ATSDR
AGENCY FOR TOXIC SUBSTANCES AND DISEASE REGISTRY

Public Health Assessment for

Evaluation of Drinking Water Well Exposures via Confirmed Off-Site Contamination

FORT DETRICK AREA B GROUNDWATER
FREDERICK, MARYLAND
EPA FACILITY ID: MDD985397249
DECEMBER 9. 2009

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

THE ATSDR PUBLIC HEALTH ASSESSMENT: A NOTE OF EXPLANATION

This Public Health Assessment was prepared by ATSDR pursuant to the Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA or Superfund) section 104 (i)(6) (42 U.S.C. 9604 (i)(6)), and in accordance with our implementing regulations (42 C.F.R. Part 90). In preparing this document, ATSDR has collected relevant health data, environmental data, and community health concerns from the Environmental Protection Agency (EPA), state and local health and environmental agencies, the community, and potentially responsible parties, where appropriate.

In addition, this document has previously been provided to EPA and the affected states in an initial release, as required by CERCLA section 104 (i)(6)(H) for their information and review. The revised document was released for a 30-day public comment period. Subsequent to the public comment period, ATSDR addressed all public comments and revised or appended the document as appropriate. The public health assessment has now been reissued. This concludes 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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Prepared by:

U.S. Department of Health and Human Services
Agency for Toxic Substances and Disease Registry
Division of Health Assessment and Consultation

Foreword

The Agency for Toxic Substances and Disease Registry (ATSDR) was established by Congress in 1980 under the Comprehensive Environmental Response, Compensation, and Liability Act, also known as the Superfund law. This law set up a fund to identify and clean up our country's hazardous waste sites. The Environmental Protection Agency (EPA) and the individual states regulate the investigation and cleanup of the sites.

Since 1986, ATSDR has been required by law to conduct public health assessment activities at each of the sites on the EPA National Priorities List. The aim of these evaluations is to find out if people are being exposed to hazardous substances and, if so, whether that exposure is harmful and should be stopped or reduced. If appropriate, ATSDR also conducts public health assessments when petitioned by concerned individuals. Public health assessments are carried out by environmental and health scientists from ATSDR and from the state, tribal, and territorial programs with which ATSDR has cooperative agreements. The public health assessment program allows the scientists flexibility in the format or structure of their response to the public health issues at hazardous waste sites. For example, a public health assessment could be one document or it could be a compilation of several health consultations—the structure may vary from site to site. Whatever the form of the public health assessment, the process is not considered complete until the public health issues at the site are addressed.

Exposure

As the first step in the evaluation, ATSDR scientists review environmental data to see how much contamination is at a site, where it is, and how people might come into contact with it. Generally, rather than collecting its own environmental sampling data, ATSDR reviews information provided by EPA, other government agencies, businesses, and the public. When there is not enough environmental information available, the report will indicate what further sampling data are needed.

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

Health Effects

If there are potential or completed exposure pathways where people have or could come into contact with hazardous substances, ATSDR scientists then evaluate whether these contacts may result in harmful effects. ATSDR recognizes that children, because of their play activities and their growing bodies, may be more vulnerable to these effects. As a policy, unless data are available to suggest otherwise, ATSDR considers children likely to be more sensitive and vulnerable to hazardous substances than adults. Thus, the health impact to the children is

considered first when evaluating the health threat to a community. The health impacts to other high-risk groups within the community (such as the elderly, chronically ill, and people engaging in high-risk practices) also receive special attention during the evaluation.

ATSDR uses existing scientific information, which can include the results of medical, toxicologic, and epidemiologic studies and the data collected in disease registries, to determine the health effects that may result from exposures. The science of environmental health is still developing, and sometimes scientific information on the health effects of certain substances is not available. ATSDR identifies those types of information gaps and documents public health actions needed in public health assessment documents.

Conclusions

If appropriate, this report presents conclusions about the public health threat, if any, posed by a site. Any health threats that have been determined for high-risk groups (such as children, the elderly, chronically ill people, and people engaging in high-risk practices) are summarized in the Conclusions section of the report. Recommendations are presented on how to stop or reduce exposure. The public health action plan describes how those recommendations will be implemented.

ATSDR is primarily an advisory agency, so its reports usually identify what actions are appropriate to be undertaken by EPA, other responsible parties, or the research or education divisions of ATSDR. However, if there is an urgent health threat, ATSDR can issue a public health advisory warning people of the danger. ATSDR can also authorize health education or pilot studies of health effects, full-scale epidemiology studies, exposure registries, surveillance studies or research on specific hazardous substances.

Community

ATSDR also needs to learn what people in the area know about the site and what concerns they may have about its impact on their health. Consequently, throughout the evaluation process, ATSDR actively gathers information and comments from the people who live or work near a site, including residents of the area, civic leaders, health professionals, and community groups. To ensure that the report responds to the community's health concerns, an early version is also distributed to the public for their comments. Comments received from the public are addressed in the final version of the report.

Comments

If, after reading this report, you have questions or comments, we encourage you to send them to us. Letters should be addressed as follows:

Manager
ATSDR Records Center (MS F-09)
4770 Buford Highway, NE
Building 106, Room 2108
Atlanta, GA 30341

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Appendices

List of Abbreviations

ATSDR	Agency for Toxic Substances and Disease Registry
CREG	cancer risk evaluation guide
CV	comparison value
EMEG	environmental media evaluation guide
EPA	U.S. Environmental Protection Agency
MCL	maximum contaminant level
MRL	minimal risk level
PCE	tetrachloroethylene or tetrachloroethene
PHA	public health assessment
PHAP	Public Health Action Plan
ppb	parts per billion
ppm	parts per million
RfD	reference dose
SVOC	semi-volatile organic compound
TCE	trichloroethylene or trichloroethene
VOC	volatile organic compound

Summary

Fort Detrick is an active U.S. Army installation operated under the Army Medical Command (MEDCOM). Fort Detrick includes six non-contiguous land parcels (designated as Areas A, B, Area C Water Treatment Plant, Area C Waste Water Treatment Plant, Forest Glen Annex, and Glen Haven Annex). Areas A, B, and C, are located within the city limits of Frederick, Maryland. Area B is approximately 399 acres and includes Fort Detrick's active municipal landfill, an animal farm (for research purposes), a former shooting range, waste disposal areas, an explosives storage facility, and open fields used for historic research activities. On April 9, 2009, the Environmental Protection Agency (EPA) added the Fort Detrick Area B Groundwater to the National Priorities List (NPL) based on tetrachloroethene (PCE) and trichloroethene (TCE) detections in off-site drinking water wells.

ATSDR is required to conduct a public health assessment (PHA) for every site proposed for listing on the NPL. Using the extensive data available from Area B site assessments and measured concentrations of contaminants in off-site drinking water wells, this PHA will determine whether people exposed to chemicals from Area B groundwater are likely to get sick from those exposures.

Doses from ingestion, inhalation, and dermal contact with TCE and PCE at the offsite well locations are estimated assuming that exposure occurred in a residential setting, that is that exposure was continuous (24 hours per day, 365 days per year) and all water consumed was from affected wells. It was also assumed that exposures occurred over a 40 year timeframe (circa 1955 to 1992). Doses from other residences are not estimated because PCE and TCE were not detected or were intermittently detected below health comparison values. The off-site drinking water well pathway is considered to be complete only for *past exposures* (although non-ingestion incidental exposures may still be occurring). All known affected off-site locations with contaminated wells have been provided with an alternate water supply beginning with the initial locations sampled in 1992. Due to source remediation, groundwater monitoring, and provision of municipal water supplies, no future exposures from off-site residential drinking water wells are expected.

The maximum measured concentration of PCE (1992) was 17 ppb (and trace or non-detectable concentrations in all other residential wells). The drinking water concentration of 17 ppb result in estimated combined (ingestion plus inhalation plus dermal contact) PCE doses of 0.00097 mg/kg/day for adults and 0.0021 mg/kg/day for children. *Based on the available measured PCE concentrations and estimated doses, adverse health effects from past exposure to PCE via contaminated drinking from the contaminated water wells around the Area B site are unlikely to produce any harmful health effects, including cancer.*

Measurements of TCE (1992) were 19 ppb in Well DWSRD-1; 18 ppb in Well DWSRD-A, and trace or non-detectable concentrations in all other residential wells. The drinking water concentrations result in estimated combined (ingestion plus inhalation plus dermal contact) TCE doses of 0.0011 mg/kg/day for adults and 0.0024 mg/kg/day for children. Estimated doses at the DWSRD-A location would be similar, but slightly lower. *Based on estimated doses to the maximum measured TCE concentration, adverse health effects from past exposure to TCE via*

exposure to contaminated water around the Area B site are unlikely to produce any harmful health effects, including cancer.

Exposures to site-related contaminants have occurred or may be occurring, but at levels unlikely to create any harmful health effects. Based on the above conclusions, ATSDR recommends continued monitoring of the groundwater contaminants. To ensure community understanding and address future public health issues related to the Area B groundwater site, ATSDR has presented the results of this PHA to the Fort Detrick community (through the Fort Detrick Restoration Advisory Board). ATSDR has also addressed the resulting public comments as appropriate, and will evaluate additional monitoring data and information and revise the public health conclusions of this health assessment as necessary.

Purpose and Health Issues

Scope of the Public Health Assessment

On April 9, 2009 the Environmental Protection Agency (EPA) added the Fort Detrick Area B Groundwater to the National Priorities List (NPL). The basis for this listing, as detailed in the *Hazard Ranking System Documentation Record* (HRS; EPA, 2008), is that

> *TCE* [trichloroethene] *and PCE* [tetrachloroethene] *have been detected in drinking water wells. Most of the drinking water wells have been closed and residents are provided with public water or bottled water…*

The HRS Record provides a detailed summary of the groundwater contaminant sources, past monitoring activities and results, and the potential for community exposures to Area B groundwater contaminants. Additional reports by the State of Maryland and the U.S. Army (and its contractors) provide extensive documentation about Area B groundwater contaminants and contaminant migration and residential well contaminant concentrations. These reports will be cited as appropriate in this public health assessment (PHA).

Although existing site reports and documents include detailed results of the many site assessment studies that have been conducted for the evaluation of Area B groundwater contamination, none of the prior reports have determined whether PCE and TCE in off-site drinking water wells represents a health hazard for people drinking that water. As stated in the foreword to this PHA, ATSDR is required to conduct a public health assessment for every site proposed for listing on the NPL. Using the extensive data available from Area B site assessments and measured concentrations of contaminants in off-site drinking water wells, this PHA will determine whether people exposed to Area B groundwater are likely to get sick from those exposures.

Following sections will provide a very brief summary of the site history and background as it relates to understanding potential exposures, a statement of the *contaminants of concern* (based on a review of contaminant concentrations and distributions in on-site and off-site wells and discharges to surface waters), and an evaluation of the public health implications of potential exposures to the contaminants of public health concern.

Community Health Concerns

In order to determine if people living around Fort Detrick have any specific health concerns related to Area B groundwater, ATSDR staff attended meetings of the Fort Detrick Restoration Advisory Board (RAB; October 29, 2008 and May 7, 2009). Meeting attendees included community representatives to the RAB, as well as local residents and representatives of local, state, and federal agencies. The results of this PHA were presented to RAB members and community attendees at the May 7 meeting.

In response to ATSDR inquiries, attendees did not identify any specific health concerns related to past drinking water exposures. In the past, residents have expressed concerns about the incidence of cancers in the surrounding communities and the safety of local drinking water in

areas adjacent to Fort Detrick. ATSDR received one set of comments on the Public Comment version of this PHA. A summary of these comments and ATSDR responses are included as Appendix C.

Background

Site Description and Operational History

Fort Detrick is an active U.S. Army installation operated under the Army Medical Command (MEDCOM). Fort Detrick includes six non-contiguous land parcels (designated as Areas A, B, Area C Water Treatment Plant, Area C Waste Water Treatment Plant, Forest Glen Annex, and Glen Haven Annex). Areas A, B, and C, are located within the city limits of Frederick, Maryland (Figure 1). Area B is approximately 399 acres and includes the Fort Detrick active municipal landfill, an animal farm (for research purposes), a former shooting range, waste disposal areas, an explosives storage area, and open fields used for historic research activities (Shaw Environmental, 2008).

The Fort Detrick Restoration Advisory Board (RAB) web site includes a brief summary of the history of Fort Detrick (http://www.detrick.mil/rab/page6.cfm). Area B was initially established as a proving ground for the Army's biological weapons program (circa 1946; test grid area). Area B was also used for buried disposal of a number of contaminants including biological materials, test animal carcasses, radiological tracer materials, phosgene cylinders, and drums containing organic solvents (TCE, PCE, etc.; http://www.epa.gov/reg3hscd/npl/MD985397249.htm). The HRS Documentation Record (EPA, 2008) and the Area B remedial investigation (USACE, 1998) both provide comprehensive evaluations of the uses, history, and current disposition of the waste sources within Area B.

All of the above cited documents indicate that disposal of drums containing PCE, TCE, and other organic solvents at waste area B-11 was the source of off-site groundwater contamination at Area B. The B-11 waste pits received waste materials between 1955 and 1970 (EPA, 2008). The contaminant source materials in Area B-11 were removed in 2004 and capping of the area will be completed in 2009 (EPA, 2008). The Area B groundwater contamination is being addressed by the ongoing remedial investigation and feasibility study to select an appropriate remedy (pers. comm.; e-mail message from Joseph Gortva to Mark Evans, 4/28/2009).

Area B is on a terrain of gently sloping hills about one mile east of Catoctin Mountain. Within Area B, the topography slopes to the east and southeast from an elevation of about 400 feet on the northwest portion of the site, to a lower elevation of about 300 feet along Carroll Creek on the southeast side of Area B (Shaw Environmental, 2008). The geology of Area B consists of a thin layer of soil or residuum (7 to 35 feet thick) underlain by fractured limestone and dolomite (EPA, 2008). Dissolution of the limestone has resulted in subsurface cavities, enlarged fractures, sinkholes, and an irregular surface topography, which are characteristic of karst terrains (Shaw Environmental, 2008).

The significance of these karst features for site hydrogeology and contaminant migration are shown in Figure 2. The proposed conceptual site model in Figure 2 (Shaw Environmental, 2008) illustrates the flow path of groundwater contaminants from the B-11 pit source to down-gradient wells and discharge of the contaminant plume in springs and Carroll Creek. The following section of this PHA will discuss the specific contaminant concentrations historically and currently occurring along the flow path and at the points where people may have been exposed to those contaminants.

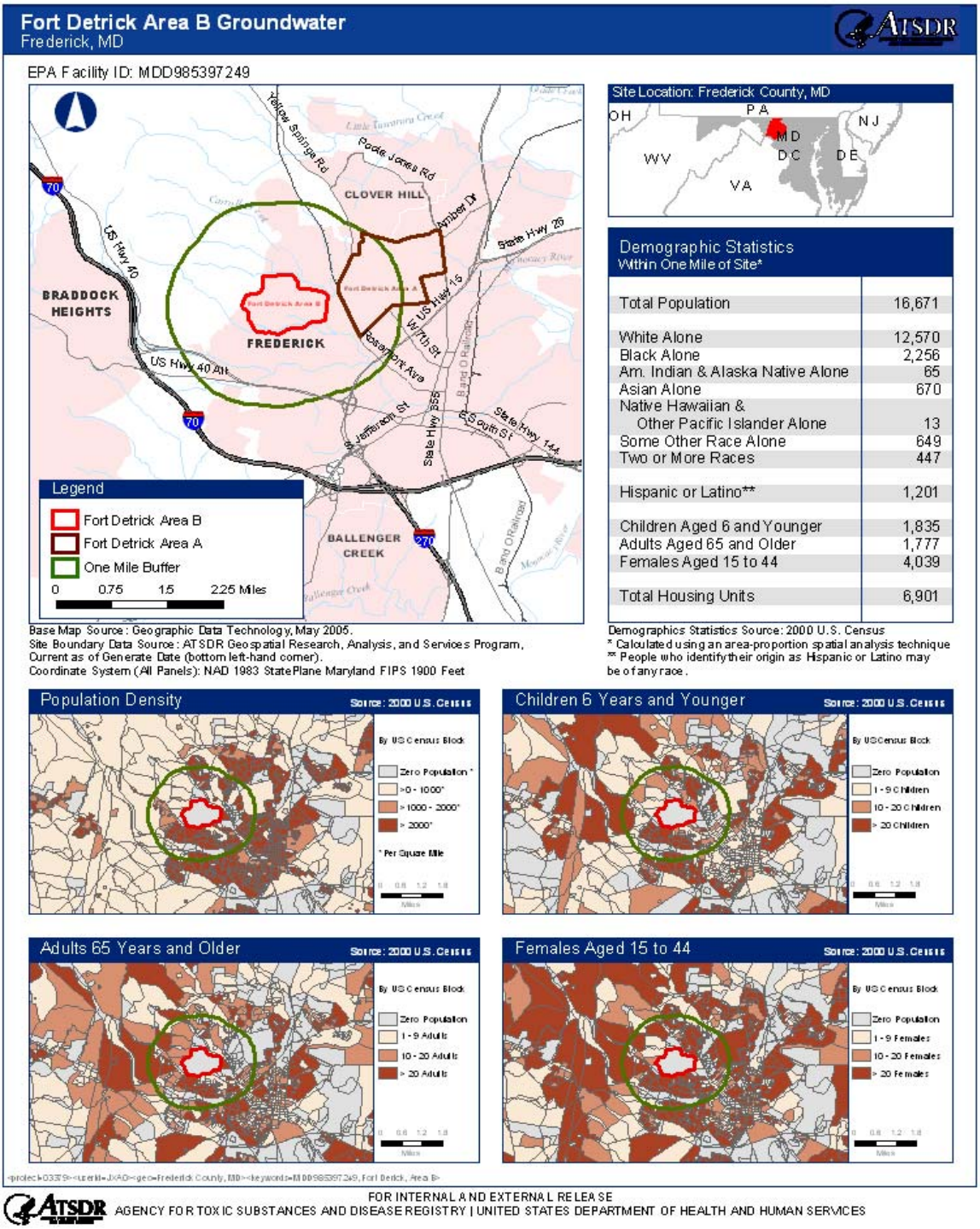

Figure 1. Location and demographic map of Fort Detrick Area B.

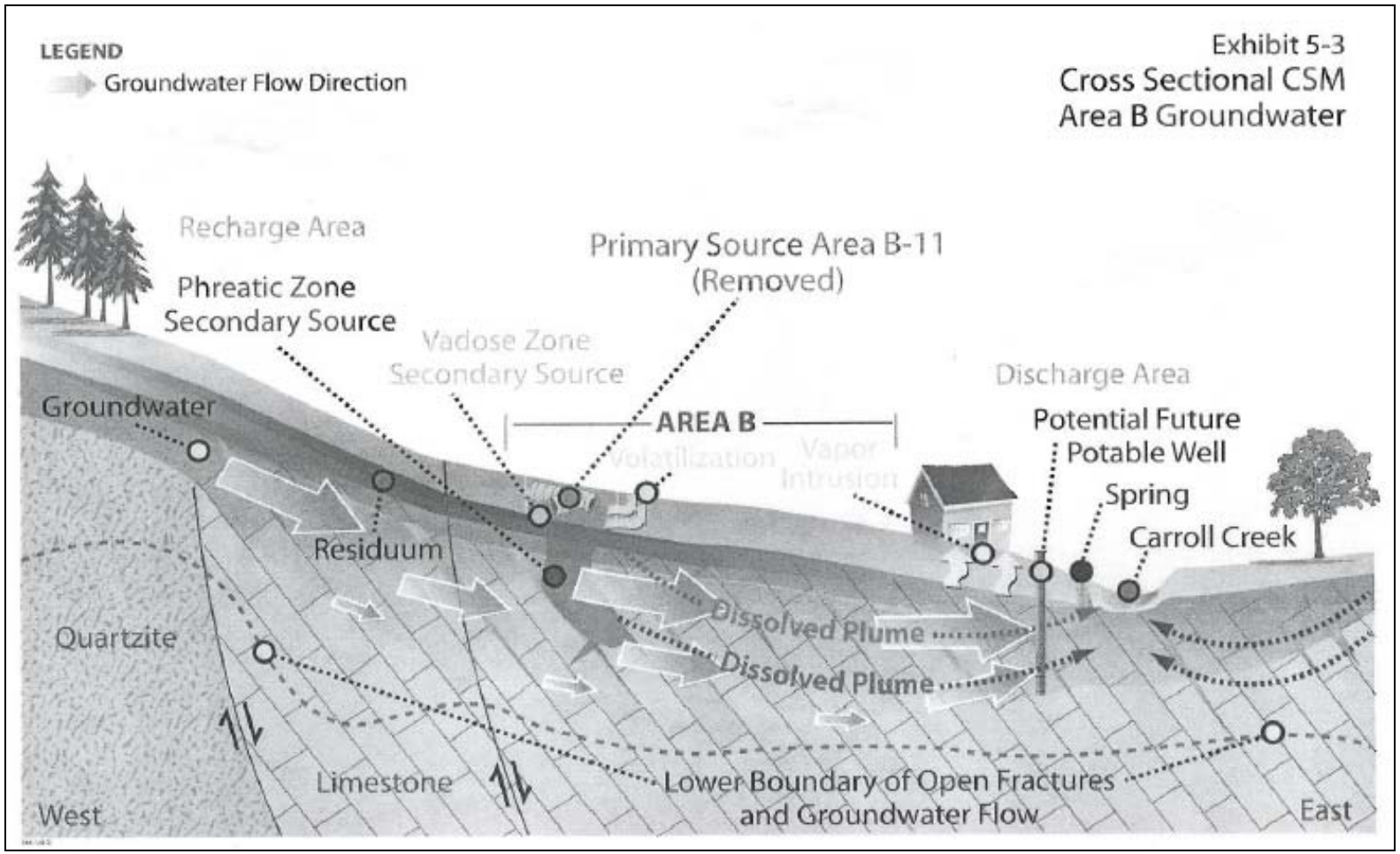

Figure 2. Proposed conceptual site model of Area B (from Shaw, Environmental, 2008). The geological and hydrogeological data underlying this conceptual model is presented and discussed in the Shaw, Environmental (2008) report.

Site Contaminants and Pathways of Exposure

Site Contaminants

There has been extensive and ongoing monitoring of groundwater, off-site drinking water wells, and surface water related to the Area B site as summarized in the "Site B Remedial Investigation" (USACE, 1998), the "Conceptual Site Model, Area B Groundwater (Shaw Environmental, 2008), and the HRS Documentation Record (EPA, 2008). Table 1 lists 17 groundwater contaminants that have been consistently detected in on-site monitoring wells (USACE, 1998; EPA, 2008). Appendix B provides a list of the analytes tested in off-site drinking water wells and also a list of tentatively identified compounds detected in on-site monitoring wells.

Nine of those 17 on-site groundwater contaminants have been measured at concentrations greater than their respective comparison values (the derivation and significance of the comparison values are described in the following section) and are considered to be "*contaminants of concern*". Table 2 lists the maximum measured concentrations of the nine contaminants of concern in off-site wells where exposure could have occurred (exposure is unlikely to occur at on-site monitor wells). Of the nine on-site contaminants, only PCE and TCE have been detected at locations of potential exposure at concentrations above their respective comparison values (Table 2). Consequently, only PCE and TCE will be evaluated for potential exposures.

Only three off-site water wells have had repeatable detections of PCE or TCE in regular monitoring beginning in 1995 (Shaw Environmental, 2008). Of the multiple measurements in each well, only one measurement each of TCE and PCE (in well DWSRD-26; Shaw Environmental, 2008) exceeded the drinking water standard (MCL) of 5 ppb. DWSRD-26 has been sampled for PCE 24 times with 15 non-detections, eight detections of 0.5 ppb or less, and one measurement of 9.3 ppb PCE in December 2001. Similarly, TCE has been detected in this well in 19 of 24 analyses at concentrations of 2.5 ppb or less and one measurement of 16 ppb (in December 2001). The DWSRD-26 residence uses municipal water so the DWSRD-26 well is not regularly used as a drinking water source.[1]

Prior to the initiation of regular well monitoring conducted by the U.S. Army (and its contractors), the MDE and the Frederick County Health Department sampled the drinking water of seven residences adjacent to Area B.[2] Two of these residences had drinking water supplies (wells) with PCE and TCE concentrations that exceeded the respective drinking water standards (maxima are listed in Table 2). Alternate water supplies were provided to all known affected locations in 1992 (USACE, 1998).

[1] Note that one other well had a single detection above the MCL, however it is located up-gradient of the Area B site and the result was not replicated in subsequent samples and probably represents sample contamination (Shaw Environmental, 2008).

[2] The 1992 sampling of off-site residential locations was conducted by the MDE and the Frederick County Health Department. The data are described and referenced in the EPA HRS documentation (EPA, 2008) from a three page facsimile from the Directorate of Safety, Health, and Environment, Fort Detrick to Steve Hirsch, Remedial Project Manager, EPA, 1992.

As illustrated in Figure 2, groundwater from Area B also discharges into several on-site and off-site springs and Carroll Creek. PCE and TCE have also been irregularly detected in those surface waters although concentrations are generally less than 1 ppb. In 1997, the PCE concentration in Robinson Pond (Box Spring) jumped from 0.54 ppb in April to 20,000 ppb in October, and back to 0.87 ppb in November (USACE, 1998). Similarly, the TCE concentrations jumped from 4.5 ppb to 5,000 ppb and back down to 11 ppb for the same sampling events. This sharp, short term increase in PCE and TCE concentrations in Robinson Pond corresponds to excavation and sampling events that were conducted in the same time frame at the Area B-11 waste pits.

It is important to note that there is no direct exposure to water from the Box Spring sample site at Robinson Pond. This water is not used as drinking water. PCE and TCE analyses in Robinson Pond proper and Carroll Creek have been mostly non-detects with only two measured values above the MCL (Table 3). Note that the geometric means for these PCE and TCE samples from surface water samples in areas of potential exposure are both below their respective health comparison values.

The presence of PCE and TCE in drinking water supplies and surface waters at concentrations above relevant comparison values does not necessarily indicate that these contaminants presented a public health hazard. The following section on exposure pathways will evaluate the conditions and timeframe of potential exposures to PCE and TCE.

Table 1. On-site groundwater contaminants at Fort Detrick Area B.

Contaminant	Maximum Value* ppb	Comparison Value ppb Source	Contaminant of Concern
Benzene	**31.5**	**5 MCL**	**Y**
Carbon tetrachloride	2.71	5 MCL	N
Chloroform	**300**	**80 MCL**	**Y**
1,4-Dichlorobenzene	2.0	700 MCL	N
1,2-Dichloroethane	**23**	**5**	**Y**
1,1-Dichloroethene	**97.5**	**7**	**Y**
1,2-Dichloropropane	**125**	**5 MCL**	**Y**
Dieldrin	0.08	0.5 EMEG cc	N
Heptachlor	0.003	0.4 MCL	N
Heptachlor epoxide	**3.2**	**0.2 MCL**	**Y**
1,1,2,2-Trichloroethane	0.97	5,000 EMEG ci	N
Tetrachloroethene (PCE)	**30,000**	**5 MCL**	**Y**
1,1,1-Trichloroethane	100	200 MCL	N
1,1,2-Trichloroethane	3.2	5 MCL	N
Trichloroethene (TCE)	**1,000**	**5 MCL**	**Y**
Trichlorofluoromethane	300	3,000 MCL	N
Vinyl chloride	**3.45**	**2 MCL**	**Y**

* Maximum values are from Exhibit 6-15, Exposure point concentrations for chemicals of potential concern (USACE, 1998; sampling occurred circa 1995-96).
MCL – EPA maximum concentration level
EMEG—ATSDR environmental media evaluation guides (cc- child chronic; ci- child intermediate).

Table 2. Off-site groundwater contaminants from Area B sources.

Contaminant	Maximum Value ppb	Comparison Value ppb Source	Evaluate Exposures
Benzene	ND	5 MCL	N
Chloroform	5	80 MCL	N
1,2-Dichloroethane	ND	5 MCL	N
1,1-Dichloroethene	0.6	7 MCL	N
1,2-Dichloropropane	ND	5 MCL	N
Heptachlor epoxide	ND?	0.2 MCL	N
Tetrachloroethene (PCE)	**17***	**5 MCL**	**Y**
Trichloroethene (TCE)	**19***	**5 MCL**	**Y**
Vinyl chloride	ND	2 MCL	N

* Results from 1992 off-site sampling as documented in facsimile from Fort Detrick Directorate of Safety, Health, and Environment to Steve Hirsch, EPA Region 3.
Other results from Excel spreadsheet "Fort Detrick Res Well Data 1995 to 2007a.xls" provided to ATSDR.

Table 3. PCE and TCE Concentrations in Surface Waters Adjacent to Area B.

Chemical	Range of Detections (ppb)	Frequency of Detections	Frequency Above Comparison Value	Geomean* ppb	Comparison Value (ppb)
Box Spring (surface flow into Robinson Pond; sample ID RISP-3)					
PCE	ND–20,000	55 of 63	2 of 63	1.0	5 MCL
TCE	4.5—5,000	63 of 63	62 of 63	11.6	5 MCL
Robinson Pond and Carroll Creek (below Robinson Pond; sample IDs SW-2, SW-4, and STR5SW-44)					
PCE	ND—4.3	3 of 10**	0 of 9	1.5	5 MCL
TCE	ND—10	6 of 10**	2 of 10	2.3	5 MCL

ND – Non-detect (concentration below measurable level)
MCL – maximum contaminant level
ppb – parts per billion
* Geomean is the geometric mean of all measured values (or average of log values).
** Note that several of the surface water analyses include "U" qualifiers which denote unconfirmed analyses.
Data are from USACE, 1998 and an EXCEL spreadsheet provided to ATSDR ("All Area B SW.xls")

Pathways of Contaminant Exposure

A pathway of exposure describes the process and timeframe by which a person is exposed to contaminants from a site. Pathways may be "complete, potentially completed, or eliminated" if all parts of the pathway are present, one or more parts are unknown, or not present, respectively. Exposure pathways also have a time component, such that they may have been completed in the past, the present, or potentially complete in the future. Figure 2 graphically illustrates where people may come into contact with contaminates from Area B groundwater. These locations include off-site drinking water wells, springs and streams, and volatization to air from the subsurface plume into houses.

These pathways of exposure are listed in Table 4. The off-site drinking water well pathway is considered to be complete only for past exposures. All affected off-site locations with contaminated wells have been provided with an alternate water supply beginning with the initial locations sampled in 1992. At that time, only three of ten locations sampled had PCE or TCE concentrations that exceeded drinking water standards (MCL). Due to source remediation, groundwater monitoring, and ongoing groundwater management, no future exposures via drinking water wells are expected.

Past exposures to PCE and TCE will be evaluated based on 1992 measured drinking water well concentrations of 17 ppb and 19 ppb (respectively; Table 2). It is not known if PCE and TCE concentrations were higher in off-site wells before 1992, but considering that TCE-containing drums were placed in the B-11 pits around 1968 (USACE, 1998) it is assumed that low levels of TCE contamination were present in these wells for several years.

Exposure to PCE and TCE in surface water is an eliminated pathway (Table 4). Although there is one set of PCE/TCE analyses from the Box Spring of Robinson Pond with high concentrations of these contaminants (in October, 1997 concentrations spiked to values of 20,000 ppb PCE and 5,000 ppb TCE; USACE, 1998), there is no direct contact with that water. Water from the Box Spring flows into Robinson Pond proper and then via an outfall stream into Carroll Creek. Most PCE/TCE analyses from Robinson Pond and Carroll Creek where exposure could occur are non-detects. As average PCE/TCE concentrations in surface waters where only incidental exposures could occur are well below the comparison values for drinking water, this pathway is not considered a health concern because contaminant concentrations at points of potential exposure, if detected, are below health comparison values.

As with surface water, exposure to sediments from Robinson Pond and Carroll Creek is not a significant pathway of human exposure. Volatile organic compounds such as PCE and TCE volatize rapidly from surface water to air and do not readily adsorb onto sediment particles (ATSDR, 1997a; 1997b; respectively). Similarly, PCE and TCE do not bioaccumulate so fish, if present in Carroll Creek, would not contain those contaminants. Carroll Creek and Robinson Pond do not represent a significant fishery resource. Volatile compounds like PCE and TCE occasionally present a vapor intrusion hazard when the gaseous compounds volatize from groundwater into confined air spaces such as basements or tunnels. As there are no buildings or confined underground spaces overlying the high concentration portion of the plume (immediately down-gradient or east of the B-11 pits) this pathway is not complete for the Area B groundwater.

Table 4. Exposure Pathway Elements for Fort Detrick Area B groundwater

Pathway Name	Source of Contamination	Fate and Transport	Point of Exposure	Route of Exposure	Potentially Exposed Population	Time Frame for Exposure
Completed Pathway Off-site drinking water wells	Area B waste pits	Limited detections of PCE/TCE above the MCL in drinking water wells	Tap water in homes or businesses that used private wells	P Ingestion P Skin contact P inhalation	People in two locations with contaminant detections	Past, locations now supplied with alternate water source
Eliminated Pathways Surface water	Emerging groundwater	PCE/TCE concentrations below comparison values in Carroll Creek	No significant exposure in downstream areas of Carroll Creek	None	None	Not applicable
Contacting sediments in Carroll Creek	Emerging groundwater	PCE/TCE not significantly adsorbed on aquatic sediment.	None. No significant sediment contamination.	None	None	Not applicable
Fish caught from Carroll Creek	Emerging groundwater	PCE/TCE not routinely detected in surface water. TCE/PCE do not bio-accumulate	None.	None	None	Not applicable
Vapor intrusion	Subsurface PCE/TCE plume	Volatization from subsurface plume.	No on-site or off-site buildings directly over high concentration portion of plume.	None	None	Not applicable

Exposure scenarios and dose calculation

Estimating an exposure dose requires identifying how much, how often, and how long a person may come in contact with some chemical in a specific medium (air, water, soil). The equation used to estimate exposure doses from ingesting contaminants in water is below (ATSDR, 2005a).

Equation 1: Exposure Dose Equation for Ingestion

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

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

For potential carcinogenic health effects, the doses are multiplied by the contaminant-specific cancer slope factor (Table 6) to determine the theoretical excess cancer risk:

Equation 2: Estimation of Theoretical Excess Cancer Risk

$$\text{Cancer Risk} = \text{Dose (mg/kg/day)} \times \text{Cancer Slope Factor (mg/kg/day)}^{-1}$$

Doses from ingestion, inhalation, and dermal contact with TCE and PCE at the offsite well locations are shown in Table 5. The exposure dose calculations assume that exposure occurred in a residential setting, that is that exposure was continuous (24 hours per day, 365 days per year) and all water consumed was from affected wells. Exposures are assumed to have occurred over a 40 year timeframe (circa 1955 to 1992; EPA, 2008). Doses from other residences are not estimated because PCE and TCE were not detected or were intermittently detected below health comparison values.

Table 5 presents the estimated doses for both adults and children based on the maximum measured PCE and TCE concentrations from the drinking water wells. The doses are in units of milligrams (PCE or TCE) per kilogram body weight per day (mg/kg/day). Adult doses are estimated assuming a person drinks 2 liters of water per day (from the household source) and weighs 70 kg (154 pounds). Doses for children assume 1 liter per day water ingestion and a weight of 16 kg (35 pounds). In addition to the oral dose from drinking water, for these VOCs a person may also absorb these compounds directly from contaminated water through the skin (dermal dose) and breathe the compound in the air (inhalation dose). These secondary exposures to the VOCs in drinking water essentially represent a doubling of the ingestion dose. The public health implications of the cumulative doses are discussed in the following section.

Table 5. TCE and PCE doses from groundwater exposure in residences near the Area B site.							
	Dose (mg/kg/day)	Concentration (ppb)	Ingestion Rate (L/day)	Exposure Factor	Conv. Factor	Body Wt. (kg)	
TCE	2.4E-03	19	1	2	1.00E-03	16	Child
	1.1E-03	19	2	2	1.00E-03	70	Adult
PCE	2.1E-03	17	1	2	1.00E-03	16	Child
	9.7E-04	17	2	2	1.00E-03	70	Adult

Notes:
Concentrations are from Table 2.
The exposure factor of 2 is used to account for a doubling of the ingestion dose due to inhalation and dermal contact.
Intake rates and body weights of children and adults are recommended values from EPA (1999).

Table 6. PCE and TCE doses, health comparison values (CVs), and excess cancer risks from exposure to groundwater at wells adjacent to Area B.

Well B Contaminant	**Adult Dose[1] mg/kg/day**	**MRL mg/kg/day**	**RfD mg/kg/day**	**CSF[2]**	**40 year Cancer Risk[3]**
PCE	0.001	0.05[6]	0.01	0.05[4]	2.8E-5[4]
TCE	0.001	NA	NA	0.02 to 0.4[5]	2.5E-4 to 1.2E-5

Notes:
[1] Adult doses are used to estimate cancer risk because the risks are based on lifetime exposures. Child exposures and intakes occur over a small portion of the assumed lifetime.
[2] Cancer slope factors (CSF) are in units of risk per unit dose 1/(mg/kg/day).
[3] Cancer risk is "excess theoretical risk" for a 40 year exposure. Standard cancer risk estimates are based on a 70 year lifetime excess risk. As the Area B-11 waste site operations began in 1955 (and it would take several years for groundwater contaminants to migrate to down-gradient locations) and exposures ceased in 1992, the 40 year exposure duration is appropriate and health protective.
[4] PCE is considered a "possible" human carcinogen and available data are not adequate to calculate quantitative risk factors (EPA, 2009). The listed CSF has been withdrawn (see following discussion on PCE in Public Health Implications section for more information).
[5] TCE cancer risks are calculated using two different slope factors. The EPA recommends using this range of CSFs and presenting a range of estimated excess cancer risks (EPA, 2001).
[6]The PCE MRL is for short term ingestion exposure (exposures occurring over a few hours to 14 days).

Public Health Implications

Historic analyses of groundwater samples from residential wells adjacent to the Area B site have detected low concentrations of several VOCs. The specific compounds, their measured concentrations, and health comparison values (CVs) are listed in Tables 2 and 5. PCE and TCE were the only compounds detected at concentrations above their respective screening values (MCLs). Note that only two wells (listed as Wells DWSRD-1 and DWSRD-A in Table 5) had VOC concentrations above the CVs and had the highest concentrations of most of the compounds.

The CVs are calculated concentrations of a substance in air, water, food, or soil that are unlikely to cause harmful (adverse) health effects in exposed people. The CV is used as a screening level during the public health evaluation process. Exposure to these compounds at concentrations greater than their comparison value does not necessarily mean that someone will get sick. Substances found in amounts greater than their CVs are selected for further evaluation by estimation of the doses that people may be exposed to via drinking or direct absorption of the contaminants from water and breathing them in air. These estimated doses are then compared with doses that have resulted in disease or sickness for people or laboratory animals. The health implications for each contaminant are presented in a discussion that relates the potential doses with the specific diseases or health effects caused by each contaminant.

Table 5 shows the two contaminants and their estimated doses that had concentrations above their respective comparison values. The estimated doses in Table 5 are based on measured concentrations of each contaminant in separate wells (Table 2). These locations had the only measured contaminant concentrations that exceeded the comparison values. Other wells had only trace concentrations or non-detections of VOCs.

It must be noted that these dose estimates and health determinations are based on the available measured VOC concentrations. Although unlikely (based on extrapolation of available concentration trends), it is possible that pre-1992 concentrations were higher than 1992 measured concentrations. If this had occurred, the resulting doses would have been commensurately higher.

Studies have shown that exposure to volatile organic compounds (VOCs) from routes other than direct ingestion might be as large as the exposure from ingestion alone. The inhalation dose due to volatization during a shower may equal the ingestion dose from 1.3 liters of water (Wan, et.al., 1990) and that 50—90% of VOCs in water may volatize during showering, laundering, and other activities (Moya et al, 1999; Giardino and Andelman, 1996). Similarly, the dermal dose has been estimated to equal 30% of the ingested dose (Maine DEP/DHS, 1992). Based on the results of these studies, combined VOC exposure doses in Table 4 include an inhalation dose that is 70% of the ingestion dose, and a dermal contact dose that is 30% of the ingestion dose.

Table 6 shows the estimated doses and theoretical excess cancer risk for TCE and PCE. These comparison values (MRLs, RfDs, and CSFs) are as defined below:

> **MRL**-- (minimal risk level) is an estimate of the daily human exposure to a substance that is likely to be without appreciable risk of adverse health effects during a specified

period of exposure (acute—minutes to 14 days; intermediate—14 to 365 days; chronic—more than 1 year);

RfD--(Reference Dose) is a daily dose that is likely to be without discernable risk of deleterious effects to human population (including sensitive subgroups) during a lifetime of exposure.

CSF--(cancer slope factor) is an estimate of age-averaged lifetime excess cancer incidence rate per unit intake.

A discussion of the estimated doses, cancer risks, and possible health effects from exposure to each to these contaminants is presented in the following sections.

Tetrachloroethylene (PCE or tetrachloroethene)

Based on the available measured PCE concentrations and estimated doses, adverse health effects from past exposure to PCE via contaminated drinking from the contaminated water wells around the Area B site are unlikely to produce any harmful health effects, including cancer.

The maximum measured concentration of PCE (1992) was 17 ppb in Well DWSRD-A (and trace or non-detectable concentrations in all other residential wells, Tables 1, 2, and 5; note that these concentrations have not been replicated in subsequent samples). The drinking water concentration of 17 ppb result in estimated combined (ingestion plus inhalation plus dermal contact) PCE doses of 0.00097 mg/kg/day for adults and 0.0021 mg/kg/day for children (Table 5). Estimated doses from Area B groundwater exposures and doses with adverse health effects are illustrated in Figure 3.

The following summary of PCE health effects is from the ATSDR Toxicological Profile of Tetrachloroethylene (ATSDR, 1997a) and from the EPA Integrated Risk Information System (http://cfpub.epa.gov/ncea/iris/index.cfm; accessed January, 2009). PCE is a manufactured compound widely used for dry cleaning fabrics and as a metal degreaser. It is also used as an intermediate in the manufacturing of other products. Summaries of both cancer and non-cancer PCE health effects for humans and laboratory animals are discussed below.

Non-cancer Effects

Liver and kidney damage have been observed in laboratory animal studies after exposure to high doses of PCE. Liver weight/body weight ratios were significantly higher than controls for animals treated with 100 mg/kg/day of PCE. At higher doses, hepatotoxic effects included decreased DNA content, increased SGPT, decreased levels of G6P and hepatocellular necrosis, degeneration and polyploidy (ATSDR, 1997a; EPA, 2009).

Groups of 20 Sprague-Dawley rats of both sexes were administered doses from 14 to 1,400 mg/kg/day (7,000-700,000 times greater than those estimated for the DWSRD-A residents). Male rats in the high-dose group and females in the two highest groups exhibited depressed body weights. Equivocal evidence of hepatotoxicity (increased liver and kidney weight/body weight ratios) were also observed at the higher doses (ATSDR, 1997a; EPA, 2009)

Relative sensitivity to man cannot be readily established, but the RfD of 0.01 mg/kg/day is protective of the most mild effects observed in humans [diminished odor perception/modified Romberg test scores in volunteers exposed to 100 ppm for 7 hours; roughly equivalent to 20 mg/kg/day].

As the maximum estimated exposure dose was about 5 times lower than the oral RfD and about 300 times lower than the No Observed Adverse Effect Level (Figure 3), non-cancer health effects are unlikely to result from these exposures. The RfD was derived from a NOAEL of 14 mg/kg/day, and a LOAEL of 71 mg/kg/day, based on hepatotoxicity in mice and weight gain in rats.

Cancer Effects

Various case-control studies were evaluated for possible associations between exposure to PCE and cancer effects in human populations. Although some of these studies indicate a possible association between exposure to PCE and various cancers, including bladder cancer, kidney cancer, and leukemia, the studies had limitations which precluded definitive conclusions. Cancer has been reported in experimental animals after oral exposure to PCE. Statistically significant increases in hepatocellular carcinomas occurred in the treated mice of both sexes. A cancer effect level (CEL) of 386 mg/kg/day was derived from a chronic mouse study (ATSDR, 1997a). The cancer effects in this study were hepatocellular carcinomas. However, the highest off-site exposure dose (0.0021 mg/kg/day; Table 5) was more than 183,000 times lower than this CEL.

An EPA workgroup is currently reassessing PCE carcinogenicity and has removed the oral slope factor. In 1987, an EPA carcinogen assessment proposed PCE as a probable human carcinogen. In light of new data, EPA findings indicate that the weight-of-evidence for PCE as human carcinogen is on a continuum between *a possible and a probable human carcinogen*. Presently, the agency has not adopted a final position on the classification of human carcinogenicity for this chemical. In order to estimate the cancer risk from exposure to PCE, the 1987 oral slope factor was used (Table 6). The estimated increased cancer risk, assuming a continuous 40 year exposure to the maximum concentration (17 ppb) of PCE in drinking water, is approximately 0.00003 (2.8E-5; Table 6), averaged over a 70 year lifetime. The actual risk is likely even less than this since this estimated exposure dose assumed inhalation, dermal, and ingestion routes of exposure for the entire 40 year time period.

All of the uncertainties and conservative exposure assumptions associated with the dose calculations are included in the risk estimation as well as the uncertainty in deriving the cancer slope factor (EPA, 2000). The risk estimates in Table 6 cannot be interpreted as evidence that any of the Area B site neighbors will develop cancer as a result of PCE exposure. *These estimates of excess risk fall within the range of low to no apparent increased risk* (ATSDR, 1991).

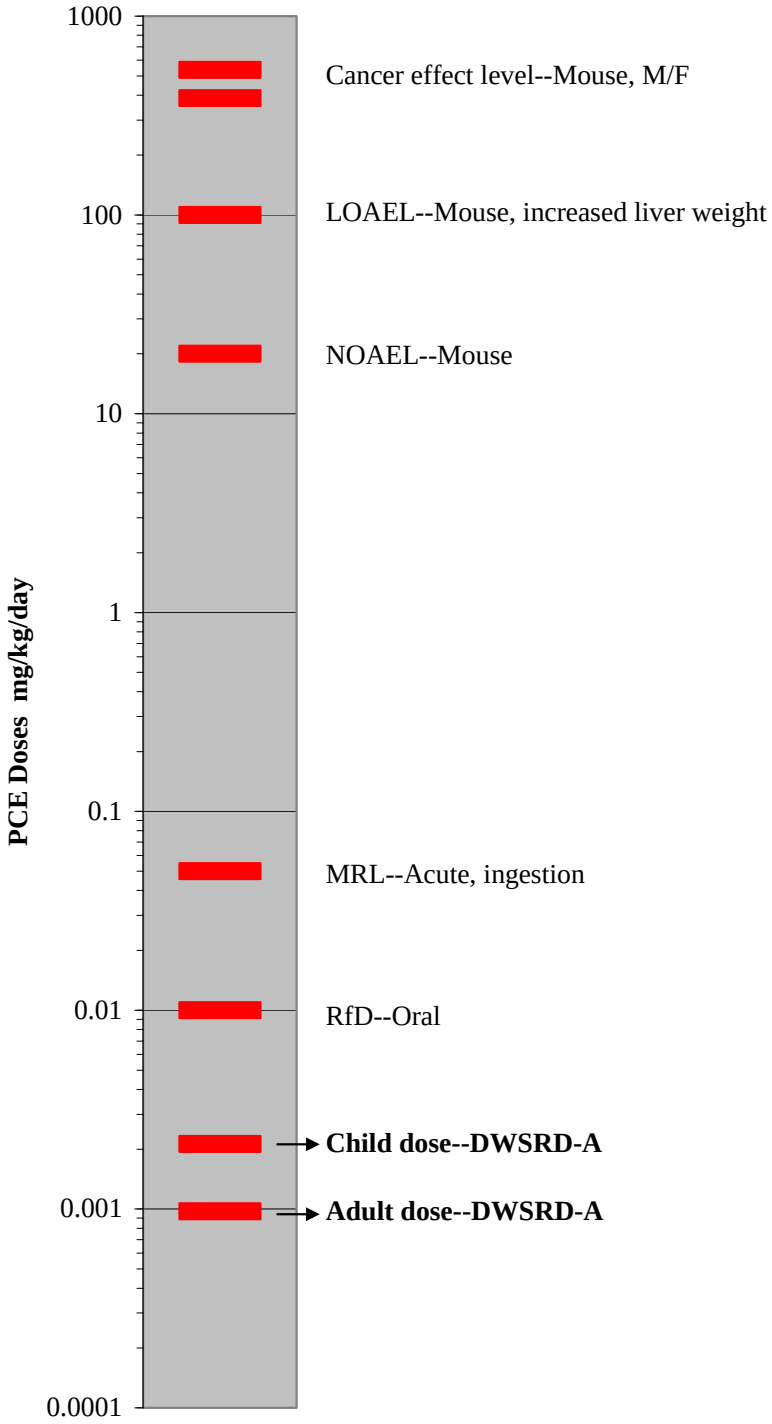

Figure 3. Estimated PCE doses from groundwater exposures at locations adjacent to the Area B site relative to health comparison values and doses associated with adverse health effects. MRL is minimal risk level; NOAEL is no observed adverse effect level; and LOAEL is lowest observed adverse effect level.

Trichloroethylene (TCE or trichloroethene)

Based on estimated doses to the maximum measured TCE concentration, adverse health effects from past exposure to TCE via exposure to contaminated water around the Area B site are unlikely to produce any harmful health effects, including cancer.

Measurements of TCE (992) were 19 ppb in Well DWSRD-1, 18 ppb in Well DWSRD-A, and trace or non-detectable concentrations in all other residential wells (Tables 1, 2, and 5; note that these concentrations have not been replicated in subsequent samples). The drinking water concentrations result in estimated combined (ingestion plus inhalation plus dermal contact) TCE doses of 0.0011 mg/kg/day for adults and 0.0024 mg/kg/day for children (Table 5). Estimated doses at the DWSRD-A location would be similar, but slightly lower. Estimated doses from Area B groundwater exposures and relevant health comparison doses (doses with adverse health effects) are illustrated in Figure 4.

The following summary of TCE health effects is from the ATSDR Toxicological Profile of Trichloroethylene (ATSDR, 1997b) and from the EPA Integrated Risk Information System (http://cfpub.epa.gov/ncea/iris/index.cfm); accessed January, 2009). TCE is a nonflammable, oily, colorless liquid that has a sweet odor and a sweet, burning taste. Years ago, TCE was used as an anesthetic. It is now used as a solvent to remove grease from metal parts and to make other chemicals. It is heavier than water and has low solubility (up to one part TCE per thousand parts of water at room temperature; ATSDR, 1997).

When present in groundwater, free-phase TCE tends to settle into a layer at the bottom of the aquifer and then continuously dissolves into the groundwater. This may result in high levels of TCE in the aquifer for years after the original release of contamination has ended. Alternatively, dissolved-phase TCE flows with groundwater. There is limited evidence of free-phase (or dense, non-aqueous phase PCE/TCE) at Area B such that most of the documented contaminant plume is present as a dissolved phase. Summaries of both cancer and non-cancer TCE health effects for humans and laboratory animals are discussed below.

Non-cancer Effects

ATSDR has derived a health guideline of 0.2 mg/kg/day for ingestion of TCE based on an acute-duration (less than 14 days) study showing developmental and behavioral changes in mouse pups administered 50 mg/kg/day of TCE (Fredriksson et al., 1993). In this study, the TCE was dissolved in oil and administered by stomach tube (gavage; ATSDR, 1997b). The findings of this study are not entirely relevant for evaluating health hazard for Area B site neighbors exposed to TCE in well water for several reasons. First, gavage doses in the animal study were administered as one large dose per day, while Area B site neighbors were likely to have been exposed to TCE in drinking water several times a day. (The body handles a single large dose much differently than it does a series of smaller doses.) Second, the total dose entering the body is higher and maintained for a longer time when TCE is dissolved in oil than when it is dissolved in water. Lastly, exposure to TCE in the animal study lasted less than 14 days, while maximum exposures to Area B site neighbors may have occurred over a period of many years. Despite these

limitations, the estimated TCE doses for Area B site neighbors are much lower than any for which adverse health effects have been documented.

ATSDR's TCE Sub-Registry reports an excessive number of children aged 9 years old or younger with speech and hearing deficits (note that these exposures are not related to this site; ATSDR, 1994). Although the exposure levels of these children were not well characterized, the findings support the types of outcome seen in animals. Several studies of workers and community residents suggest a possible association between exposure to TCE (and other chemicals) and developmental outcomes (ATSDR, 1998; Fagliano et al., 1990; Bove et al., 1995; MDPH, 1997). However, none of the studies provide conclusive evidence for a causal relationship, largely because information about TCE exposure was incomplete and exposure to other chemicals was likely (ATSDR, 1997b). Collectively, the scientific data indicate that the developing nervous system in young animals and humans may be sensitive to the toxic effects of TCE (ATSDR, 1997b), although the dose levels at which these effects occur has not been established. The available TCE measurements indicate that past exposures to TCE by Area B site neighbors were many orders of magnitude below TCE doses that have been shown to cause neurotoxic effects in animals.

Also, as the estimated TCE doses are much lower than any doses that have been shown to produce other, non-cancer health effects, such as liver disease (Figure 4), past exposures to TCE, via drinking water wells are unlikely to produce any non-cancer adverse health effects. The highest estimated doses are above the proposed RfD (Figure 4). However, these exposures would not be expected to cause adverse health effects because the proposed RfD has an uncertainty factor of 3,000 (this means the RfD is 3,000 times lower than doses which have caused adverse health effects in laboratory animals).

Cancer Effects

There are several reports of an increased occurrence of cancer from ingestion and inhalation of TCE by animals and humans (ATSDR, 1997b; 1998). Human health studies *suggest* an increased incidence of cancer of various types (e.g., bladder, lymphoma, kidney, respiratory tract, cervix, skin, liver, and stomach) from exposure to TCE; however, no studies provide clear, unequivocal evidence that exposure is linked to increased cancer risk in humans (ATSDR, 1997b; 1998). The available studies suffer from inadequate characterization of exposure, small numbers of subjects, and the fact that subjects were likely exposed to other potentially carcinogenic chemicals. There is, however, sufficient evidence that TCE exposure results in cancer development in animals, although animal studies may not be relevant for evaluating health hazard to humans (ATSDR, 1997b).

In 1989, EPA withdrew its cancer assessment for TCE, which was based primarily on animal studies, because more recent pharmacokinetic and mechanistic data for TCE became available (EPA, 2006; Cogliano, 1999). An updated approach to TCE cancer assessment using existing animal data and state-of-the-science papers has been proposed (Cogliano, 1999). This approach, which is supported by high-dose animal studies, does not appear entirely relevant for evaluating the health hazard of low-dose human environmental exposures for several reasons. First, cancer in animals appears to result from species-specific mechanisms that are not entirely relevant to humans (ATSDR, 1997b). Second, the animals used in these studies were exposed to very high

doses of TCE, several orders of magnitude *higher* than estimated for Area B site neighbors, and the overall death rate in the animal studies was high. The surviving animals were not likely to have been in good health and, therefore, would have been more susceptible to adverse effects from TCE exposure (like infections and illnesses) than healthy animals. Third, the overall findings from animal studies are inconsistent: some studies report an increased incidence of cancer, while an equal number do not report an increase at similar levels of exposure (ATSDR, 1997b). Fourth, the studies used pure TCE and did not evaluate the effect of exposure to stabilizers and impurities in TCE; these things may also be carcinogenic.

The excess cancer risks in Table 6 represent an estimate of the theoretical increase in cancer risk due to exposure to TCE. Note that the TCE cancer risks in Table 6 include estimated excess risk calculated with two different cancer slope factors. The EPA TCE Health Risk Assessment (EPA, 2001) has identified several cancer slope factors, with most between $2x10^{-2}$ and $4x10^{-1}$ per mg/kg-d (Table 6). As there is no scientific consensus on a specific CSF, the EPA recommends using a range of CSFs and presenting a range of estimated excess cancer risks. Consequently, the highest estimated excess cancer risks due to forty years of TCE exposure range from about 0.0003 (2.5E-04) to about 0.00001 (1.2E-5; Table 6).

All of the uncertainties and conservative exposure assumptions associated with the dose calculations are included in the risk estimation as well as the uncertainty in deriving the cancer slope factor (EPA, 2000). The risk estimates in Table 6 cannot be interpreted as evidence that any of the Area B site neighbors will develop cancer as a result of TCE exposure. *These estimates of excess risk fall within the range of low to no apparent increased risk* (ATSDR, 1991). Note that the highest theoretical excess cancer risk (calculated using the highest cancer slope factor and assuming 40 years of exposure) of about 0.0002 (1.9E-04) is "outside the EPA acceptable risk range." These low risk estimates indicate that TCE exposure from Fort Detrick Area B sources is not likely to cause an observable increase in cancer.

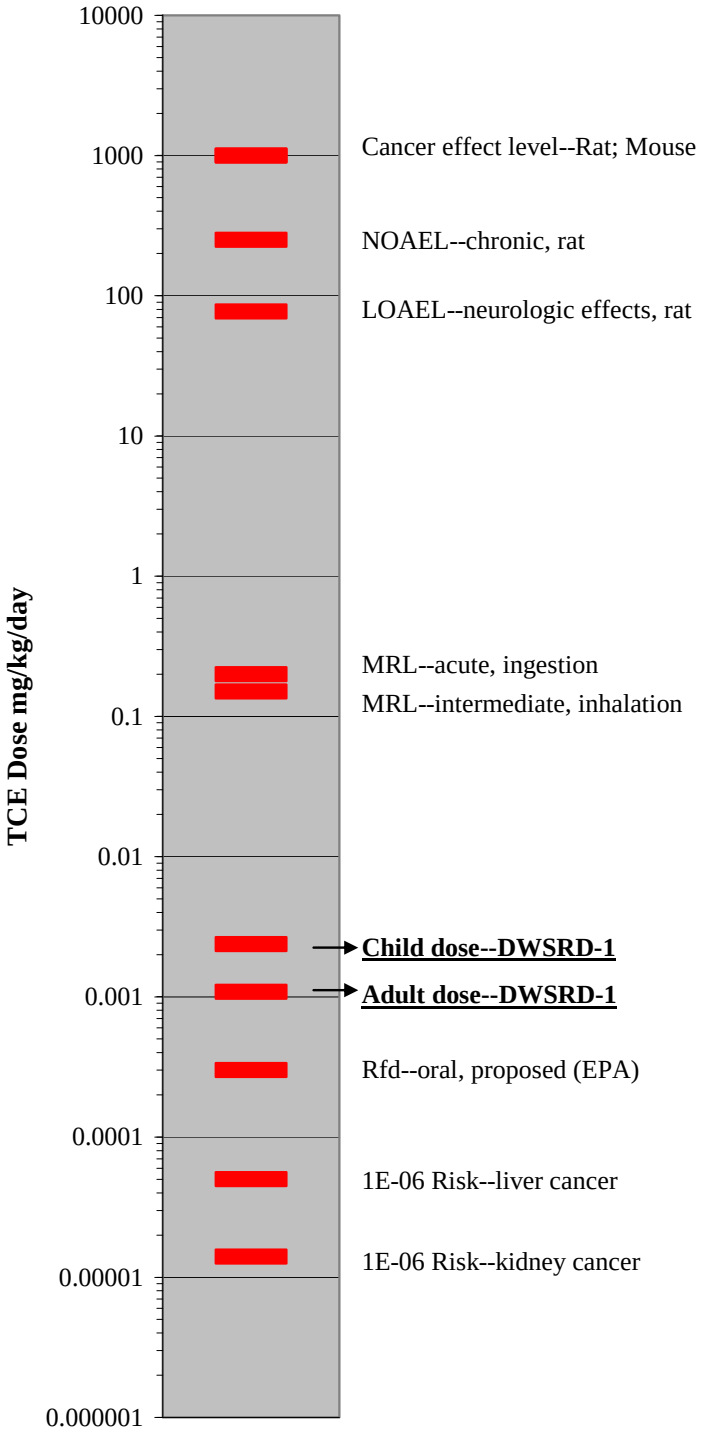

Figure 4. Estimated TCE doses from groundwater exposures at locations adjacent to the Area B site relative to health comparison values and doses associated with adverse health effects. MRL is minimal risk level; NOAEL is no observed adverse effect level; and LOAEL is lowest observed adverse effect level.

Adequacy of Available Data

The groundwater and well monitoring data in off-site drinking water wells indicate that at least two households may have been exposed to VOCs via ingestion of water, direct skin contact with the water, and inhalation of vapors from the water. The concentrations for two different VOCs (PCE and TCE) are listed in Tables 1, 2, and 5. Comparison of the estimated VOC doses based on those measured concentrations at the private well locations with health comparison values and doses associated with adverse health effects in humans and laboratory animals indicates that observable harmful health effects are unlikely.

However, the contaminant concentrations on which these dose estimates are based reflect only the data available at each location. There are no historic monitoring data available to determine when the private wells first became contaminated or if pre-1992 concentrations were higher than post 1992 analyses. Available monitoring data indicate that VOC concentrations are decreasing from 1999 onward (Shaw Environmental, 2008). Additionally, data from 1995 onward indicate only non-detections or trace concentrations of PCE and TCE in the affected drinking water wells.

Existing data suggest that past concentrations were not any higher than those measured in the 1990's. Groundwater sampling was conducted using EPA protocols with EPA and MDE oversight and analyses were conducted at EPA-approved laboratories (USACE, 1998). Sampling protocols and quality control procedures for the groundwater samples appear adequate for use of the resulting data in public health determinations.

Child Health Considerations

In communities faced with air, water, or food contamination, the many physical differences between children and adults demand special emphasis. Children could be at greater risk than are adults from certain kinds of exposure to hazardous substances. Children play outdoors and sometimes engage in hand-to-mouth behaviors that increase their exposure potential. Children are shorter than are adults; this means they breathe dust, soil, and vapors close to the ground. A child's lower body weight and higher intake rate results in a greater dose of hazardous substance per unit of body weight. If toxic exposure levels are high enough during critical growth stages, the developing body systems of children can sustain permanent damage. Finally, children are dependent on adults for access to housing, for access to medical care, and for risk identification. Thus adults need as much information as possible to make informed decisions regarding their children's health.

In this PHA, doses to children have been estimated using child-specific intake rates and body weights. As the estimated child-specific doses are somewhat greater than adult doses, the resulting public health determinations are based on the doses and potential adverse health effects to children.

Conclusions, Recommendations, and Public Health Action Plan

Conclusions

Based on the above findings, past operations at the Area B site have led to releases of several VOCs, including PCE and TCE into groundwater underlying the Area B site. Off-site migration of these contaminants, via groundwater flow, has caused the contamination of two residential drinking water wells. People living in those residences with private wells were exposed to the PCE and TCE by drinking water from the wells, direct skin contact with the VOCs in the household water, and breathing the gaseous VOCs that escaped into the household air.

This PHA provides an estimate of the VOC exposure doses to residents of those houses using the maximum measured concentrations of the VOCs in 1992 (when potable use of the wells ceased). Evaluation of the contaminant distributions and estimated doses, using both children and adult body weights and intake rates for PCE and TCE leads to the following public health determinations;

- Harmful health effects are unlikely for users of the contaminated private wells based on maximum measured concentrations of PCE and TCE.

- Residences with contaminated wells are currently being provided with alternate water. Current exposure to VOCs at these locations is limited to incidental use of the wells for irrigation or other outside uses. These exposures are unlikely to result in any harmful health effects.

- The available information on the historic use and release of VOCs at the Area B site and patterns of groundwater flow is not adequate to determine how long (prior to 1992) the residential wells were contaminated.

- The current distribution of groundwater VOCs suggests that concentrations in residential wells were not higher in the past.

- Based on the above findings, *past, current and future* exposures to VOCs via contaminated groundwater are not expected to cause adverse health effects. This determination means that exposures to site-related contaminants have occurred, or may be occurring, but at levels unlikely to create any harmful health effects.

Recommendations

Based on the above conclusions, ATSDR recommends continued monitoring of the groundwater contaminants and completion of ongoing site remediation activities.

Public Health Action Plan

To ensure community understanding and address future public health issues related to the Area B groundwater site, ATSDR will conduct the following public health actions:

- ATSDR has presented the results of this PHA to the Fort Detrick community (via the Fort Detrick Restoration Advisory Board) and addressed public comments as appropriate (Appendix C).
- ATSDR will evaluate additional monitoring data and information provided by the U. S. Army, MDE, and the EPA and, if requested, revise the public health conclusions of this health assessment as necessary.

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Appendix A. ATSDR Glossary of Terms

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

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

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

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

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

Aerobic
Requiring oxygen [compare with anaerobic].

Ambient
Surrounding (for example, ambient air).

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

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

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

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

Background level
An average or expected amount of a substance or radioactive material in a specific environment, or typical amounts of substances that occur naturally in an environment.

Biodegradation
Decomposition or breakdown of a substance through the action of microorganisms (such as bacteria or fungi) or other natural physical processes (such as sunlight).

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

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

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

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

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

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

CAP [see Community Assistance Panel.]

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

Cancer risk
A theoretical risk 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 [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. This law was later amended by the Superfund Amendments and Reauthorization Act (SARA).

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 an injury that happens as a result of exposures that might have occurred in the past.

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

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

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

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

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

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

DOD
United States Department of Defense.

DOE
United States Department of Energy.

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

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

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

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

Environmental media and transport mechanism
Environmental media include water, air, soil, and biota (plants and animals). Transport mechanisms move contaminants from the source to points where human exposure can occur. The environmental media and transport mechanism is the second part of an exposure pathway.

EPA
United States Environmental Protection Agency.

Epidemiologic surveillance [see Public health surveillance].

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

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

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

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

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

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

Exposure registry
A system of ongoing followup of people who have had documented environmental exposures.

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

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

Grand rounds
Training sessions for physicians and other health care providers about health topics.

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

Half-life ($t_{½}$)
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 evaluate 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.

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/cm^2
Milligram per square centimeter (of a surface).

mg/m^3
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, a condition, or an 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.

National Toxicology Program (NTP)
Part of the Department of Health and Human Services. NTP develops and carries out tests to predict whether a chemical will cause harm to humans.

No apparent public health hazard
A category used in ATSDR's public health assessments for sites where human exposure to contaminated media might be occurring, might have occurred in the past, or might occur in the future, but where the exposure is not expected to cause any harmful health effects.

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

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

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

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

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

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

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

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

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

ppb
Parts per billion.

ppm
Parts per million.

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

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

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

Public 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 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 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 health 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.

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 an 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 [see Comprehensive Environmental Response, Compensation, and Liability Act of 1980 (CERCLA) and Superfund Amendments and Reauthorization Act (SARA)]

Superfund Amendments and Reauthorization Act (SARA)
In 1986, SARA amended the Comprehensive Environmental Response, Compensation, and Liability Act of 1980 (CERCLA) and expanded the health-related responsibilities of ATSDR. CERCLA and SARA direct ATSDR to look into the health effects from substance exposures at hazardous waste sites and to perform activities including health education, health studies, surveillance, health consultations, and toxicological profiles.

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

Surveillance [see public health 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 that, under certain circumstances of exposure, can cause harmful effects to living organisms.

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

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

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

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

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

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

Volatization

The process of evaporation of a liquid into the air; VOCs such at PCE and TCE readily evaporate into air at normal ambient or room temperatures.

Other glossaries and dictionaries:
Environmental Protection Agency (http://www.epa.gov/OCEPAterms/)
National Library of Medicine (NIH)
(http://www.nlm.nih.gov/medlineplus/mplusdictionary.html)

For more information on the work of ATSDR, please contact:
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Appendix B:
Analytes tested in Off-Site Wells and TICs in On-Site Wells

Table B-1: Analytes Tested in Off-site Wells

Note that most of the analytes tested in off-site wells were not detected. Table 2 (main document) lists those analytes that were detected along with their respective concentrations, etc.

Table B-2: Tentatively Identified Compounds in On-site Wells

Note that most of these tentatively identified compounds (TICs) do not have specific comparison values. This may be because the compounds are non-specific, such as “acid esthers” or “aldehydes”, or “non-specific ether” or are sufficiently rare such that their specific toxicity has not been quantified (by the EPA or ATSDR). Although concentrations of TICs should be considered only as “estimates of true concentration”, the concentrations of compounds that do have applicable comparison values are well below levels of public health concern. Based on the relative on-site/off-site dilution of concentrations of TCE and PCE, it is unlikely that any of the listed tentatively identified compounds were present at off-site drinking water wells at levels of public health concern.

TICs or Tentatively Identified Compounds are those which can be detected by an analytical method but concentration cannot be confirmed without additional analytical testing. For instance, a gas chromatograph/mass spectrometer instrument can be calibrated to identify and quantify the concentrations of a number of target compounds. However, additional compound spectra may be detected for which instrument was not calibrated. Their identity can be confirmed with a search of the spectral library of compounds to find a match, but the concentration cannot be confirmed without running a known standard of the tentative matched compound. Sometimes no good match for the compound can be found, so only the class of compound can be identified (i.e. it's an alkane; http://www.caslab.com/Tentatively_Identified_Compounds_TICs_Meaning/).

Table B-1. List of analytes measured in off-site drinking water wells. Note that most of these compounds were not detected in any off-site wells.

Ethylbenzene
Styrene
cis-1,3-Dichloropropene
1,4-Dichlorobenzene
1,2-Dibromoethane / Ethyl dibromide
1,2-Dichloroethane
4-Methyl-2-pentanone
Toluene
Chlorobenzene
Dibromochloromethane
PCE
cis-1,2-Dichloroethylene
trans-1,2-Dichloroethylene
1,3-Dichlorobenzene
Carbon tetrachloride
2-Hexanone
Acetone
Chloroform
Benzene
1,1,1-Trichloroethane
Methyl bromide
Chloromethane
Bromochloromethane
Chloroethane
Vinyl chloride
Methylene chloride
Carbon disulfide
Bromoform
Bromodichloromethane
1,1-Dichloroethane
1,1-Dichloroethylene
1,2-Dichloropropane
2-Butanone
1,1,2-Trichloroethane
TCE
1,1,2,2-Tetrachloroethane
o-Xylene
1,2-Dichlorobenzene
1,2-Dibromo-3-chloropropane
m- and/or p-Xylene (undifferentiated) / 1,3- and/or 1,4-Dimethylbenzene (undifferentiated)
trans-1,3-Dichloropropene

Table B-2. Tentatively identified compounds in on-site monitor wells (Area B)

Well No.	Sample Date	CAS No.	Substance Name	Conc.	Unit	Qual	Comparison Value	CV Reference
BMW17	3/24/97	76-13-1	1,1,2-Trichloro-1,2,2-trifluoroethane	**3**	ppb	N	300,000	RMEG-C
BMW56D	8/26/97	78-99-9	1,1-Dichloropropane / Propylidene chloride	**1**	ppb	N		
BMW57D	8/28/97	78-87-5	1,2-Dichloropropane	**4.3**	ppb	S	900	EMEG-CC
MW36D-0	1/24/95	1009-61-6	1,4-Diacetylbenzene / 1,1'-(1,4-Phenylene)bis(ethanone)	**5**	ppb	S		
BMW58D	4/15/97	111-76-2	2-Butoxyethanol	**20**	ppb	N	700	EMEG-IC
BMW32	4/25/95	80-40-0	4-Methylbenzenenesulfonic acid ethyl ester / ethyl-p-toluenesulfonate	**4**	ppb	S		
BMW56D	4/15/97		Acid Esters	**20**	ppb	N		
BMW56D	8/26/97		Aldehydes	**2**	ppb	N		
DWSRD-23	8/27/97		Aliphatic hydrocarbons	**2.9**	ppb	Y		
BMW58D	4/15/97	314-40-9	Bromacil	**30**	ppb	N	90	LTHA
BMW57D	8/28/97	108-86-1	Bromobenzene	**3.5**	ppb	N		
BMW31D	5/2/95	123-95-5	Butyl stearate / Octadecanoic acid butyl ester	**29**	ppb	S		
BMW58D	8/26/97		Chlorinated benzenes	**8**	ppb	N		
BMW17	3/24/97	000075-69-4	Chlorofluoromethane (Tri-)	**1**	ppb	N	3,000	RMEG-C
BMW57D	8/28/97	60-29-7	Diethyl ether	**65**	ppb	N	2,000	RMEG-C
BMW31D	5/2/95	2440-22-4	Drometrizole / Tinuvin P / 2-(2H-Benzotriazol-2-yl)-4-methylphenol	**13**	ppb	S		
BMW31D	5/2/95	112-95-8	Eicosane	**8**	ppb	S		
BMW57D	8/28/97		Ether - nonspecific	**91**	ppb	N		
BMW56D	4/15/97	143-07-7	Lauric acid / Dodecanoic acid	**6**	ppb	N		
BMW36	5/1/95	25154-52-3	Nonyl phenol (any isomer)	**4**	ppb	S		
BMW56D	4/15/97	124-07-2	Octanoic acid / Caprylic acid	**10**	ppb	N		
BMW56D	8/26/97	95-47-6	o-Xylene / 1,2-Dimethylbenzene	**1.5**	ppb	S		
BMW31D	5/2/95	646-31-1	Tetracosane	**11**	ppb	S		
BMW57D	8/28/97	109-99-9	Tetrahydrofuran / Diethylene oxide / Tetramethylene oxide	**73**	ppb	Y		

Appendix C

Comments and Responses

ATSDR has received one set of written comments from a member of the Fort Detrick Restoration Advisory Board (RAB). This appendix presents a summary of these comments and the ATSDR responses.

1) "…the Comment period be extended further so that the appropriate individuals at EPA and MDE can weigh in based on their technical review of the Assessment."

ATSDR Response: The comment period has been extended and comments from EPA and MDE representatives have been directly solicited and the revised version the PHA reflects their comments.

2) "I strongly object to how ATSDR arrived at its conclusion that there is a "*low level of community health concern*"…In fact there is plenty of community health concern."

ATSDR Response: The relevant portion of the PHA has been revised and the described health concerns have been included in the document.

3) "At page 4 of its Assessment, the ATSDR states: "*Area B was also used for buried disposal of the number of contaminants including biological materials (no live biological agents from the old weapons program have ever been detected)*"… Wrong. The discovery of live agents at Area B was well publicized in the media."

ATSDR Response: The above ATSDR reference relates to the disposal of historical biological weaponized agents that occurred at Area B. However, as you point out, because live pathogenic agents have been discovered, the statement is incorrect and has been deleted.

4) "It seems questionable to me, especially given the geology of Area B, that the sampling of ten off-site wells establishes the full extent of possible public exposures. Furthermore, given that none of these off-site wells is deeper than 180 feet, it seems beyond questionable for ATSDR to conclude that there is no groundwater problem."

ATSDR Response: More than 75 off-site wells and springs have been sampled for Area B site contaminants. The vast majority of these analyses have been non-detections. With regard to contaminant migration in the karst terrain, the dye tracing study (Ozark Underground Laboratory, 1997) concluded that "about 90% of all eosine and fluorescein dyes recovered during the groundwater tracing study discharged from this spring" (re: Robinson Spring). This indicates that the subsurface flow path for contaminants from the Area B site is well defined with discharge in the area along the southeastern boundary of the Area B site.

It should also be noted that Rhodamine WT dye introduced up-gradient of several disposal trenches was not recovered. However, Rhodamine WT is adsorbed onto clays and granular materials and is not recommended for use in granular aquifers (USEPA, 1988). Although the natural aquifer materials in this area are not "granular", the down-gradient landfill materials through which this dye would have to migrate are comprised of granular materials. The OUL 1997 report likewise concluded that the failure to recover the Rhodamine WT dye was due to adsorption onto aquifer materials.

As the above comments allude, there is also some potential for contaminants to sink below this shallow karstic flow system. VOCs, such as PCE and TCE, have the potential to exist

as "dense non-aqueous phase liquids" (DNAPLs). This type of subsurface flow only occurs when the VOCs are not dissolved in water. If the TCE and PCE are dissolved in water, they flow with the water rather than flowing as a separate liquid. Extensive contaminant monitoring indicates that this type of two phase flow is not prevalent at the Area B site (a single DNAPL detection has been attributed to a waste removal action and has not been replicated in subsequent sampling.)

In summary, there is more than twenty years worth of surface and subsurface water monitoring and hydrogeological studies at the Area B site. Although it is always possible to invent some hypothetical process by which contaminants from Area B are migrating to some unmonitored area, the collective monitoring and hydrogeological data uniformly indicate that subsurface flow from the Area B waste sites follow a short, shallow flow path towards Carroll Creek and the southeastern boundary of Site B.

5) "I do not accept ATSDR's assertion that the only contaminants of public health concern arising from Area B are TCE and PCE. In a telephone conversation of May 20, 2009, Robert Thomson of the EPA stated to me that other contaminants of concern to the EPA include pesticides and herbicides that evidently were dumped in substantial quantities at Area B."

   "Thus, both the start and end dates for the "30 year exposure duration" upon which so much of ATSDR's analysis is based, are probably erroneous."

ATSDR Response: The contaminant screening process, as described in the PHA, is initially based on evaluation of contaminant source materials and contaminants detected in on-site monitor wells. Of the hundreds of potential compounds placed in the Area B landfills (and tested for in on-site wells), a relatively small number have been regularly detected in groundwater. This screening process identified 17 groundwater contaminants in on-site monitor wells. Of these 17 contaminants, only 9 were present above health comparison values in on-site wells and only PCE and TCE have been measured above comparison values in off-site wells. In order for a substance to be identified as a "contaminant of public health concern" it must be present at a location of potential exposure at a concentration above an applicable health comparison value. The results of this screening process are confirmed by the EPA NPL documentation record which similarly identifies PCE and TCE as the primary contaminants of concern.

A list of "tentatively identified compounds" from on-site wells has been included in Appendix B. Although this list does include some herbicides and pesticides, the overall concentrations are low (see Appendix B) and are not present at levels of public health concern. There are several reasons why these contaminants may not present in significant concentrations. First, some of these compounds may not be very soluble. That means the compounds are more likely to remain as a solid or adsorb onto solid particles and not occur in significant quantities in groundwater. Secondly, these compounds may be present only as break down products of the original materials and thus occur in relatively minute quantities.

Herbicides, pesticides, and semi-volatile compounds have been analyzed in Area B groundwater samples. They are not tested on a routine basis because the results, which are primarily non-detections, indicate that ongoing analyses are not warranted.

With regard to the exposure duration for excess cancer risk calculation, disposal in the B-11 waste pits began around 1955 and ended in 1970. The specific locations for which these doses are calculated were provided with bottled water beginning in 1992. We have revised the theoretical excess cancer risk calculations to account for a forty year exposure duration. This change results in a small increase in the estimated risk but does not change the overall conclusions of this assessment.

6) "Another linchpin of ATSDR's analysis is the EPA's MCL figures, referred to as the 'Comparison Values' for both PCE and TCE in the amount of 5 ppb…"

ATSDR Response: ATSDR is aware of the 2006 NRC report and the "TCE Reduction Act". From 2006 NAS/National Research Council (NRC) report on scientific issues related to assessing health risks of trichloroethylene:
http://dels.nas.edu/dels/rpt_briefs/trichloroethylene_brief_final.pdf

Trichloroethylene is metabolized in the body by two major pathways (the oxidative pathway and the glutathione-conjugation pathway). There are many animal studies that show that trichloroethylene and its metabolites (products of metabolism) are associated with several health effects, including cancer. Studies of human populations (epidemiologic studies) suggest that trichloroethylene may also affect human health, but less is known about the exposures needed to induce effects and physiologic responses. In all risk assessments, it is very difficult to assess the relevance of the findings of animal studies to humans. To do so requires an understanding of which metabolites are responsible for observed health effects and their "mode of action," or how the metabolites cause health effects. The following are highlights of the committee's findings:

Kidney Toxicity and Cancer
Trichloroethylene and some of its metabolites in the glutathione-conjugation pathway have been shown to be both toxic and carcinogenic to the kidneys. There is concordance between animal and human studies, which supports the conclusion that trichloroethylene is a potential kidney carcinogen. Studies with experimental animals and human tissues indicate a genotoxic mode of action. The metabolite S-dichlorovinyl-L-cysteine has been linked with the development of kidney cancer, but there are no studies of the carcinogenic potential of this metabolite. The magnitude of exposure needed to produce kidney damage is not clear. Thus, it is not possible to predict whether humans are more or less susceptible than other animals to trichloroethylene induced kidney cancer. However, it should be noted that exposure levels associated with kidney cancer in animals and humans are orders of magnitude higher than levels found in off-site drinking water wells (adjacent to Ft Detrick Area B).

Liver Toxicity and Cancer
The epidemiologic evidence is mixed; some studies show an excess of liver cancer in trichloroethylene exposed populations while other studies do not. Animal data on trichloroethylene indicate that relatively high doses are needed to induce liver toxicity and

cancer, even in susceptible strains of mice. Three major oxidative metabolites (trichloroacetic acid, dichloroacetic acid, and chloral hydrate) can contribute to liver toxicity and cancer in rodents. The mode of action of trichloroacetic acid as a rodent liver carcinogen is not a likely mode of action in the human liver. For the metabolite chloral hydrate, differing rates of oxidation and conjugation in rats and humans make it unlikely that the mode of action in mice is relevant to humans. The mode of action for the metabolite dichloroacetic acid in rodents is understood, but whether this metabolite is formed in humans has not been established and differences between mice and human suggest that humans would be much less susceptible to liver carcinogenesis. Thus, exposure to trichloroethylene at concentrations relevant to the general public is not likely to induce liver cancer in humans. However, it is possible that much higher exposure to trichloroethylene, such as in certain high-risk occupations or in heavily contaminated locales, could result in increased risks of liver toxicity and cancer.

Respiratory Toxicity and Cancer
Trichloroethylene has been shown to induce lung tumors in rodents. The mode of action for this effect is localization of trichloroethylene metabolites in the Clara cells of the lungs. The collective evidence indicates that rodents and humans are significantly different in their capacity to metabolize trichloroethylene in the lungs, with humans having less capacity. Results of most epidemiologic studies of occupational exposure to trichloroethylene do not show a strong association between trichloroethylene exposure and increased incidence of lung tumors. Thus, pulmonary cancer does not appear to be a critical end point in assessing human health risks to trichloroethylene.

Additional information regarding TCE/PCE can be obtained from the following 2009 NRC report "Contaminated Water Supplies at Camp Lejeune: “Assessing Potential Health Effects." In their review of epidemiologic evidence, "the committee has concluded that the epidemiologic studies give some reason to be concerned that sufficiently high levels of the chemical may cause the disease, but the studies do not provide strong evidence that they actually do." http://dels.nas.edu/dels/viewreport.cgi?id=5793

In summary, there has been extensive recent scientific review of the toxicity and human health effects of exposure to TCE and PCE. These scientific reviews by the National Academy of Sciences indicate that the current health comparison values (such as the EPA MCL and the ATSDR MRL) are protective of human health.

Other Comments; 7/8) Two additional comments concerning other ATSDR site evaluations or non-specific statements were received.

ATSDR Response: Comments noted, no response necessary.