of Toxicology, 1600 Clifton Road NE, Mailstop F-32, Atlanta, GA 30333. Phone: 1-888-422-8737, FAX: 770-488-4178. ToxFAQs Internet address via WWW is <http://www.atsdr.cdc.gov/toxfaq.html> ATSDR can tell you where to find occupational and environmental health clinics. Their specialists can recognize, evaluate, and treat illnesses resulting from exposure to hazardous substances. You can also contact your community or state health or environmental quality department if you have any more questions or concerns.



Appendix E

ATSDR Public Health Hazard Levels

ATSDR categories for exposure pathways at hazardous waste sites are as follows:

Urgent Public Health Hazard: This category applies to exposure pathways and sites that have certain physical features or evidence of short-term (less than 1 year), site-related chemical exposure that could result in adverse health effects and require quick intervention to stop people from being exposed

Public Health Hazard: The category applies to exposure pathways and sites that have certain physical features or evidence of chronic (long-term), site-related chemical exposure that could result in adverse health effects.

Indeterminate Public Health Hazard: The category applies to exposure pathways and sites where important information is lacking about chemical exposures, and a health determination cannot be made.

No Apparent Public Health Hazard: The category applies to pathways and sites where exposure to site-related chemicals may have occurred in the past or is still occurring, however, the exposure is not at levels expected to cause adverse health effects.

No Public Health Hazard: The category applies to pathways and sites where there is evidence of an absence of exposure to site-related chemicals.

Appendix F
ATSDR Glossary

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 functions 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.

Epidemiologic surveillance

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 are 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 ground water); 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.

Ground water

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

Half-life ($t_{1/2}$)

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 remain.

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 that 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 ground water.

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.

Resource Conservation and Recovery Act (1976, 1984) (RCRA)

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]**SARA** [see Superfund Amendments and Reauthorization Act]**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 ground water].

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].

USEPA

United States Environmental Protection Agency.

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.