Health Consultation

PUBLIC COMMENT VERSION

An Exposure and Health Effects Evaluation of Former Workers and Residents to Chemical Contamination at 4825 Glenbrook Road

Within the Spring Valley Formerly Used Defense Site (FUDS)
Washington, District of Columbia

APRIL 19, 2016

COMMENT PERIOD ENDS: JUNE 20, 2016

U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES
Agency for Toxic Substances and Disease Registry
Division of Community Health Investigations
Atlanta, Georgia 30333

Health Consultation: A Note of Explanation

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

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

ATSDR is primarily an advisory agency, so its reports usually identify what actions are appropriate to be undertaken by USEPA or state regulatory agencies, federal or state public health agencies (including the research or health/community education divisions of ATSDR), the military, and/or other organizations. As part of being an advisory agency, ATSDR relies on information and data from other agencies and organizations.

You May Contact ATSDR Toll Free at

1-800-CDC-INFO

or

Visit our Home Page at: http://www.atsdr.cdc.gov

HEALTH CONSULTATION

PUBLIC COMMENT RELEASE

An Exposure and Health Effects Evaluation of Former Workers and Residents to Chemical Contamination at 4825 Glenbrook Road

Within the Spring Valley Formerly Used Defense Site (FUDS)
Washington, District of Columbia

Prepared By:

Eastern Branch
Division of Community Health Investigations (DCHI)
Agency for Toxic Substances and Disease Registry (ATSDR)
Atlanta, Georgia

Table of Contents

APPENDICES

FIGURES

TABLES

TABLES in APPENDICES

List of Abbreviations and Acronyms

ABP	agent breakdown product
ATSDR	Agency for Toxic Substances and Disease Registry
AU	American University
AUES	American University Experiment Station
bgs	below ground surface
CERCLA	Comprehensive, Environmental, Response, Compensation, and Liability Act
CSF	Cancer slope factor
CV	comparison value
CVAA	2-chlorovinyl arsonous acid
CVAO	lewisite oxide or chlorovinyl arsenous oxide
CWA	chemical warfare agent
CWM	chemical warfare materiel or monogram
DC DOH	District of Columbia Department of Health
DDOE	District (of Columbia) Department of the Environment
EMEG	environmental media evaluation guide
EMS	Environmental Management Services
FUDS	Formerly Used Defense Site
HCl	hydrochloric acid
HBESL	health based environmental screening level
LOAEL	lowest observed adverse effect level
MRL	minimal risk level
NOAEL	no observed adverse effect level
ppb	parts per billion
ppbv	parts per billion by volume
ppm	parts per million
RfD	Reference dose
TP	test pit
USACE	United States Army Corps of Engineers
USACHPPM	United States Army Center for Health Promotion and Preventive Medicine
USEPA	U.S. Environmental Protection Agency
VOC	volatile organic compound
WV	West Virginia
WWI	World War I

Summary

Introduction

The 4825 Glenbrook Road property is within the Spring Valley Formerly Used Defense Site (FUDS) in northwest Washington, D.C. in an area formerly known as the American University Experiment Station (AUES). At AUES, the U.S. government researched and developed chemical warfare agents (CWAs) and protective masks, and trained soldiers during the World War I (WWI) era.

In 2010, the United States Army Corps of Engineers (USACE) requested assistance from the Agency for Toxic Substance and Disease Registry (ATSDR) after finding additional WWI era munitions, glassware, debris, and soil containing or contaminated with CWAs and other chemicals at 4825 Glenbrook Road. USACE asked ATSDR to evaluate past exposure scenarios for two groups: construction and other workers who developed the property (1992-1993) and the family who lived at the residence (1994-1999). The USACE provided ATSDR with excerpts of worker transcripts from video recorded interviews conducted in 1993 and results from environmental sampling at the site. ATSDR evaluated these exposure scenarios to determine if workers or residents could have health effects from any past exposures.

However, our evaluation was limited because most of the available data are insufficient for public health evaluation purposes. Most environmental samples available for this health consultation were collected from 2007-2010, 14 to 18 years after potential exposures to workers and 8 to 11 years after potential exposures to residents. The time between potential exposure and sample collection results in samples that may not be representative of exposure levels. Because no indoor air samples were collected in the residence while it was occupied, we do not know if the residents were exposed to contaminants in their indoor air.

Because of these data gaps for both residents and workers, except for arsenic in soil, which is persistent in the environment and a breakdown product for many of the CWAs, ATSDR was not able to evaluate the public health implications using our standard public health assessment process (see Appendix B for a description of ATSDR's evaluation approach). Although ATSDR evaluated the worker's exposures to arsenic in soils using our standard approach, in the final analysis, ATSDR relied on an occupational medical officer evaluation of the worker transcripts provided by the USACE as the basis for our conclusions regarding the former workers. By using this alternative approach, we were able to conclude that the workers were likely exposed to other chemicals in addition to arsenic and to

provide some public health perspective to these exposures (see Basis for Conclusion 1—Former Workers below).

In a separate analysis, using ATSDR's standard health assessment approach, we do provide an evaluation of the available environmental data in relation to published health comparison values. In addition, in an attempt to be as responsive to the USACE and the public as possible, we have researched and provide general public health implications of several of the major CWAs found at the site. Because of the uncertainties in the exposure parameters such as not knowing what levels or for how long either former workers or residents were exposed, ATSDR did not draw health conclusions from this part of the evaluation (except from the arsenic concentrations remaining on the property after its development).

Conclusions

ATSDR reached two main conclusions in this health consultation.

Conclusion 1

Former Workers Who Developed the Property

Some construction workers likely experienced harmful short-term (or acute) health effects while working at the property because of exposure to arsenic, CWAs (such as lewisite and sulfur mustard), and other chemicals. Workers, especially those who worked on the property for a year or more, may have an increased cancer risk; however, ATSDR cannot quantify this risk with any certainty. Workers who had intensive soil contact and repeated exposures are more likely to have experienced harmful health effects. Except for arsenic, no exposure information is available to determine if workers could also have long-term non-cancer health effects.

Basis for Conclusion 1 Former Workers

In May 1992, several construction workers were reportedly overcome during soil excavation activities at 4825 Glenbrook Road and required emergency care for respiratory problems. Transcripts of interviews with workers indicate exposure to chemicals during home construction in 1992 and 1993. Based on a medical officer evaluation of these transcripts, exposure to arsenic, arsenicals, irritants, and possibly sulfur mustard was likely. Irritation of mucous membranes and skin effects were associated with these exposures.

Because of the lack of sampling for most worker exposures, most exposure parameters (concentrations, frequency, duration, etc.) are unknown. Site workers with intensive soil contact for a year or longer (ingesting 330 milligram/kilogram (mg/kg) or more) could have been exposed to arsenic at levels that cause hyperpigmentation and hyperkeratosis of the skin. Based on estimates for the ingestion of soil arsenic, the increased cancer risk to workers was low (estimated at about 2-4 additional cases per 100,000 exposed

	persons). However, this cancer risk estimate is based on soil arsenic levels remaining on the property after it was developed and is the minimum increased cancer risk (as soil removed during development is likely to have reduced soil arsenic concentrations). Although workers may have an increased cancer risk because of their chronic exposure to arsenic and sulfur mustard in soil, we cannot estimate their total increased cancer risk because of the uncertainty associated with the exposures parameters. Because workers could have been exposed to subsurface soil and chemical releases from broken containers, they could also have been exposed to pesticides, herbicides, and other chemicals on the property. However, the lack of sampling for most worker exposures prevents ATSDR from quantifying the health implications of exposures to these chemicals.
Conclusion 2	**Former Residents of the Property** Except for arsenic in soil, insufficient past exposure information is available to determine if former residents of 4825 Glenbrook Road had (or have) harmful health effects related to exposures. Dose estimates indicate that children with pica behavior could have had harmful non-cancer health effects from soil ingestion. Former residents may have a low increased cancer risk because of chronic exposure to arsenic in soil (estimated at 4.5 in a million for adults to 4.4 in one hundred thousand risk for children). Residents may have been exposed to volatile contaminants in indoor air, but no data are available to evaluate this possibility. One former resident was reportedly diagnosed with keratosis (actinic), which has been associated with chronic arsenic exposures in the scientific literature, and another resident with a reported benign brain tumor. Without data for all possible past exposures, particularly indoor air and dust and more medical information about the keratosis case, ATSDR is unable to speculate on any possible causal relationship between the former residents' reported health conditions and their potential exposures while residing at 4825 Glenbrook Road.
Basis for Conclusion 2 Former Residents	Residents could have been exposed to contaminants in surface soil. Estimated arsenic doses for children who exhibit pica behavior (persistent and compulsive ingestion of non-food items such as soil) exceeded doses for hyperpigmentation and hyperkeratosis. ATSDR does not know whether a child with pica behavior lived or played at the residence. However, for a child with pica behavior, estimated doses associated with exposures to the highest arsenic levels detected on the property would be in the range at which symptoms

characteristic of acute arsenic "poisoning" (e.g., facial swelling, nausea, vomiting, and diarrhea) have been reported. However, this would happen only if relatively large amounts of the most contaminated soil were ingested in a short amount of time.

Based on the arsenic levels in surface soil and a residential occupancy period of five years, we concluded that there was a low increased risk of developing cancer for adults and children.

Soil gas samples, collected 2007 to 2009, indicate potential for chronic exposure to low levels of chlorinated solvents in indoor air, but neither soil gas samples nor indoor air samples were taken while the residents occupied the home, so we cannot estimate exposures to contaminants in indoor air.

Next Steps

ATSDR will attempt to notify former workers and residents about their potential for past exposure to chemicals at this site and share this health consultation with them.

If former workers or former residents have experienced symptoms they feel are related to past potential environmental exposures at this property, their healthcare providers can contact ATSDR for further assistance. ATSDR's regional director, Lora Werner, can be contacted by phone at 215-814-3141 or by email at lkw9@cdc.gov.

Former workers and residents should continue their routine preventive cancer screenings and health check-ups and any additional screening recommended by their private medical professionals based on personal health issues.

ATSDR recommends that the property at 4825 Glenbrook Road not be used until appropriate remediation is completed and sampling data show it is safe for residential purposes.

Background and Statement of Issues

The United States Army Corps of Engineers (USACE) requested Agency for Toxic Substances and Disease Registry (ATSDR) assistance in 2010 after they found additional WWI-era munitions, glassware, debris, and soil contaminated with chemical warfare agents (CWAs) and other chemicals on the 4825 Glenbrook Road property. USACE asked ATSDR to evaluate exposures to construction and other workers who developed the property (1992-1993) and the family who lived at the residence (1994-1999). In many cases, ATSDR must rely on information and data from other agencies and organizations to perform our health evaluations. For this request from USACE, ATSDR environmental scientists and medical staff evaluated data and information provided from USACE including a review of transcripts of interviews with some of the former construction workers at the site. Appendix E presents detailed information on environmental investigations and data considered in this evaluation.

The 4825 Glenbrook Road property is within the Spring Valley Formerly Used Defense Site (FUDS) in northwest Washington, D.C. (Appendix A, Figure 1) in an area formerly known as the American University Experiment Station (AUES). At AUES, the U.S. Government researched and developed chemical warfare agents (CWAs) and protective masks, and trained soldiers during the World War I (WWI) era. The AUES was located on the grounds of the current American University (AU) and included additional properties in the vicinity.

The 4825 property is located between 4835 and 4801 Glenbrook Road. According to the U.S. Army Corp or Engineers (USACE), the properties of 4825, 4835, and part of 4801 Glenbrook Road were within the former AUES fence line and border AU (Appendix A, Figures 2 and 3). The home at 4825 Glenbrook Road was built in 1992 - 1993 and was purchased in 1994. The family moved out of the house in 1999 and it remained vacant. In 2001, AU regained ownership of the property. The upper structure of the house was demolished in 2012. In 2014, USACE continued to investigate and remove a debris field in the front yard. In 2015 and 2016, the USACE plans to remove the basement foundation and any contamination in accordance with USACE's Decision Document for 4825 Glenbrook Road [USACE 2012].

Three disposal pits (referred to as Burial Pits) were discovered south of the 4825 Glenbrook Road home in the late 1990s and early 2000s where laboratory items and chemicals were buried (Appendix A, Figure 3). Burial Pits 1 and 2 and part of Burial Pit 3 were next door at 4801 Glenbrook Road. The remainder of Burial Pit 3 was on the 4825 property. In all, many hundreds of pounds of glass fragments were found at 4825 Glenbrook Road [USACE 2011e]. The USACE plans further excavation at 4825 Glenbrook Road to remove any remaining AUES-related contamination and debris.

Historical Use of the Property and its Surroundings

The 4825 Glenbrook Road property is located in Baker Valley which includes the current AU athletic field area and other residential properties [USACE 1995]. The process for making lewisite (organoarsenical compound used as chemical warfare agent) was perfected at AUES [DC Gov 1996] and there was a documented release of lewisite in Baker Valley in the early 1900's [USACE 1995]. The eastern 4825 property boundary abuts AU near the current Watkins and Kreeger buildings, where numerous AUES buildings were located (Appendix A, Figure 2)

[USACE 1995]. According to the USACE, the 4825 property lies within the fenced area of the former AUES (Appendix A, Figures 2 and 3).

Figure 2 (Appendix A) shows multiple buildings on the former AUES that were located behind the 4835 property. Building #82 is believed to be the site of a February 16, 1918 explosion resulting from an experiment using a mixture of arsenic and magnesium [USACE 1995]. The AU archives also showed tests with arsine and magnesium arsenide and a release of 60 pounds of arsine [DC Gov 1996]. The three burial pits south of the 4825 Glenbrook Road home (Appendix A, Figure 3) are in the approximate location of a pit shown in old records [USACE 1995].

Former Worker and Resident Health Concerns

Former Worker Health Issues

The home and property at 4825 Glenbrook Road was under development and construction from about spring of 1992 through fall of 1993. In early May 1992 (first reported on May 7), during soil excavation activities at 4825 and 4835 Glenbrook Road, a rotten, acrid odor was detected coming from the soil. Later that month, on May 27, 1992, AU was notified that on-site construction workers were experiencing irritation to their eyes and faces when loading dirt into a dump truck [Apex 1996]. Several workers were overcome during excavation procedures and were taken to the emergency room because of respiratory problems [DC Gov 1996; Albright 2008].

After the initial excavation work for property development, construction crews were hired by the developer to build the homes at 4825 and 4835 Glenbrook Road. Anecdotal information about worker health concerns was gleaned from three transcripts from the November 6, 1993 interviews between three WV workers and a documentary filmmaker, Virginia Durrin that was provided to ATSDR by USACE [Durrin 1993]. Like the May 1992 property excavators, the WV workers also had odor complaints about the site and reported a few incidents of acute exposures from vapors released from broken bottles that caused more immediate irritation to the eyes, nose, and throat. Since the WV workers were on-site for about one year, they also reported longer term health complaints. These health concerns can be broadly grouped into skin concerns and nose and sinus irritation. More details of these and other health concerns are discussed in the Public Health Implications Section (Occupational Medical Officer Evaluation of Worker Exposures) below.

Former Resident Health Complaints

The residents of 4825 Glenbrook Road lived there for approximately five years (1994-1999). The family had young children [ATSDR 1999; Moeller 2005]. Health complaints from residents of 4825 Glenbrook Road and those in other areas of Spring Valley were recorded in 2002 by the Washington, D.C. Department of Health (DC DOH) [DC DOH hotline 2002; ATSDR 2005]. One resident of 4825 Glenbrook Road who reportedly occupied a room on the basement level, had been diagnosed with keratosis (which has been associated in scientific studies with chronic arsenic exposures). Another resident reportedly was diagnosed with a benign brain tumor [Current Newspapers 2004; USCOA, 2004]. ATSDR previously evaluated the DC hotline records for the Spring Valley site [ATSDR 2005]. Without data for all possible past exposures, particularly indoor air and dust and more medical information about the keratosis case, ATSDR

is unable to speculate on any possible causal relationship between the former residents' reported health conditions and their potential exposures while residing at 4825 Glenbrook Road.

Discussion

Site-Specific Evaluation Approach and Limitations

This health consultation focuses on contamination and exposures at 4825 Glenbrook Road. It includes a review of the WV construction workers health concerns by an ATSDR occupational medical officer based on the excerpts of the transcript information. Historical information was included because it was relevant to use of the property and past exposures. A description of ATSDR's standard evaluation process for our assessments is included in Appendix B and our methods for determining arsenic doses are presented in Appendix C along with additional specifics on potential health effects from arsenic exposure.

Most environmental samples for CWA analyses in this review were collected from 2007 through 2010 during the remedial investigation, 14 to 18 years after worker exposures during property development and 8 to 11 years after residential exposures. Generally, too much time had elapsed between potential exposures and environmental sample collection. This resulted in environmental data that are not representative of past exposure concentrations (except soil concentrations for arsenic which is a persistent breakdown product of lewisite and other arsenicals present at the site).

Some contaminated soil was removed from the property in May 1992 (near the beginning of property development). Additionally, truckloads of contaminated soil were reportedly removed from the property at the end of development so the remaining soil concentrations may not accurately represent exposure scenarios for workers. Also, the site is not fully characterized because removal of the house foundation has not yet occurred.

Additional data and information gaps affecting this evaluation include:

- No indoor air or dust samples were taken in this home while it was occupied, leaving exposure data gaps. There was potential for vapor intrusion of volatile contaminants into the home as numerous volatile organic compounds (VOCs) were detected in test pits near the home. However, no appropriate data on VOCs were available to quantify potential exposures during occupancy, which occurred in the 1990s.
- No ambient air samples were taken in the 1990s to quantify contamination in outdoor air at the time potential exposures were occurring.
- In 2010, twelve surface soil samples (taken at 6 inches below ground surface) were analyzed for sulfur mustard, lewisite, 1,4-dithiane, and 1,4-oxathiane. These CWAs were not detected in surface soil samples from the backyard. Although no samples were available for the rest of the yard, this is not likely an important data gap for residential exposures to soils as these CWAs are not likely to persist in surface soils due to their physical properties.

- The increased cancer risk estimate for workers is based on soil arsenic levels remaining on the property after it was developed and is the minimum increased cancer risk (as soil removed during development is likely to have reduced soil arsenic concentrations).

Except for arsenic, determining the extent of potential past exposure and potential health effects in workers and former residents is difficult without applicable data from the time period in which those exposures may have occurred. Therefore, the approach taken in this health consultation was to evaluate arsenic exposures for both workers and former residents (due to the persistent nature of this metal and that it is a breakdown product for several CWAs found at the site) and to rely on the occupational medical officer evaluation of the WV workers interview transcripts to further evaluate their exposures. For all other contaminants and pathways where exposure point data are not available to evaluate the public health implications of worker exposures using ATSDR standard evaluation approach, ATSDR discussed the data in relation to available health comparison values and we provide general health effects information but we could not conclude if exposure occurred or whether those health effects are likely or unlikely (Appendices E and F).

Site-Specific Exposure Pathways

ATSDR evaluates site conditions to determine if people could have been (a past scenario), are (a current scenario), or could be (a future scenario) exposed to site-related contaminants. When evaluating exposure pathways, ATSDR identifies whether exposure to contaminated media (soil, water, air, waste, or biota) has occurred, is occurring, or might occur through ingestion, dermal (skin) contact, or inhalation. ATSDR also identifies an exposure pathway as *completed* or *potential,* or *eliminates the pathway from further evaluation*. Completed exposure pathways exist if all elements of a human exposure pathway are present. A potential pathway is one in which one or more of the pathway elements cannot be definitely proved or disproved. A pathway is eliminated if at least one element is absent.

A summary of exposure pathways for this site is presented in Table D-1 (Appendix D). Below is a discussion of the exposure pathways for former workers and residents. For both former workers and residents, the exposure pathways are considered by activities and categorized as having a low, medium, or high potential for exposure. A table of past activities that would involve contact with soil can be found in Table D-2 (Appendix D). The groundwater pathway was eliminated because groundwater was not used as drinking water or for any other household purposes. The surface water and food pathways were also eliminated because there is no surface water on the property and there were no known gardens used for food consumption.

<u>Exposure Pathways for Former Workers</u>

Chemicals, including chemical warfare agents, in munitions or bottles were buried in soil in Spring Valley. During the development of the 4825 Glenbrook Road property, activities that disturbed the soil, such as excavation and regrading increased the chance of exposure to these chemicals.

Home construction occurs in several phases and involves multiple trades. Each phase presents a different likelihood for worker exposure to soil contaminants or material buried in the soil. Direct contact, inhalation of soil dust or vapors, and incidental soil ingestion (while eating or

smoking at the site) are the likely routes of exposure. Exposure duration and frequency also vary by the worker's task. Workers include skilled tradespeople, laborers, and management. Often the specialty work is performed by subcontractors who are present only to complete their specific tasks. For example, delivery men may be present for hours, foundation layers may be there for only a week or two, while carpenters may be on site for months. A backhoe operator will likely have less direct contact with soil than a laborer using a shovel.

No construction plans or schedules for 4825 Glenbrook Road were provided; discussion of work activities performed at the address assumed general residential building construction practices and also relied on recollections from the WV workers (interview transcripts from Durrin, 1993). The construction workers at this site generally worked 5 day, 40 hour work weeks. The WV workers indicated working 4 days per week for 10 to 12 hours per day, wearing work boots and nail aprons, and in warm weather, a couple of the WV workers occasionally took off their shirts while working. Wearing boots and aprons would decrease the chance for dermal exposure; not wearing shirts would increase contaminant exposures.

The first two phases of construction would pose the greater chance for exposure to contaminants in soil. In the first phase, site work involving clearing the lot of trees, shrubbery, rocks, and debris; stripping and stockpiling top soil; rough grading the lot; and excavating for the foundation would be performed. A backhoe and bulldozer would be used along with some manual labor. This phase may last about a week or two, depending on weather and the size of the lot. The next phase of home construction would involve laying the footings, installing underground utilities, and building or pouring the foundation; this phase could last a few weeks as well. The foundation is usually subject to an inspection to determine if it meets building codes.

The majority of site clearing work and foundation work at 4825 Glenbrook Road appeared to have been performed in May 1992. On May 2, 1992, workers reported a strong odor during soil excavation at the site; one soil sample was taken and analyzed for metals and pesticides and it was concluded that there were no hazardous substances present at the site [Apex 1996]. On May 27, 1992, several workers were taken to the hospital for eye irritation and respiratory problems they experienced while excavating the property [DC Gov 1996; ATSDR 1997a]. A sample was collected and analyzed for priority pollutants: volatile organic compounds (VOCs), semi-volatile organic compounds (SVOCs), metals, polychlorinated biphenyls (PCBs), pesticides and herbicides. The results showed that the VOCs methylene chloride (at 74 ppb) and toluene (at 2 ppb) were present as well as the herbicide Silvex (at 13 ppb) and some metals (below comparison levels with arsenic reported at less than 10 ppm). Based on the analysis, Environmental Management Services concluded that white granular layers observed throughout the excavation were Silvex [2-(2,4,5-trichlorohenoxyl)propionic acid or 2,4,5-TP], a banned herbicide [Apex 1996]. The WV workers reported 25 to 30 truckloads of soil and debris (one worker mentioned 50 to 75 truckloads) had been removed from site prior to them starting work there in late May or early June 1992 (Durrin, 1993).

Although EMS concluded that silvex was in the excavation when the workers were overcome, Silvex is unlikely to have been the odorous chemical released into the air which exposed the construction workers in May 1992; no analyses were done for CWAs at that time.

The herbicide Silvex (2,4,5-TP) is a white powder with little odor that strongly adsorbs to soils. This acid will not evaporate into air because of its very low vapor pressure. The average half-life for biodegradation of Silvex in soils ranges from 12 to 17 days [USEPA 2011].

During the intermediate phases of construction, workers would have less exposure to soil. These phases would include rough carpentry and framing of the floors, walls, and roof; installation of the heating and cooling system, plumbing lines, and electrical wiring in the walls, floors, and ceilings; installation of the roof and siding; and masonry. These activities occur over the course of several months, and there are usually building inspections after the completion of these activities. This rough work is followed by installation of insulation and drywall. Workers would be exposed to soil in the course of accessing supplies such as plywood, shingles, and drywall that had been unloaded on the property.

At the 4825 Glenbrook Road construction site, the WV workers described participating in many of the intermediate phase activities. They discussed doing carpentry and roofing. They stated that while they were paid as skilled laborers, their skills and job activities included plumbing, masonry, electrical work, and carpentry. The WV workers reported non-skilled laborers present on the site performing manual tasks including clean-up.

It appeared that the early phase activity of grading the property and building retaining walls at 4825 Glenbrook Road continued into the intermediate phases. Early on in their work, some of the WV workers assisted in soil compaction. This was done concurrently with framing activities. The latter took place when the machine operator, an employee of the developer, was away from the site. While compacting soil, the WV workers reported 7 or 8 instances when an uncovered bottle was broken, releasing a liquid and a strong smelling, irritating blue-green to black colored "smoke". In some areas, the soil was moved around, compacted, or used as backfill. The workers mentioned that while they were on-site, 15 to 20 loads of soil and trash were removed and replaced with clean fill to build up retaining walls. In other areas, the workers reported that the soil on the property remained untouched.

Final phases of construction include interior and exterior work. The interior work such as painting; detailing; finishing; and installing hard-surface flooring, cabinets, fixtures, and appliances would pose a low risk for exposure to soil. The exterior work involves final grading, landscaping, and installing the driveway and sidewalks and poses a greater risk for exposure to primarily surface soils. The final phases occur over a few months. A final building inspection is usually required before residents can occupy the home.

The former WV workers reported one construction worker who was there once a week to perform trim work. This worker was on site for only a couple of months. The WV workers were on-site for over a year: construction work was completed in the summer or fall of 1993. Residents moved into the home in 1994. In June 1996, landscapers at the adjacent property (4835 Glenbrook Road), complained of strong odors and eye and respiratory irritation after unearthing a broken bottle while planting a tree on the property [ATSDR 1997a,b]. Arsenic, volatile organic chemicals, and releasable sulfides were found in the soil [ATSDR 1997a,b]. After reviewing the sampling data, ATSDR concluded that no adverse health effects were expected from exposure to the soil. However, the discarded laboratory glassware could pose a health threat if unearthed [ATSDR 1997b].

During the course of the construction of 4825 Glenbrook Road, a foreman was present on the site. A trailer was located on the property as an office. The developer occasionally visited the work site and reportedly changed in and out of work shoes when visiting the property. Delivery

men (plywood, cement, etc.) spent limited time on the property. The WV workers reported that one cement delivery driver did not return to the site after his initial delivery reportedly because of burning eyes.

Thus, the pathways that had the greatest potential to cause harm to former workers who were potentially exposed to CWAs and other chemicals were:

- Direct contact with broken containerized waste in soil (waste released from a container)--***Potential***
- Dermal contact with and incidental ingestion of contaminated soil or dust--***Completed***
- Inhalation or skin/eye absorption of gases/vapors from release of chemicals during excavations and other soil disturbing activities—***Completed***.

Exposure Pathways for Former Residents

Although residents could have been exposed to chemicals in the pathways described for former workers, they are likely to have had no or less direct exposure to wastes and deeper contaminated soil. Former residents who lived at the property from 1994-1999 are most likely to have been exposed to contaminants through their contact with surface soil or possibly indoor air. Additionally, former residents would have been exposed to different levels of contaminants based on their activities, as compared to former workers. Residents likely had mostly low with some medium soil contact due to gardening or outdoor recreational activities.

Vapor intrusion refers to the transport of vapors from the subsurface into buildings (ATSDR 2008). Different building construction techniques and conditions may have varying impacts on the ability of vapors to enter indoor space. Basements may have more surface area through which vapors can move inside and may be closer to subsurface sources. Vapors can migrate from soil gas and enter below-grade basements through cracks in walls and floors. Basements usually have lower air pressures than the surrounding soil gas which drives the flow of vapors into residences.

The possible pathways to result in exposure to former residents were:

- Contact with and accidental ingestion of contaminated surface soil or dust (indoor and outdoor scenarios)--***Completed***.
- Inhalation or skin/eye absorption of gases/vapors from release of chemicals from soil to air—***Completed.***
- Contamination of indoor air by entrance of outdoor air contaminants (through openings such as garage door, windows, etc.)—***Completed.***
- Contamination of indoor air by vapor intrusion of contaminants in soil gas beneath/beside the house—***Completed.***

Summary of Findings from Investigations and Environmental Data

This section focuses on the environmental data (2000-2010) in completed exposure pathways; therefore, it covers findings in laboratory and munitions waste in soil (waste released from a container) and other soil samples since these were both completed exposure pathways for either

former workers and/or residents. More detailed information on the findings from various environmental investigations and environmental data can be found in Appendix E.

During property development, some of the bottles and other containers were broken and chemicals were released to the soil and/or air. No sampling occurred during these events. However, in 2010, during site investigations, hydrochloric acid (HCl) was detected in air near an arsenic trichloride discovery. The contents of intact containers are summarized in this section because they indicate which chemicals may have been released during property development and contributed to soil and air contamination. Many of the chemicals in intact containers were VOCs that could have contributed to vapors in soil gas and air. Similarly, CWAs in buried munitions or drums could have leaked their contents into soil and air.

Test pit (TP) investigations (excavating soil in an area with suspected contamination and debris) were used to determine the extent of contamination and to find munitions, glassware and debris. They were located in areas where there were inconclusive geophysical results and elevated arsenic concentrations in soil. The two TPs mentioned here are the high probability test pits in the front yard where USACE determined they were likely to uncover AUES-related items or debris.

This section also provides summary information on soil sampling for arsenic. Preliminary evaluation of the data determined that arsenic was a contaminant of concern at the site because arsenic was present at the site in both surface and sub-surface samples above ATSDR's health based comparison values. Arsenic is an element and does not undergo decomposition. Therefore, some of the limitations of evaluation based on time elapsed between potential exposures to workers and residents and sampling time for other chemicals are not a concern for arsenic. It is important to note that arsenic is a naturally occurring element widely distributed in the earth's crust in forms of inorganic and organic arsenic compounds. Inorganic arsenic compounds are mainly used to preserve wood. In the past, arsenic was also used for pigment in paint. Some inorganic arsenical compounds such as arsenic trichloride were used during WWI to make organic arsenicals such as lewisite. Organic arsenic compounds are primarily used as pesticides; some organic arsenicals were produced during WWI as CWAs. Arsenic is a persistent breakdown product of lewisite and other arsenicals. Please see Appendix C for more information on arsenic and ATSDR's evaluation of it. ATSDR assumed that all the arsenic was in the inorganic form.

Public health implications of soil arsenic and dose estimates are provided below.

Chemicals in Intact Containers

Sulfur mustard, lewisite and agent breakdown products (ABPs) were detected in intact containers uncovered in test pits (TP) 120 and 134 as well as in soil from these front yard test pits (43 intact containers were found in 2010 during these investigations). Most of the intact glass containers in these test pits contained multiple chemicals or CWAs (i.e., chemical mixtures); although most were chemical residues, there were some containers with more substantial liquid/solid content. Approximately 74% of the intact containers (32 of 43 containers) recovered from the property

contained CWAs or their ABPs. Approximately 63% (27 of 43 containers) of this glassware contained sulfur mustard or its ABPs, 19% (8 of 43 containers) contained chlorodiphenylarsine, and 14% (6 of 43 containers) contained lewisite. Hexachloroethane (HCE) and phosgene oxime (CX) were found in several containers. One jar contained arsenic trichloride.

At least three recovered intact containers had enough content to warrant concern for potential spills/releases (more than residues) such as a half-liter of sulfur mustard of unknown purity and bottle 7/8 full with clear liquid of carbon tetrachloride with phosgene oxime (a CWA) and 1,2,4-trichlorobenzene (a pesticide and solvent); both bottles were found in the front yard. A release of arsenic trichloride from an intact container occurred during USACE removals in March 2010. Specific information on some of these discoveries is provided in Appendix E.

Arsenic in Soil

This section presents and summarizes arsenic in soil from investigations that occurred in 2000 through 2010. Most data can be found in the Final Remedial Investigation Report for 4825 Glenbrook Road [USACE 2011d, Appendix E] as well as other sources. As a result of these investigations, thousands (3,000-4,000) of tons of arsenic-contaminated soil have been removed from the property. In all, approximately 43% of the 249 soil samples exceeded 20 parts per million (ppm) arsenic (the Spring Valley USACE arsenic action level). This value also exceeds ATSDR's recommended soil comparison value of 15 ppm for arsenic (chronic environmental media evaluation guide (EMEG) for a child). Therefore, arsenic was evaluated further in this health consultation. Table 1 below contains a summary of arsenic concentrations in soil.

In September 2000, soil samples were collected from 4825 Glenbrook Road to determine the lateral extent of arsenic in surface soils [USACE 2000]. A grid pattern and test pits were used to establish arsenic soil removal. USACE sampled soil on a 20-foot by 20-foot grid and collected soil at the center of each grid at a maximum depth of six inches. From December 2000 to March 2001, arsenic-contaminated soil from 24 grids at the 4825 Glenbrook Road property was excavated under a non-time critical removal action. Eighteen grids had soil arsenic levels exceeding the Spring Valley arsenic action level of 20 ppm and ranging from 21.6 to 620 ppm [USACE 2011d, Appendix E, Table E.1; USACE 2011f]. Sampling of arsenic at greater depths (2 and 4 feet) was also conducted. The overall results were arsenic ranging from 2.36 to 694 ppm (Table 1). Samples from the driveway area ranged from 2.46 to 520 ppm (5 of 6 samples exceeded 20 ppm). Contaminated soil was removed from the property in 2001 with additional soil being removed during the investigation of Burial Pit 3 and in 2009 [USACE 2011d, Appendix E, Table E.1].

A soil sampling investigation of the driveway area was conducted in 2007, concurrently with work on Burial Pit 3 [USACE 2009a, 2011c]. Arsenic soil samples were collected in June 2007 in driveway area; their concentrations ranged from approximately 0.33 to 68.6 ppm. In 2008 and 2009, soil samples were taken for arsenic in Burial Pit 3 and extensions as well as Trench 2

Table 1. Summary of Arsenic in Soil [surface, subsurface, and mixed]

Range of Arsenic in Soil for sampling event, (or ppm)	Location of Maximum Sample ID, Location	Number of Detections > 20 ppm/ Number of Samples Analyzed (%)	Comment (Depths are specified if that information was available)‡	Date data collected [Reference]
260-**2,500 D**	SW-4825GB-CWM-161. TP-134, Front yard.	15/15 (100%)	Composite samples of drummed soil from TPs 134 and 120, front yard.	2010 [USACE 2011d, Appendix E, Table E.11]
140 D-1,100 D	CWM-025 ESW-4825GB-074	14/14 (100%)	Composite samples of drummed soil from TP-138, back yard.	2010 [USACE 2011b]
1.1 J -601	SW-4825GB-FLOOR-(6.5). TP-138, Back yard	1/5 (20%)	Characterization samples of TP-138, backyard.	Dec. 2009 [USACE 2011d, Appendix E, Table E.10]
7.1- **4,280 D**	SW-4825GB-GRAB-01(TP-120). Front yard.	3/4 (75%)	TP-120 and Grab samples (GS11-13).	Apr. 2009/Mar. 2010 [USACE 2011d, Appendix I; Appendix E, Table E.8]
0.086 J – 3,250 J	SW-4825GB-(-70, 30)-T2-SW-S-4.5: Trench 2.	14/49 (29%)	Pre-confirmation & grab samples: Trench, BP3 and east ext. Depths 0.5-6 feet.	May-Aug. 2009 [USACE 2011d, Appendix E, Table E.7]
BDL of 1- 184	SW-BP3-EFL-(-10)	4/16 (25%)	BP3, east ext., East 2nd ext.	March/July 2008 [USACE 2011d, Appendix E, Table E.6]
18.2-298	SW-BP3-GS-03	2/4 (50%)	BP3 and east ext. grab samples	Jan./May 2008 [USACE 2011d, Appendix E, Table E.5]
0.33 JK - 68.6 K	4825GR-ABP5 (2'), Trench 2.	4/8 (50%)	Trench 2: Driveway area. Depths 0.5-4 feet.	June 2007 [USACE 2009a, Table B-3], [USACE 2011d, Appendix E, Table E.4]
2.36-**694***	Grid (-10, 110)@ 4'	25/75 (33%)	Depths 0-4 feet.	2000-2001 [USACE 2011d, Appendix E, Table E.1]
2.46-**620**†	Grid(-10, 70)@ 0.5'	20/44 (45%)	Depths 0-6 inches.	
2.46-520	Grid(-90, 10)@ 0.5'	5/6 (83%)	Driveway area, 0-6 inches.	

Qualifiers: D-Sample was diluted, J- Result is an estimated value, K-biased high--the actual value may be lower. BDL= below detection limit.

* The average concentration from this soil sampling was 56 ppm arsenic.

†The average concentration from this surface soil sampling was **73 ppm arsenic** (Used as an exposure point concentration for residents/gardeners).

‡ Samples from Test Pits (TPs) are mixed soil/composite samples (surface and subsurface).

Bolded values indicate use as exposure point concentrations in risk evaluation (Appendix C)

(sample depths up to about 6 feet) in the driveway area (Table 1). The highest arsenic concentration and the largest number of samples were taken in 2009 during the pre-confirmation and grab soil sampling of these areas. In 2009, arsenic concentrations ranged from approximately 0.086 to 3,250 ppm [USACE 2011d, Appendix E, Table E.7]. The maximum arsenic concentration was found in Trench 2 near the driveway area. Contaminated soil in the trench was removed [USACE 2011c]. The excavation of the 4825 Glenbrook Road portion of TP-23 (also called Burial Pit 3) started in October 2007 and was completed in March 2009 including extensions of Burial Pit 3 [USACE 2011d, Appendix R]. Approximately 20 tons of contaminated soil was removed from Burial Pit 3 and two East extensions of TP-23/Burial Pit 3 [USACE 2011c].

Between June and July 2009, approximately 750 tons of soil, containing arsenic exceeding the Spring Valley action level of 20 ppm, was removed from the driveway. In 2009, soil was excavated on the north side of the house to the northern property line. Arsenic grids were removed from the northern property boundary and excavated to 5 and 6 feet below ground surface (bgs). The grids closer to the home were excavated to 3 feet bgs.

In 2009 and 2010, soil was sampled for arsenic in the high probability test pits (TPs 120, 134, and 138). Arsenic was detected on the floor (depth of 6.5 feet) of TP-138 at 601 ppm in June 2009 [USACE 2011d, Appendix E, Table E.10]. None of the geotechnical borings from under the home exceeded 20 ppm for arsenic from the 2009 sampling. The maximum arsenic concentration of 4,280 ppm (a diluted sample and therefore likely underestimated) was found in grab sample GRAB-01 of TP-120 in the front yard near the steps [USACE 2011d, Appendix I & Appendix E, Table E.8]. This concentration is the highest on record for the Spring Valley area. Composite soil samples were taken from drummed soil which originally came from TPs 120 and 134 in the front yard (Table 1 below). Arsenic was analyzed in fifteen soil samples and found to range from 260 to approximately 2,500 ppm [USACE 2011d, Appendix E, Table E.11]. In 2010, composite soil samples were taken from drummed soil which originally came from TP-138 in the back yard. Arsenic was analyzed in fourteen soil samples and ranged from approximately 140 to 1,100 ppm [USACE 2011b]. Approximately 2,326 tons of arsenic-affected soil, from the high probability test pits, along with non-hazardous soil was disposed off-site at the King and Queen County Landfill as non-RCRA hazardous waste [USACE 2011c].

Public Health Implications

Approach

The effects of exposure to arsenic, CWAs, and other chemicals found at the site depend on the dose, duration, frequency of exposure, exposure route (ingestion, inhalation, or dermal contact), personal traits and habits, and concurrent exposure to other chemicals. At 4825 Glenbrook Road, as previously indicated, with the exception of soil arsenic concentrations, too much time had elapsed between potential exposures and environmental sample collection, resulting in environmental data that are not representative of past exposure conditions for former workers and residents. Thus, our evaluation for chemicals other than arsenic is primarily qualitative rather than quantitative.

Information on arsenic is provided below. This section is followed by the evaluation of the excerpts of the worker transcripts by an ATSDR occupational medical officer. Information on the general public health implications of specific chemicals or chemical groups (arsenic trichloride and hydrochloric acid vapors, sulfur mustard and lewisite, and VOCs and other chemicals) can be found in Appendix F.

Arsenic Exposure Dose Estimates and Health Implications for Former Workers and Residents

This section presents a summary of the findings of ATSDR's health effects assessment for potential ingestion to arsenic in soil; general public health implications of arsenic and methods used by ATDSR to calculate the exposure doses are presented in Appendix C. ATSDR estimated exposure doses for former workers who developed the property for about a 1.5 year timeframe and former residents who resided there for approximately 5 years

Tables C-1 and C-2, Appendix C, show the estimated exposure doses for ingestion of arsenic and comparison levels. As part of ATSDR's evaluation process, we first compared the calculated exposure doses to ATSDR's Minimal Risk Levels (MRLs). ATSDR has a provisional acute (up to two weeks) oral MRL of 0.005 mg/kg/day and a chronic (a year or more) oral MRL of 0.0003 mg/kg/day for arsenic. The MRL is an exposure level below which non-cancerous harmful effects are unlikely. The acute MRL is based on several transient (i.e., temporary) effects including nausea, vomiting, and diarrhea. The most sensitive endpoints related to chronic arsenic exposure are skin lesions. When an estimated acute exposure dose of arsenic is below 0.005 mg/kg/day, non-cancerous harmful effects are unlikely. It should be noted that:

1) The acute MRL is 10 times below the levels that are known to cause harmful effects in humans;
2) The acute MRL is based on people being exposed to arsenic dissolved in water instead of arsenic in soil – a fact that might influence how much arsenic can be absorbed once ingested; and
3) The MRL applies to non-cancerous effects only and is not used to determine whether people could develop cancer [ATSDR 2007a].

If an estimated exposure dose is above the MRL, ATSDR further evaluates the public health implication of the exposure dose by comparing them to effect levels from scientific studies (also see Tables C-1 and C-2, Appendix C).

Former Workers

To evaluate chronic exposures, ATSDR used the average soil arsenic concentration of 876 ppm (taken from subsurface soil removed from a test pit near the front of the home and placed in a storage drum; composite samples were taken from the drum) for chronic/sub-chronic exposure to workers. For acute exposures, ATSDR assumed exposure to the highest detected concentration (4,280 ppm) in a single spot (discrete sample) from test pits in the front yard for workers (Table 1).

ATSDR estimated doses for sub-chronic or chronic (an intermediate to long-term duration) and acute (short-term duration) exposures (Appendix C). Based on ATSDR's analysis, arsenic doses associated with possible soil ingestion exposures for workers are above the chronic ATSDR MRL (0.0003 mg/kg/day); however, except for workers with intensive soil contact, they are below doses shown in the scientific literature to cause harmful health effects. Former workers with intensive soil contact (ingesting 330 mg/kg or more of soil) could have been exposed to amounts approaching the threshold dose for hyperpigmentation and hyperkeratosis of the skin (see Table C-1, Appendix C and the ATSDR Occupational Medical Officer Evaluation of Workers Exposures section below for more discussion on these health outcomes).

Based on estimates for the ingestion of soil arsenic, the increased cancer risk to workers was low (estimated at about 2-4 additional cases per 100,000 exposed persons)--see Appendix C, Table C-3. The cancer risk to workers who worked less than 1.5 years in arsenic-contaminated soil would be less. The cancer risk to workers could have included many other carcinogens if they were exposed to chemicals in broken containers or spilled from containers, however, we cannot quantify these exposures or increased cancer risks (also see Occupational Medical Officer Evaluation of Workers Exposure below).

For workers with intensive soil contact, exposure doses were above the acute ATSDR MRL of 0.005 mg/kg/day; however, they were below levels shown to result in harmful effects (see Table C-2, Appendix C). Moreover, for workers with less intensive soil contact, exposure doses were below the ATSDR MRL; therefore, based on this evaluation, we do not expect harmful effects to workers from their acute exposures to arsenic.

Former Residents

Past resident's exposure doses to arsenic were calculated, as specified in Appendix C, using soil concentrations from the 1990s through 2010. ATSDR used the average surface soil (from a depth of 0-6 inches) arsenic concentration of 73 ppm to estimate chronic/sub-chronic exposure for residents and people gardening after property development. The average concentration of arsenic in grid samples, 0-4 feet for gardeners, was 56 ppm; since deeper soils lowered the concentration, we used the higher concentration of 73 ppm for gardeners as well. Additionally, ATSDR assumed acute exposure to 620 ppm, the maximum concentration in a grid sample taken from surface soil for residents and 694 ppm, the maximum concentration in a grid sample taken from subsurface soil at four feet for gardeners (Table 1). Exposure doses calculated from arsenic soil concentrations are presented in Appendix C: Tables C-1 and C-2 and are discussed below.

ATSDR does not know if former residents had gardens or worked in the soil at the residence. If they did work in the soil, they could have been or may be at risk of adverse health effects from their exposures. However, most of the higher arsenic concentrations (exceeding 600 ppm) were in the deeper subsurface soil where the residents should have had limited contact.

Ingestion or inhalation of contaminated soils or dusts was a plausible exposure route for children at 4825 Glenbrook Road because young children occupied the home in the 1990s. If the yard was well vegetated during their occupancy and/or they had minimal contact with the soil, then their exposures may have been insufficient to cause harm. However, arsenic in surface soil was wide-spread and at high enough concentrations (maximum of 620 ppm) to warrant concern over exposures, particularly to children. However, even if exposed to arsenic, the body can often eliminate it before damage occurs or, if damage does occur, the body is often able to repair itself

[ATSDR 2007a]. The maximum arsenic concentration of 4,280 ppm was found in subsurface soil and that soil is unlikely to be accessible by children. Many contaminants and items were buried deeper than surface soil and were unlikely to be available to children playing in the yard.

ATSDR also included exposure dose estimates for children with pica behavior, which is the tendency to ingest large quantities of soil and other non-food items. Children with pica behavior could conceivably consume a teaspoon or more of contaminated soil each day. ATSDR does not know whether a child with pica behavior lived or played at the residence.

ATSDR estimated doses for chronic or sub-chronic (an intermediate to long-term duration) and acute (short-term duration) exposures (Appendix C).

The most sensitive endpoints related to chronic arsenic exposure are skin lesions. For the former adult resident and gardener, acute and chronic dose estimates are lower than ATSDR's MRLs indicating that harmful effects are not expected (Appendix C, Tables C-1 and C-2). For children who do not exhibit pica behavior, exposure doses were below ATSDR's acute MRL but above ATSDR's chronic MRL. However, chronic doses for non-pica children were well below effect levels. For a child with pica behavior, however, the estimated dose of 0.089 mg/kg/day (Table C-2) is greater than ATSDR's acute MRL and the Lowest observed Adverse Effect Level (LOAEL) of 0.014 mg/kg/day. This estimated dose associated with exposures to the highest arsenic levels detected on the property would be in the range at which symptoms characteristic of acute arsenic "poisoning" (e.g., facial swelling, nausea, vomiting, and diarrhea) and an increased incidence of dermal lesions have been reported. These symptoms would only happen if relatively *large* amounts of the *most* contaminated soil were ingested in a short amount of time.

Based on ATSDR's analysis, arsenic doses associated with possible soil exposures for residents are below doses shown in the scientific literature to cause harmful health effects except for children who might ingest a large amount of soil (exhibiting pica behavior). Estimated exposure doses for children with pica behavior exceeded doses for hyperpigmentation and keratosis of the skin. It is unknown whether children exhibiting pica behavior resided at this home and even if so, whether they were exposed to contaminated soil.

Although the mechanism of arsenic carcinogenicity is not known, arsenic is believed to function mainly as a cancer promoter or co-carcinogen [ATSDR 2007a]. Therefore, evaluating human cancer risks from inhalation or oral exposure needs improvement and may not adequately characterize these risks. ATSDR estimated the theoretical increased cancer risk for residents from 4 to 5 years of potential exposure (period of occupancy) to the average detected concentration of arsenic in surface soil samples (73 ppm). Using this soil arsenic concentration and conservative assumptions for soil ingestion, the estimated increased cancer risk (upper percentile) was approximately 4.4×10^{-5} (4.4 in 100,000) for children and 4.5×10^{-6} (4.5 in 1,000,000) for adults. Based on a residential occupancy period of approximately 5 years and arsenic in surface soil, their cancer risk was estimated as a low increased risk for children and low increased risk for adults. The estimated increased cancer risk to former residents has been qualitatively described in Table C-3 in Appendix C.

Occupational Medical Officer Evaluation of Worker Exposures

This section provides an occupational medical perspective on non-clinical or medical documents. The material reviewed for this evaluation primarily include the preceding sections of this health

consultation that discuss site history and environmental sampling, previous ATSDR health consultations [ATSDR 1997a,b], the Government of the District of Columbia's "Final Report on World War I Poison Gas Production at the American University Experiment Station" [DC Gov 1996], and three transcripts from November 6, 1993 of worker interviews with a documentary filmmaker, Virginia Durrin, that were provided by the USACE [Durrin 1993]. (As in previous sections, references to the transcript anecdotal information provided by three of four construction workers who commuted from West Virginia are referred to as WV workers). Appendix F provides information on the general public health implications of important contaminants detected in several media at the 4825 Glenbrook Road site.

The WV worker transcripts included descriptions of health effects that four WV workers experienced while employed at the site and immediately afterward. The interviewer was not a health care provider. No formal review of systems was asked; presumably, the workers commented only on health issues that were more obvious or concerning to them. However, the absence or presence of various signs and symptoms and the chronology of onset are important in constructing a differential diagnosis on possible chemical exposures and in inferring the duration, frequency, and dose received; specific signs and symptoms would be expected with certain chemical exposures. It should be noted that without a formal review of systems administered by a health care provider, some pertinent positive or negative findings may not have been mentioned by the workers.

The predominant symptoms reported by workers were either skin related issues or involved mucous membrane and eye irritation. The WV workers interviewed, who were present at the site for possibly 15 months, all reported itchy, scaly skin rashes.

The WV worker, who reportedly was the first to experience a rash, also had the most severe rashes that were still present at the time of the interview. His skin was described as being dry and scaly and having "red places". During the interview, he showed a healing blister on his arm. He stated that when the blisters first appeared they felt "hot" and when the blisters healed they left scars and a scaly skin area. A relative of this worker recalled one incident seeing the worker with watery blisters on his legs similar to blisters resulting from scalding. He reported peeling of his toes, ankles, and lower limbs. He discussed brown spots on his arms, hands, and feet. He stated the brown spots first appeared as if he had been "touched by an ink pen", but then they increased in size. He reported that exposed areas were the primary site for rashes, which he attributed to areas with sweat and dirt.

Another WV worker reported dry skin as his first symptom and that his ankles were affected first. He stated his ankles itched for a period of two weeks. Later, his knees also itched but this had cleared up. He described secondary lesions and scabs from scratching. He reported dry and scaly arms and hands. His wife reported that he had rough skin that felt "weird like goose". He mentioned that his skin problems started after he returned to West Virginia. He also reported his back was covered with small light brown spots. He pointed out that some brown spots on his hand were next to a blister. He mentioned having had callouses on his hands, water blisters the size of a cigarette burn, and "warm spots". He reported being "broken out" in skin areas where he wore his nail apron and on his back side. At the time of the interview, he reportedly had a new red lesion on his nose and one red lesion at his temple.

The third WV worker interviewed reported itching from the top of his head down to his feet. He reported having the top of his feet turn red and itch. He also reported a scaly, itchy rash on his thigh. He described scratching his skin until it bled. He reported "breaking out" from beltline and below. At the time of the interview he stated that the only thing bothering him was intermittent itchiness including around his navel, his chest, thigh, and feet. He also reported having had peeling skin.

The WV workers reported frequent rhinitis (inflamed lining inside the nose). They stated that there was a constant strong odor coming from the soil that gave them a burning sensation in their sinuses and eyes; the smell was described as a "hot smell", "orange-peel", "Vick's vapor (rub)", and something "you'd put on your skin to take the stiffness out." There was no mention of a garlic, horseradish, mustard, or geranium smelling odor. One worker stated he avoided rubbing his eyes to prevent any burning sensation. The acute exposure incidents involving broken unearthed bottles also resulted in complaints of watery eyes, rhinitis, and sinus congestion.

In addition to the previously mentioned skin complaints, for some of the WV workers, rhinitis was reported to be present at the time of their interviews (based on transcripts, inferred to be about 2 to 4 months after leaving the job site). Two reported the onset of snoring after working at the site. One worker, who had a case of pneumonia in the winter of 1992/93, reportedly had continued intermittent hoarseness and bronchitis with accompanying chest soreness. There were no eye complaints mentioned at the time of the interview.

Fatigue was mentioned by the WV workers in the context of working 12 hour days and driving 4 hours home at the end of the four day work week. One of the WV workers reported nausea and vomiting each morning he worked at the construction site. One reported difficulty in concentration and memory at the time of the interview.

The WV workers did not mention weight loss or gain, loss of appetite, weakness, numbness, tremors, difficulty with coordination or balance, conjunctivitis, blurred eyes, vision changes, photophobia (sensitivity to light), swollen eyelids, itchy eyes, nose bleeds, change in the sense of smell, nasal polyps, sneezing, itchy nose, change in the sense of taste, garlic taste, diarrhea, fingernail changes, painful blisters, excessive hair growth at inappropriate locations, or dark urine. Facial, axilla (armpit), perineal (area between the anus and genital organs), or eyelid irritation was not mentioned. There were no cardiovascular, reproductive, renal, or blood-related symptoms reported.

The WV workers reported only a few instances of missing work and/or seeking medical care. These instances involved one worker with a case of pneumonia in the winter of 1992/93 and another worker with a sprained ankle in December 1992. The worker with the sprained ankle received worker's compensation during his absence from work. One WV worker reported visiting his doctor about his dry skin. For the WV workers, there was no mention of visiting an urgent care center or emergency room for any other symptoms or exposures from the site.

Based on workers reported symptoms and the determination of the presence of multiple chemicals buried on the property, workers appear to have been exposed to some unspecified chemical or chemicals during the construction of 4825 Glenbrook Road that resulted in some health effects. Based on environmental sampling results described in this health consultation,

general categories of contaminants found either in the soil or in laboratory equipment include various acids, arsenic containing compounds, sulfur mustard and its degradation products, and lewisite.

The varying degrees of health effects can be due to different exposures (for example, from different job duties, duration of time spent at the property, presence at the time of a bottle being broken, etc.), different susceptibilities (prior medical history and family medical history is unknown), smoking history (at least two WV workers were smokers), and individual variation. Since the work was performed prior to completion of landscaping the properties, workers would be exposed to soil contaminants by direct contact, dust, and incidental ingestion while smoking or eating on site. Workers would also be exposed to any vapors coming from the property.

Health effects with respect to chemicals found at the site
Workers appear to have had some acute and sub-chronic (medium duration) exposures to site-related contaminants. The predominant symptoms reported were either skin related or involved mucous membrane and eye irritation. There were several acute incidents related to the breaking of bottles on the property that contained an irritating chemical. Symptoms from these acute exposure incidents did not persist. No emergency care was sought for these symptoms. Some symptoms, such as rhinitis, point toward a more sub-chronic exposure to lower concentrations of chemicals. With the exception of the acute pneumonia and sprained ankle, workers reported to work daily for their 12 hour shifts. This suggests less severe, non-debilitating symptoms.

From the worker reports, there appears to have been an irritating odor that was constantly present at the site. The WV workers described something more similar to either a citrus-like or a menthol or camphor smell, while in the May 1992 excavation incident, the excavation workers reported the odor as acrid. These descriptions suggest an irritant. Arsenic trichloride, which was found in the soil, has an acrid odor. Both hydrochloric acid and sulfuric acids have pungent odors. Sulfur mustard is an irritant; its odor is described as smelling like garlic, horseradish, or mustard. Individuals sometimes report a garlic-like taste in their mouth, as well. Lewisite, a blistering agent, has the odor of geraniums.

Many of the symptoms described by the WV workers (for example eye, respiratory, and skin irritation) are suggestive of exposure to chemical irritants. Unless there is a high concentration exposure that resulted in severe health effects, removal from exposure generally resolves irritant symptoms without after effects. For example, the WV workers did not report continued eye irritation or vision problems after the broken bottle incidents. Water soluble chemicals, such as acid aerosols, irritate mucous membranes on contact leading to tearing, runny nose, and a burning throat. Less water soluble chemicals typically have a more delayed response and result in lower respiratory tract injuries.

Exposure for several months to low level concentrations of irritants can result in mucosal inflammation and the development of rhinosinusitis (inflammation of the nose and sinuses). Symptoms reported by the workers suggest that they may have had occupational rhinosinusitis while working at the site and also present at the time of the interview in 1993. The absence of sneezing or itchy nose points away from an allergic response. Acid aerosols are water soluble irritants that impact the upper airways. Arsenic dusts cause irritation of the nose and throat which can lead to rhinitis, laryngitis, and bronchitis [ATSDR 2007A]. Early symptoms of sulfur

mustard exposure following a symptom-free period are irritation of the nose and throat resulting in runny nose, loss of smell and taste, sneezing, cough, and hoarseness [Kehe 2005]. In occupational studies of workers in sulfur mustard plants, chronic low-level sulfur mustard exposure resulted in similar symptoms [Perrotta 1996]. Lewisite causes immediate nasal and upper airway irritation including a burning sensation, nose bleed, and sore throat. Symptoms of rhinosinusitis would be expected to improve unless additional irritants, such as cigarette smoke, are present. Removal from exposure and symptomatic treatment are the mainstay of treatment for chemical exposures.

Exposure to irritating vapors, fumes, or dusts could lead to eye irritation. This symptom, described as a burning sensation, was reported by one worker, especially after having rubbed his eyes. These eye symptoms were limited and appear to have been resolved when removed from exposure. Immediate tearing of the eyes was reported only in connection to the bottle breakage events. This would suggest a water soluble chemical. Acidic chemicals can cause an immediate burning sensation of the eyes and tearing. Itchy, watery eyes are associated with allergens. Arsenic dusts can cause a chemical conjunctivitis (inflammation on the inside of the eyelid), which is usually accompanied by facial skin rashes. The eyes are one of the most sensitive organs to sulfur mustard exposure. After a delay of several hours, pain, tearing, conjunctivitis, and photophobia are reported even with low level exposures [Kehe 2005]. Even at very low doses, lewisite causes fairly immediate eye pain, followed by conjunctivitis and swelling of the eyelids. During the broken bottle events and while performing other work on the site, workers did not report conjunctivitis, blurred eyes, vision changes, photophobia, itchy eyes, or swollen eyelids. Vision problems were not mentioned to be present at the time of the WV worker interviews.

Dermatological symptoms appeared to have two patterns. One was more focal and appeared to be related to direct contact irritation. Ankles and the top of the feet were often the first areas affected. These areas were described as pruritic (itchy). Scratching seems to have exacerbated the rashes. Workers also reported “breaking out” along the belt line and nail apron straps. These symptoms seem consistent with an irritant contact dermatitis. One possible explanation is that dirt containing some irritant chemicals may have collected in shoes or work boots or along belt/strap lines. The “breaking out” may also be related to friction and rubbing or blocked follicles. One worker’s recollection suggested that the soil was not immediately irritating, “I love to get my hands all muddy and patted (the building developer) on the back of that white shirt.” A second pattern reported by the workers was a generally dry, scaly, itchy dermatitis covering larger areas of the body. Mild hyperkeratosis (overgrowth of the outer layer of the skin) sometimes appears as rough, scaly skin. The workers reported scratching these areas often until they bled. Workers also occasionally had some red patches distinct from excoriations (eroded skin areas caused by scratching or rubbing). Some of the workers commented on peeling skin. Because of thinner skin, some areas of itchiness reported that are consistent with irritant dermatitis include the back of the hands and the inner thighs. Airborne chemical irritants typically affect more exposed areas such as the face, hands, and arms; occasionally, some workers did not wear shirts, so both their chests and backs would be exposed. These exposed areas would also be impacted by any photosensitivity responses to a chemical. Of note, no facial irritation was reported in the WV workers. Airborne deposition of chemically contaminated soil would also more typically affect exposed body parts, and would adhere to wet, sweaty skin.

Irritant vapors that can penetrate thin clothing could affect non-exposed areas as well. Areas of the body with more moisture, such as the axilla and perineum, may be more severely affected.

A general pattern for irritant dermatitis is erythema (redness) with some swelling followed by vesicle or blister formation, erosion of the vesicle, crusting, and then scaling. Itching or burning sensations are associated with this reaction. Phototoxic reactions are similar and appear as an exaggeration of the sun-burn response. Repeated exposures result in scaling, and prolonged rubbing and scratching leads to thickening of the skin. Typically in occupational settings the chemical irritants are acids, alkalis, solvents, or detergents. Unlike allergic irritants which are dependent on an individual's immune response, these irritant chemicals are a non-specific response. At this site, repeated exposure to various acid aerosols could have resulted in some of the skin symptoms reported. The treatment for irritant dermatitis is removal from the causative agent.

In itself, dry skin is associated with itching. There are some systemic diseases, including some of the liver and kidneys, that cause generalized pruritis (itchiness), but these would be less likely considering three workers are involved. Generalized pruritis has been reported with arsenic. Hyperkeratosis is a common skin manifestation of arsenic exposure. An exfoliative (scaling and shedding) dermatitis, small blisters, and peeling of the skin on the fingers and toes were noted in some acute arsenic poisonings [Uede 2003]. At a threshold dose for effects, sulfur mustard causes itching and erythema 4 to 8 hours after exposure. The erythema caused by sulfur mustard exposure is similar to sunburn or first or second degree burns, and almost all exposed patients develop skin burns [Davis 2001]. A latent effect (i.e., months after recovery of the acute injury) in people with previous substantial exposures to sulfur mustard is dry skin.

The presence of small brown spots, or pigmented macules, was noted by two of the WV workers. As explained by the transcriptionist, in the Durrin film, a close up of some of the lesions was shown. The distribution of the lesions appeared to be on the back, trunk, and extremities. Hyperpigmentation is sometimes seen in chronic irritant dermatitis. Pigmented lesions are associated with sun exposure. Hyperpigmentation is one of the early non-malignant health effects associated with longer-lasting arsenic exposure. There can be a lag of development by six to eight weeks with arsenic exposure. Both hypopigmented and hyperpigmented skin changes are a late effect after sulfur mustard lesions. Pigmentary changes in covered areas of skin have been reported in sulfur mustard munition workers after longer-term moderate exposures [Perrotta 1996].

Formation of blisters that were water-filled and "hot" or "warm", but not described as painful were reported by two WV workers. One of these workers also reported onset of a new blister occurring several weeks before the interview but weeks after leaving the site. One blister was described as being the size of a cigarette burn. As stated previously, vesicle and blister formation occurs with irritant contact dermatitis. Other conditions which result in vesicles and blisters are allergic contact dermatitis, acute high exposure arsenic intoxication, and acquired porphyria cutanea tarda (PCT) (a disorder marked by abnormal blood pigment production). The latter two conditions can both cause hyperpigmentation and elevated urinary uroporphyrin excretion. In susceptible individuals, acquired PCT can be triggered by alcohol, smoking, and sunlight. In PCT, the fluid filled vesicles and blisters are pruritic but not painful and occur on sun-exposed areas; individuals often have increased facial hair and thickened chest and back skin [Fitzpatrick

2000]. No laboratory testing results were provided for any of the workers and no worker reported having reddish-brown urine. Pseudoporphyria, a phototoxic reaction which mimics PCT, presents with skin fragility, vesicles and bullae of sun-exposed skin, especially the dorsal hands, chest and neck without the excess hair growth, thickened skin changes, and uroporphyrin excretion. While pseudoporphyria has been associated with various medications and excessive sun exposure, there are no reports of any links with arsenic exposure [Stein 2007]. Both Lewisite and sulfur mustard are vesicants (blister producers), but their onset is either immediate or within the first few days of exposure; they are reportedly very painful.

The daily morning nausea and vomiting described by one WV worker may more likely be related to a response to the odors at the site, as opposed to suggesting a more high level exposure to some toxic chemical or a vomiting agent. The lack of other severe symptoms, such as diarrhea and abdominal pain, and the periodic nature of the symptoms, favors the former interpretation. These gastrointestinal symptoms did not persist after leaving the work site.

The non-specific and more subjective symptoms reported by one worker of difficulty concentrating, poorer memory, and daytime sleepiness are less easily attributable to any chemical exposures, especially in light of there being no neurological symptoms such as peripheral neuropathy (damage to nerves outside of the brain, such as muscle weakness or tingling and burning sensations) reported. One possible explanation is that these symptoms do not have a neurological origin, but may result from poor sleep. Since new onset snoring is reported, these symptoms may be indirectly related to the site secondary to the sinusitis.

Chemicals found at the site with respect to health effects
Some acidic chemicals that are associated with skin, eye, and upper respiratory irritation were found at the site. When exposed to moisture, arsenic trichloride converts to hydrochloric acid. Similarly, chemical reactions with water and sulfur mustard leads to the formation of thiodiglycol and hydrochloric acid. Hydrochloric acid was detected at the site. Releasable sulfides were found in the soil samples taken after the landscaper incident in 1996 [ATSDR 1997a,b]. Both hydrochloric acid and sulfuric acids have pungent odors. Response to exposure to these acids is fairly immediate, with signs and symptoms related to the concentration and duration of exposure. The symptoms of the WV workers are consistent with low to moderate exposures. The rhinosinusitis symptoms reported suggests a low-level longer term exposure. With either higher concentrations or duration of exposure, there would be lower respiratory injury, more severe eye burns, and more severe skin responses. Resolution of symptoms would be expected in low or moderate exposures once an individual is removed from exposure.

Arsenic trichloride, arsenic containing lewisite degradation products, arsine, and arsenic were present at the site. As mentioned previously, arsenic trichloride has an acrid smell and can be an irritant when combined with any moisture. Some of these arsenic compounds are readily absorbed into the body and could result in arsenism. Dermatologic findings of hyperpigmentation and hyperkeratosis are common with chronic arsenic exposures and are one of the earliest non-malignant health effects. Melanosis (excessive production of dark skin pigment) has been found in chronic drinking water cases at doses below the minimum contaminant level [Yoshida 2004]. A diffuse or spotty melanosis of the trunk and limbs and mild thickening of the palms and soles would be indicative of a mild exposure. Melanosis typically appears before hyperkeratosis. The latter might be associated with a grit-like sensation or scaly

skin. An exfoliative dermatitis can be seen with arsenic poisoning along with small blisters. The presence of Mee's lines (white bands on the fingernails) is noted in acute poisonings and in chronic exposure. More wart -like lesions and buccal (inside the cheek) melanosis would indicate a more severe arsenic exposure. Smoking and sun exposure have been found to increase the risk of skin lesions [Chen 2009]. Some symptoms of some moderate or high level arsenic exposures, such as anemia and peripheral neuropathy, were not reported.

Given that other health findings associated with higher dose arsenic exposures (such as anemia and peripheral neuropathy) were not reported, the worker exposure was more likely a lower dose arsenic exposure received during the course of their employment. Skin manifestations from arsenic exposure may be chronic even after removal from exposure, although with low dose arsenic exposures, clearance of symptoms or decrease of pigmentation are expected over time. Low and moderate doses of arsenic from drinking water is associated with increased blood pressure. Other risk factors for hypertension would influence this health outcome. Arsenic is a known carcinogen and exposure increases the risk for skin, lung, and bladder cancer [ATSDR 2007A].

Although sulfur mustard and its degradation products were reported at low levels in some soil samples and in laboratory equipment, it is less likely that the workers were exposed to sulfur mustard by inhalation. The minor mucosal symptoms and lack of eye involvement point away from exposure to this chemical. No garlic, onion, or mustard odor was reported. The eye is very sensitive to sulfur mustard, yet the only eye symptom reported was tearing, and this was an immediate and not a delayed effect of exposure to the broken bottles. No photophobia or eyelid swelling was reported. A review of long term health effects with subclinical exposures to sulfur mustard concluded that long term eye damage would not occur in the absence of an initial injury [Perrotta 1996].

The respiratory system is also a prime target organ of sulfur mustard exposure. While irritation of the nose and sinuses was reported by the WV workers, in occupational studies at sulfur mustard factories, workers developed sore throats and hoarseness, lost their sense of taste and smell, and became hypersensitive to fumes and dusts [Hurst 2001].

Sulfur mustard is a strong irritant and blistering agent. It was used as an incapacitating agent because exposures required hospitalizations for extended periods of time for treatment of mustard burns or respiratory distress. Breakdown products of sulfur mustard such as thiodiglycol, 1,4-dithiane, and 1,4-oxathiane have a much lower toxicity [Munro 1999]. While mustard agents may have a delayed response of a few days, especially with lower dose exposure, they would not result in the continued appearance of blisters that the workers reported months after exposure had ceased. Blistering of other sensitive areas from vapor exposure, such as the axilla, scrotum, and anal region, was not reported by the workers. Of note, no pain was associated with the blisters. A relative reported that one worker returned home one week with a scalded looking leg with water blisters. Of the various reports of skin problems by the WV workers, this one incident more closely resembles a direct contact with sulfur mustard or its degradation products, however, other chemicals can result in burns and subsequent blister formation. Hyperpigmentation was found to be a late skin effect from Iraqi battlefield exposures [Balali-Mood 2008].

Sulfur mustard is a known human carcinogen. A review found that for exposed occupational workers and soldiers, there was an increased risk of respiratory and skin cancers related to moderate to high exposures [Perrotta 1996]. A 50-year follow-up mortality study of WWII navy veterans who participated in sulfur mustard gas chamber tests and who experienced skin reactions such as erythema, vesicles, and ulceration found no increased risk of cause specific mortality, including cancer [Bullman 2000].

Lewisite, a vesicant, was ruled out as a cause of the chemical exposures to the workers at the site. The odor ascribed to lewisite is that of geraniums. Even at low concentrations, lewisite is immediately highly irritating to the skin, eye, and other mucous membranes. Exposure would result in rapid onset of vesicle or blister formation and burning pain [Karalliedde 2000]. This description is inconsistent with the symptoms reported by the workers.

Conclusions

Former Workers Who Developed the Property

Some construction workers likely experienced harmful short-term (or acute) health effects while working at the property because of exposure to arsenic, CWAs (such as lewisite and sulfur mustard), and other chemicals. Workers, especially those who worked on the property for a year or more, may have an increased cancer risk; however, ATSDR cannot quantify their total increased cancer risk with any certainty. Workers who had intensive soil contact and repeated exposures are more likely to have experienced harmful health effects. Except for arsenic, no exposure information is available to determine if workers could also have long-term non-cancer health effects.

In May 1992, several construction workers were reportedly overcome during soil excavation activities at 4825 Glenbrook Road and required emergency care for respiratory problems. Transcripts of interviews with workers indicate exposure to chemicals during home construction in 1992 and 1993. Based on an occupational medical officer's evaluation of these transcripts, exposure to arsenic, arsenicals, irritants, and possibly sulfur mustard was likely. Irritation of mucous membranes and skin effects were associated with these exposures.

Because of the lack of sampling during worker exposures, most exposure parameters (concentrations, frequency, duration, etc.) are unknown. Site workers with intensive soil contact for a year or longer (ingesting 330 milligram/kilogram (mg/kg) or more) could have been exposed to arsenic at levels that cause hyperpigmentation and hyperkeratosis of the skin. Based on estimates for the ingestion of soil arsenic, the increased cancer risk to workers was low (estimated at about 2-4 additional cases per 100,000 exposed persons). However, this cancer risk estimate is based on soil arsenic levels remaining on the property after it was developed and is the minimum increased cancer risk (as soil removed during development is likely to have reduced soil arsenic concentrations). Although workers may have an increased cancer risk because of their chronic exposure to arsenic and sulfur mustard in soil, we cannot estimate their total increased cancer risk because of the uncertainty associated with the exposures parameters. Because workers could have been exposed to subsurface soil and chemical releases from broken containers, they could also have been exposed to pesticides, herbicides, and other chemicals on

the property. However, the lack of sampling for most worker exposures prevents ATSDR from quantifying the health implications of exposures to these chemicals.

Former Residents of the Property

Except for arsenic in soil, insufficient past exposure information is available to determine if former residents of 4825 Glenbrook Road had (or have) harmful health effects related to exposures. Dose estimates indicate that children with pica behavior could have had harmful non-cancer health effects from soil ingestion. Former residents may have a low increased cancer risk because of chronic exposure to arsenic in soil (estimated at 4.5 in a million for adults to 4.4 in one hundred thousand risk for children). Residents may have been exposed to volatile contaminants in indoor air, but no data are available to evaluate this possibility. Without data for all possible past exposures, particularly indoor air and dust and more medical information about the keratosis case, ATSDR is unable to speculate on any possible causal relationship between the former residents' reported health conditions and their potential exposures while residing at 4825 Glenbrook Road.

One former resident was reportedly diagnosed with keratosis (actinic), which has been associated with chronic arsenic exposures in the scientific literature, and another resident with a reported benign brain tumor. Without data for all possible past exposures, particularly indoor air and dust and more medical information about the keratosis case, ATSDR is unable to speculate on any possible causal relationship between the former residents' reported health conditions and their potential exposures while residing at 4825 Glenbrook Road.

Residents could have been exposed to contaminants in surface soil. Estimated arsenic doses for children who exhibit pica behavior (persistent and compulsive ingestion of non-food items such as soil) exceeded doses for hyperpigmentation and hyperkeratosis. ATSDR does not know whether a child with pica behavior lived or played at the residence. However, for a child with pica behavior, estimated doses associated with exposures to the highest arsenic levels detected on the property would be in the range at which symptoms characteristic of acute arsenic "poisoning" (e.g., facial swelling, nausea, vomiting, and diarrhea) have been reported. However, this would happen only if relatively large amounts of the most contaminated soil were ingested in a short amount of time.

Based on the arsenic levels in surface soil and a residential occupancy period of five years, we concluded that there was a low increased risk of developing cancer for adults and children. Soil gas samples, collected 2007 to 2009, indicate potential for chronic exposure to low levels of chlorinated solvents in indoor air, but neither soil gas samples nor indoor air samples were taken while the residents occupied the home, so we cannot estimate exposures to contaminants in indoor air.

Recommendations

ATSDR will attempt to notify former workers and residents about their potential for past exposure to chemicals at this site and share this health consultation with them.

If former workers or former residents are experiencing symptoms they believe are related to potential environmental exposures at this property, ATSDR recommends that they contact their

health care provider. Their healthcare providers can contact ATSDR for further assistance. Former workers and residents should continue their routine preventive cancer screenings and health check-ups, and any additional screening recommended by their private medical professionals based on other personal health issues.

ATSDR recommends that the property at 4825 Glenbrook Road not be used until remediation is complete and appropriate sampling data show it is safe for residential purposes.

Public Health Action Plan

ATSDR physicians will speak with health care providers for former residents and former workers, if desired. To get in touch with an ATSDR physician, first contact ATSDR's regional representative, Lora Werner, at 215-814-3141 or by email at lkw9@cdc.gov .

USACE plans to complete the Comprehensive, Environmental, Response, Compensation, and Liability Act (CERCLA) process of the Spring Valley FUDS.

ATSDR will review additional data as they become available to determine if new data and information may affect the conclusions and recommendations of this health consultation. If necessary, a new report may be written.

Authors and Reviewers

Authors

Laura Frazier, MS
Geologist, Environmental Health Scientist
Eastern Branch
Division of Community Health Investigations (DCHI)
Agency for Toxic Substances and Disease Registry (ATSDR)
Atlanta, Georgia

Michelle Watters, MD, PhD, MPH
Division Medical Officer, Office of the Director
DCHI, ATSDR

Reviewers

Gregory Ulirsch, MS, PhD
Environmental Health Scientist
Associate Director for Science
Eastern Branch
DCHI, ATSDR

Lisa Hayes, MS
Team Lead for the Eastern Branch
Environmental Engineer
Eastern Branch
DCHI, ATSDR

References

Albright 2008. Richard D. Albright. Cleanup of Chemical and Explosive Munitions: Locating, Identifying Contaminants, and Planning for Environmental Remediation of Land and Sea Military Ranges and Ordnance Dumpsites. Publisher: William Andrew Inc. 325 pp.

Apex 1996. Apex Environmental, Inc. Final Report conducted on President's Residence, 4835 Glenbrook Road, Washington, D.C. 20016. Apex Project No. 153.09. Prepared for American University, 4400 Massachusetts Avenue, Washington, D.C. August 6, 1996.

ATSDR 1997a. Agency for Toxic Substances and Disease Registry. Health Consultation. Spring Valley/American University Experiment Station, Washington, District of Columbia. U.S. Department of Health and Human Services, Atlanta, Georgia, June 3, 1997. http://www.atsdr.cdc.gov/sites/springvalley/consult_060397.html

ATSDR 1997b. Agency for Toxic Substances and Disease Registry. Public Health Consultation. Assessment of Soil Sampling Results at the American University, Washington, D.C. U.S. Department of Health and Human Services, Atlanta, Georgia, August 26, 1997. http://www.atsdr.cdc.gov/sites/springvalley/consult_082697.html

ATSDR 1999. Oct. 8, 1999 ATSDR Record of Activity on 4825 Glenbrook Road. Conference call with USEPA Region 3.

ATSDR 2002. Agency for Toxic Substances and Disease Registry. Blister Agents: Lewisite (L), Mustard-Lewisite Mixture (HL) Sulfur Mustard Agent H/HD and HT. Fact sheet. Division of Toxicology ToxFAQs. Internet address is http://www.atsdr.cdc.gov/toxfaqs/tfacts49.pdf

ATSDR 2003. Agency for Toxic Substances and Disease Registry. Toxicological profile for sulfur mustard (update). Atlanta, GA: U.S. Department of Health and Human Services. September 2003.

ATSDR 2005. Agency for Toxic Substances and Disease Registry. Public Health Evaluation for the Spring Valley Community. Spring Valley Chemical Munitions, Washington, District of Columbia. Health Consultation. September 7, 2005. Atlanta, GA: U.S. Department of Health and Human Services. Internet address is http://www.atsdr.cdc.gov/sites/springvalley.

ATSDR 2007a. Agency for Toxic Substances and Disease Registry. Toxicological Profile for Arsenic. U.S. Department of Health and Human Services, Atlanta, Georgia, August 2007. http://www.atsdr.cdc.gov/ToxProfiles/tp.asp?id=22&tid=3

ATSDR 2007b. Agency for Toxic Substances and Disease Registry. Toxicological Profile for Benzene. Atlanta, GA: U.S. Department of Health and Human Services. August 2007.

ATSDR 2008. Agency for Toxic Substances and Disease Registry. Evaluating Vapor Intrusion Pathways at Hazardous Waste Sites, Atlanta, GA: US Department of Health and Human Services, Feb 6, 2008. Available online at: http://www.atsdr.cdc.gov/document/evaluating_vapor_intrusion.pdf .

ATSDR 2010. Agency for Toxic Substances and Disease Registry. Site visit by Greg Zarus of ATSDR with partial video/audio record in December 2010. Site tour given by Dan Noble of USACE.

ATSDR 2014. Agency for Toxic Substances and Disease Registry. The Division of Community Health Investigations (DCHI) Soil Dose & Cancer Risk Estimator (Excel spreadsheets). Excel file that goes along with the Exposure Dose Guidance for soil ingestion.

AU 1919. American University. Index of technical reports dated 1918 and 1919. American University Technical Reports. Includes Bureau of Mines, War Gas Investigations (WGI) Monographs (Old Series) and reports from other Sections.

Balali-Mood, M, SH Mousavi, and B Balali-Mood 2008. Chronic health effects of sulfur mustard exposure with special reference to Iranian veterans. Emerging Health Threats Journal 1:e7. Doi 10.3134/ehtj.08.007.

Bullman, T and H Kang 2000. A fifty year mortality follow-up study of veterans exposed to low level chemical warfare agent, mustard gas. Annals of Epidemiology 10(5):333-338.

Chen, Y, F Parvez, M Gamble, T Islam, A Ahmed, M Argos, JH Graziano, and H Ahsan 2009. Arsenic exposure at low-to-moderate levels and skin lesions, arsenic metabolism, neurological functions, and biomarkers for respiratory and cardiovascular diseases: Review of recent findings from the Health Effects of Arsenic Longitudinal Study (HEALS) in Bangladesh. Toxicology and Applied Pharmacology 239(2):184-192.
Davis, KG and G Aspera 2001. Exposure to liquid sulfur mustard. Annals of Emergency Medicine 37 (6):653-656.

Current Newspapers 2014. Spring Valley: At Risk From WWI Poisons?. A Supplement to the Current Newspapers, Washington, DC. Wednesday, November 10, 2004.

DC DOH 2002. Government of the District of Columbia, Department of Health, Fax from Dr. Lynette Stokes (Bureau of Hazardous Material & Toxic Substances, UST/LUST Program, Washington, D.C.) to Laura Frazier on January 29, 2002. Disorders and streets are listed. Health complaints of Spring Valley residents recorded by the DC DOH on their hotline (phone line specifically made available to record health concerns of residents during the late 1990s through early 2000s).

DC Gov 1996. Government of the District of Columbia, Department of Consumer and Regulatory Affairs, Environmental Regulation Administration, Hazardous Waste Branch. Final Report on World War I Poison Gas Production at the American University Experiment Station. July 1996.

Delepine 1923. S. Delepine. Journal of Industrial Hygiene. Vol. IV, May 1922- April 1923, pages 346-464 and 410-423. Published by Harvard Medical School.

Durrin 1993. Transcripts of interviews conducted by Virginia Durrin with three West Virginia construction workers who built the 4825 Glenbrook Road house, November 6, 1993. Three

Word Documents provided by USACE: #084interviewWVworkers.doc, #085interviewWVworkers.doc, #086interviewWVworkers.doc. Durrin Productions, Inc. 1993. Durrin 2013. SV transcript #1189, transcript only. Phone conversation between G. Durrin and former Spring Valley worker on burials at 4825 and 4835 Glenbrook Road NW conducted on March 13, 2013. Durrin Productions, Inc. 2013.

Fitzpatrick, TA, K Wolff, and R Johnson 2000. Color Atlas and Synopsis of Clinical Dermatology, 4th Edition. McGaw Hill, New York, NY.

Garrett et al. 2007. Benjamin Garret, John Hart. Historical Dictionary of Nuclear, Biological, and Chemical Warfare: *Issue 33 of Historical dictionaries of war, revolution, and civil unrest*. Scarecrow Press, Lanham, Maryland, p. 16.

GuideChem MSDS. GuideChem. Material Safety Data Sheet. Accessed online at http://img1.guidechem.com/msdspdf/12044-49-4.pdf on 2/25/2015.

Hurst, CG and WJ Smith 2000. Chronic effects of acute low-level exposure to the chemical warfare agent sulfur mustard. In Chemical Warfare Agents: Toxicity at Low Levels, SM Somani and JA Romano, Jr., editors. CRC Press, Boca Raton, FL.

Hurst et al. 2008. C.G. Hurst, J.P. Petrali, D.J. Barillo, J.S. Graham et al. Vesicants. In *Textbooks of Military Medicine, Medical Aspects of Chemical Warfare*. Washington, DC, Office of the Surgeon General (OSG) at TMM publications. Chapter 8, pp. 259-309.

IARC 1987. International Agency for Research on Cancer. Monograph on Arsenic and Arsenic Compounds. Accessed online at http://monographs.iarc.fr/ENG/Monographs/vol100C/mono100C-6.pdf

Karalliedde, L, H Wheeler, R Maclehose, and V Murray 2000. Possible immediate and long-term health effects following exposure to chemical warfare agents. Public Health 114:238-248.

Kehe,K and L Szinicz 2005. Medical aspects of sulphur mustard poisoining. Toxicology 214:198-209.

Marrs et al. 2007. Marrs, T. C., R.L. Maynard, and F.R. Sidell. Chemical Warfare Agents, Toxicology and Treatment. Second Edition. John Wiley and Sons, Ltd. England. 738 p.

Kyle RA and Pease GL. 1965. Hematologic aspects of arsenic intoxication. New Engl J Med 273(1):18–23.

MARB 2014. Material Assessment Review Board. JPM E (P) Conference Notes. Material Assessment Review Board (MARB) Recommendation for Spring Valley (SVM), Washington, D.C., Item SVM-13-195. December 18, 2013.

NICNAS 2001. National Industrial Chemicals Notification and Assessment Scheme. Sydney NSW, Australia. Polychlorinated Naphthalenes. Available at http://www.nicnas.gov.au/__data/assets/pdf_file/0015/4920/S48_PCN_July02.pdf

Mizuta et al. 1956. Mizuta N, Mizuta M, Ito F, et al. An outbreak of acute arsenic poisoning caused by arsenic-contaminated soy-sauce (shoyu): A clinical report of 220 cases. Bull Yamaguchi Med Sch 4(2-3):131-149. Cited in ATSDR toxicological profile for arsenic, 2007.

Moeller 2005. James W. Moeller, Arsenic and an Old Base: Legal Issues Associated with the Environmental Restoration of Defense Sites in Washington, D.C., Used for the Development and Disposal of World War I Chemical Munitions, 54 Cath. U. L. Rev. 879 (2005).

Munro, NB, SS Talmage, GD Griffin, LC Waters, AP Watson, JF King, and V Hauschild 1999. The sources, fate, and toxicity of chemical warfare degradation products. Environmental Health Perspectives 107(12):933-974.

NIOSH 2011. National Institute for Occupational Safety and Health. International Chemical Safety Card (ICSC) 0221 for Arsenic Trichloride. Accessed on-line in Jan. 2011.

NIOSH 2015a. National Institute for Occupational Safety and Health. Emergency Response Safety and Health Database, Arsine (SA): Systemic Agent. Accessed online at http://www.cdc.gov/niosh/ershdb/emergencyresponsecard_29750014.html on 02/26/2015.

NIOSH 2015b. National Institute for Occupational Safety and Health. Worker Health Study Summaries. Research on long-term exposure, Navy Cable Manufacturers (Chlorinated Naphthalenes Exposure). Accessed online at http://www.cdc.gov/niosh/pgms/worknotify/chloronapth.html#doctor on 2/25/2015.

OSHA 2015. Occupational Safety and Health Administration. U.S. Department of Labor, Hydrogen Chloride Exposure Limits and Health Effects. Accessed online at: https://www.osha.gov/dts/chemicalsampling/data/CH_246300.html

Oomen AG, Hack A, Minekus M, Zeijdner E, Cornelis C et al. 2002.Comparison of five in vitro digestion models to study the bioaccessibility of soil contaminants. Environ Sci Technol Aug 1;36(15):3326-34.

ORNL 2007. Oak Ridge National Laboratory. Re-evaluation of 1999 Health-Based Environmental Screening Levels for Chemical Warfare Agents. May 2007. Accessed Dec. 2012 at http://apps.ornl.gov/~pts/prod/pubs/ldoc6607_watson_tm_reevaluation_of_1999_health_5_23_07.pdf.

Perrotta, DM 1996. Long-term health effects associated with sub-clinical exposures to GB and mustard. A review conducted by the Environment Committee Armed Forces Epidemiological Board. July 18, 1996. http://www.gulflink.osd.mil/agent.html

Ruby MV, Schoof R, Brattin, W et al. 1999. Advance in evaluating the oral bioavailability of inorganics in soil for use in human health risk assessment. Environ Sci Technol 33(21):3697–705.

Sittig, Marshall. 1991. Handbook of Toxic and Hazardous Chemicals and Carcinogens. Third edition. Volume 1. Noyes Publications. Park Ridge, NJ, USA.

Smith et al. 2010. Milton G. Smith, William Stone, Ren-Feng Guo et al. Vesicants and Oxidative Stress. In *Chemical Warfare Agents: chemistry, pharmacology, toxicology, and therapeutics.* Editors, James A. Romano,Brian J. Lukey and Harry Salem. 2nd ed. Informa Healthcare. New York NY. ISBN 1-4200-4661-6. Chapter 12, pp.247-292.

Stein, KR and NS Scheinfeld 2007. Drug-induced photoallergic and phototoxic reactions. Expert Opinions Drug Safety 6(4):431-443.

Tseng WP, Chu HM, How SW, et al. 1968. Prevalence of skin cancer in an endemic area of chronic arsenicism in Taiwan. J Natl Cancer Inst 40:453–63. Cited in ATSDR toxicological profile for arsenic, 2007.

Uede, K and F Furukawa 2003. Skin manifestations in acute arsenic poisoning from the Wakayama curry-poisoning incident. British Journal of Dermatology 149(4):757-762.

Yoshida, T, J Yamauchi, and GF Sun 2004. Chronic health effects in people exposed to arsenic via the drinking water: dose-response relationships in review. Toxicology and Applied Pharmacology 198:243-252.

USACHPPM 1998. U.S. Army Centers for Health Promotion and Preventive Medicine. 1998. The Deputy for Chemical Services' Publications—detailed chemical fact sheets. General Facts About Vomiting Agent Adamsite (DM). General Facts About Blister Agent Mustard-Lewisite Mixture (HL). General Facts About Blister Agent Lewisite (L). Last accessed June 23, 2003 but no longer available at: http://chppm-www.apgea.army.mil/dts/dtchemfs.htm . Not accessible, March 2011.

USACHPPM 1999. The US Army Center for Health Promotion and Preventative Medicine. Derivation of Health-Based Environmental Screening Levels for Chemical Warfare Agents. A Technical Evaluation. Prepared by USCHPPM, Aberdeen Proving Ground, Maryland in conjunction with ORNL for the US Dept. of Energy. March 1999. Available at: http://chppm-www.apgea.army.mil/hrarcp/CAW/HBESLcover.pdf . Last accessed Feb 2013.

USACE 1995. Final Remedial Investigation Report for the Operation Safe Removal Formerly Used Defense Site, Washington, D.C. Prepared for U.S. Army Corps of Engineers, Baltimore District. Prepared by Parsons of Fairfax, VA. June 1, 1995.

USACE 2000. Revised Final Removal Action Design. 4801, 4825, and 4835 Glenbrook Road. Prepared for U.S. Army Corps of Engineers, Baltimore District. Prepared by Parsons of Fairfax, VA. Nov. 1, 2000.

USACE 2002. Technical memorandum—arsenic bioavailability study. Spring Valley Operable Unit 4, Washington, D.C. Prepared for U.S. Army Corps of Engineers, Baltimore District. Prepared by Parsons of Washington, DC.

USACE 2003. [Parsons] Parsons Engineering Science, Inc. 2003. Engineering Evaluation/Cost Analysis (EE/CA) for Arsenic and Other Selected Contaminants in Soil, Spring Valley Operable Units 4 and 5, Washington, D.C. Volumes 1, 2, and 3. July 2003.

USACE 2007c. United States Army Corps of Engineers. Fenceline Presentation from Nov. 2007. AUES Fenceline Study.

USACE 2009a. Soil Gas and Driveway ABP, Soil Sampling Report, 4825 Glenbrook Road. Spring Valley Formerly Used Defense Site, Operable Unit 3, Washington D.C. Prepared for U.S. Army Corps of Engineers, Baltimore District. Prepared by Parsons of Washington, DC. April 15, 2009.

USACE 2009b. Site-Specific Work Plan for High Probability Test Pit Investigations at 4825 Glenbrook Road. Prepared for U.S. Army Corps of Engineers, Baltimore District. Prepared by Parsons of Washington, DC. October 16, 2009.

USACE 2011a. USACE's Spring Valley website at http://www.nab.usace.army.mil/Projects/Spring%20Valley/pit3.htm. Accessed July 2011.

USACE 2011b. Final 4825 Glenbrook Road Human Health Risk Assessment. Spring Valley Formerly Used Defense Site, Operable Unit 3, Washington D.C. Prepared for U.S. Army Corps of Engineers, Baltimore District. Prepared by Parsons of Washington, DC. July 29, 2011.

USACE 2011c. Final Remedial Investigation Report for 4825 Glenbrook Road. Spring Valley Formerly Used Defense Site (SVFUDS), Operable Unit 3, Washington D.C. Prepared by Parsons Washington D.C. for U.S. Army Engineering and Support Center, Huntsville and U.S. Army Corps of Engineers, Baltimore District. July 29, 2011.

USACE 2011d. Appendices A-S of the Final Remedial Investigation Report for 4825 Glenbrook Road. Spring Valley Formerly Used Defense Site (SVFUDS), Operable Unit 3, Washington D.C. Prepared by Parsons Washington D.C. for U.S. Army Engineering and Support Center, Huntsville and U.S. Army Corps of Engineers, Baltimore District. (Specific appendices referenced in this consultation include: Appendix E, Data Summary Tables. Appendix I, Lab Report. Appendix L, Additional Arsenic Trichloride Analysis. Appendix N, Air Monitoring Reports, Arsine Monitor Datalog folder. Appendix P, Destruction and munitions certificates. Appendix R, MEC-HA Analysis. Revised September 27, 2011.

USACE 2011e. Comments on the Draft Spring Valley ATSDR Health Consultation dated October 14, 2011.

USACE 2011f. Final Work Plan for Human Health Risk Assessment at 4825 Glenbrook Road. May 27, 2011.

USACE 2012. Final Decision Document 4825 Glenbrook Road Spring Valley Formerly Used Defense Site, Operable Unit 3, Prepared by Baltimore District, U.S. Army Corps of Engineers, June 13, 2012.

USACE 2014. United States Army Corp of Engineers. Report entitled, "MINICAMS Interferences on March 25, 26 and 28, 2014 and April 1, 2014.

USCOA, 2004. United States Court of Appeals for the District of Columbia Circuit. Argued 10/18/04 and Decided 12/21/04. No. 03-5252. Available online at: http://www.cadc.uscourts.gov/internet/opinions.nsf/E35A98743FC1411E85256F82006D6C3F/$file/03-5284a.pdf.

USEPA 1994. U.S. Environmental Protection Agency. Spring Valley Munitions Site Analytical Summary. TDD# 9310-23D. PCS# 5299.

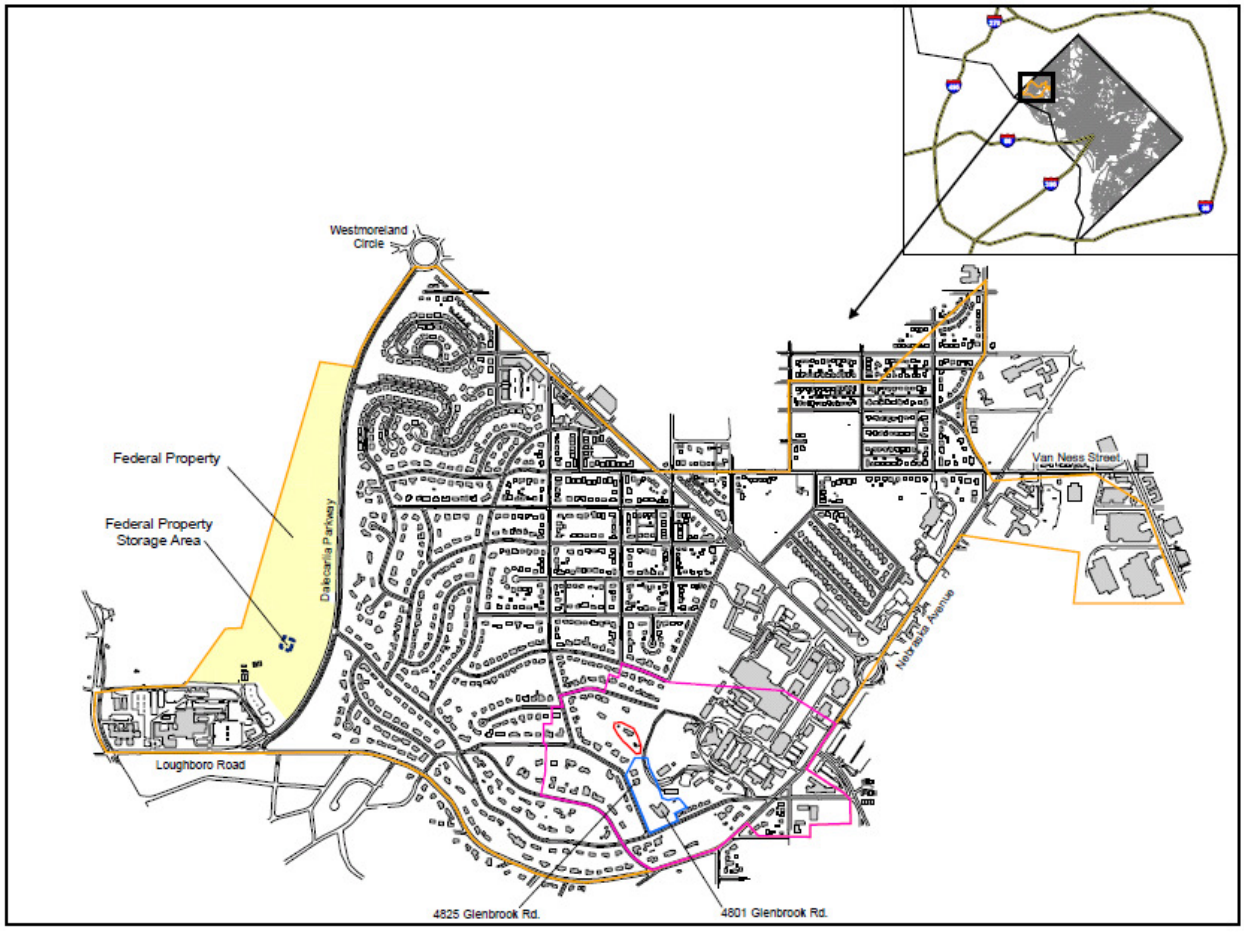
USEPA 2003. US Environmental Protection Agency. Integrated Risk Information System. Hydrogen chloride (CASRN 7647-01-0). Last revision 2003. Accessed Nov. 2012 at http://www.epa.gov/iris/subst/0396.htm.

USEPA 2011. US Environmental Protection Agency. Technical Factsheet on 2,4,5-TP. Accessed at www.epa.gov/ogwdw/pdfs/factsheets/soc/tech/245-tp.pdf on 9/23/2011.

WHO 2001a. World Health Organization. Arsenic and arsenic compounds. Environmental health criteria series, no. 224. 2nd ed. Geneva, Switzerland.

WHO 2001b. World Health Organization. Concise International Chemical Assessment Document 34: Chlorinated Naphthalenes. 2001. Available online at http://www.who.int/ipcs/publications/cicad/en/cicad34.pdf. Last accessed on 3/10/2015.

Appendix A. Figures



Legend

Buildings
Road
Federal Property
Federal Property Storage Area

Operable Unit
OU-2
OU-3
OU-4
OU-5

Notes:
1. OU-1 encompasses all of the areas depicted as OU-2, 3, 4, and 5.
2. OU-4 and OU-5 do not include the smaller operable units shown within their boundaries (e.g., OU-4 does not include the areas shown as OU-2 and OU-3).

Figure 1. The location of the Spring Valley FUDS in northwest Washington, D.C. and the 4825 Glenbrook Road site in OU-3 of the FUDS are shown [USACE 2011d, portions of Figure 1-1]. In addition to the 4825 property, the properties of 4835 and 4801 Glenbrook Road are also in OU-3. OU-2 includes the area where the Spaulding and Captain Rankin Area shell pits were built. Notes on the OUs are included in the legend.

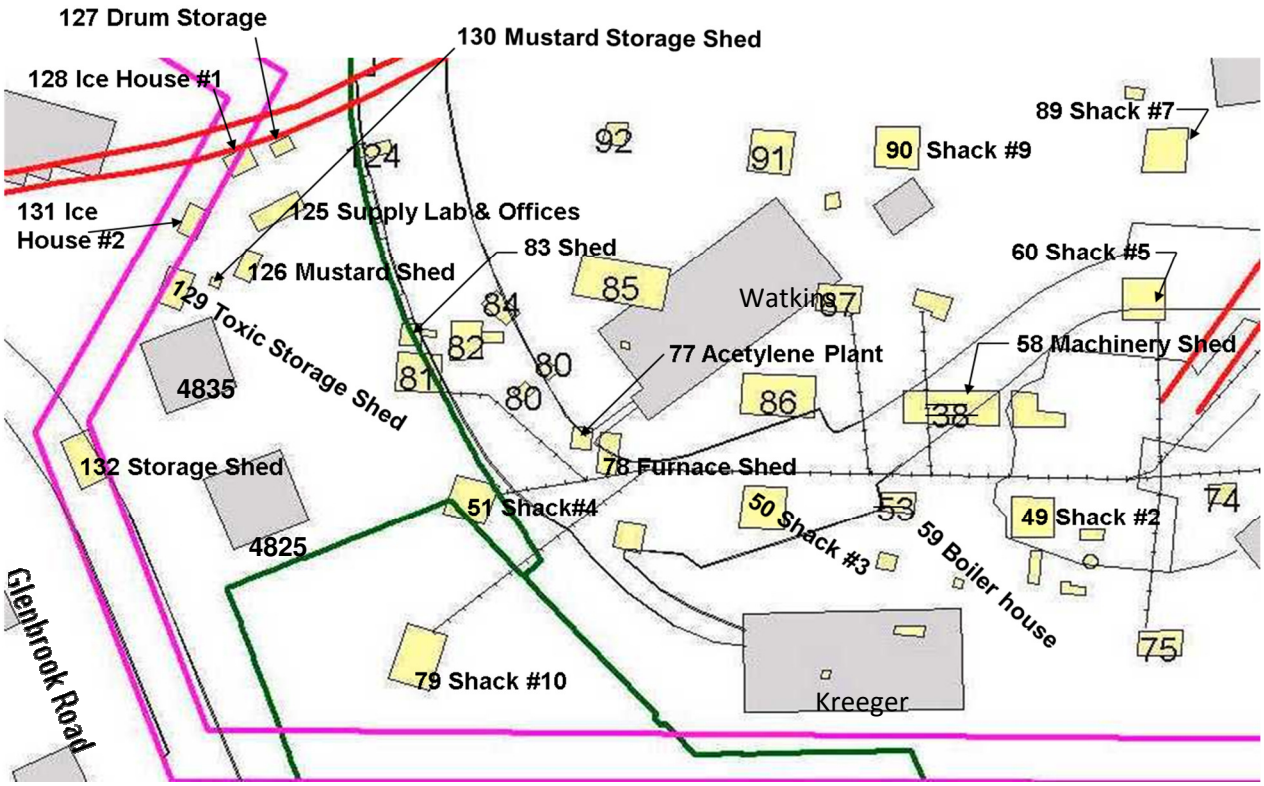
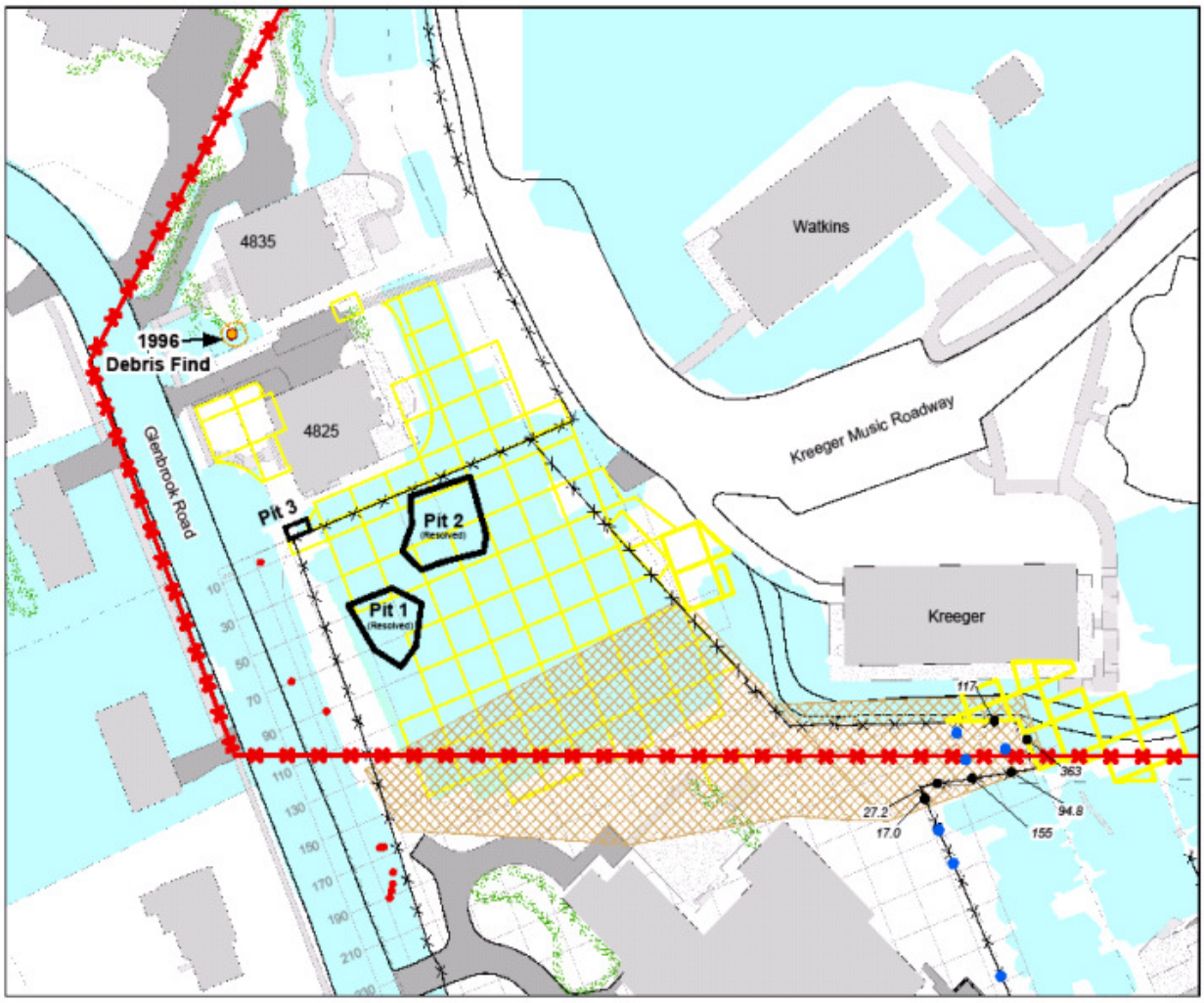


Figure 2. The location of former AUES buildings (shaded yellow) on American University are shown [USACE 2007c, slide 7, modified by adding addresses to 4825 and 4835 and labeling Glenbrook Road]. Current buildings are shaded gray. Note that building 126 was a mustard shed, Shed 130 a mustard Storage Shed, and Shed 129 a toxic storage shed. Building 82 is believed to be the location of a 1918 explosion involving arsenic and magnesium [USACE 1995].



Legend

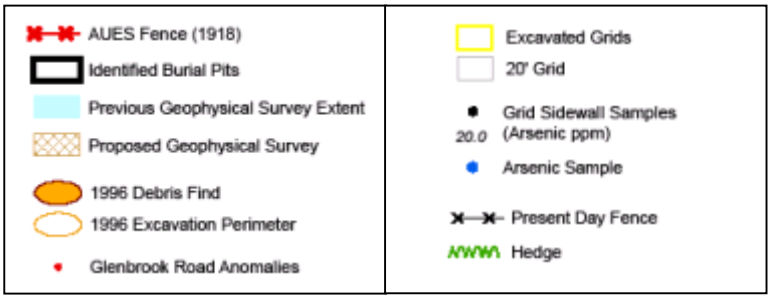


Figure 3. The properties of 4825, 4835, and part of 4801 are within the former AUES fence line. Pits 1 through 3 are burial pits which were located south of the home at 4825 Glenbrook Road. Part of Burial Pit 3 was located on the 4825 Glenbrook Road property, at the property line with 4801 Glenbrook Road [Part of Burial Pit 3 and two other burial pits (1 and 2) were on the 4801 Glenbrook Road property]. Not all of Burial Pit 3 and its extensions are shown [USACE 2007c, slide 11, portions of legend]. Note the 1996 debris find, on 4835 Glenbrook Road, where landscapers encountered buried chemical wastes and laboratory glassware; they experienced irritation to their eyes and respiratory system.

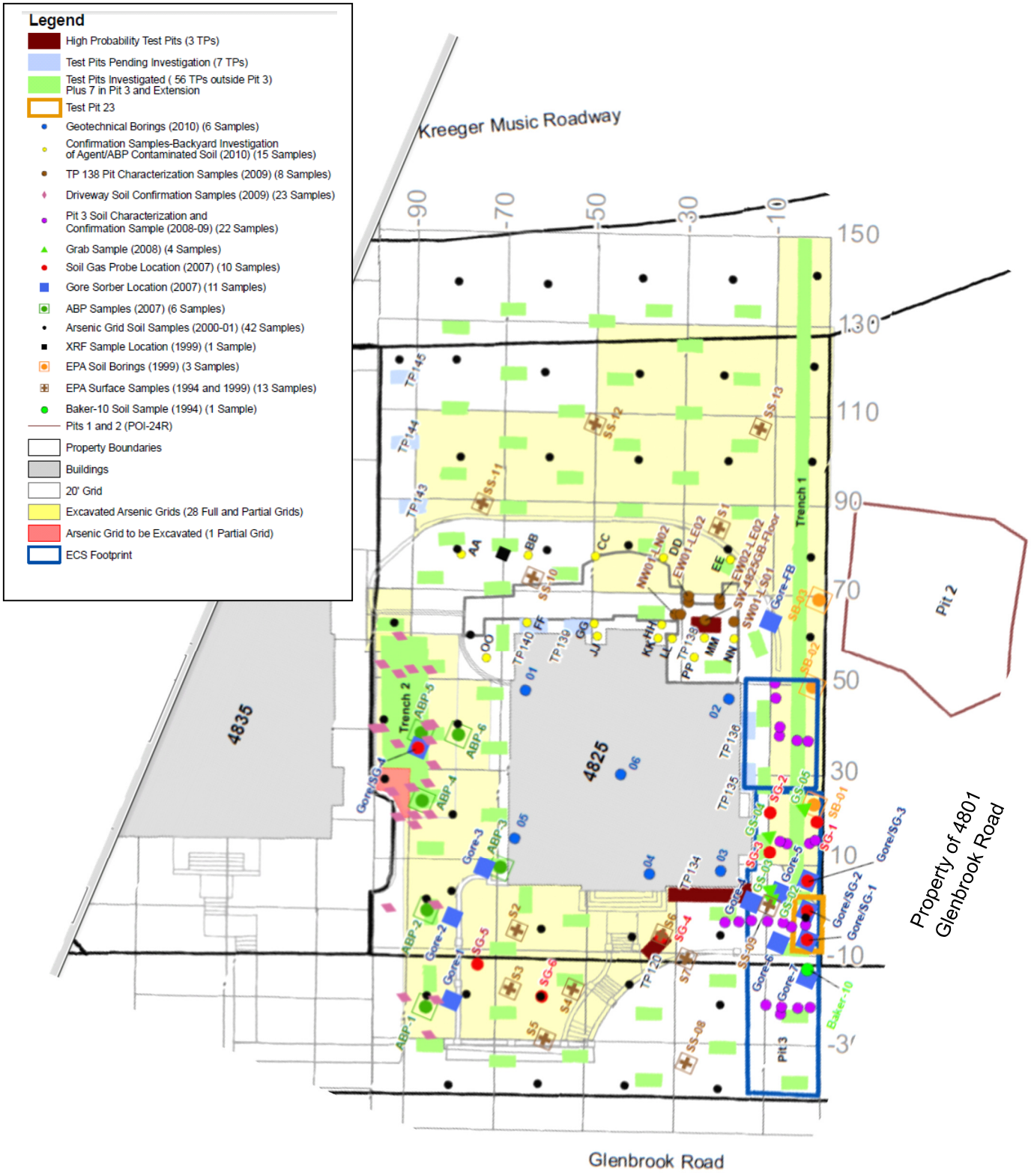


Figure 4. Sampling locations at 4825 Glenbrook Road [USACE 2011e, Figure 1-3: The map has been rearranged and cropped].

Note: Additional Sampling Not Shown: 1992 EMS Investigation (Specifics Unavailable) and the 2000 Quadrant Sampling for Sulfur Mustard ABPs (Composited Locations).

Appendix B.
ATSDR's Evaluation Process

Identifying Exposure

ATSDR's health assessments are exposure (or contact) driven. People in the area of an environmental release can only be exposed to a contaminant if they come in contact with it. Exposure might occur by breathing, eating, or drinking a substance containing the contaminant or by skin contact with a substance containing the contaminant. Therefore, *a release does not always result in exposure*. Exposure can only occur when a person has contact with a contaminant.

ATSDR evaluates site conditions to determine if people could have been (a past scenario), are (a current scenario), or could be (a future scenario) exposed to site-related contaminants. When evaluating exposure pathways, ATSDR identifies whether exposure to contaminated media (soil, water, air, waste, or biota) has occurred, is occurring, or might occur through ingestion, dermal (skin) contact, or inhalation. ATSDR also identifies an exposure pathway as *completed* or *potential,* or *eliminates the pathway from further evaluation*. Completed exposure pathways exist if all elements of a human exposure pathway are present. A potential pathway is one in which one or more of the pathway elements cannot be definitely proved or disproved. A pathway is eliminated if at least one element is absent.

As defined by ATSDR, an *exposure pathway* is 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 elements: a source of contamination (such as burial or disposal pits); an environmental media and transport mechanism (such as movement through soil as vapors into ambient or indoor air); a point of exposure (such as a residential yard or home); a route of exposure (eating, drinking, breathing, or touching), and a receptor population (people potentially or actually exposed). When all five elements are present, the *exposure pathway* is termed a completed exposure pathway.

Interested persons can learn more about the ATSDR evaluation process by reading ATSDR's Public Health Assessment Guidance Manual (available at http://www.atsdr.cdc.gov/hac/PHAManual/toc.html) or by contacting ATSDR at 1-800-CDC-INFO.

Exposure and Health Effects

Given sufficient exposure levels, chemicals in the environment can cause harmful health effects. The type and severity of effects is influenced by several complex factors including the concentration (how much), the frequency and/or duration of exposure (how often and how long), the way(s) the chemical enters the body, and combined exposure to other chemicals. Once exposure occurs, characteristics of the exposed person such as age, sex, nutritional status, genetics, and health status can affect how a person responds to an exposure and experiences any subsequent health harm.

Once a completed exposure pathways is identified, ATSDR selects chemicals in that pathway for further evaluation by comparing them against health-based screening values. Screening values are developed from the available scientific literature on the magnitude of exposure and health effects. They are derived for each of the different environmental media (e.g. air, water, soil), and each reflects an estimated contaminant concentration that is *not expected* to cause adverse health effects for a given chemical, assuming a standard daily contact rate (e.g., amount of water or soil consumed or amount of air breathed) and body weight. To be conservative and protective of public health, screening values are generally based on contaminant concentrations *many times lower than levels at which no effects were observed* in experimental animals or human studies. ATSDR does not use screening values to predict the occurrence of adverse health effects, but rather to serve as a health protective first step in the evaluation process.

Identifying Chemicals of Concern

Screening values include ATSDR's comparison values (CVs): environmental media evaluation guides (EMEGs), reference dose media evaluation guides (RMEGs), and cancer risk evaluation guides (CREGs). CREGs, EMEGs, and RMEGs are non-enforceable, health-based CVs developed by ATSDR for screening environmental contamination for further evaluation. In addition,to ATSDR's screening values, or in their absence, Health Based Environmental Screening Levels (HBESL), developed to address potential long-term or chronic exposures to residual concentrations [ORNL 2007], were used in this evaluation. HBESL were used as screening values for sulfur mustard and lewisite in various media (soil and non-containerized waste in soil).

If contaminant concentrations are above these environmental screening values, ATSDR analyzes exposure variables (for example, duration and frequency), the toxicology of the contaminant, and epidemiology studies for possible health effects. During this part of the public health assessment process, ATSDR estimates site-specific exposure *doses and* compares them to health guideline values. This health guideline comparison allows health assessors to study possible public health implications of site-specific conditions. Health guidelines are derived based on data drawn from the epidemiologic and toxicological literature with many uncertainty or safety factors applied to ensure that they are amply protective of human health. ATSDR's minimal risk levels (MRLs) and USEPA's reference doses, reference concentrations, and cancer slope factors are the health guidelines most commonly used in the public health assessment screening process. Estimated doses that are below health guidelines are not expected to cause adverse health effects.

Appendix C.

Exposure Dose Estimates and Parameters Used to Evaluate Ingestion Exposures to Arsenic

At sites such as 4825 Glenbrook Road, where contaminants have weathered over a long period of time, the presence of stable degradation products are relevant for health risk assessment. For very volatile or reactive contaminants, the residence time in the soil may be so short that the potential for chronic exposures will be very low. The 1992 worker exposure incident indicates exposure to chemicals in air from broken bottles and contaminated soil. Broken bottles with chemical content could have released solids/liquids to the soil creating hot spots of contamination in soil during development of the property. Although chemical spills/releases could have created localized hot spot areas with high initial soil concentrations, the concentrations of non-persistent chemicals would have degraded and are likely to have been much lower to non-existent when samples were taken years to decades later. Only the persistent chemicals and degradation products would have remained in soil. For CWAs, no soil concentrations at 4825 Glenbrook Road are available in the decade when exposures occurred.

Due to the lack of historical data relevant to exposures, no dose estimates for ingestion and skin contact based on environmental levels have been made for sulfur mustard and lewisite. Containerized CWAs and even CWAs recently spilled from their containers into environmental media, if contacted, could be at high enough doses to result in harmful health effects. Former outdoor workers, if exposed to soil containing waste in the hot spot areas, may have experienced harmful health effects. Some discussion of exposure doses has been provided in the main text for additional perspective. In this appendix, ATSDR estimates exposure doses based on arsenic, a persistent breakdown product of lewisite and other arsenicals.

Evaluating Potential Health Hazards

ATSDR performs an in-depth evaluation to determine whether exposures might be associated with adverse health effects (non-cancer and cancer). As part of this process, ATSDR examines relevant toxicologic and epidemiologic data to determine whether estimated doses are likely to result in adverse health effects. As a first step in evaluating non-cancer effects, ATSDR compares estimated exposure doses to standard health guideline values, including ATSDR's minimal risk levels (MRLs) and the U.S. Environmental Protection Agency's (EPA's) reference doses (RfDs). The MRLs and RfDs are estimates of daily human exposure to substances that are unlikely to result in non-cancer effects over a specified duration. Estimated exposure doses that are less than these values are not considered to be of health concern. To be very protective of human health, MRLs and RfDs have built in "uncertainty" or "safety" factors that make them much lower than levels at which health effects have been observed. Therefore, if an exposure dose is much higher than the MRL or RfD, it does not necessarily follow that adverse health effects will occur.

To evaluate carcinogens, ATSDR compares the exposure levels to cancer effect levels that have been shown to cause cancer in animals or humans. In addition, ATSDR may calculate quantitative estimates of risk using EPA's cancer slope factors. These cancer estimates are based

on conservative models and assumptions, so the actual risk may be substantially less than the calculated value.

If health guideline values are exceeded, ATSDR examines the effect levels seen in the literature and more fully reviews exposure potential to help predict the likelihood of adverse health outcomes. Specifically, ATSDR examines "no-observed-adverse-effect levels" (NOAELs) or the "lowest-observed-adverse-effect levels" (LOAELs) for the most sensitive outcome for a given route of exposure (e.g., ingestion or skin contact). ATSDR looks at human studies, when available, as well as experimental animal studies. In the case of arsenic, a great deal of human data is available, though most is related to water and air exposures versus soil exposures. This information is used to describe the disease-causing potential of a particular contaminant and compare site-specific dose estimates with doses shown to result in illness in applicable studies (known as the margin of exposure). For cancer effects, ATSDR also reviews genotoxicity studies to further understand the extent to which a contaminant might be associated with cancer outcomes. This process enables ATSDR to weigh the available evidence, in light of uncertainties, and offer perspective on the plausibility of adverse health outcomes under site-specific conditions. Reviewing the scientific literature in this way enabled ATSDR to evaluate the range of dose levels that may be associated with the substance being evaluated and the characteristics of that substance that may make adverse health effects less or more likely.

Arsenic

This appendix presents the methods and findings of ATSDR's health effects assessment. It describes how ATSDR estimated exposure doses for former workers who developed the property during an approximately one year timeframe and former residents who resided there for approximately 5 years. We discuss what estimated doses mean—that is, how do the doses compare to those shown in the scientific literature to result in adverse health effects? Past exposure doses are calculated using environmental concentrations from the 1990s through 2010. Some children have a much higher tendency to ingest soil and other non-food items. This is known as pica behavior. Pica children could conceivably consume a teaspoon or more of contaminated soil each day. No documentation of this type of exposure has been identified at 4825 Glenbrook Road.

Outdoor workers with high soil contact would be involved with activities such as packing soil behind retaining walls and landscaping. Outdoor workers with low contact spend less time actively working in the soil and come into contact with subsurface soils less often.

Deriving Exposure Doses for Arsenic

ATSDR estimated exposure doses, which are estimates of how much contaminant a person may be exposed to on a daily basis. Variables considered when estimating exposure doses include the contaminant concentration in the environmental media, the exposure amount (how much of the substance the person was actually exposed to), the exposure frequency (how often), and the exposure duration (how long). Together, these factors influence an individual's physiological response to chemical contaminant exposure and potential outcomes. Where possible, ATSDR used site-specific information about the frequency and duration of exposures. In cases where site-specific information was not available, ATSDR applied several conservative exposure

assumptions to estimate exposures for former workers and residents at 4825 Glenbrook Road. The equations and parameters are summarized in this appendix.

For ingestion of contaminants, a gastrointestinal absorption factor (AF) of 0.5 or 50% was used for arsenic. The absorption factor is based on bioavailability. We assumed that 50% of the arsenic in soil would actually be absorbed in the body once ingested. The selected value represents the high end of the range of "bioavailability factors" reported in the scientific literature and from site-specific studies (ATSDR 2007A; Oomen et al. 2002; Ruby et al. 1999; USACE 2002, WHO 2001a). Using the high end of this range could overestimate exposures. See the text box for more information on the bioavailability of arsenic in soils.

Understanding Bioavailability of Arsenic

Arsenic in water has been shown to be very well absorbed across the gastrointestinal tract (ATSDR 2007). However, this is not so with arsenic in soil. Fairly extensive studies of arsenic bioavailability reveal that the human body absorbs only a portion of the arsenic that is present in a soil matrix. Bioavailability is dependent on arsenic form and soil type. The best measure of bioavailability is testing designed to quantify uptake under site-specific conditions. Such testing occurred at Spring Valley. USACE tested 11 soil samples and reported bioavailability factors ranging from 3% to 50% (USACE 2002). To be conservative, ATSDR chose the highest reported factor when calculating exposure doses. Recognize, however, that only one Spring Valley sample yielded bioavailability as high as 50%. The bioavailability in the remaining samples was considerably lower, ranging from 3% to 22%, with a mean of 10%. Site-specific doses are probably lower than those used in our analysis because our dose estimates could be overestimated by a factor of approximately 2 to 16.

Evaluating Incidental Ingestion

The Division of Community Health Investigations (DCHI) Soil Dose & Cancer Risk Estimator (Excel spreadsheets) was used to estimate potential exposure doses and cancer risk (ATSDR 2014). The methods used follow ATSDR's Exposure Dose Guidance for Soil Ingestion 2014. Standard exposure parameters such as ingestion rates and body weights follow USEPA guidance [USEPA 2002 and 2011]. Based on the Spring Valley bioavailability study for arsenic, a 50% relative bioavailability factor (50% of the soil concentration) was applied to the calculations and estimates. For workers, we calculated doses for outdoor workers (construction type) and for residents we calculated doses for both children and adults. The following equation was used to estimate exposure doses for contaminants detected in soil at 4825 Glenbrook Road:

DOSE = [CONCENTRATION x Bioavailability Factor x INTAKE x EXPOSURE FACTOR x Conversion Factor] / [BODY WEIGHT].

Parameters that went into the spreadsheet calculations in this dose equation were:

C: Contaminant concentration in soil (mg/kg or ppm)

IR: Ingestion rate or intake:

IRowh: 330 mg/day for an outdoor worker with high contact (0.00033 kg/day)

IRowl: 100 mg/day for an outdoor worker with low contact (0.0001 kg/day)
IRa: 100 mg/day for an adult (0.0001 kg/day) resident or gardener
IRc: 200 mg/day for a child (for various age groups within 1-21 years of age) (0.0002 kg/day)
5,000 mg/day for a pica child (0.005 kg/day)

EF: Exposure frequency or factor (exposure events per year of exposure): 365 days/year for residents (exposure factor of 1: 7/7 days) and 260 days for workers (exposure factor of 0.7143: 5/7 days for chronic) and (exposure factor of 1: 5/5 days for acute). For children exhibiting pica behavior a one-time exposure (exposure factor of 1) and 3/7 days (exposure factor of 0.4286) were used for acute exposure.

ED: Exposure duration (the duration over which exposure occurs):

Outdoor Worker = 1 year (approx. building time of residence)
Adult = 5 years (approx. duration at residence)
Child = 5 years (approx. duration at residence)

AF: Bioavailability or absorption factor (gastrointestinal) for arsenic: 0.5 or 50% site-specific AF [USACE 2002].

BW: Body weight:

BWow: Outdoor Worker = 80 kg (176 pounds)
BWa: Adult = 80 kg (176 pounds)
BWc: Child = 17.4 kg (38 pounds; average weight of child 2–6 years)
BWpc Child exhibiting pica behavior = 11.4 kg (25 pounds; average weight of child 1 to < 2 years)

AT: Averaging time or the period over which cumulative exposures are averaged [ED x 365 days/year non-carcinogen; 1825 days or 5 years (residential occupancy; a fraction of a lifetime of 78 years) carcinogen].

Cancer risk calculated for a residential occupancy of 5 years; also based on age of child.

Oral Cancer Slope Factor for Arsenic: 1.5 $(mg/kg/day)^{-1}$

ATSDR has a provisional acute oral MRL of 0.005 mg/kg/day and a chronic oral MRL of 0.0003 mg/kg/day for arsenic. The MRL is an exposure level below which non-cancerous harmful effects are unlikely. The acute MRL is based on several transient (i.e., temporary) effects including nausea, vomiting, and diarrhea. When an estimated acute dose of arsenic is below 0.005 mg/kg/day, non-cancerous harmful effects are unlikely. It should be noted that:

1) The acute MRL is 10 times below the levels that are known to cause harmful effects in humans;

2) The acute MRL is based on people being exposed to arsenic dissolved in water instead of arsenic in soil – a fact that might influence how much arsenic can be absorbed once ingested; and
3) The MRL applies to non-cancerous effects only and is not used to determine whether people could develop cancer [ATSDR 2007A].

Tables C-1 and C-2 show the estimated exposure doses for ingestion of arsenic and comparison levels. For a former child resident exhibiting pica behavior, an acute exposure dose was calculated (Table C-2) but not a chronic exposure dose, as this is not a chronic or long-term behavior. Additionally, a landscaper is included in Table B-2 and depending on the depths and areas landscaped, etc. could be in the Gardener or Landscaper category or the Former Outdoor Worker with Low Soil Contact category.

Table C-3 gives a quantitative estimate and qualitative description of the increased cancer risk for residents based on ingestion of arsenic in surface soil (the average of 73 ppm was used as the exposure point concentration). The estimate is based on an oral cancer slope factor for arsenic of 1.5 $(mg/kg/day)^{-1}$. Although workers would have an increased cancer risk due to arsenic in soil, we cannot quantify the risk. Additionally, no cancer risk was quantified for a former child resident exhibiting pica behavior because this is not a chronic or long-term behavior.

The cancer risk equation used for the calculation was:

CANCER RISK = [Age-specific Annual Dose x Cancer Slope Factor (CSF) x Age-specific Number of Years of Exposure] / [Lifetime in Years] or CR = Age-specific dose x CSF x Age-specific # years / 78 years.

Table C-1. Exposure Doses for Non-cancer Effects for Ingestion of Inorganic Arsenic (Chronic or long-term exposure).

Category of People, Time, and Contact	*Incidental Ingestion of Soil Using an Average Concentration to determine Dose, (mg/kg/day)*	*Is the Dose Above the Comparison Value? Yes or No (mg/kg/day)*			
		Chronic MRL, 3.0E-4 or 0.0003	**NOAEL** 8.0E-4 or 0.0008	Threshold dose for hyperpigmentation and hyperkeratosis 2.0E-3 or 0.002*	**LOAEL****, *1.4E-2 or 0.014*
Former Outdoor Worker with High Soil Contact	1.3E-03	**Yes**	**Yes**	**Approaches threshold dose**	No
Former Outdoor Worker with Low Soil Contact	5.5E-04	**Yes**	No	No	No
Former Adult Resident	4.6E-05	No	No	No	No
Former Adult Gardener	4.6E-05	No	No	No	No
Former Child Resident	4.2E-04	**Yes**	No	No	No

For chronic exposures (365 days or longer), the average soil concentration of **876 ppm arsenic** from composite (mixed) samples from TPs 134 and 120 was used in the exposure calculation for **outdoor workers** (2010 data). The soil concentration of **73 ppm arsenic** was chosen to represent a worst case scenario for **residents** as this concentration was the average found in surface soil (2000 data). The average concentration of arsenic in grid samples, 0-4 feet for gardeners, was 56 ppm; since deeper soils lowered the concentration, we used the higher concentration of 73 ppm for gardeners as well. Table 1 in the main health consultation contains a summary of arsenic in soil.

MRL = ATSDR's minimal risk level
NOAEL = no-observed-adverse-effect level
LOAEL = lowest-observed-adverse-effect level

*The threshold dose for hyperpigmentation and hyperkeratosis of skin: The estimated threshold for skin effects to oral ingestion of inorganic arsenic is based on the lowest LOAEL and rounded to 2 µg/kg/day or 0.002 mg/kg/day.

** hyperpigmentation and keratosis of the skin. Screening levels and observed effect levels are based on the following principle studies: Mizuta et al. 1956, Tseng et al. 1968, and others [ATSDR 2007A].

Examples of calculations for Table C-1:

DOSE = [CONCENTRATION x Bioavailability Factor x INTAKE x EXPOSURE FACTOR x Conversion Factor] / [BODY WEIGHT] where the exposure factor for workers was 0.7143 and for residents was 1. Concentration x 0.5 is due to 50% bioavailability. Conversion factor = 10^{-6}.

Former Outdoor Worker with High Soil Contact: dose= 876 mg/kg x 0.5 x 330 mg/day x 0.7143 x 10^{-6}/80 kg= 1.3 x 10^{-3} mg/kg/day.

Adult Resident: dose = 73 mg/kg x 0.5 x 100 mg/day x 1 x 10^{-6}/80 kg=4.6 x 10^{-5} mg/kg/day.

Table C-2. Exposure Doses for Non-cancer Effects for Inorganic Arsenic (Acute or short-term exposure).

Category of People, Time, and Contact	*Incidental Ingestion of Soil Using a Maximum Concentration to determine Dose, (mg/kg/day)*	*Is the Dose Above the Comparison Value? Yes or No (mg/kg/day)*	
		Acute MRL, **5.0E-3 or 0.005**	**LOAEL**, **5.0E-2 or 0.05**
Former Outdoor Worker with High Contact	8.8E-03	**Yes**	No
Former Outdoor Worker with Low Contact	2.7E-03	No	No
Former Adult Resident	3.9E-04	No	No
Former Adult Gardener/ Landscaper	4.3E-04	No	No
Former Child Resident	3.6E-03	No	No
Former Child Resident exhibiting Pica Behavior	1.4E-01 to 8.9E-02 EF= 1*	**Yes**	**Yes**
	3.8E-02 to 5.8E-02 EF 3/7 days	**Yes**	**Yes** for a child 1 to < 2 years. No for a child 2 to < 6 years.

For an acute exposure of several weeks, we used the maximum soil concentration of 4,280 ppm, a grab sample taken from subsurface soil in TP 120 for workers; 620 ppm, maximum concentration. in a grid sample taken from surface soil for residents; and 694 ppm, maximum concentration in a grid sample taken from subsurface soil at four feet for gardeners (Table 1 in the main health consultation).

MRL = ATSDR's minimal risk level
LOAEL = lowest-observed-adverse-effect level
*EF = 1 time soil ingestion
Screening levels and observed effect levels are based on the following principle studies (ATSDR 2007A): Mizuta et al. 1956 and Tseng et al. 1968.

Examples of calculations for Table C-2:

DOSE = [CONCENTRATION x Bioavailability Factor x INTAKE x EXPOSURE FACTOR x Conversion Factor] / [BODY WEIGHT] where the exposure factor for workers and and residents is 1. Conc x 0.5 is due to 50% bioavailability. Conversion factor = 10^{-6}.

Former Outdoor Worker with High Soil Contact: dose= 4,280 mg/kg x 0.5 x 330 mg/day x 1 x 10^{-6}/80 kg= 8.8 x 10^{-3} mg/kg/day. (note: for acute exposure for workers the EF changed from 5/7 to 5/5 or 1)

Former Outdoor Worker with Low Soil Contact: dose= 4280 mg/kg x 0.5 x 100 mg/day x 1 x 10^{-6}/80 kg= 2.7 x 10^{-3} mg/kg/day.

Adult Resident: dose = 620 mg/kg x 0.5 x 100 mg/day x 1 x 10^{-6}/80 kg=3.9 x 10^{-4} mg/kg/day.

Former Child Resident exhibiting Pica Behavior:

Former Child Resident exhibiting pica behavior (one time exposure for a child 1 to < 2 years) = 620 mg/kg x 0.5 x 5,000 mg/day x 1 x 10^{-6}/11.4 kg= 1.4 x 10^{-1} mg/kg/day.

Former Child Resident exhibiting pica behavior (one time exposure for a child 2 to < 6 years) = 620 mg/kg x 0.5 x 5,000 mg/day x 1 x 10^{-6}/17.4 kg= 8.9 x 10^{-2} mg/kg/day.

Former Child Resident exhibiting pica behavior (EF=3/7 for a child 1 to < 2 years) = 620 mg/kg x 0.5 x 5,000 mg/day x 0.4286 x 10^{-6}/11.4 kg= 5.8 x 10^{-2} mg/kg/day.

Former Child Resident exhibiting pica behavior (EF=3/7 for a child 2 to < 6 years) = 620 mg/kg x 0.5 x 5,000 mg/day x 0.4286 x 10^{-6}/17.4 kg= 3.8 x 10^{-2} mg/kg/day.

Table C-3. Estimated Increased Cancer Risk from Ingestion of Arsenic in Soil (Former Residents and Workers)

Former Resident	*Estimated Increased Cancer Risk***
Child	1 to 4.4* in 100,000 (see table below on cancer risk based on age of child)
Adult	4.5 in 1,000,000
Former Outdoor Worker with Low Soil Contact	1.6 in 100,000
Former Outdoor Worker with High Soil Contact	3.8 in 100,000
*Residents assume to have lived there approximately 5 years. Based on combining the upper percentile risk for a child 1 to < 2 years and 2 to < 6 years, for a total of five years. ** Based on the cancer risk upper percentile. The soil concentration of **73 ppm arsenic,** the average found in surface soil (2000 data), was used to estimate the risk. For chronic exposures (365 days or longer), the average soil concentration of **876 ppm arsenic** from composite (mixed) samples from TPs 134 and 120 was used in the exposure calculation for **outdoor workers** (2010 data).	

Based on estimates for the ingestion of soil arsenic, the increased cancer risk to workers was low. The cancer risk to workers who worked less than 1.5 years in arsenic-contaminated soil would be less than the above estimates. However, the cancer risk to workers could have included many other carcinogens if they were exposed to chemicals in broken containers or spilled from containers, however, we cannot quantify these exposures or increased cancer risks.

Reminder: Other carcinogens found at the site included arsine, benzene, carbon tetrachloride, sulfur mustard, chloroform, 1,2 DCA, PCE, TCE, and VC.

Cancer Risk Based on Age of Child:

Age Group	Mean Ingestion Rate (mg/day)	Upper Percentile Ingestion Rate (mg/day)	Age-Specific Body Weights (kg)	Years Exposed	Cancer Risk Upper Percentile	Cancer Risk Mean
Child 6 wks to < 1 yr	60	100	9.2	0.88	6.7E-06	4.0E-06
Child 1 to < 2 yr	100	200	11.4	1	1.2E-05	6.2E-06
Child 2 to < 6 yr	100	200	17.4	4	3.2E-05	1.6E-05
Child 6 to < 11 yr	100	200	31.8	5	2.2E-05	1.1E-05
Child 11 to <16 yr	100	200	56.8	5	1.2E-05	6.2E-06
Child 16 to <21 yr	100	200	71.6	5	9.8E-06	4.9E-06

Example Calculations for Table C-3:

Residents:

CANCER RISK = [Age-specific Annual Dose x Cancer Slope Factor (CSF) x Age-specific Number of Years of Exposure] / [Lifetime in Years] or CR = Age-specific dose x CSF x Age-specific # years / 78 years.

Adult: CANCER RISK =4.6 x 10^{-5}x 1.5 x 5/78 = 4.5E-06

Child 6 to < 11 yr:

Child Resident (6 to < 11 yrs): upper percentile dose = 73 mg/kg x 0.5 x 200 mg/day x 1 x 10^{-6}/31.8 kg=2.3 x 10^{-4} mg/kg/day.

CANCER RISK =2.3 x 10^{-4} x 1.5 x 5 /78 = 2.2E-05

Outdoor workers:

Low soil contact cancer risk = 5.5 x 10^{-4} mg/kg/day x 1.5 x 1.5/78 = 1.6 x 10^{-5}

High soil contact cancer risk = 1.3 x 10^{-3} mg/kg/day x 1.5 x 1.5/78 = 3.8 x 10^{-5}

See reference section of this health consultation for citations used in this appendix.

Appendix D—Exposure Pathways Tables

Table D-1. Exposure Pathways for Contaminants in Residential Yard and Inside Residence (Points of Exposure)

Exposure Pathways and Routes of Exposure to Contaminants:	**Exposed Population: Former Workers who developed the property**	**Exposed Population: Former Residents**
Potential Exposure Pathway		
Spillage from Containers		
Direct Contact with liquids/solids spilled from containers	No such exposure reported in excerpts of the transcripts or other records.	No known Exposure
Completed Exposure Pathways		
Soil		
Inhalation of soil and dusts	Exposure likely to mixed surface and subsurface soils and dust	Exposure likely to surface soils and dusts (indoor and outdoor)
Ingestion of soil and dusts	Exposure likely to mixed surface and subsurface soils and dusts	Exposure likely to surface soils and dusts
Skin Contact/Dermal absorption	Exposure likely to mixed surface and subsurface soils and dusts	Exposures likely to surface soils and dusts (indoor and outdoor)
Air		
Container to air releases	Some exposure during breakage of containers on the property	No known Exposure
Inhalation of outdoor or ambient air	Soil to air releases from chemicals in burial pits areas during construction of retaining walls, particularly along southern property boundary near former Burial Pits 2 and 3. Soil to air releases from other areas with contaminated debris (such as Test Pits in the yard).	Soil to air releases from chemicals in burial pits along southern property boundary and from other contaminated areas on the property.
Inhalation of indoor air (vapor intrusion and entry of outdoor air)	No to limited exposure because home was under construction.	Via cracks in foundation walls and floor of basement leading to vapor intrusion into the home. Entry of outdoor air into indoor air of home via drive-under garage.*

Eliminated Pathways		
Groundwater	Not used as drinking water or for any other household purposes.	Not used as drinking water or for any other household purposes.
Surface water	None on property	None on property
Food	Not applicable	No known gardens used for food consumption.
*USACE theorizes that a former pit was at the house location prior to it being constructed there and that the builder may have removed some or most of it during construction; This lead to the house being built in a depression/pit and the basement ceiling to being unusually high. Additionally, USACE theorized (based on investigative findings) that workers disturbed Pit 3 and having found some of the munitions and other debris, placed them near the house foundation [ATSDR 2010].		

Table D-2. Past Activities Associated with Soil Contact and Potential for Exposure

	Potential for Exposure to Contaminated Soil		
	Low	Medium	High
Potential Past Activities	• Living in Home. • Gardening in surface soil. • Using yard for recreational activities. • Finishing work	• Building home or developing the property after foundation work but during construction phase. • Laying utility lines, trenching. • Planting shrubbery or small trees.	• Constructing the foundation of the home. • Building retaining walls, particularly through or near burial pits. • Packing contaminated dirt behind retaining walls. • Planting large trees involving digging large holes.

Appendix E. Detailed Information on Environmental Investigations and Data

This section focuses on the environmental data (2000-2010), in potential and completed exposure pathways, in relation to contaminants found at the site (except for arsenic) and compares the levels found to ATSDR and other health comparison values. However, ATSDR was not able to further evaluate any contaminant detected above these health values, using our standard evaluation approach, because of the uncertainty as to whether they represent exposure point concentrations to either the former workers or residents. Appendix F provides general public health implications of the major contaminants detected at the site.

This section also provides more detail about CWA found in intact containers. Over 500 pounds of laboratory glassware/debris including dozens of intact bottles/containers, and tons of contaminated soil were recovered and removed from the property. The chemicals most often associated with these WWI items and contaminated soil were sulfur mustard, dichlorophenylarsine, lewisite, and arsenic (from organic and inorganic arsenicals). Additionally, volatile organic compounds (VOCs) present in soil gas are discussed.

Arsenic Trichloride Discovery

On March 29, 2010, an open clear-glass jar with a broken top containing dark oily soil was collected from test pit (TP)-134 at 6.5 feet below ground surface (bgs) [USACE 2011c]. A vapor sample identified arsenic trichloride. Sulfur mustard and 1,4-dithiane were detected in the solid sample. A gas chromatography/mass spectrometry analysis indicated arsenic trichloride and arsenic trioxide. The sample also contained other minor constituents: hexachloroethane (used in smoke munitions) and octasulfur (consistent with the presence of sulfur mustard), chlorinated aromatics, chlorodiphenylarsine, phosgene oxime, and triphenylarsine [USACE 2011d, Appendix L].

On April 5, 2010, hydrochloric acid (HCl) was detected in ambient air at a maximum of approximately 2,400 ppb on a HCl monitor in the vicinity of the arsenic trichloride discovery. The "smoking" was believed to be a reaction occurring between the vaporizing arsenic trichloride and moisture in the air which produces HCl and arsenic oxides. Arsenic trichloride is used to make lewisite. One hundred ninety-two (192) pounds of arsenic trichloride were reportedly produced at AUES [DC Gov 1996].

Test Pits Discoveries—Containers, munition, waste

Specifics on the contents of some of the intact containers were:
An intact bottle containing approximately a half-liter of sulfur mustard (75% mustard of unknown purity –although both sulfur mustard and 1,4-Dithiane were present) was found in the front yard. A bottle 7/8 full with clear liquid which was identified as carbon tetrachloride with phosgene oxime (a CWA) and 1,2,4-trichlorobenzene (a pesticide and solvent). p-Nitrochlorobenzene or PNCB, which was investigated at AUES as toxic smoke [AU 1919: CWM42] was found in an intact glass test tube recovered from the front yard along with other

contaminants. A bottle 75% full of liquid containing chloroacetic acid was found [USACE 2011d, Appendix E, Table E.11]. Benzoic acid was tentatively identified in multiple intact containers at the site. Although no concentrations were given, acids (benzoic acid, benzenecarboxylic acid, 2-chlorobenzoic acid and chloroacetic acid) and other chemicals were identified or tentatively identified in the intact containers [USACE 2011d, Appendix E, Table E.11].

Sulfur mustard and its ABPs were identified in a flask glassware item in TP-138 [USACE 2009b]. Another flask, which was found later in the general area, contained lewisite. The contents of these intact containers were analyzed. The highest quantitative result for sulfur mustard was 3,100,000 ppb in sample SVS-10-006 [USACE 2011d, Appendix E, Table E.11]. The maximum concentrations for the sulfur mustard ABPs 1,4 dithiane and 1,4-oxathiane in this sample were estimated at 17,000,000 and 660,000 ppb, respectively. The highest concentration of lewisite was estimated at 900,000 ppb in sample TP-134-TE-019 which also contained sulfur mustard [USACE 2011d, Appendix E, Table E.11]. It is reasonable to assume that if product levels of sulfur mustard and lewisite from intact containers were spilled into soil, some soil concentrations would exceed our health based comparison values. No soil samples for these contaminants were collected and analyzed during property development [Soil samples analyzed approximately 18 years later indicated that maximum concentrations of sulfur mustard and lewisite did exceed our comparison values (Table E-1)]. The general public health implications of these contaminants are discussed in Appendix F and they are also discussed in body of the report in relation to our occupational medical officer's evaluation of the worker interview transcripts.

Sulfur mustard agent was detected in glassware excavated from TP-138 in the back yard. A confirmed detection of sulfur mustard agent and sulfur mustard agent breakdown products was reported for a substance inside of a flask glassware item in TP-138 [USACE 2009b]. The white solid in the flask contained 1,600 ppb sulfur mustard, 800 ppb 1,4-dithiane, and 1,000 ppb 1,4-oxathiane [USACE 2011d, Appendix D, Table D.9].

CWAs in Munitions

Items excavated from the 4825 Glenbrook Road side of Burial Pit 3 and its extensions included 508 munition-related items [including munitions and explosives of concern and munitions debris] and 23 Chemical Warfare Materiel (CWM)-related items (projectiles/munitions containing arsine, lewisite, or sulfur mustard and other chemicals). Some headspace samples, collected from munitions and explosives along with their debris, tested positive for sulfur mustard and lewisite.

Items recovered from Burial Pit 3 (TP-23 area) included an arsine gas shell, the contents of which were confirmed and two additional shells which are believed to have contained arsine (Additionally, three arsine gas shells were found in Test Pit 23 on the 4801 Glenbrook Road property.)

Dozens of WWI-related items were found touching the concrete foundation of the house and/or retaining walls. Munitions and their debris were buried next to the house foundation. The amount of AUES-related glassware recovered in the 2007-2010 timeframe was approximately 510 pounds.

Chemicals in Soil

In addition to waste in containers which were buried in the yard, discolored soil and solids were found outside of containers (non-containerized waste). Twenty-two grab samples were taken from front yard test pits and the results are summarized in Table E-1 [USACE 2011d, Appendix E, Table E.11]. Lewisite was detected in seven samples ranging from approximately 61 to 14,000 ppb with the maximum concentration detected in TP-134. Sulfur mustard was detected in seven samples ranging from 21 to 150 ppb with the maximum concentration detected in TP-120. Some sulfur mustard and lewisite concentrations in these grab samples exceeded HBESLs which were developed to address potential long-term or chronic exposures to residual concentrations [ORNL 2007]. In addition, a comparison value (intermediate EMEG) for a child exhibiting pica behavior was exceeded for sulfur mustard. Sulfur mustard ABP, 1,4-dithiane, was found in 10 samples having concentrations less than the soil comparison value. Chloroacetophenone (CN), a tearing agent, was found in two samples but the concentrations were not quantified.

In 2010, composite soil samples were taken from drummed soil which originally came from TPs 120 and 134 in the front yard (Table E-1). Lewisite was detected in 20% (in 50 soil samples out of 250 samples analyzed) of the soil samples at concentrations ranging from approximately 26 to 1100 ppb. Sulfur mustard and/or its ABPs (1,4-dithiane and/or 1,4-oxathiane) were detected in 28% of the soil samples. Some sulfur mustard and lewisite concentrations in these composite samples exceeded HBESLs. In addition, a comparison value (intermediate EMEG) for a child exhibiting pica behavior was exceeded for sulfur mustard. As previously indicated, the general public health implications for these contaminants can be found in Appendix F. However, due to lack of exposure point concentrations for the former residents, ATSDR could not further evaluate the implications of these levels found in soil.

In addition to some CWAs and their breakdown products, other chemicals were present in soil such as carbon tetrachloride and chloroform. However, they were below ATSDR CVs. There were traces of other VOCs in soil below ATSDR CVs: acetone, benzene, chlorobenzene, chloroform, ethylbenzene, methyl tertbutyl ether, methylene chloride, toluene and total xylenes. There were traces of semi volatile organic compounds (SVOCs) that did not have ATSDR CVs: bis (2-ethylhexl) phthalate, diethyl phthalate, and phenyl isocyanate. Other contaminants in trace levels included fluoride, iodine pentafluoride, and perchlorate.

Lewisite was detected in soil samples in the back yard porch area (TP-138). In 2010, fifteen soil samples were taken in the backyard of 4825 Glenbrook Road to determine if CWA or their ABPs were present in the backyard. Lewisite, estimated at 70 and 47 µg/kg (ppb), was detected in two backyard samples (MM2 and PP2) collected in August 2010 [USACE 2011c and d, Appendix E, Table E.12]. These concentrations were not above soil comparison values. Lewisite, estimated at 70 and 47 µg/kg (ppb), was detected in two backyard samples (MM2 and PP2) collected in August 2010 (collected at 3 feet below ground surface) [USACE 2011c and d, Appendix E, Table E.12]. These concentrations were not above soil comparison values. Twelve surface soil

Table E-1. Non-containerized Waste and CWAs in Soil [mixed surface and subsurface] of the Front Yard (2010) [USACE 2011d, Appendix E, Table E.11].

Contaminant	Soil Comparison Values (ppb)	Range of Waste in Soil Concentrations (ppb) in grab samples*	Range of Soil Concentrations (ppb) in composite samples‡	Maximum Conc. Exceeds a Comparison Value? Yes or No
Lewisite	No ATSDR CV 300 HBESL*	61 J-14,000 D 32% (7 detections/22 samples analyzed)	26 J-1,100 D§ 20% (50 detections/250 samples analyzed)	**Yes** for both waste in soil and soil
Sulfur mustard	1,000 acute EMEG Pica Child, 140 int. EMEG Pica Child, 10 HBESL *	21-150 D† 32% (7 detections/22 samples analyzed)	1.9 J-590 D 9% (23 detections/250 samples analyzed)	**Yes** for both waste in soil and soil
Sulfur Mustard ABP: 1,4-Dithiane	500,000 RMEG Child, 78,000 RBC†	24 J-4,000 D	20 J-5,200 D	No for both waste in soil and soil
Sulfur Mustard ABP: 1,4-Oxathiane	No ATSDR CV 78,000 RBC†	22 J-1500 D	20 J-260	No for both waste in soil and soil
Sulfur Mustard ABP: Thiodiglycol	No ATSDR CV 39,000 RBC†	Not Analyzed	<250	No for soil
Sum of sulfur mustard and its 3 primary ABPs for a total %.	As previously indicated above.	As previously indicated above. 68% (15 detections/22 samples analyzed)	As previously indicated above. 28% (71 detections/250 samples analyzed)	

*Grab Samples, SW-4825GB-GS01 through GS22, from TPs 120 and 134 in the front yard (2010). Chloroacetophenone (CN), a tearing agent, found in 2 samples (GS-08 a chalky white substance and GS-14 with chunks of wax or chalky substance) but not quantified.

†4 samples show detection of sulfur mustard in headspace analysis results although the concentration was not quantified.

‡Soil (Drum Composite Samples) from TP-120 and TP-134 in the front yard (2010).

§Most samples containing lewisite also contained sulfur mustard or one or more of its ABPs, only 3 samples contained just lewisite.

Qualifiers: J- Result is an estimated value, D-Sample was diluted Qualifiers.

ATSDR's comparison values (CV) are screening levels used to determine if further evaluation is needed:

CREG: Cancer Risk Evaluation Guide

EMEG: Environmental Media Evaluation Guide (acute, intermediate (int.), or chronic exposures)

RMEG: Reference Dose Media Evaluation Guide

HBESL: USACHPPM's Health-Based Environmental Screening Level (1999 HBESL): developed to address potential long-term or chronic exposures to residual concentrations of CWAs [ORNL 2007].

RBC: USACE's Risk-Based Concentration [USACE 2003]

samples (taken at 6 inches below ground surface) were analyzed for sulfur mustard, lewisite, 1,4-dithiane, and 1,4-oxathiane. These CWAs were not detected in surface soil samples from the backyard. Although no samples were available to the rest of the yard, this is not likely an important data gap for residential exposures to soils as these CWAs are not likely to persist in surface soils due to their physical properties.

Sulfur mustard ABPs were detected in a soil sample in the back yard porch area (TP-138). A 2009 grab sample from TP-138 contained 510 ppb of 1,4-dithiane and approximately 50 ppb of 1,4-oxathiane [USACE 2009b]these concentrations were not above soil comparison values.

Soil Gas Sampling Results

ATSDR summarized soil gas results (Table E-2) because of the potential for soil gas to enter the home through vapor intrusion and entry of outside air via the drive-in basement (with bedrooms and other living spaces). Soil gas results were compared with ATSDR air comparison values; these results were gathered 18 to 20 years after development of the property so ATSDR does not know if these represent actual exposures concentrations to the residents. Given local soil excavations of burial pits and the time period between exposure and sampling, VOC data are unlikely to represent worse case concentrations for soil gas and ambient air exposures.

The house was reportedly poorly constructed and there were obvious routes for soil gas infiltration into the home [Information from Dan Noble (Project Manager for the USACE) sent to L. Frazier via email on 7/10/2014]. The home was topographically in an open-ended depression such that CWAs that were heavier than air would have lingered. Potential existed for release of gases to ambient air on the property via retaining walls near burial pits (leaking munitions, containers, etc.) and other retaining walls on the property. The basement level was a drive-in garage with bedrooms on this level. The opening of the garage door could have allowed entry of volatile contaminants and dust into the home. No indoor air samples were taken in the home during or after occupancy by the residents.

Soil gas was sampled in three areas: Trench 2, Burial Pit 3 and its extensions, and TP-120. The sampling locations are shown on Figure 4 Appendix A and Table E-2 is a summary of the soil gas sampling results collected by summa canister in 2007 and 2009. The highest concentrations of soil gas were found in TP-120 (which also contained some glassware). During investigation of Burial Pit 3, there were unconfirmed detections of arsine or another gas or gases at the Command Post (east of 4825 on the AU Kreeger Music Roadway) but there was no known or identified source (no excavation was occurring in Burial Pit 3). Low levels of VOCs were detected in various areas of Burial Pit 3; however, other locations usually had higher concentrations. Therefore, Table E-2 does not contain Burial Pit 3 data.

An ambient air sample was collected nearby (on the other side of the sidewalk to the front door of the home) [Figure 4Appendix A] [USACE 2011c]. There were traces of methane compounds and toluene in ambient air. Benzene was detected above its comparison value (Table E-2). Although these levels were above ATSDR's comparison value, it could not be further evaluated because of the uncertainty as to whether the former residents were exposed to these levels.

Trench 2

A soil gas and soil sampling investigation of the driveway area was conducted in March 2007, concurrently with work on Burial Pit 3 [USACE 2009a, 2011c]. Sulfur mustard agent breakdown products (ABPs), 1,4-oxathiane at 2.93 parts per billion by volume (ppbv) and 1,4-dithiane at 11.21 ppbv, were detected in a soil gas sample collected within Trench 2 [near the driveway and 4835/4825 property boundary, at a depth of 2 feet (sample Gore/SG-4(2'), Figure 4 Appendix A] [USACE 2009a, 2011c]. However, sulfur mustard ABPs were not detected in a soil sample taken near the soil gas location.

Summa canister soil gas results (Table E-2) indicated several VOCs for which ATSDR had a comparison value that were exceeded: benzene, chloroform, 1,2 Dichloroethane (1,2 DCA), tetrachloroethene (PCE), trichloroethene (TCE), and vinyl chloride [USACE 2011c]. Most of these contaminants were at low levels and may be breakdown products or impurities in other chemicals. VOCs were present for which there were no comparison values such as 2,2,4-trimethylpentane.

TP-120

Summa canister soil gas samples were collected from TP-120 in 2007 and 2009 (Table E-2). VOCs that had concentrations of several hundred ppbv in soil gas were benzene, ethylbenzene, toluene, 1,2,4-trimethylbenzene, and xylenes (Table E-2). These contaminants exceeded comparison values for air except for 1,2,4-trimethylbenzene for which there is no ATSDR comparison value.

Samples Beneath the Home

Concurrently collected with the backyard samples, samples from six geotechnical borings were taken beneath the basement slab [USACE 2011c]. These sub-slab grab samples were analyzed for VOCs, SVOCs, metals, explosives, total cyanide, iodine, fluoride, and perchlorate [USACE 2011c]. The geotechnical borings did not appear to contain sulfur mustard or lewisite or their breakdown products. However, some VOCs, SVOCs, and other contaminants (such as acetone, chlorobenzene, methylene chloride, bis(2-ethylhexyl)phthalate, fluoride, cyanide, and mercury) were identified [USACE 2011f]. Arsenic concentrations were less than 20 ppm.

Table E-2. VOCs in Soil Gas and Ambient Air (Summa Canister 2007 & 2009)

Contaminant: **VOCs**	**Air Comparison Value (CV) (ppb)**	**Max. Soil Gas Concentration (ppbv)*>CV at given location**	**Location of Maximum Conc.**	**Ambient Air† (ppbv)**
Benzene	CREG 0.04	2.3 313	Trench 2 TP-120	0.31
Carbon tetrachloride	CREG 0.026	80	TP-120	ND
Chloroform	CREG 0.0089	1.6 26	Trench 2 TP-120	ND
1,2 Dichloroethane (1,2 DCA)	CREG 0.0095	1.5	Trench 2	ND
Ethylbenzene	Chronic EMEG, 60	282	TP-120	ND
Tetrachloroethene (PCE)	CREG 0.57	15 34	Trench 2 TP-120	ND
Toluene	Chronic EMEG, 80	1,200	TP-120	0.3
Trichloroethene (TCE)	CREG 0.045	4.4 6.2	Trench 2 TP-120	ND
1,2,4-Trimethylbenzene	No ATSDR CV	610	TP-120	ND
2,2,4-Trimethylpentane	No ATSDR CV	25	Trench 2	ND
Vinyl Chloride	CREG 0.044	2.4	Trench 2	ND
m&p-xylene	Chronic EMEG, 50 ppb total xylenes	1,510	TP-120	ND
o-xylene	Chronic EMEG, 50 ppb total xylenes	561	TP-120	ND

*Summa Canister samples were collected in 2007 [USACE 2011c, Tables D.2 and D.3]. TP-120 samples were also collected in 2009 by summa canister [USACE 2011d, Appendix I.] Summa canister results from 2007 indicate TICs (tentatively identified compounds, qualitative data) for 12 volatile organics which have no ATSDR comparison values and 15 unknown compounds.
†Ambient Air was sample 4825 GR-SG-6.
ND = not detected.
Isopropyl Alcohol (no ATSDR CV; a solvent) was found on the property at a maximum of approximately 120 ppb.
ATSDR's comparison values are screening levels used to determine if further evaluation is needed: CREG: Cancer Risk Evaluation Guide, EMEG: Environmental Media Evaluation Guide.

Many of the contaminants found beneath the home were also found in TP-138, in the backyard. Chlorobenzene, used in many pesticides, was found in both places. It can be used to make chloronitrobenzene (also called p-Nitrochlorobenzene or PNCB), which was investigated at AUES as toxic smoke [AU 1919: CWM42]. PNCB was found in an intact glass test tube recovered from the front yard (Sample SVS-10-018) along with other contaminants.

2013-2014 Sampling during remedial activities

From February 2013 to August 2014, the USACE collected additional environmental samples while performing ground intrusive activities at the 4825 Glenbrook Road NW property. The 2013/14 sampling data included the following:

- air data from open air digs, headspace (vapor screening) clearance prior to sample shipment, and the filtration system;
- soil data (disposal and confirmation);
- data from intact containers found at the site;
- sampling data from the concrete wall prior to demolition; and
- data from wipe samples collected from the surfaces of structural beams inside the Engineering Control Structure.

The chemical detects and concentrations in the soil, concrete and wipe samples were similar to those in previous samples. Because the chemical constituents and concentrations were similar, the potential health impacts are similar to those previously described. However, the testing did reveal the presence of two contaminants - magnesium arsenide and dichloronaphthalene - not previously detected in environmental samples from the site. The sampling data only showed that the chemicals were detected, but *did not include the concentrations of the chemicals in the environmental media*. Therefore, ATSDR is only able provide general health effects information about the chemicals, but cannot conclude whether those health effects are likely or unlikely (see Appendix F).

Approximately 50 intact containers (e.g., glass bottles, sealed test tubes/vials, projectiles, copper vessels) were recovered during remedial operations at the property. Nineteen (19) of the containers contained a chemical agent or a chemical agent breakdown product.

Magnesium arsenide in Artillery Projectile

In January 2014, a 75mm artillery projectile recovered from the site was found to contain an unknown arsenical compound consistent with the profile for magnesium arsenide. [MARB 2014] Magnesium arsenide had not been previously detected in any samples from the site. Magnesium arsenide was detected in an intact munition, but former workers or residents may have come into contact with the content of the munition in the past if a container was broken during disturbances of the soil. ATSDR does not know to what extent, if any, former workers or residents may have been exposed to magnesium arsenide.

Historical reports indicate that chemists at AUES were interested in using metallic arsenides in weapons. [Information from Dan Noble (Project Manager for the USACE) sent to T. Foster via email on 2/13/2015].

Dichloronaphthalene in Pre-filter Air

During soil sampling in March and April 2014, USACE's near real time air monitors indicated the presence of Lewisite during ground intrusive activities under one of the tents. The air monitors indicated the presence of Lewisite on four different days; each alarm was below the action level for Lewisite. The USACE collected the air samples to perform additional testing to confirm the presence of Lewisite. The tests came back negative for Lewisite; the alarms were deemed false positives. Further investigation determined that the chemical detected was dichloronaphthalene [USACE 201]. ATSDR does not know to what extent, if any, former workers or residents may have been exposure to dichloronaphthalene.

See reference section of this health consultation for citations used in this appendix.

Appendix F: General Public Health Implications of Several Agents found at the 4825 Glenbrook Road

This appendix provides information on the general public health implications of important contaminants detected in several media at the site. As stated in the main body of this health consultation, except for arsenic, ATSDR was not able determine site-specific public health implications for these contaminants using our standard evaluation approach. ATSDR's site-specific conclusions regarding past exposures to workers is based on our occupational medical officer's evaluation of the interview transcripts and our evaluation of arsenic exposures for former workers and residents (see Public Health Implications in main body of this health consultation).

Spills and Worker Exposure Incidents

Chemical warfare agents in concentrated forms can be highly toxic upon direct contact. Most intact containers recovered on the property contained sulfur mustard or its ABPs. Based on chemicals found in containers at 4825 Glenbrook Road [USACE 2011d, Appendix E, Table E.11], other CWAs (examples are chlorodiphenylarsine, lewisite, arsenic trichloride, and hexachloroethane) and chemicals (solvents and chemicals used in pesticides/herbicides) were mixed with sulfur mustard at the AUES. Many of these chemicals as well as the acids could also have affected the eyes, airways, and skin and contributed to injury if exposure occurred. One purpose of experimentation at AUES was to increase the effectiveness of the chemicals to cause injury. Exposure to CWAs, without proper protective equipment, is a health hazard with the possibility of short and long-term health effects.

There was an acute health risk to workers from concentrated areas of chemicals on the 4825 Glenbrook Road property due to the breakage of glass containers and release of their contents into the environment. Depending upon the properties of the chemical or chemical mixture and environmental parameters, these spills would have created hot spots of concentrated chemicals in the soil and lead to soil gas and ambient air concentrations with potential for health impact. In ambient air, lewisite is about ten times more volatile than sulfur mustard [Smith et al. 2010] However, using the less volatile example of sulfur mustard: if a spill of liquid sulfur mustard remained in surface soil and was not immediately covered or was later uncovered, it is reasonable that workers, in the immediate vicinity, who were exposed to sulfur mustard in air could have had effects such as irritation to the eyes, skin, and possibly lungs. Airborne concentrations of chemicals from spills/releases from intact containers could have been at harmful levels.

Arsenic Trichloride and Hydrochloric Acid Vapors

Pure arsenic trichloride is colorless but if impure, it is a yellowish to slight brownish oily liquid with an acrid odor [Delepine 1923]. When exposed to air, it emits fumes and evaporates fairly rapidly [Delepine 1923]. It decomposes in moist air to hydrochloric acid (HCl) and arsenious oxide [Marrs et al. 2007]. It decomposes by water to form arsenic hydroxide and hydrogen

chloride. For spills, some precautions are to keep out of low areas and ventilate closed spaces before entering them [Sittig 1991].

During the 2010 excavations, a release of HCl occurred within the engineering controls set up by USACE and was filtered before any outside venting occurred. However, this indicates that former workers could have experienced similar releases with resultant exposures to HCl. HCl is a sensory irritant which can cause pulmonary irritation and other effects at certain levels of exposure. The maximum concentration detected by the HCl monitor at 4825 Glenbrook Road was 2,400 ppb and it only lasted for a short time (the exposure duration indicates the possibility of an acute exposure but not a chronic one).

Based on a chronic study which used only one dose and limited toxicological measurements, the LOAEL for HCl is 10,000 ppb (15 mg/m^3) [USEPA 2003]. The site concentration of 2,400 ppb is less than the LOAEL based on a chronic study and less than the LOAEL of 4,086 ppb (6.1 mg/m^3) based on hyperplasia of laryngeal-tracheal segments in HCl exposed rats [USEPA 2003]. The LOAEL of 4,086 ppb was calculated from a rat chronic inhalation study for a gas: respiratory effect in the extrathoracic and tracheobronchial regions. However, 2,400 ppb exceeds the LOAEL (Adjusted for longer term exposures) of 1,810 ppb (2.7 mg/m^3), calculated from the LOAEL of 10,000 ppb (15 mg/m^3* 6hrs/24hrs*5 days/7days). USEPA has a Reference Concentration (RfC) – an estimate of a daily inhalation exposure that is likely to be without an appreciable risk of deleterious effects during a lifetime –of 13.4 ppb (0.02 mg/m^3) for HCl. Neither ATSDR nor USEPA have a comparison value for short-term inhalation or acute exposure; however, OSHA and NIOSH have an occupational acute value of 20 ppm [OSHA 2015] and the National Oceanic and Atmospheric Administration or NOAA has several short-term Emergency Response and Planning Guidelines or ERPGs [USDOC 2015].

A controlled human exposure study of HCl was conducted on ten asthmatics exposed to 800 or 1,800 ppb HCl for 45 minutes. Pulmonary function tests performed immediately after exposure was compared to baseline levels. No exposure-related effects were observed in subjective symptoms or in pulmonary function tests [USEPA 2003].

Although some short-lived irritation could have been experienced by an exposed worker, long-term health effects are not anticipated based on the maximum concentration of 2,400 ppb HCl as detected by an HCl monitor for a limited duration exposure. Consideration of the source of the HCl, arsenic trichloride, indicates that workers in the immediate vicinity could have experienced irritation to the nose, throat, and lungs. It also indicates an exposure to inorganic arsenic. If an arsenic trichloride release resulted in doses or durations that were higher, longer, or more repetitious than previously described, they could have had a greater health impact.

Long-term or repeated exposure to arsenic trichloride can lead to harmful health effects. Chronic poisoning can lead to peripheral nerve damage, skin conditions, and liver damage. According to the International Agency for Research on Cancer (IARC), arsenic trichloride is carcinogenic to humans [IARC 1997]. It has been implicated in the induction of skin and lung cancer. Long-term or repeated exposure may result in pigmentation disorders, hyperkeratosis, perforation of the nasal septum, neuropathy, liver impairment and anemia [NIOSH 2011].

Sulfur Mustard

Sulfur mustard and its degradation products were found at low levels in the 2000s in soil and waste in soil such that workers performing work tasks in contaminated areas of the yard would have been likely to come in contact with this contaminant; however, we do not know what the doses would have been in the 1990s. Although sulfur mustard was found at maximum concentrations of several thousand ppm (maximum of 3,100 ppm or more) in intact containers at the site, the soil concentrations (0.59 ppm or less from sampling in 2010) were much lower. Sulfur mustard in soil had degraded to breakdown products and even those concentrations were low such as a maximum of 5.2 ppm 1,4-dithiane. Additionally, no quantitative results are reported for sample SVS-10-22 (approx.1/2 liter of sulfur mustard of unknown purity recovered in the front yard) in which sulfur mustard and 1,4-dithiane were present in a container at concentrations that saturated the detector for a prolonged period. Although sulfur mustard in soil of test pits and burial pits was found during the investigations, surface soil data for this contaminant or its ABPs were not available.

Product levels of sulfur mustard can be in the tens to hundreds of thousands of ppm. Exposure to product-type levels, in hot spot areas, could have resulted in adverse health effects, particularly if a worker with high soil contact were to ingest and dermally contact very contaminated soil. For example, if the 3,100 ppm sulfur mustard in the intact container (the maximum concentration quantified from the 2010 data) spilled into the soil and workers were exposed to a similar concentration, the acute MRL of 0.0005 mg/kg/day would be exceeded for outdoor workers with high soil contact ingesting soil in an acute exposure scenario. If product levels of hundreds of thousands of ppm sulfur mustard were ingested by outdoor workers with high soil contact, the LOAEL of 0.5 mg/kg/day would be exceeded. However, the actual historical levels of sulfur mustard in soil in the 1990's are not known.

The active ingredient in sulfur mustard is bis(2-chlorethyl)sulfide. Sulfur mustard can remain in soil for months to years and is usually present in the form of encapsulated globules, the coatings of which prevents further dissolution and degradation. If these capsules are broken, the potential for an acute hazard is high [USACPPM 1999]. However, the actual historical levels of sulfur mustard in soil at 4825 Glenbrook Road in the 1990's are not known.
Inhalation of sulfur mustard can cause damage to the respiratory tract within a few days of exposure and can cause coughing, inflammation of the airways (nose, throat, trachea, bronchi, and lung), and hoarseness. Over the long-term, after substantial exposures, it can cause bronchitis, pneumonia, respiratory disease, and occasionally death. Exposure to sulfur mustard can also~~,~~ cause nausea~~,~~ and vomiting.

Sulfur mustard is a known human carcinogen such that long-term exposure may lead to cancer of the upper respiratory airways [ATSDR 2003]. IARC has determined that sulfur mustard is carcinogenic to humans. The Department of Health and Human Services (DHHS) has also determined that sulfur mustard is a known carcinogen. It may cause cancer in your airways, lungs, skin, and maybe other areas of your body later in life [ATSDR 2003]. It can suppress the immune system.

Skin/eye absorption of sulfur mustard may occur after contact with liquid or vapor (in the immediate vicinity of the liquid). Mild eye irritation and redness is the first effect from air

exposure. Warm, moist areas with thin skin, the moist linings of body passages and cavities (mucous membranes), and perspiration-covered skin are more sensitive to the blistering (vesicant) effects of sulfur mustard. Short-term exposure to sulfur mustard can cause eye and skin burns, eyelid swelling, and fluid blisters within a few days. There may be increased pigmentation or a darkening and de-pigmentation or lightening of exposed skin. Skin injury can resemble sunburn or small rash-like dots and is usually accompanied by itching, burning, or stinging [Hurst et al. 2008].

After a significant amount of sulfur mustard has been absorbed through the skin or inhaled, sulfur mustard can also affect a part of the nervous system responsible for everyday bodily function, causing "cholinergic toxicity," marked by excessive saliva, tears and urine; gastrointestinal cramping and diarrhea; vomiting (emesis); and constricted or pinpoint pupils (miosis). Long-term effects of high concentration exposures to sulfur mustards include scarring of skin, eye damage, and possibly cancer [ATSDR 2003)].

In experimental studies, application of 2.5 µg of sulfur mustard to human skin resulted in erythema (redness of the skin) in 87 of 209 individuals and blistering in 5 of 209 [USACHPPM 1999]. Human and animal data indicate that sulfur mustard doses of only a few micrograms (received in a single discrete exposure) are likely to cause erythema in a large percentage of exposed individuals and may even cause blistering in some sensitive individuals. In skin contact studies conducted on rats, a dose of 0.03 mg/kg/day (about 0.01 mg/animal) caused no toxic effects or produced only mild signs of toxicity after repeated exposures for 13 weeks [USACHPPM 1999].

ATSDR's MRL for acute-duration oral exposure is 0.5 µg/kg/day (0.0005 mg/kg/day) and 0.07 µg/kg/day (0.00007 mg/kg/day) for intermediate-duration oral exposure; ATSDR has no oral MRL for chronic exposure [ATSDR 2003]. Additionally, a LOAEL of 0.5 mg/kg/day is based on inflamed mesenteric lymph nodes (membrane encircling the small intestines) in the female rat and reduced ossification (formation of bone) in the fetuses of rats (dose used for acute MRL derivation). No information is available on minimum effect levels for ingestion of sulfur mustard in humans. In rats, severe damage to the gastric mucosa occurred from a daily dose of 2.5 mg/kg (about 0.8 mg/animal) for 14 days. A LOAEL of 0.03 mg/kg/day is based on gastric lesions in rats which can be translated to potential for tissue damage in the gastrointestinal tract for humans (dose used for intermediate MRL derivation).

Lewisite

We do not know if workers developing the property in the 1990s came in contact with lewisite or its ABPs in soil. From sampling in 2010: Six containers from TP-134 in the front yard, next to the home, contained lewisite and sulfur mustard; the maximum concentration of lewisite was 900,000 ppb. The maximum concentration of lewisite in soil visually contaminated with waste was 14,000 ppb and the maximum in soil was 1,100 ppb. Given that lewisite in a container could have broken and released its content into soil, the soil concentration could have been high for some workers but ATSDR does not have actual exposure levels to verify this. Based on this scenario, if a former worker had contacted or ingested highly contaminated soil, they could have experienced adverse health effects as previously described. However, the actual historical levels of lewisite in soil in the 1990's are not known.

Lewisite, classified as an organic arsenical, is a complex mixture of several compounds, with dichloro(2-chlorovinyl)arsine predominating. Inhalation of lewisite vapor can cause immediate burning pain of the respiratory tract, burning in the nose and sinuses, laryngitis, coughing, and shortness of breath. Tissue damage to the airways and the accumulation of fluid in the lungs could result in death. Inhalation of vapors from a sulfur mustard and lewisite mixture induces immediate respiratory tract irritation and severe inflammation after a few hours. Both agents are readily absorbed from the lungs.

Skin/eye absorption may also occur after contact with liquid or vapor lewisite. Dermal contact with lewisite results in local pain, swelling, and a rash, followed by blistering that might be delayed for hours. If it contacts the eyes, it will cause immediate pain and rapid swelling, as well as serious damage to the cornea and other parts of the eye.

Ingestion of lewisite or its sulfur mustard mixture can lead to local effects and systemic absorption [ATSDR 2002]. It will burn the mouth and throat and cause severe stomach pain, nausea, vomiting, and bloody stools. If some of the lewisite and mustard-lewisite that you breathe, touch, or ingest pass into your blood stream, it can cause bone marrow damage and fluid loss from the blood vessels, which could results in low blood pressure and damage to the rest of the body.

Due to extremely rapid hydrolysis, the toxicological data associated with lewisite may be more representative of its degradation products: CVAA (2-chlorovinyl arsonous acid; soluble in water and non-volatile) and CVAO (lewisite oxide or chlorovinyl arsenous oxide; a dehydration product of CVAA) [USACHPPM 1999]. Since CVAO is a solid, exposure to particulates containing arsenic is possible (Exposure doses for arsenic have been estimated and detailed in Appendix C. Exposure to arsenic via particulates would be a much smaller dose compared to ingestion and has not been estimated). Skin contact with liquid lewisite or CVAA is possible when these chemicals are present in containers or soil. Lewisite breakdown products, lewisite oxide and CVAA, are considered to be as toxic as lewisite itself [USACHPPM 1998]. Lewisite presents a potential systemic poisoning hazard due to inorganic arsenic released from metabolism of the organoarsenical agent. The oral RfD for lewisite is 1.0E-04 mg/kg/day; however, the RfD of 3.0E-04 mg/kg/day for inorganic arsenic can be used. The median threshold dose for blistering has been reported to be 14 µg. A dose as low as 3.5 µg was reported to cause erythema in 27 out of 93 individuals and blisters in 8 of the 93 [USACHPPM 1999]. A dermal reference dose of 0.0000017 mg/kg/day has been derived from acute toxicity data [USACHPPM 1999]. On the skin, the amount of liquid lewisite needed to cause harmful effects is minimal. No epidemiological or experimental data indicate that lewisite is a carcinogen; however, the lewisite breakdown product, inorganic arsenic is considered to be carcinogenic.

Volatile Organic Compounds (VOCs)

Workers packing soil behind the retaining wall near TP-120 may have been exposed to soil gases during that activity (anticipated to last 2 weeks or less and to represent an acute exposure duration). In 1992, when most reported exposures occurred, the soil gas concentrations were likely to have been higher. ATSDR considered the likelihood of health effects from this

potential short exposure duration. The chemicals exceeding comparison values are listed in Table E-2, Appendix E. With respect to these contaminants and health effects, benzene is a primary VOC of health concern due to its toxicity. Benzene was found in soil gas and ambient air above its comparison value (Appendix E, Table E-2). The soil gas concentration of benzene in TP-120 was 313 ppb. Much higher levels of benzene in air (700,000 ppb -3,000,000 ppb) can cause drowsiness, dizziness, rapid heart rate, headaches, tremors, confusion, and unconsciousness [ATSDR 2007b]. Breathing fresh non-contaminated air helps to alleviate effects. The concentrations in TP-120 would have had to be significantly higher in the past for workers to experience these health effects. The concentrations of VOCs in soil gas of TP-120, if similar to those in the early 1990s, are not likely to have impacted the health of workers spending a limited amount of time there. However, no sampling for VOCs occurred during the 1990s.

With regard to residential exposures, soil gas could have migrated into the basement. However, without indoor air sampling prior to soil disturbance and removal of contaminants, we do not know how much contamination might have entered the home given that no indoor air samples were taken during occupation of the home and prior to removals of contaminated soil and WWI-related items. VOCs (benzene, carbon tetrachloride, chloroform, etc.) were present at 4825 Glenbrook Road in TP-120 near the walkway to the front door (Appendix E, Table E2). Most of these contaminants were detected in soil gas at low levels (ppb by volume). Soil gas data collected in 2007 and 2009 (Appendix E, Table E-2) indicate potential for chronic exposure to low levels of chlorinated solvents in indoor air. The house was reportedly poorly constructed and there were obvious routes for soil gas infiltration into the home [Information from Dan Noble (Project Manager for the USACE) sent to L. Frazier via email on 7/10/2014]. Although the soil gas concentrations in the 1990s are not known, they are likely to have been higher in the past; however, as stated before, we do not have indoor air measurements so we cannot determine what exposures and subsequent public health implications might have been. Benzene is a known human carcinogen where long term exposure can lead to anemia and a compromised immune system [ATSDR 2007b]. Given these potential exposures to benzene and other site carcinogens (such as sulfur mustard and carbon tetrachloride), the residents may have an increased cancer risk if there were carcinogens in indoor and/or ambient. However, as previously stated, these risks cannot be estimated.

Magnesium Arsenide

Arsine was developed as a chemical war agent (CWA) by other countries and by the U.S. in World Wars I and II, respectively. The process involved producing arsine in a projectile by breaking vessels containing magnesium arsenide and sulfuric acid and allowing their contents to mix. The method adopted by the US involved dispersing the arsenides, which would react with atmospheric moisture or rain to liberate arsine. [Garrett *et al*. 2007]. The goal was to spread the arsenides in the immediate environment of the release.

Metal arsenides, such as magnesium arsenide, react with acids and/or moisture to form highly toxic arsine gas. Arsine is a colorless gas with a distinct, garlicky odor. Exposures to arsine induce vomiting, and can produce convulsions, loss of consciousness, paralysis and respiratory compromise at high exposures. Exposure to arsine in sufficient quantities can be fatal [NIOSH 2015a].

Arsenides are toxic because of the inherent toxicity of arsenic. Acute arsenic poisoning from ingestion results in marked irritation of the stomach and intestines with nausea, vomiting and diarrhea. In severe cases, the vomitus and stools are bloody and the exposed subject goes into collapse and shock with weak, rapid pulse; cold sweats; coma; and death. Chronic arsenic poisoning may cause disturbances of the digestive system such as loss of appetite, cramps, nausea, constipation or diarrhea. Some magnesium salts have produced muscle weakness, cardiac arrhythmias, respiratory effects and changes in blood chemistry following ingestion [ATSDR 2007A].

Dichloronaphthalene

Dichloronaphthalene belongs to the chemical group of chlorinated naphthalenes, or polychlorinated naphthalenes (PCNs). There are 75 possible congeners of PCNs. PCNs are structurally similar to polychlorinated biphenyls (PCBs) [NICNAS 2001]. A number of commercial products containing various concentrations of PCNs were used in the past. Halowax, the trade name of a commercial product used at this site, may be the source of the dichloronaphthalene [Information from Dan Noble (Project Manager for the USACE) sent to T. Foster via email on 2/13/2015].

Chlorinated naphthalenes can be absorbed via oral, inhalation, and dermal routes, with the liver and fat tissue (besides kidneys and lungs) being the main target organs [WHO 2001b]. In 1994, the National Institute for Occupational Safety and Health (NIOSH) completed a study of the long-term effects of exposure to PCNs by studying navy cable manufacturers. [NIOSH 2015b] The study plant used PCNs (sold under the trade name Halowax) to insulate electrical cable from 1939 to 1944. In addition to Halowax, other possible toxic exposures included asbestos, carbon tetrachloride, and polychlorinated biphenyls (PCBs). The major study finding was that workers exposed to the chlorinated naphthalene product Halowax during World War II had an increased risk of dying from cirrhosis of the liver. Mortality from other cancers (including lung, oral and rectal cancers) was also slightly elevated.

Severe skin reactions (chloracne) and liver disease have both been reported after occupational exposure to chlorinated naphthalenes. [WHO 2001b] Chloracne was common among workers handling PCN in the 1930s and 1940s. Other symptoms described in workers exposed to PCN include irritation of the eyes, fatigue, headache, anemia, hematuria, impotence, impotentia, anorexia, nausea, vomiting, and occasional severe abdominal pain [WHO 2001b].

See reference section of this health consultation for citations used in this appendix.

Greetings,

You are receiving a document from the Agency for Toxic Substances and Disease Registry (ATSDR). We are very interested in your opinions about the document you received. We ask that you please take a moment now to complete the following ten question survey. You can access the survey by clicking on the link below.

Completing the survey should take less than 5 minutes of your time. If possible, please provide your responses within the next two weeks. All information that you provide will remain confidential.

The responses to the survey will help ATSDR determine if we are providing useful and meaningful information to you. ATSDR greatly appreciates your assistance as it is vital to our ability to provide optimal public health information.

https://www.surveymonkey.com/r/ATSDRDocumentSatisfaction

LCDR Donna K. Chaney, MBAHCM
U.S. Public Health Service
4770 Buford Highway N.E. MS-F59
Atlanta, GA 30341-3717
(W) 770.488.0713
(F) 770.488.1542