# **Health Consultation**

U.S. Department of Energy Oak Ridge Reservation: Off-Site Groundwater in Melton Valley and Bethel Valley, Tennessee

Oak Ridge, Anderson County, Tennessee

EPA Facility ID: TN1890090003

MARCH 4, 2015

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

#### Health Consultation: A Note of Explanation

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

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

You May Contact ATSDR Toll Free at 1-800-CDC-INFO or Visit our Home Page at: http://www.atsdr.cdc.gov

#### HEALTH CONSULTATION

U.S. Department of Energy Oak Ridge Reservation: Off-Site Groundwater in Melton Valley and Bethel Valley, Tennessee

Oak Ridge, Anderson County, Tennessee

EPA Facility ID: TN1890090003

Prepared By:

Agency for Toxic Substances and Disease Registry (ATSDR) Division of Community Health Investigations Central Branch

### Table of Contents

Foreword		i			
Summary	and Statement of Issues	i			
I. Back	sground	1			
I.A.	Statement of Issues	1			
I.B.	Site Description and History	1			
I.C.	Environmental Data	2			
II. Disc	ussion	3			
II.A.	Radiological Evaluation	3			
II.B.	Chemical Screening Methodology	4			
II.C.	Chemical Screening Results	6			
II.D.	Public Health Implication Evaluation	9			
III. Co	onclusions4	6			
IV. Re	ecommendations4'	7			
V. Publ	ic Health Action Plan	8			
V.A.	Completed public health actions:	8			
V.B.	V.B. Scheduled public health actions:				
VII. Authors, Technical Advisors					
VIII. Re	eferences	0			
Appendix	A. Chemical Screening Methodology	1			
Appendix	x B. TablesB-	1			
Appendix	x C Figure 1C-2	L			

### Foreword

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

Since 1986, ATSDR has been required by law to conduct a public health assessment at each of the sites on the USEPA National Priorities List. The aim of these evaluations is to find out if people are being exposed to hazardous substances and, if so, whether that exposure is harmful and should be stopped or reduced. If appropriate, ATSDR also conducts public health assessments when petitioned by concerned individuals. Public health assessments are carried out by environmental and health scientists from ATSDR and from the states with which ATSDR has cooperative agreements. The public health assessment process allows ATSDR scientists and public health assessment cooperative agreement partner's flexibility in document format when presenting findings about the public health impact of hazardous waste sites. The flexible format allows health assessors to convey to affected populations important public health messages in a clear and expeditious way.

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

**Health Effects:** If the review of the environmental data shows that people have or could come into contact with hazardous substances, ATSDR scientists evaluate whether or not these contacts may result in harmful effects. ATSDR recognizes that children, because of their play activities and their growing bodies, may be more vulnerable to these effects. As a policy, unless data are available to suggest otherwise, ATSDR considers children to be more sensitive and vulnerable to hazardous substances. Thus, the health impact to the children is considered first when evaluating the health threat to a community. The health impacts to other high-risk groups within the community (such as the elderly, chronically ill, and people engaging in high risk practices) also receive special attention during the evaluation.

ATSDR uses existing scientific information, which can include the results of medical, toxicologic and epidemiologic studies and the data collected in disease registries, to evaluate the possible health effects that may result from exposures. The science of environmental health is still developing, and sometimes scientific information on the health effects of certain substances is not available.

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



early version is also distributed to the public for their comments. All the public comments related to the document are addressed in the final version of the report.

**Conclusions:** The report presents conclusions about the public health threat posed by a site. Ways to stop or reduce exposure will then be recommended in the public health action plan. ATSDR is primarily an advisory agency, so usually these reports identify what actions are appropriate to be undertaken by U.S. EPA or other responsible parties. However, if there is an urgent health threat, ATSDR can issue a public health advisory warning people of the danger. ATSDR can also recommend health education or pilot studies of health effects, full-scale epidemiology studies, disease registries, surveillance studies or research on specific hazardous substances.

**Comments:** If, after reading this report, you have questions or comments, we encourage you to send them to us.

Letters should be addressed as follows:

Attention: Manager, ATSDR Record Center Agency for Toxic Substances and Disease Registry, 1600 Clifton Road (F-09), Atlanta, GA 30333.

### Summary and Statement of Issues

Introduction	In 2012, the DOE Oversight Division of the Tennessee Department of Environment and Conservation (TDEC) asked the Agency for Toxic Substances and Disease Registry (ATSDR) to evaluate the public health issues with the level of contaminants detected in the off-site groundwater samples. Specifically, TDEC asked ATSDR to look at groundwater samples from off-site residential and monitoring wells in Melton and Bethel Valleys adjacent to the U.S. Department of Energy (DOE) Oak Ridge Reservation (ORR). TDEC and DOE had gathered the samples in 2010 and 2011 from off-site residential wells and from off-site DOE monitoring wells. These wells are to the west of and downgradient from DOE's legacy waste disposal areas. The DOE disposal areas contain chemical and radioactive waste from the Oak Ridge National Laboratory and from off-site generators. But no one has ever tested off-site groundwater from some of these active private wells for chemicals or radionuclides.
	In July 2006, the ATSDR finished a public health assessment entitled <i>Evaluation of Potential Exposures to Contaminated Off-Site Groundwater from the Oak Ridge Reservation.</i> In it, ATSDR scientists focused on possible off-site exposures to contaminated groundwater from the ORR. ATSDR reviewed off-site groundwater monitoring data and groundwater modeling data, as well as area
	<ul> <li>Demographics,</li> <li>Land uses, and</li> <li>Groundwater uses.</li> </ul>
	ATSDR scientists concluded in the 2006 public health assessment that off-site groundwater did not pose a public health hazard. ATSDR determined that no human exposures to contaminated groundwater outside the ORR boundary occurred in the past, were currently occurring, or were likely to occur in the future. Therefore, ATSDR did not expect any health effects from contaminated off-site groundwater. But ATSDR scientists did state that the complex nature of karst groundwater systems generally raised questions about the reliability of any conclusions drawn from the monitoring and modeling data.
	Off-site private wells in Melton and Bethel Valleys are located across the Clinch River and downgradient of DOE's legacy waste disposal areas. In 2009, DOE offered to connect Melton Valley and then Bethel Valley residences to the Watts Bar Utility District water supply. Most Melton and Bethel Valley area residents accepted DOE's offer. But some Melton and Bethel Valley residences remain unconnected to Watts Bar Utility District water supply; these residents continue to use the groundwater from private wells for home purposes.



	<ul> <li>In this 2014 health consultation, ATSDR looks at off-site groundwater data in the Melton and Bethel Valley areas. ATSDR also looks at any possibly harmful health effects related to contaminant levels found in those off-site groundwater samples. Specifically, ATSDR</li> <li>Looks at harmful health effects from drinking or using contaminated off-site groundwater,</li> <li>Looks at whether people are drinking or using contaminated groundwater that could cause harmful health effects, and</li> <li>Recommends public health actions for contaminated groundwater</li> </ul> Note that this health consultation does not look into the sources or the environmental fate and transport of contaminants found in groundwater from off-site wells.
Conclusions	
Conclusion 1	Currently, Melton Valley and Bethel Valley residents are not exposed to any known chemicals and radionuclides in off-site groundwater at public health hazard levels.
Conclusion 1 Basis	Although ATSDR did find elevated chemical levels in some off-site wells in Melton and Bethel Valleys, those wells —and the groundwater that supplies them—are not currently used for home purposes.
Conclusion 2	Long-term exposure (chronic, more than 1 year) to groundwater from off- site DOE <i>monitoring</i> wells in Melton Valley would be a public health hazard.
Conclusion 2 Basis	Groundwater samples from off-site DOE monitoring wells in Melton Valley showed elevated concentrations of lead, lithium, fluoride, and trichloroethylene. These concentrations were at levels that could cause harmful health effects from long-term ingestion (drinking), dermal contact (skin contact), and inhalation (breathing). But these DOE monitoring wells are not used for home purposes; no one is actually in contact with chemicals in the groundwater from these monitoring wells. The source of the contaminants is unknown, as is the extent of the contaminant plumes in Melton Valley groundwater. See Table 5, Figure 1, and the discussion of the specific chemicals in the Public Health Implications section of this health consultation for information on certain wells, exposure routes, potentially affected population, and potential health effects. Potential health impacts from combined action of chemical mixtures are not evaluated because there is no known exposure to chemicals at public health hazard levels.
Conclusion 2 Next Steps	Continue monitoring the off-site groundwater in Melton Valley. Specifically, collect quarterly groundwater samples. Monitoring will help to characterize the potential exposure to chemicals over an extended period of time and will help to determine the temporal and spatial

	(vertical and lateral) extent of contaminant plumes in the off-site Melton Valley groundwater.
Conclusion 3	Long-term exposure (chronic, more than 1 year) to off-site groundwater from one former residential Bethel Valley <i>monitoring</i> well could cause harmful health effects.
Conclusion 3 Basis	TDEC used off-site, former residential wells in Bethel Valley as monitoring wells. Groundwater samples from one former residential well contained concentrations of fluoride and benzene at levels which, given long-term ingestion (drinking), dermal contact (skin contact), or inhalation (breathing), might cause harmful health effects. At this time that former residential well is not used for home purposes. We do not know where these contaminants in the well came from, nor do we know the extent of the contaminant plumes in Bethel Valley groundwater. Note that Table 6, Figure 1, and the discussion of the specific chemicals in the Public Health Implications section has facts on the exact wells, the possibly affected populations, and any potential health effects. Potential health impacts from combined action of chemical mixtures are not evaluated because there is no known exposure to chemicals at public health hazard levels.
Conclusion 3 Next Steps	Continue monitoring the off-site groundwater in BethelValley. Specifically, collect quarterly groundwater samples. Such monitoring will help to characterize the potential exposure to chemicals over an extended period of time and will help to determine the temporal and spatial (vertical and lateral) extent of contaminant plumes in the off-site Bethel Valley groundwater.
Conclusion 4	ATSDR can not fully characterize health hazards posed by chemicals in off-site, private well groundwater sampled in Bethel Valley.
Conclusion 4 Basis	Too few samples were collected from off-site private wells in Bethel Valley to characterize fully any chronic exposure to chemicals in the groundwater of off-site private wells.
Conclusion 4 Next Steps	Continue collecting quarterly groundwater samples from off-site private wells previously sampled in Bethel Valley.
Conclusion 5	ATSDR can not conclude whether chemicals and radionuclides in unsampled Melton Valley and Bethel Valley private wells could harm the health of residents now using those private wells for home purposes.
Conclusion 5 Basis	Groundwater data are not available for some off-site private wells now used for home purposes. Some of the off-site, active private wells in Melton Valley and Bethel Valley have neither been sampled nor tested for chemical or radioactive contaminants. Without groundwater test results from all private residential wells used for home purposes, ATSDR can not say whether the groundwater in these wells contains chemicals and radionuclides at public health hazard levels.



Conclusion 5 Next Steps	ATSDR recommends to the parties of the Federal Facilities Agreement for the ORR: TDEC, DOE, and U.S. Environmental Protection Agency (U.S. EPA).						
	1. Conduct a complete well use survey of all off-site private wells in Melton Valley and Bethel Valley. The survey area should include the area from the Clinch River toward the west and downgradient of the ORR for at least 1 mile.						
	<ol> <li>Inventory all off-site private wells in Melton Valley and Bethel Valley survey area.</li> </ol>						
	3. Monitor the groundwater in private wells within the Melton and Bethel Valley survey area that are used for home purposes. If elevated levels of chemicals are found in the groundwater the residents should find an alternative source of water for home use, such as connecting to the Watts Bar Utility District						
	4. Monitor groundwater quarterly. Monitoring should involve testing for:						
	a. Metals,						
	b. Volatile organic chemicals,						
	c. Gross beta, and						
	d. Gross alpha.						
	arterly monitoring should allow for adequate characterization of posure over an extended time and the temporal and spatial (vertical and eral) extent of contaminant plumes in the off-site Melton and Bethel alley groundwater.						
Conclusion 6	Persons undergoing lithium treatment need to be cautious about drinking any Melton Valley and Bethel Valley groundwater that has high levels of lithium.						
Conclusion 6 Basis	Drinking groundwater with high levels of lithium can raise the lithium dose level of persons undergoing lithium treatment. Drinking such groundwater can also increase the risk of nephrogenic diabetes insipidus—a form of diabetes insipidus primarily due to pathology of the kidney—and other lithium intoxication side effects.						

### I. Background

### I.A. Statement of Issues

The DOE Oversight Division of the Tennessee Department of Environment and Conservation (TDEC) asked the Agency for Toxic Substances and Disease Registry (ATSDR) to evaluate offsite groundwater data. TDEC and the U.S. Department of Energy (DOE) collected these data in areas across the Clinch River from the DOE Oak Ridge Reservation (ORR). The data, in the form of groundwater samples, were collected in 2010 and 2011 from off-site residential wells and off-site DOE monitoring wells in Melton and Bethel Valleys. These areas lie west and downgradient from DOE's legacy waste disposal areas, which contain chemical and radioactive waste from the Oak Ridge National Laboratory and off-site generators (TDEC 2011). ATSDR prepared this health consultation in response to the TDEC request for an evaluation of the potential public health issues related to the detected levels of contaminants in the off-site groundwater samples. The health consultation's specific goals were to

- 1. Evaluate the public health implications of exposure to the concentrations of contaminants detected in the off-site groundwater,
- 2. Determine whether members of the community are exposed to off-site groundwater contaminants at levels of health concern, and
- 3. Recommend needed public health actions regarding contaminated off-site groundwater.

Note that this health consultation neither evaluates nor determines the source of any contaminants or the environmental fate and transport of contaminants detected in the evaluated off-site groundwater samples.

### I.B. Site Description and History

In 1942, as the United States continued its entry into World War II, the government developed the Oak Ridge Reservation (ORR) in Anderson and Roane Counties in Tennessee. ORR was part of the Manhattan Project initiative to research, develop, and produce special radioactive materials for nuclear weapons. The government built four facilities at ORR: the Y-12 plant, the K-25 site, and the S-50 site to enrich uranium, and the X-10 site as a pilot plant to demonstrate plutonium production and chemical separation. At the end of World War II, the Y-12 plant became the Y-12 National Security Complex. The K-25 site expanded to include the former S-50 plant, all of which became the East Tennessee Technology Park (ETTP). The X-10 site, formerly known as the Clinton Laboratories, became the Oak Ridge National Laboratory (ORNL), which broadened its role to include a variety of nuclear research and production projects vital to national security.

Over the years, Oak Ridge Reservation operations generated a variety of radioactive and nonradioactive wastes. Some wastes remain in old disposal sites, but others were released into the environment. Consequently, in 1989, the U.S. EPA added the ORR to its National Priorities List (NPL). Under a Federal Facility Agreement with U.S. EPA and TDEC, DOE conducts ORR cleanup activities. But all the agencies work together to investigate and to take remedial action on hazardous wastes generated from both past and present ORR activities.

The Oak Ridge National Laboratory lies within two watersheds: Bethel Valley and Melton Valley. The main ORR plant, key research facilities, and primary administrative offices, as well



as various waste sites, are in Bethel Valley. Remote facilities and waste storage areas are in Melton Valley. The Clinch River forms the southern and western borders of the Oak Ridge National Laboratory.

In July 2006, ATSDR completed an ORR public health assessment entitled *Evaluation of Potential Exposures to Contaminated Off-Site Groundwater from the Oak Ridge Reservation* (ATSDR 2006). ATSDR scientists focused solely on evaluating potential off-site exposures to contaminated groundwater migrating from the Oak Ridge Reservation. The goal was to determine the potential public health hazard posed by releases of contaminants to off-site groundwater. ATSDR evaluated available off-site groundwater monitoring data as well as demographic, land use, and groundwater use information. ATSDR scientists did not identify any completed exposure pathway to contaminated groundwater migrating from ORR (ATSDR 2006). ATSDR scientists concluded that off-site groundwater did not pose a public health hazard (ATSDR 2006). Nevertheless, the public health assessment did note that the conclusions were based on data available in 2006 and were limited by the uncertainties inherent in both the modeling data and the general nature of the karst groundwater systems in and near the Oak Ridge Reservation (ATSDR 2006).<sup>1</sup>

In 2009 DOE decided to offer public water connections to residents of Melton Valley and then Bethel Valley. DOE offered to pay for connecting residences to the Watts Bar Utility District public water supply. Most Melton and Bethel Valley area residents accepted DOE's offer and currently have Watts Bar Utility District water for home use. But some Melton and Bethel Valley residences remain unconnected to the Watts Bar Utility District water supply and continue to use private well groundwater for home purposes.

In 2010 and 2011, DOE and TDEC sampled groundwater from off-site residential wells and monitoring wells across the Clinch River and downgradient from the ORR legacy waste disposal areas in Bethel and Melton Valleys. At a number of locations, DOE and TDEC off-site groundwater sample analysis identified the presence of radionuclides, volatile organic chemicals (VOCs), metals, nonmetallic inorganics, and elevated pH (TDEC 2011). These findings prompted TDEC's request for ATSDR evaluation of the TDEC and DOE data.

#### I.C. Environmental Data

ATSDR used groundwater sampling data collected from off-site residential wells and from offsite monitoring wells in Melton and Bethel Valleys, across the Clinch River from the Oak Ridge National Laboratory (See Figure 1). Groundwater data sources were the TDEC Environmental Monitoring & Compliance Program and the DOE Oak Ridge Environmental Information System (OREIS)—a centralized and standardized, quality-assured and configuration-controlled environmental data management system. OREIS contains data from all key surveillance activities and environmental monitoring efforts for compliance and environmental restoration at

<sup>&</sup>lt;sup>1</sup> A fact sheet, a summary, and the full report on ATSDR's 2006 public health assessment evaluation of off-site groundwater, including the release of contaminates into the Bethel Valley and Melton valley watershed, are available on the ATSDR Oak Ridge Reservation Web site at

<sup>&</sup>lt;u>http://www.atsdr.cdc.gov/sites/oakridge/contaminated\_groundwater.html</u>. An extensive compilation of all of ATSDR's public health activities at the DOE Oak Ridge Reservation since 1991 is on the ATSDR Oak Ridge Reservation Web site at <u>http://www.atsdr.cdc.gov/sites/oakridge/</u>.

the Oak Ridge Reservation. On May 25, 2012, ATSDR scientists queried the OREIS database. The query was for all off-site groundwater samples collected since the 2006 health assessment from wells across the Clinch River from the Oak Ridge National Laboratory. ATSDR combined the TDEC and OREIS data then sorted them into two datasets based on well location—either Melton Valley or Bethel Valley. Within each valley, scientists further separated the chemical data (VOCs, metals, and general inorganic parameters) and the radiological data into two additional datasets.

### I.C.1. Melton Valley Data

The TDEC and OREIS databases contain more than 11,500 records on chemicals analyzed in off-site Melton Valley groundwater samples. TDEC and DOE collected the samples from February 11, 2010 to March 8, 2011. The samples came from 16 DOE monitoring wells (four monitoring well cluster locations) and 12 former residential wells used as monitoring wells. The samples were analyzed for 119 chemicals, including volatile organic chemicals (VOCs), metals, general inorganic parameters, and radionuclides. Groundwater from a few private residential wells in Melton Valley currently used for domestic purposes have not been sampled and analyzed for chemicals or radionuclides (see Figure 1).

### I.C.2. Bethel Valley Data

The TDEC database contained over 1,380 records on chemicals analyzed in off-site Bethel Valley groundwater samples collected from 13 residential wells between January 26, 2010 and October 28, 2010. Four of these residential wells were still used for home purposes, and nine were former residential wells converted to monitoring wells. Most of the residential wells were sampled only once, four wells were sampled twice, and one well was sampled 10 times. The groundwater samples were analyzed for 108 chemicals, including volatile organic chemicals (VOCs), metals, general inorganic parameters, and radionuclides. Groundwater from a few private residential wells in Bethel Valley still used for home purposes have not been sampled or analyzed for chemicals or radionuclides (see Figure 1).

### **II.** Discussion

### II.A. Radiological Evaluation

ATSDR evaluated the radionuclide concentrations in Bethel Valley and Melton Valley separately. ATSDR evaluates radioactive contaminants in groundwater by calculating committed effective dose from annual intakes (drinking water ingestion) for various age groups under conservative, site-specific scenarios. Initially, however, ATSDR performed a screening of potential contaminant radionuclide concentrations in each sample using U.S. EPA's Maximum Contaminant Levels found in its Drinking Water Regulations: Radionuclides (40 CFR Part 141). Although this regulation pertains to public water systems, ATSDR compared the radionuclide concentrations in private well groundwater with these levels. The regulation includes the requirements that 1) if gross beta analysis detects greater than 50 picocuries per liter (> 50 pCi/L), then isotopic analyses need to be performed, and 2) the concentrations of man-made beta or gamma-emitting radionuclides in drinking water must not produce a dose of more than 4 millirem per year (mrem/yr) [pathway specific]. This dose suggests no increase in cancer risk.



ATSDR also has a comparison value (CV) for noncancer effects from radiation exposure of 100 mrem/yr.<sup>2</sup>

No radionuclide sampling results for off-site Bethel Valley groundwater exceeded the U.S. EPA's Drinking Water standards for radionuclides, and no sampling results for man-made radionuclides exceeded detection limits. ATSDR did not analyze Bethel Valley samples for cesium-137, but the gross beta activities were very low.

For off-site Melton Valley groundwater, no well sample results exceeded U.S. EPA's Drinking Water standards for man-made radionuclides. Still, on four out of five sampling dates, results for well OMW-1D did exceed U.S. EPA's standard for gross beta concentrations. After review of all the results for well OMW-1D on these dates, the only beta emitter concentrations that appeared elevated were for potassium-40, a naturally occurring isotope often found in fertilizers and geological formations.

The potassium content in the human body is under homeostatic control. That means the body actively regulates the amount of potassium retained to achieve the normal range required for the body systems to function—potassium is not influenced by variations in environmental levels. This well also had one uranium concentration that exceeded U.S. EPA's Drinking Water standard of 0.030 milligrams per liter (mg/L). The reported concentration was 0.200 mg/L, which might be an error or anomaly given that this well was sampled five other times with results of 0.000135 mg/L, 0.000080 mg/L, 0.000077 mg/L, 0.000067 mg/L, and 0.000072 mg/L. None of the isotopic uranium analyses were elevated.

Therefore, radionuclides in groundwater samples collected from off-site monitoring and residential wells in Melton and Bethel Valleys do not appear present at levels that would cause harmful health effects.

#### II.B. Chemical Screening Methodology

To evaluate chemicals in off-site groundwater, ATSDR looked at the Melton Valley and Bethel Valley data separately. Melton Valley and Bethel Valley have separate hydrogeologic formations and different potential soursces of contamination. ATSDR screened the chemical concentrations in all the groundwater sampling data using a two-step chemical screening process to identify chemicals of potential public health concern. To determine exposure implications, chemicals of concern required further, in-depth evaluation of the contaminate concentrations. See Appendix A, "Methodology" for a detailed discussion of the chemical screening process, equations, and exposure parameters.

Note that in this evaluation of chemicals in off-site groundwater ATSDR *assumed* exposure to contaminated groundwater. For chemicals determined to be of potential public health concern, in the Public Health Implications section ATSDR evaluates and documents whether anyone is actually exposed.

In this consultation, ATSDR did not evaluate essential nutrients (e.g., calcium, magnesium, phosphorus, potassium, and sodium). Essential nutrients are minerals that maintain basic life functions; therefore, health agencies recommend certain doses on a daily basis. Because these

<sup>&</sup>lt;sup>2</sup> Note that ATSDR's CVs are not thresholds for adverse health effects but are concentrations many times lower than levels at which no effects were observed in experimental animals or human epidemiologic studies.

chemicals are necessary for life, we neither have—nor need—comparison values for them. They're in many foods, such as milk, bananas, and table salt. For example, the Food and Nutrition Board of the Institute of Medicine of the National Academy of Sciences has recommended adequate intakes (AI) and recommended dietary allowance (RDA) for these essential nutrients.

### II.B.1. Children's Health

In communities faced with air, water, or food contamination, the many physical differences between children and adults demand special emphasis. Children and infants could be at greater risk than are adults from certain kinds of exposure to hazardous substances. Children's lower body weight and high intake rate results in a greater dose of hazardous substance per unit of body weight. An infant who drinks formula prepared with contaminated groundwater is likely to have a higher exposure dose because of the large volume of water they consume relative to their body size. If toxic exposure levels are high enough during critical growth stages, the developing body systems of children can sustain permanent damage. Finally, children and infants are dependent on adults for access to housing, for access to medical care, and for risk identification. Thus adults need as much information as possible to make informed decisions regarding their children's health.

### II.B.2. Comparing Environmental Concentrations with Comparison Values

ATSDR evaluated the chemical data by comparing the maximum concentration of each chemical detected in off-site groundwater against ATSDR's conservative (protective), chemical-specific, drinking water comparison value. These drinking water comparison values are environmental concentration guidelines set well below levels that are known or anticipated to result in adverse health effects. We do not expect that chemicals with maximum concentrations at or below the ATSDR groundwater comparison value to cause health effects in people. We do not consider them a health hazard, and we won't further evaluate them.

### II.B.3. Comparing Estimated Exposure Doses with Screening Guideline Values

### II.B.3.a. Noncancer Screening

ATSDR next evaluated each chemical with a maximum concentration above a comparison value. For each well, ATSDR calculated a chronic (1-year annual) exposure dose for an infant and adult, using the mean concentration of chemical in the groundwater and a reasonable maximum exposure (RME) scenario. We used the mean concentration because people are more likely to be exposed to a range of concentrations over time; the mean estimates a more probable exposure dose. For each well, ATSDR followed Helsel's recommended methods for estimation of summary statistics (Helsel 2012). For many wells, the mean chemical concentration was based on the maximum concentration detected—fewer than three samples were collected from those well stations. The RME scenario refers to people who are at the high end of the exposure distribution (approximately the 95<sup>th</sup> percentile) with higher than average water-intake rates.

ATSDR believes the RME scenario is a health-protective assumption. It overestimates the average groundwater consumption but remains within a realistic exposure range. The infant scenario is included in this evaluation because infants can be more sensitive to exposure than are adults. And because of the large volume of water consumed relative to body size, infants who drink formula prepared with contaminated groundwater are likely to have a higher exposure



dose. ATSDR calculated chronic exposure doses for these chemicals using the equation and RME parameters described in Appendix A, Methodology.

ATSDR then compared these estimated exposure doses to conservative (protective), chemicalspecific, health-based noncancer screening guidelines, including ATSDR's minimal risk levels (MRLs) and the U.S. EPA's reference doses (RfDs) (See results in Appendix B, Table B1 and Table B3). An ATSDR MRL is a dose estimate of daily human exposure to a hazardous substance likely without an appreciable risk of adverse noncancer health effects over a specified route and duration of exposure. The U.S. EPA RfD is a dose estimate of the human population's daily exposure to a potential hazard likely without risk of deleterious effects during a lifetime. Estimated exposure doses lower than MRLs or RfDs are not expected to cause health effects, are not considered a health hazard, and are not further evaluated.

Note that while estimated exposure doses at or below a respective conservative (protective), chemical-specific, health-based noncancer screening guideline values can be considered safe, estimated exposure doses above these screening guideline values do not necessarily imply adverse health effects. Rather, if the estimated exposure dose is higher than the noncancer screening guideline value, that is only an indication that ATSDR should further evaluate exposure to that chemical and include the results in the health consultation's Health Implications section.

#### II.B.3.b. Cancer Screening

To screen for cancer effects, ATSDR evaluated potentially carcinogenic chemicals by calculating the potential excess cancer risk for a child and an adult. ATSDR multiplied the estimated chronic exposure doses and the U.S. EPA cancer slope factors (CSFs) (See results in Appendix B – Table B2 and Table B4). ATSDR calculated potential excess cancer risk using the equation and exposure parameters described in Appendix A, Methodology.

Because conservative models are used to derive CSFs, the exposure doses associated with these potential cancer risks are typically orders of magnitude lower than doses reported in the toxicology literature to cause cancer effects. As such, estimated cancer risk less than (below) 1 in 10,000 (less than  $1 \ge 10^{-4}$ ) indicate that the toxicology literature would support a finding that an observable increase in cancer is not expected and that there is a low or no apparent risk of cancer. Therefore, chemicals with estimated excess cancer risk lower than the cancer screening guideline of 1 in 10,000 (less than  $1 \ge 10^{-4}$ ) are not a health hazard and are not further evaluated. While estimated excess cancer risk below the cancer screening guideline (less than  $1 \ge 10^{-4}$ ) supports a finding that excess cancer risk is unlikely, estimated excess cancer risk higher than these screening guideline values does not automatically imply that adverse health effects will occur. Rather, it is an indication that ATSDR should further evaluate exposure to these chemicals in the Public Health Implications section of this document.

#### II.C. Chemical Screening Results

For each chemical detected in off-site Melton and Bethel Valley groundwater, ATSDR compared the maximum concentration detected with ATSDR's conservative (protective), chemical-specific, drinking water comparison values. For 18 chemicals in Melton Valley groundwater (Table 1) and 13 chemicals in Bethel Valley groundwater (Table 2) maximum concentrations were detected above comparison values.

## Table 1 - Chemicals Detected in Off-Site Melton Valley Groundwater with a Maximum ConcentrationGreater than ATSDR Drinking Water Comparison Values

Metals	Organics	General Inorganic Parameters
Antimony	Benzene	Fluoride
Arsenic	Bromodichloromethane	
Barium	Cis-1,2-Dicholormethane	
Cadmium	Trichloroethylene	
Chromium	Vinyl chloride	
Lead		
Lithium		
Manganese		
Selenium		
Strontium		
Thallium		
Uranium		

# Table 2 - Chemicals Detected in Off-Site Bethel Valley Groundwater with a Maximum ConcentrationGreater than ATSDR Drinking Water Comparison Values

*Metals* Arsenic Chromium Lithium Organics Benzene Bromodichloromethane Bromoform Carbon tetrachloride Chloroform Dibromochloromethane 1,2-Dichloroethane 1,2,3-Trocholopropane *General Inorganic Parameters* Fluoride

At each well station, ATSDR estimated chronic (1-year annual) exposure doses for each of the 18 chemicals in Melton Valley groundwater and the 13 chemicals in Bethel Valley groundwater with a maximum concentration higher than ATSDR's groundwater comparison value. ATSDR used the mean concentration and RME scenario parameters and compared these estimated doses with ATSDR chemical-specific health-based noncancer guideline values (See estimated exposure doses and noncancer health-based guideline values in Appendix B, Table B1 and Table B3). For carcinogenic chemicals, ATSDR estimated the cancer risk at each well station using the mean concentration and RME scenario parameters. ATSDR then compared the cancer risk with the cancer screening guideline (See cancer risks in Appendix B, Table B2, and Table B4). Because estimated exposure doses or cancer risk exceeded either or both noncancer or cancer screening guidelines, thirteen chemicals in Melton Valley groundwater (Table 3) and six chemicals in Bethel Valley groundwater (Table 4) required further in-depth evaluation. We further evaluate these chemicals in the Public Health Implications section.



Table 3 - Chemicals Detected in the Off-Site Melton Valley Groundwater with a Mean ConcentrationResulting in Estimated Exposure Doses Greater than Chemical-Specific Health-Based NoncancerScreening Guideline Values or Estimated Cancer Risks Higher than Cancer Screening Guideline

Metals	Organics	General Inorganic Parameters
Antimony	Cis-1,2-Dichloroethene	Fluoride
Arsenic	Trichloroethylene	
Barium	-	
Cadmium		
Chromium		
Lead		
Lithium		
Manganese		
Strontium		
Thallium		

Table 4 - Chemicals Detected in the Off-Site Bethel Valley Groundwater with a Mean ConcentrationResulting In Estimated Exposure Doses Greater than Chemical-Specific Health-Based NoncancerScreening Guideline Values or Estimated Cancer Risks Higher than Cancer Screening Guideline

*Metals* Chromium Lithium *Organics* Benzene Bromodichloromethane Chloroform *General Inorganic Parameters* Fluoride

### II.D. Public Health Implications

ATSDR determines public health implications by further evaluating chronic exposure to concentrations of chemicals in Melton and Bethel Valley groundwater that exceed screening guidelines (i.e., estimated screening exposure doses exceeding the MRLs or RfDs, or the estimated excess cancer risk is greater than  $1 \times 10^{-4}$ ). See Table 3 and Table 4 for the chemicals evaluated for public health implications. The in-depth discussion of each chemical includes current scientific information on the chemical's disease-causing potential. The discussion also compares estimated site-specific exposure dose with doses shown to cause harmful health effects. This section offers a perspective on the plausibility of harmful health outcomes from exposure to each chemical in off-site Melton and Bethel Valley groundwater.

ATSDR further analyzes site-specific exposure variables (e.g., exposure intake rates, duration, frequency). ATSDR considers multiple chemical factors, including physical properties, form, and bioavailability. ATSDR considers characteristics of the exposed population—such as age, sex, genetics, lifestyle, nutritional and health status—that influence how people absorb, distribute, metabolize, and excrete contaminants. Where appropriate, we've included these characteristics in the chemical-specific discussions.

To evaluate health implications of chronic exposure to the contaminants in the groundwater, ATSDR, if appropriate, will base its health evaluation on a more realistic, site-specific exposure using a central tendency exposure (CTE) scenario. The central tendency exposure scenario refers to persons who have average or typical water intake rates. The likelihood that adverse health outcomes will actually occur depends on the concentration of the chemical, site-specific exposure conditions, individual differences, and factors that affect the route, magnitude, and duration of actual exposure. See Appendix A, "Methodology" for a detailed discussion of the public health implications evaluation and CTE parameters.

ATSDR uses a chemical's current scientific information. This information can include the results of medical, toxicologic, and epidemiologic studies, and data collected in disease registries. ATSDR reviews the weight-of-evidence of toxicologic and epidemiologic data and health effects variables to obtain information about the toxicity of the chemicals. In this way, we more completely understand the public health implications of exposure. The weight-of-evidence is the extent to which the available scientific information supports the hypothesis that a specific dose of a substance causes an adverse effect in humans. We use this information to determine the likelihood of health effects that might result from exposure by understanding a chemical's disease-causing potential. We also use the information to compare site-specific exposure dose estimates with doses shown to cause health effects. This process enables us to weigh available evidence in light of known uncertainties and offer perspective on the plausibility of harmful health outcomes under site-specific conditions. The science of environmental health is still developing. Sometimes scientific information on the health effects of certain substances is not available. In this case, we recommend further needed public health actions such as substance-specific applied research to fill important data needs.

Tables 5 and 6 summarize the public health implications of 13 chemicals in Melton Valley groundwater and 6 chemicals in Bethel Valley groundwater that exceed noncancer or cancer screening guidelines. The tables identify for each chemical the specific wells where they were found and the following additional information:



- Potential exposure routes evaluated,
- Potentially affected populations,
- Whether an exposure pathway is complete, and
- Any public health implications.

A chemical identified as a *potential public health hazard* indicates that chronic (more than 1 year) exposure to the chemical at levels reported in the Melton or Bethel Valley groundwater could result in adverse health effects. Given, however, that at the time of this health consultation no one was exposed to the chemical at levels of health concern, the chemical posed no current health threat. A more detailed discussion of each chemical follows the tables.

Note that at the time of this health consultation in Melton and Bethel Valleys, we knew of no one exposed to chemicals in off-site groundwater at public health hazard levels. In Melton Valley, four chemicals—lead, lithium, fluoride, and trichloroethylene—were identified in Table 5 as potential public health hazards. These chemicals were detected in seven off-site DOE monitoring wells (See Figure 1). They are considered a potential public health hazard because chronic (i.e., more than 1 year) exposure to these chemicals at concentrations detected in Melton Valley groundwater would pose a public health hazard. But no one was exposed to the contaminated groundwater from these seven off-site DOE monitoring wells. The other nine DOE monitoring wells and all the former private residential wells in Melton Valley sampled contained concentrations of chemicals in the groundwater at levels unlikely to cause harmful health effects. Chemicals were detected at concentrations below the groundwater comparison value, or estimated exposure doses were below noncancer and cancer screening guidelines or doses associated with adverse health effects. The potential health impacts from combined action of chemicals as a result of exposure to chemical mixtures are not evaluated because there is no know exposure to chemicals at public health hazard levels.

Table 6 shows two chemicals in Bethel Valley groundwater (fluoride and benzene) as potential public health hazards. These two chemicals were detected in one former private residential well used as a monitoring well (See Figure 1). These chemicals are a potential public health hazard because chronic (more than 1year) exposure to concentrations detected in Bethel Valley groundwater would pose such a hazard. But this former residential well is not used for home purposes, and no one is exposed to the contaminated groundwater from this well.

Beginning in 2009, most Melton and Bethel Valley residents chose to stop using their private residential wells. They chose instead to accept DOE's offer to connect their residences to the Watts Bar Utility District public water supply. We have no historic groundwater monitoring data to determine whether these former private residential wells were contaminated before the 2010 groundwater sampling. The source of contamination and the temporal and spatial (vertical and lateral) extent of the contaminants plumes are both unknown. And as stated, some Melton and Bethel Valley residents remain unconnected to the Watts Bar Utility District water supply; they continue to use groundwater from private residential wells for home purposes (See Figure 1). ATSDR does not have any results of chemical or radiological analysis of groundwater from some of these wells. And without analytical results from continued monitoring of the groundwater from these residential wells currently used for home purposes, ATSDR can not determine whether the groundwater in these wells contains chemical and radioactive contaminants at public health hazard levels. As a result, ATSDR will discuss the public health

implications of exposures to contaminants at the levels detected in monitoring wells. While we are not aware of any current exposures to the contaminants at the levels detected, exposures could be occurring to those using residential wells that have not been monitored.

Chemical In Groundwater	Wells with Elevated Levels	Exposure Routes Evaluated	Potentially Affected Populations	Completed Exposure Pathway?	Public Health Implications
Antimony	None	Ingestion	None	No	None expected
Arsenic	None	Ingestion	None	No	None expected
Barium	None	Ingestion	None	No	None expected
Cadmium	None	Ingestion	None	No	None expected
Chromium	None	Ingestion	None	No	None expected
Lead	OMW-1C OMW-1D	Ingestion	Infant & Child	No	Potential public health hazard
Lithium	OMW-1B OMW-1D OMW-2C OMW-2D	Ingestion	Infant & Adult	No	Potential public health hazard
Manganese	None	Ingestion	None	No	None expected
Strontium	None	Ingestion	None	No	None expected
Thallium	None	Ingestion	None	No	None expected
Fluoride	OMW-1B OMW-1C OMW-2B, OMW-2C OMW-3C	Ingestion	Infant, Child, & Adult	No	Potential public health hazard
1,2-Dichloroethene, cis	None	Ingestion Inhalation Dermal		No	None expected
Trichloroethylene	OMW-1B	Ingestion Inhalation Dermal	Child & Adult	No	Potential public health hazard

Table 5. Summary of Public Health Implications of Chemicals in Melton Valley Groundwater Exceeding
Noncancer or Cancer Health-Based Screening Guidelines

OMW – Off-site Monitoring Well



Table 6. Summary of Public Health Implications of Chemicals in Bethel Valley Groundwater ExceedingNoncancer or Cancer Health-Based Screening Guidelines

Chemical In Groundwater	Wells with Elevated Levels	Exposure Routes Evaluated	Potentially Affected Population	Completed Exposure Pathway?	Public Health Implications
Chromium	None	Ingestion	None	No	None expected
Lithium	None	Ingestion	None	No	None expected
Fluoride	RWA-104	Ingestion	Infant, Child, & Adult	No	Potential public health hazard
Benzene	RWA-104	Ingestion Inhalation Dermal	Infant, Child & Adult	No	Potential public health hazard
Bromodichloromethane	None	Ingestion Inhalation Dermal	None	No	None expected
Chloroform	None	Ingestion Inhalation Dermal	None	No	None expected

RMW – Residential Monitoring Well

#### II.D.1. Antimony

As shown in Appendix B - Table B1, the estimated exposure dose for an infant drinking Melton Valley groundwater from monitoring well OMW-1D exceeds the noncancer screening guideline for antimony. As such, ATSDR further examined the public health implications of antimony in groundwater from OMW-1D if used for home purposes. The antimony concentrations detected in the groundwater samples from the other monitoring wells and residential wells in Melton Valley and Bethel Valley (Appendix B - Table B3) were not at health hazard levels (i.e., the concentrations detected were below the groundwater comparison value) and were not further evaluated in this section.

Antimony is a silvery white metal naturally found in the environment. Small amounts of antimony in the earth's crust (ATSDR 1992a). Antimony is usually mixed in small amounts with other metals such as lead and zinc to form mixtures of metals called alloys (ATSDR 1992a). These alloys are then incorporated into lead storage batteries, solder, sheet and pipe metal, bearings, castings, type metal, ammunition, and pewter (ATSDR 1992a).

Naturally occurring antimony is at very low levels. So low, in fact, that often it can not be measured. Soil usually contains very low concentrations of antimony, fewer than 1 part per million (ppm or one part of antimony in a million parts of soil) (ATSDR 1992a). Still, concentrations close to 9 ppm have been found in some soil (ATSDR 1992a). The concentration of antimony dissolved in rivers and lakes is also low, usually fewer than 5 parts per billion (pbb or one parts of antimony in 1 billion parts of water) (ATSDR 1992a). Food contains small amounts of antimony. The average concentration of antimony in meats, vegetables, and seafood is 0.2–1.1 ppb (ATSDR 1992a).

People are exposed to antimony by breathing air, drinking water, and eating foods that contain it. People eat and drink about 5 micrograms (5 millionths of a gram) of antimony every day (ATSDR 1992a). A few hours after ingestion, a small amount enters the bloodstream and mostly distributes to the liver, lungs, intestines, and spleen (ATSDR 1992a). Using animal data, gastrointestinal absorption of antimony into the bloodstream was estimated at 2 to 7 percent (ATSDR 1992a). Antimony then leaves the body in urine and feces over several weeks (ATSDR 1992a).

### II.D.1.a. Comparison of Estimated Antimony Doses to Health Effect Levels

The estimated antimony exposure dose in Table B1 for an infant ingesting Melton Valley groundwater from OMW-1D exceeds the noncancer screening guideline (USEPA RfD) when assuming 100 percent gastrointestinal absorption. But the gastrointestinal tract absorbs antimony slowly. To account for this slow absorption, ATSDR recalculated the estimated exposure dose and assumed the gastrointestinal tract absorbed only 10 percent of the antimony. The recalculated, estimated antimony exposure dose of  $1.2 \times 10^{-4}$  mg/kg/day for an infant chronically ingesting groundwater from OMW-1D under a RME scenario and an absorption value of 10 percent is below the U.S. EPA RfD of  $4.0 \times 10^{-4}$  mg/kg/day. Remember that the U.S. EPA RfD (noncancer screening guideline) is an estimate of the daily human exposure to a hazardous substance likely to be without appreciable risk of adverse (harmful) noncancer health effects for the most sensitive people. Therefore, we do not expect an estimated exposure dose below the screening guideline value to result in adverse health effects. In addition, the USEPA RfD for antimony is based on a rat study with a lowest-observed-adverse-effect level (LOAEL) of  $3.5 \times$  $10^{-1}$  mg/kg/day and applying an uncertainty factor of 1000 (10 for interspecies conversion, 10 to protect sensitive individuals, and 10 because the effect level is a LOAEL). This LOAEL is base on a shorter lifespan of rats administered antimony in water compared to the controls. A LOAEL is the lowest tested dose of a substance reported in studies to cause harmful (adverse) health effects in people or animals. The recalculated, estimated antimony exposure dose for an infant drinking Melton Valley groundwater is 2,900 times lower than the LOAEL and more than 3 times lower than the RfD. As such, we do not expect that ingesting antimony at the reported levels in the off-site Melton Valley groundwater to cause harmful health effects.

#### II.D.2. Arsenic

As shown in Appendix B - Table B1, the estimated arsenic exposure doses from ingesting groundwater exceed the noncancer screening guideline. In Melton Valley, 12 wells exceeded the infant guidelines and 4 wells exceed the adult guidelines. As such, ATSDR further examined the potential health effect of arsenic from ingesting groundwater from these Melton Valley wells. In Table B3, the arsenic concentration in the groundwater from only one Bethel Valley residential well (RWA-22) exceeded the ATSDR comparison value. Nevertheless, the estimated arsenic exposure dose from ingesting groundwater from RWA-22 is below the noncancer screening guideline for arsenic; we do not expect it to produce any harmful health effects. The arsenic concentrations detected in the groundwater samples from the other monitoring wells and residential wells in Melton Valley and Bethel Valley were not at levels that constituted a noncancer health hazard, and we do not evaluate them further in this section.

In Appendix B- Table B2 and Table B4, the estimated cancer risks for an infant and adult ingesting groundwater from each Melton Valley and Bethel Valley well were below the cancer



screening guidelines. The cancer screening guideline is a cancer risk of 1 in 10,000 ( $1 \times 10^{-4}$ ), which means out of 10,000 persons exposed, one additional cancer might occur. The arsenic levels detected in the groundwater from these wells were not at levels exceeding the cancer risk (i.e., the estimated cancer risk is below the cancer screening guideline) and we won't discuss further any cancer risk from arsenic exposure.

Arsenic occurs naturally in the environment and usually combines with other elements such as oxygen, chlorine, and sulfur (ATSDR 2007a). When combined with these elements we call it inorganic arsenic. When arsenic combines with carbon and hydrogen, we call it organic arsenic. The concentration of arsenic in natural surface and groundwater is generally about 1 ppb, but it might exceed 1,000 ppb in contaminated areas or where arsenic levels in soil are high (ATSDR 2007a). Groundwater is far more likely to contain high levels of arsenic than is surface water (ATSDR 2007a). Surveys of U.S. drinking water indicate that about 80% of water supplies have fewer than 2 ppb of arsenic, but 2% of supplies exceed 20 ppb of arsenic (ATSDR 2007a).

People normally take in small amounts of arsenic from the air they breathe, the water they drink, and the food they eat (ATSDR 2007a). Ingestion is the primary way arsenic enters the body. Once arsenic is in the body, the liver changes some of it into a less harmful organic form (ATSDR 2007a). Both inorganic and organic forms of arsenic leave the body in urine. Studies have shown that the body eliminates 45 to 85 percent of the arsenic within 1 to 3 days; some, however, remains for several months or longer (ATSDR 2007a).

The scientific literature indicates that the single most characteristic effect of long-term oral exposure to inorganic arsenic is a pattern of skin changes (ATSDR 2007a). These changes include patches of darkened skin and the appearance of small "corns" or "warts" on the palms, soles, and torso, often associated with changes in the blood vessels of the skin (ATSDR 2007a). Tseng et al. (1968) and Tseng (1977) investigated the incidence of Blackfoot Disease and dermal lesions (hyperkeratosis and hyperpigmentation) in a large number of poor farmers in Taiwan (both male and female) exposed to arsenic in well water (ATSDR 2007a). These dermal (skin) health effects could have resulted from chronic ingestion of low levels of arsenichyperkeratosis and hyperpigmentation were reported in humans exposed a LOAEL of  $1.4 \times 10^{-2}$ mg/kg/day arsenic in their drinking water for more than 45 years (Tseng et al. 1968). ATSDR's MRL for chronic (more than one year) oral exposure to inorganic arsenic is the noncancer screening guideline for arsenic. ATSDR derived it by applying an uncertainty factor of 3 (for human variability) to the no-observed-adverse-effect level (NOAEL) of  $8 \times 10^{-4}$  mg/kg/day (Tseng 1977). A NOAEL is the highest tested dose of a substance reported to have no harmful (adverse) health effects on studied people or animals. A large number of well-conducted epidemiological studies that identify reliable NOAELs and LOAELs for dermal effects in humans support ATSDR's MRL for arsenic (ATSDR 2007a).

#### II.D.2.a. Comparison of Estimated Arsenic Doses to Health Effect Levels

ATSDR made the public health-protective assumption that all arsenic to which people were exposed in Melton and Bethel Valleys was the more toxic and harmful inorganic form. We made this assumption because inorganic arsenic is the most abundant in the environment, and the analytical method used did not determine which form of arsenic was present in the groundwater.

In Table 7 the estimated chronic arsenic exposure doses for an infant using the RME scenario exceeded the arsenic NOAEL of  $8 \times 10^{-4}$  mg/kg/day in five Melton Valley monitoring wells

(OMW-1A, OMW-1B, OMW-1D, OMW-2B, OMW-2D). At four monitoring wells (OMW-1A, OMW-1B, OMW-1D, OMW-2D), the chronic arsenic exposure doses for an infant were 6 times less than the  $1.4 \times 10^{-2}$  mg/kg/day LOAEL. Using the RME scenario, all the estimated adult exposure doses were below the NOAEL.

But the more realistic exposure scenario uses a central tendency exposure (CTE), which refers to persons who have average or typical water intake rates. For four DOE monitoring wells (OMW-1A, OMW-1B, OMW-1D, OMW-2D), the estimated chronic infant exposure doses were between 14 and 20 times below the LOAEL (See Table 7). The estimated chronic exposure dose for an adult and child (6 to 11 years of age) using the CTE scenario were below the NOAEL for all five DOE monitoring wells (See Table 7). Thus, we do not expect chronic ingestion of arsenic at the reported levels in off-site Melton Valley groundwater to cause harmful health effects, including cancer. That said, if an infant chronically ingests a higher than average intake of groundwater containing the reported levels of arsenic (8.1–16 ppb), then he or she might have a slight increased risk of developing reversible skin changes. Note again, however, that no one uses any of these monitoring wells for home purposes.

Most Melton Valley area residents have chosen to stop using their private wells; they've switched to the Watts Bar Utility District water supply. Still, the source of the arsenic and the vertical and lateral extent of the arsenic contaminant plume are unknown, and a few Melton Valley residents with private wells continue to use the groundwater for home purposes. Moreover, for some of these off-site private wells, ATSDR does not have any analyses of groundwater for chemical or radioactive contaminants, and we are unable to determine the health implications of using the groundwater from these private wells for home purposes.

Melton Valley Well		Mean	ng/kg/day)		
		Concentration (ppb)	Infant	Child	
			Injuni	(6 to 11 years)	Adult
	RME scenario	14.1	2.0E-03		5.0E-04
OIVIV-TA	CTE scenario		9.0E-04	2.1E-04	2.0E-04
OMW-1B	RME scenario	13.2	1.9E-03		4.7E-04
	CTE scenario		8.0E-04	2.0E-04	1.9E-04
OMW-1D	RME scenario	16.2	2.3E-03		5.7E-04
	CTE scenario		1.0E-03	2.4E-04	2.3E-04
OMW-2B	RME scenario	0.1	1.1E-03		2.9E-04
	CTE scenario	0.1	5.2E-04	1.2E-04	1.1E-04
OMW-2D	RME scenario	15 5	2.2E-03		5.5E-04
	CTE scenario	15.5	1.0E-03	2.3E-04	2.2E-04

Table7. Estimated Chronic Exposure Doses for Arsenic In Melton Valley Groundwater

ppb – part per billion = micrograms per liter ( $\mu$ g/L)

mg/kg/day - milligram per kilogram per day

RME scenario – higher than average water intake rates, high end (approximately 95<sup>th</sup> percentile) of the exposure distribution

CTE scenario – average or typical water intake rates

OMW – Off-site Monitoring Well



#### II.D.3. Barium

In Appendix B - Table B1, the estimated barium exposure dose for an infant drinking groundwater from one Melton Valley monitoring wells OMW-2D exceeds the ATSDR noncancer screening guideline for barium. But the estimated adult barium exposure dose for monitoring well OMW-2D is below the noncancer screening guideline. The mean barium concentration of 1754.7 ppb detected in Melton Valley monitoring well OMW-2D is based on six groundwater samples with barium concentrations ranging from 295 ppb to 3430 ppb. Two OMW-2D groundwater samples with the highest concentrations of 3,430 ppb and 3,340 ppb were collected on the same day. Two groundwater samples contained barium concentrations of 1,448 ppb and 1,710 ppb, and two others contained fewer than 300 ppb barium. As such, ATSDR further examined the potential effect levels of barium from ingesting groundwater from this monitoring well. The barium concentrations detected in groundwater from the other Melton Valley wells and all of the Bethel Valley wells were not at levels constituting a health hazard (i.e., the barium concentrations were well below the ATSDR groundwater comparison value) and we won't discuss them further.

Barium is a silvery-white metal found in nature but only in ores containing mixtures of elements (ATSDR 2007b). The amount of barium found in soil ranges from about 15 to 3,500 ppm (ATSDR 2007b). Barium combines with other chemicals such as sulfur or carbon and oxygen to form barium compounds. Barium compounds become part of paint, bricks, ceramics, glass, and rubber (ATSDR 2007b). Doctors sometimes use barium sulfate to perform medical tests and to take x-rays of the gastrointestinal tract (ATSDR 2007b).

Barium enters your body when you breathe air, eat food, or drink water that contains barium. Most surface water and public water supplies contain on average 30 ppb or less (ATSDR 2007b). But persons residing in certain regions of Kentucky, northern Illinois, New Mexico, and Pennsylvania who rely on groundwater for their home use might be exposed to barium concentrations as high as 10 times the U.S. EPA Maximum Contaminant Level (MCL) of 2,000 ppb of barium in drinking water (ATSDR 2007b). The MCLs are legally enforceable standards that apply to public water systems under the National Primary Drinking Water Regulations. Some foods, such as Brazil nuts, seaweed, fish, and certain plants, might also contain high amounts of barium (ATSDR 2007b). Still, the amount of barium found in food and water is usually not high enough to become a health concern (ATSDR 2007b).

The amount of barium that enters the bloodstream after breathing, eating, or drinking it depends on the barium compound (ATSDR 2007b). An important factor affecting the development of adverse health effects is the barium compound's solubility. Soluble barium compounds are generally expected to be of greater health concern than insoluble barium compounds; soluble compounds have greater potential for gastrointestinal absorption (ATSDR 2007b). Some soluble barium compounds can enter bloodstream more easily than can insoluble barium compounds such as barium sulfate, which does not easily dissolve in water and causes few harmful health effects (ATSDR 2007b). The health effects associated with exposure to different barium compounds depend on how well the specific barium compound dissolves in water or in the stomach (ATSDR 2007b). The body eliminates most of the barium within 1–2 weeks, mainly in feces and urine (ATSDR 2007b). Most of the small amount of barium that stays in the body goes into bones and teeth (ATSDR 2007b).

We reviewed the scientific literature on barium toxicity. We learned animal data provide strong evidence that renal (kidney) toxicity is the most sensitive adverse effect of oral exposure to barium (ATSDR 2007b). Researchers have observed nephropathy (damage to or disease of a kidney) in rats and mice following long-term oral barium exposure (ATSDR 2007b). Some case reports indicate renal effects in persons ingesting high doses of barium (ATSDR 2007b). ATSDR used a benchmark dose (BMD) analysis of the incidence data for renal (kidney) nephropathy from barium exposure in a 2-year, mouse drinking water study (NTP 1994). The nephropathy was characterized by extensive regeneration of cortical and medullary tubule epithelium, tubule dilatation, multifocal interstitial fibrosis, and glomerulosclerosis in some kidneys (ATSDR 2007b). ATSDR derived the lowest benchmark dose limit (BMDL05) value of 80.06 mg/kg/day for barium (ATSDR 2007b). BMDL05 is the lowest barium dose expected to be associated with a 5 percent increase in the incidence of renal nephropathy (ATSDR 2007). The ATSDR chronic oral MRL of  $2.0 \times 10^{-1}$  mg/ kg/day for barium is based on dividing the BMDL05 by a composite uncertainty factor of 100 (10 to account for extrapolation from animals to humans and 10 for human variability) (ATSDR 2007b). A modifying factor of 3 was included to account for deficiencies in the developmental oral toxicity database (ATSDR 2007b).

II.D.3.a. Comparison of Estimated Barium Doses to Health Effect Levels

ATSDR had no quantitative information regarding the solubility or extent of gastrointestinal absorption of barium compounds in the Melton Valley groundwater. ATSDR thus assumed all the barium in the groundwater was present as barium chloride, the soluble and toxic barium compound used in the 1994 NTP mouse study. In this health consultation, ATSDR then assumed the gastrointestinal tract absorbed 100 percent of the barium. In Table B2 in Appendix B, the chronic barium exposure dose is  $2.5 \times 10^{-1}$  mg/ kg/day for an infant ingesting Melton Valley groundwater containing the mean barium concentration in the OMW-2D. This dose is 320 times less than the BMDL05 of 80.06 mg/kg/day associated with a 5 percent increase in the incidence of renal nephropathy. Using the CTE scenario for average water intake and the mean concentration of barium in OMW-2D, the estimated chronic exposure doses for an infant ( $1.0 \times 10^{-1}$  mg/ kg/day) and an adult ( $2.6 \times 10^{-1}$  mg/ kg/day) were over 800 times less than the BMDL05 for barium. Considering

- Only one monitoring well had elevated barium levels,
- Only 2 out of 159 Melton Valley groundwater samples (fewer than 2 percent) had barium levels higher than the ATSDR groundwater comparison value,
- The conservative assumptions regarding the form of barium and gastrointestinal absorption,
- The health protective RME parameters used to estimate the infant exposure dose, and
- The estimated barium exposure doses of infants and adults were more than two orders of magnitude lower than the benchmark dose for barium,

ATSDR does not expect ingestion of barium at the levels reported in off-site Melton Valley groundwater to cause harmful health effects.

### II.D.4. Cadmium

In Table B1 in Appendix B, the estimated cadmium exposure doses for an infant and an adult drinking groundwater from two Melton Valley monitoring wells (OMW-1C, OMW-1D)



exceeded the ATSDR noncancer screening guideline for cadmium. These exposure doses were based on one groundwater sample from each well. The analysis of cadmium in the other five groundwater samples from OMW-1C and the other eight groundwater samples from OMW-1D did not detect any cadmium above the detection limits of 0.1 to 1.1 ppb. As such, ATSDR further examined the potential health effect of cadmium in the groundwater from these two Melton Valley monitoring wells. The cadmium concentrations detected in groundwater from the other Melton Valley wells and all the Bethel Valley wells were not at levels constituting a health hazard (i.e., the cadmium concentrations are below the ATSDR groundwater comparison value) and we won't further discuss them.

Cadmium is a soft, silver-white metal that occurs naturally in the earth's crust. All soils and rocks, including coal and mineral fertilizers, contain some cadmium (ATSDR 2012a). For the general population, food and cigarette smoke are the largest potential sources of cadmium exposure (ATSDR 2012a). Average cadmium levels in U.S. foods range from 2 to 40 ppb of cadmium in food (ATSDR 2012a). Average cadmium levels in cigarettes range from 1,000 to 3,000 ppb (ATSDR 2012a). The current U.S. average dietary intake of cadmium in adults is about  $4.0 \times 10^{-4}$  mg/kg/day; cigarette smokers receive an additional amount—about  $4.0 \times 10^{-4}$  mg/kg/day (ATSDR 2012a). The gastrointestinal tract does not absorb most of the ingested cadmium that passes through it (Kjellstrom et al. 1978). The GI tract only absorbs about 1-10% of ingested cadmium (ATSDR 2012a). Cadmium that is absorbed goes to the kidney and the liver and can remain there for many years (ATSDR 2012a). A small portion of the cadmium that enters the body leaves slowly in urine and feces (ATSDR 2012a).

The scientific literature examining the chronic toxicity of cadmium following oral exposure is extensive (ATSDR 2012a). The majority of the studies examine the relationship between urinary cadmium levels (or cumulative cadmium intake) and adverse health effects in the general population or in populations living in cadmium-polluted areas (ATSDR 2012a). ATSDR derived the chronic oral MRL ( $1.0 \times 10^{-4}$  mg/kg/day) for cadmium using meta-analysis of environmental exposure studies —this MRL is based on the whole dose-response curves from several studies rather than data from a single study (ATSDR 2012a). ATSDR used several large-scale environmental exposure studies to calculate the dietary cadmium intake ( $3.3 \times 10^{-4}$  mg/kg/day) resulting in a urinary cadmium level corresponding to a probability of 10% excess risk of kidney effects (e.g., proteinuria, or protein in the urine) (ATSDR 2012a). We divided this dietary cadmium intake by an uncertainty factor of 3 for human variability, resulting in the ATSDR MRL for chronic oral exposure to cadmium (ATSDR 2012a). Note, however, that because dietary cadmium intake is derived from the cadmium dietary exposure model—a model that estimates food cadmium concentrations from national survey data and from food consumption patterns—the MRL is not a precise value (ATSDR 2012a).

Still, ATSDR's chronic oral MRL  $(1.0 \times 10^{-4} \text{ mg/kg/day})$  is comparable to the U.S. EPA RfD  $(5.0 \times 10^{-4} \text{ mg/kg/day})$  for ingestion of cadmium in water (USEPA 1994). U.S. EPA calculated an oral chronic RfDs for cadmium based on the critical effect of significant proteinuria in humans chronically exposed to cadmium (USEPA 1994). U.S. EPA derived a NOAEL  $(5 \times 10^{-3} \text{ mg/kg/day})$  based on 200 mg of cadmium per gram wet weight in the renal cortex (highest renal cadmium level not associated with significant proteinuria) and a kinetic model assuming 5 percent cadmium absorption from water and 0.01 percent cadmium excretion per day (USEPA

1994). The RfD is based on an estimated NOAEL for cadmium in drinking water and an uncertainty factor of 10 (USEPA1994).

II.D.4.a. Comparison of Estimated Cadmium Doses to Health Effect Levels

When assuming 100 percent gastrointestinal absorption, the estimated cadmium exposure doses in Table B1 for an infant ingesting Melton Valley groundwater from monitoring wells OMW-1C and OMW-1D exceeded the noncancer screening guideline. Nevertheless, cadmium is only slowly absorbed (1 to 10%) from the gastrointestinal tract (ATSDR 2012a). To represent more realistic exposure scenarios in this evaluation. ATSDR recalculated the estimated cadmium exposure doses to account for poor cadmium absorption. ATSDR used the mean cadmium concentrations detected in the groundwater sample from each well-the RME scenario-and assumed absorption of 10 percent of the cadmium in the ingested groundwater. The recalculated cadmium exposure doses for an infant  $(5.7 \times 10^{-5} \text{ mg/ kg/day})$  and adult  $(1.4 \times 10^{-5} \text{ mg/ kg/day})$ drinking OMW-1C groundwater and for an adult  $(5.6 \times 10^{-5} \text{ mg/ kg/day})$  drinking OMW-1D groundwater were lower than the ATSDR MRL and the U.S. EPA RfD. The recalculated cadmium exposure dose  $(2.2 \times 10^{-4} \text{ mg/ kg/day})$  for an infant at OMW-1D was 2 times less than the U.S. EPA RfD and 22 times less than the NOAEL. As such, estimated cadmium exposure doses from drinking groundwater from these two wells were less than a MRL, RfD, and NOAEL and should not result in adverse health effects. ATSDR does not expect ingestion of cadmium at the reported levels in the off-site Melton Valley groundwater to cause harmful health effects.

Note too that cadmium was detected in only one out of six groundwater samples from OMW-1C and in one out of nine groundwater samples from OMW-1D. Cadmium was only detected in only two groundwater samples out of a total of 159 Melton Valley groundwater samples (1.3% of the groundwater samples) collected from 28 monitoring wells. These findings support further the conclusion that ingestion of cadmium at reported levels in off-site Melton Valley groundwater is not expected to cause harmful health effects.

### II.D.5. Chromium

In Appendix B - Table B1, the estimated chromium exposure doses for infants drinking groundwater from four Melton Valley wells (OMW-1AA, OMW-1B, OMW-1C, OMW-1D, and OMW-2C) exceeded the chromium VI screening guideline. In Table B3, the estimated chromium exposure doses from an infant and an adult ingesting Bethel Valley groundwater from one residential well (RWA-104) exceeded the screening guideline. As such, ATSDR further examined the potential health effect of chromium exposure from these monitoring wells. The chromium levels detected in the groundwater from the other Bethel Valley and Melton Valley wells were not at levels constituting a health hazard (i.e., the estimated doses were below the noncancer screening guideline) and we won't further discuss them.

Chromium is a naturally occurring element found in rocks, animals, plants, soil, and volcanic gases. The general population is exposed to chromium by inhaling ambient air, ingesting food, and drinking water containing chromium (ATSDR 2012b). The primary route of nonoccupational workers, however, is food ingestion (ATSDR 2012b). Typical drinking water supplies in the United States contain total chromium levels within a range of 0.2 to 35 ppb, but most U. S. drinking water supplies contain fewer than 5 ppb of chromium (USEPA 1984a, WHO 2004). Recent monitoring data of drinking water supplies in California indicated that 86 percent



of the sources tested had levels (reported for chromium(VI)) below 10 ppb (ATSDR 2012b). On the other hand, present-day workers in chromium-related industries can be exposed to chromium concentrations two orders of magnitude higher than the general population (ATSDR 2012b).

Chromium occurs in the environment in several chemical forms, depending on the valance state of the chromium metal (e.g., trivalent [III] chromium or hexavalent [VI] chromium). Trivalent chromium (chromium III)—an essential nutrient—is more likely found in the environment and in in the body than is hexavalent chromium. Chromium III helps regulate how the body uses insulin (ATSDR 2012b). Hexavalent chromium (chromium VI) is considerably more toxic to humans than is trivalent chromium. U.S. EPA has not identified any critical studies to determine the carcinogenic potential of chromium VI via oral exposure (USEPA1998a). But gastrointestinal absorption of orally ingested chromium is relatively poor, with absorption rates for the trivalent and hexavalent forms at below 10 percent (ATSDR 2012b).

After examining the scientific literature, ATSDR has not established a screening guideline for ingestion of chromium III. We did not find any chronic-duration studies on oral exposure of humans to chromium III compounds. The several animal studies we did find showed no adverse effects associated with chronic-duration oral exposure to chromium III compounds, even at high daily doses (ATSDR 2012b). Given that chromium III is an essential nutrient required for normal energy metabolism, the National Research Council has established an adequate intake level of 20–45 ppb chromium III for adolescents and adults, equivalent to  $2.8 \times 10^{-4}$  to  $6.4 \times 10^{-4}$  mg/kg/day, assuming a 70-kg body weight (IOM 2001).

For chromium VI compounds, however, the scientific literature contains at least limited data on chromium VI's chronic oral toxicity in humans (ATSDR 2012b). In more extensive animal studies, nonneoplastic lesions (abnormality in the tissue) of the duodenum (short part of the small intestine that connects to the stomach) are the most sensitive effect following long-term oral exposure to chromium VI (NTP 2008). ATSDR used a benchmark dose (BMD) analysis of lesions of the duodenum in female mice in a 2-year mouse drinking-water study to derive the lowest benchmark dose limit (BMDL10) value of  $9.0 \times 10^{-2}$  mg/kg/day for chromium VI (NTP 2008). This BMDL10 is the lowest chromium VI dose expected to be associated with a 10 percent increase in the incidence of duodenum lesions. The ATSDR chronic oral MRL of  $9.0 \times 10^{-4}$ mg/ kg/day for chromium VI is based on dividing the BMDL10 by a composite uncertainty factor of 100 (10 for extrapolation from animals to humans and 10 for human variability) (ATSDR 2012b).

In workers, inhalation of chromium (VI) has been shown to cause lung cancer. Mixed results have been found in studies of populations living in areas with high levels of chromium (VI) in the drinking water (ATSDR 2012b). In laboratory animals, chromium (VI) compounds have been shown to cause tumors to the stomach, intestinal tract, and lung (ATSDR 2012b). The oral carcinogenicity of Cr(VI) cannot be determined (USEPA 1998). Data in the available literature do not suggested that chromium (VI) is carcinogenic by the oral route of exposure.

II.D.5.a. Comparison of Estimated Chromium Doses to Health Effect Levels

To represent more realistic exposure scenarios for this level of evaluation, ATSDR recalculated the estimated exposure doses to account for chromium's poor gastrointestinal absorption. ATSDR assumed that the gastrointestinal tract absorbed 10 percent of the chromium. Further, because the environmental data are not specific as to valence, ATSDR estimated exposure doses

on the assumption that all chromium in the groundwater was present as the more toxic chromium VI form. When using the RME scenario and assuming 10 percent gastrointestinal absorption, all the recalculated chromium exposure doses for Melton Valley wells (OMW-1AA, OMW-1B, OMW-1C, OMW-1D, and OMW-2C) and the Bethel Valley well (RWA-104) are below the ATSDR chronic oral MRL of  $9.0 \times 10^{-4}$ mg/ kg/day for chromium (VI). The maximum recalculated chromium exposure dose of  $6.7 \times 10^{-4}$  mg/ kg/day for an infant drinking Melton Valley groundwater from monitoring well OMW-2C is 1.3 times below the MRL for chromium VI and 134 times below the BMDL10 for the chromium VI that's associated with a 10 percent increase in the incidence of nonneoplastic lesions of the duodenum. The maximum chromium exposure dose for an infant drinking Bethel Valley groundwater from RWA-104 is 2 times less than the MRL and 219 times less than the BMDL10. Therefore, ATSDR does not expect chromium ingestion at the levels reported in off-site Melton Valley and Bethel Valley groundwater to cause harmful health effects.

### II.D.6. Lead

The concentrations of lead in two Melton Valley monitoring wells (OMW-1C, OMW-1D) exceeded U.S. EPA's 15-ppb "action level" for lead in drinking water. As such, ATSDR further examined the health effect of lead in the groundwater from these two monitoring wells. In the other Melton Valley wells, the mean lead concentrations ranged from 0.4 to 2.9 ppb, with maximum lead concentrations ranging from 0.9 to 7.7 ppb. Only two wells had groundwater samples with lead concentrations higher than 5 ppb. Based on the upper mean lead concentration of 2.9 ppb in groundwater, a 100 ppm lead in soil, and other default exposure variables, the integrated exposure uptake biokinetic (IEUBK) model predicts the exposed population would have a mean blood lead concentration of  $1.7\mu$ g/dL (micrograms of lead per deciliter of blood) over 84 months of exposure. In addition, the IEUBK model predicts 99 percent of the exposed population would have a blood lead concentration below 5  $\mu$ g/dL. Therefore, the groundwater from these other Melton Valley and all Bethel Valley monitoring wells<sup>3</sup> were not at levels expected to constitute a health hazard and we won't further discuss them.

Lead is a heavy metal that occurs naturally in the earth's crust and is usually found combined with two or more other elements to form lead compounds. Most of the high levels of lead found throughout the environment come from human activities (ATSDR 2007c). Lead enters the environment through releases from mining lead or other metals and from factories that make or use lead, lead alloys, or lead compounds. Lead releases into the air from burning coal, oil, or waste. In former times, most of the lead released into the U.S. environment came from vehicle exhaust, before lead as a gasoline additive was gradually phased out and, by 1995 in the United States, completely banned (ATSDR 2007c).

The greatest potential for human exposure to lead arises from its previous use as an additive in gasoline—resulting in widespread dispersal throughout the environment—and its previous use as a pigment in both interior and exterior paints (ATSDR 2007c). Human exposure to lead continues; lead does not degrade to other substances (ATSDR 2007c). Leaded paint is still prevalent in many older homes in the United States, and peeling or flaking paint contributes to indoor and outdoor lead-dust levels.

<sup>&</sup>lt;sup>3</sup> Maximum concentration ranged from 0.2 to 2.6 ppb.



In general, only small amounts of lead appear in lakes, rivers, or groundwater used in public drinking water supplies (ATSDR 2007c). More than 99% of all publicly supplied drinking water contains fewer than 5 ppb (ATSDR 2007c). Still, in communities with acidic water supplies, the amount of lead the body takes in through drinking water can be higher. Acidic water makes it easier for the lead in pipes, leaded solder, and brass faucets to dissolve and mix with drinking water. The amount of lead contained in pipes and plumbing fittings has been strictly regulated since 1988; but human exposure to lead from drinking water still occurs because of leaching of lead from corroding pipes and fixtures or lead containing solder (ATSDR 2007c). The U.S. EPA requires public water distribution systems to reduce the corrosiveness of water if more than 10 percent of the water samples exceed 15 ppb of lead (USEPA 2012).

Most exposure to lead occurs through swallowing food, drinking liquids, dust from hand-tomouth activity, and paint that contains lead (ATSDR 2007c). Little of the lead swallowed actually enters the blood and other parts of the body (ATSDR 2007c). The amount that gets into the blood from the stomach partially depends on when the last meal was eaten, someone's age, and how well the lead particles eaten dissolved in the stomach juices (ATSDR 2007c). Experiments using adult volunteers showed that for adults who had just eaten, the amount of lead that got into the blood from the stomach was only about 6% of the total amount taken in (ATSDR 2007c). In adults who had not eaten for a day, about 60–80% of the lead from the stomach got into their blood (ATSDR 2007c). In general, if adults and children swallow the same amount of lead, a bigger proportion of the amount swallowed will enter the blood in children than in adults. Children absorb about 50% of ingested lead (ATSDR 2007c).

Shortly after lead enters the blood it travels to the "soft tissues" and organs (e.g., the liver, kidneys, lungs, brain, spleen, muscles, and heart) (ATSDR 2007c). After several weeks, most of the lead moves into bones and teeth. In adults, the bones and teeth contain about 94% of the total amount of lead in the body (ATSDR 2007c). About 73% of the lead in children's bodies is stored in their bones (ATSDR 2007c). Some of the lead can stay in bones for decades; some lead, however, can leave the bones and reenter blood and organs under certain circumstances (e.g., during pregnancy and periods of breast feeding, after a bone is broken, and during advancing age) (ATSDR 2007c).

The lead not stored in bones leaves the body in urine or feces (ATSDR 2007c). About 99% of the amount of lead taken into the body of an adult will leave in the waste within a couple of weeks, but only about 32% of the lead taken into the body of a child will leave in the waste (ATSDR 2007c). Under conditions of continued exposure, not all of the lead that enters the body will be eliminated; this might result in accumulation of lead in body tissues, especially bone (ATSDR 2007c).

Analysis of lead in whole blood is the most common and accurate method of assessing lead exposure. As regulations have reduced exposure over the past three decades regarding lead paint, leaded fuels, and lead-containing plumbing materials, blood lead levels (BLL) in the U. S. population have decreased (ATSDR 2007c). BLL measured as a part of the National Health and Nutrition Examination Surveys (NHANES) indicate that from 1976 to 1991, the mean BLL of the U.S. population aged 1 to 74 years dropped from 12.8 to 2.8  $\mu$ g/dL (ATSDR 2007c). The prevalence of BLL higher than10  $\mu$ g/dL in the U.S. population also decreased sharply from 77.8 to 4.3% (ATSDR 2007c). From the sampling data conducted for 1999–2002, 1.6% of children

aged 1–5 years had BLL higher than 10  $\mu$ g/dL, with a geometric mean BLLs of 1.9  $\mu$ g/dL (ATSDR 2007c).

The scientific literature contains an abundance of information on the health effects of lead on human health (ATSDR 2007c). In fact, although the toxic effects of lead have been known for centuries, discoveries in the just the past few decades have shown that relatively low levels of lead in blood are associated with adverse health effects. The most sensitive targets for lead toxicity are the developing nervous system, the hematological and cardiovascular systems, and the kidney (ATSDR 2007c).

The preponderance of the evidence indicates that lead exposure can impair cognitive function in children and adults, but children are more vulnerable than are adults (ATSDR 2007c). The increased vulnerability is due in part to children more likely contacting lead-contaminated surfaces; children play on the ground and engage in hand-to-mouth activities, and children absorb a larger fraction of ingested lead than do adults (ATSDR 2007c). But perhaps more important is the fact that the developing nervous system is especially susceptible to lead toxicity (ATSDR 2007c). During brain development, lead interferes with the trimming and pruning of synapses, migration of neurons, and neuron/glia interactions (ATSDR 2007c). Alterations of any of these processes may result in failure to establish appropriate connections between structures and eventually in permanently altered functions (ATSDR 2007c). Because different brain areas mature at different times, the final outcome of exposure to lead during development (i.e., *in utero* vs. pediatric exposure) will vary depending on the time of exposure (ATSDR 2007c). Many studies have associated with decrements in cognitive function (IQ decline of 1–5 points) with an increase in BLL of 10 µg/dL (ATSDR 2007c).

In 2012, the Centers for Disease Control and Prevention's (CDC) Advisory Committee on Childhood Lead Poisoning Prevention (ACCLPP) concluded BLLs lower than 10  $\mu$ g/dL harm children (ACCLPP 2012). This conclusion is based on a growing body of studies with a large and diverse group of children with low BLLs and associated IQ deficits (ACCLPP 2012). Effects at BLLs below 10  $\mu$ g/dL are also reported for attention-related deficit behaviors, specifically impulsivity and decreased academic achievement such as reading and writing (ACCLPP 2012). These effects do not appear confined to lower socioeconomic status populations (ACCLPP 2012). Also, studies suggest that the adverse health effects of BLLs below 10  $\mu$ g/dL in children extend beyond cognitive function to include cardiovascular, immunological, and endocrine effects (ACCLPP 2012). The ACCLPP underscores the critical importance of primary prevention at an early age, given the absence of an identified BLL without deleterious effects and the evidence that BLL effects appear irreversible (ACCLPP 2012).

ACCLPP's review of the scientific evidence has resulted in a recommendation of a childhood BLL reference value based on the 97.5th percentile of the population BLL in children ages 1–5 (currently 5  $\mu$ g/dL); ACCLPP recommends that reference value to identify children and environments associated with lead-exposure hazards [ACCLPP 2012]. CDC should update the reference value every 4 years based on the most recent population-based blood lead surveys among children [ACCLPP 2012]. This reference value is useful to characterize a person's BLL results as "elevated" or "not elevated" in comparison with the population average [ACCLPP 2012]. These values have also been used to set health policy goals and to interpret results from measures of chemical exposure [ACCLPP 2012].



U.S EPA's RfD Work Group considered developing a RfD for inorganic lead; but the work group concluded that was inappropriate given the medical observations and scientific research obtained over the decades (USEPA 2012). This extensive information indicates the degree of uncertainty is low about health effects of lead, and that the effects of children's neurobehavioral development could occur at blood levels so low as to be without a threshold (USEPA 2012).

U.S. EPA has developed a number of lead exposure levels in support of regulatory decisionmaking. Under the National Primary Drinking Water Regulations, which are legally enforceable standards that apply to public water systems, U.S. EPA established an action level of 15 ppb for lead in drinking water for treatment techniques (USEPA 2012). The action level is set as close to the Maximum Contaminant Level Goal (MCLG) of 0 ppb for lead as feasible, using the best available treatment technology and taking cost into consideration (USEPA 2012). This action level is the highest level of lead allowed in drinking water and is regulated by a treatment technique. If more than 10% of tap water samples exceed the 15 ppb action level for lead, water systems must take additional steps to control the corrosiveness of their water (USEPA 2012).

#### II.D.6.a. Comparison of Estimated Lead Doses to Health Effect Levels

In the Melton Valley monitoring well OMW-1C, lead was detected in only one of the six groundwater samples at a concentration of 23.1 ppb, which is 1.5 times the U.S. EPA action level. Lead was detected in eight of the nine groundwater samples from monitoring well OMW-1D, with a mean lead level of 12.7 ppb (close to the U.S. EPA action level) and a range of lead levels from 0.63 ppb to 100.0 ppb. One OMW-1D groundwater sample contained a lead level of 7.5 ppb, two OMW-1D samples contained lead levels between 1 and 2 ppb, and the other five OMW-1D samples contained fewer than 1 ppb lead.

Recognizing no identified blood lead level is without effects on children's neurobehavioral development, and recognizing that such effects appear irreversible, lead has no safe level, and lead exposures should be as low as possible (ACCLPP 2012; USEPA 2012). Thus, chronic ingestion of groundwater from these two off-site Melton Valley DOE monitoring wells with elevated lead levels could result in lead exposure that could impair cognitive function in infants and children and are a potential public health hazard. Yet neither of these two DOE monitoring wells (OMW-1C, OMW-1D) are used for home purposes. No one drinks the lead-contaminated groundwater from these wells. ATSDR thus considers the groundwater in these two DOE monitoring wells to be a *potential* rather than *actual* public health hazard.

The source of the lead in the Melton Valley groundwater in unknown as is the vertical and lateral extent of lead contaminate in the groundwater. As noted previously, most residents in the Melton Valley area have chosen to stop using their private well and connect their homes to the Watts Bar Utility District water supply. But a few Melton Valley residents with private wells continue to use the Melton Valley groundwater for home purposes. For most of these private wells, ATSDR does not have any results of chemical or radiological analysis of groundwater from these private Melton Valley wells and we are unable to determine the health implications of using the groundwater from these private wells for home purposes.

#### II.D.7. Lithium

In Tables B1 and B3 of Appendix B, the mean level of lithium in the groundwater of 21 Melton Valley wells and three Bethel Valley residential wells resulted in estimated infant exposure doses

exceeding the lithium screening guideline. Nine of these Melton Valley wells and one Bethel Valley well contain estimated adult lithium exposure doses above the screening guideline. As such, ATSDR examined further the health effects of lithium from ingestion of these wells' groundwater. The lithium levels detected in the groundwater from the other Bethel Valley and Melton Valley wells were not at levels constituting a health hazard (i.e., the estimated exposure doses of lithium in groundwater were below the screening guideline).

Lithium is the lightest metal in the periodic system. It's a soft silvery white metal which, when exposed to air, quickly becomes covered with a gray oxidation layer (Lagerkvist 2002). Lithium belongs to the sodium and potassium group and, accordingly, these elements have similar physical, chemical, and some biological properties (VDH 1997).

Lithium and its compounds are widely used in a variety of applications. These applications include:

- Special alloys in metallurgy,
- Welding and brazing fluxes,
- Metallic lithium in batteries,
- Specialized glass and ceramics,
- Moisture absorber in air conditioning,
- Pharmaceutical manufacture of psychiatric drugs,
- Thickener or gelling agent for lubrication grease, and
- Lithium-6 isotope used to produce tritium for thermonuclear weapons and as a breeding material for nuclear-fusion reactors (USEPA 2008; VDH 1997; Lagerkvist 2002).

Lithium is moderately abundant element widely distributed in the environment, although unevenly and in low concentrations (Lagerkvist 2002). The lithium content in the earth's crust is 50–65 ppm (Lagerkvist 2002). Lithium levels in soils range from 10 to 100 ppm in the United States and from 10 to 50 ppm in, for example, Russia (Lagerkvist 2002). Lithium levels in water span a wide range worldwide, with surface water at levels between 1 and 10 ppb (Schrauzer 2002). The average lithium level in seawater is around 200 ppb, which is about 100 times higher than the 2-ppb median fresh water level (Gillman 2012, Lagerkvist 2002). High concentrations of lithium also occur in water from hot springs and in certain mineral waters.

Natural background exposure to lithium from food and drinking water varies with geographical location and consumption patterns. Lithium appears in variable amounts in foods; primary food sources are grains and vegetables. In some areas, drinking water provides significant amounts (Schrauzer 2002). Studies report different countries' lithium intake levels from food vary from 0.02 to 0.54 mg/day (Lagerkvist 2002). The intake from drinking water varies from below 0.001 to approximately 0.3 mg/day (Lagerkvist 2002). Studies have reported very high lithium intakes of more than 5 mg/day from drinking water in mineral-rich soil areas of northern Chile (Lagerkvist 2002). A U.S. assessment of daily lithium intake from food ranged from 0.58–2.8 mg/day, with the range based on variation in lithium levels in vegetables and grains (Lagerkvist 2002). The assessment reported that consumption of mineral supplements could result in an additional internal dose of 5–6 mg/day (Lagerkvist 2002). Intake of lithium from municipal drinking water was calculated as up to 1.4 mg/day (Lagerkvist 2002). One study reported the average body burden of lithium in an adult as 2.2 mg (Lagerkvist 2002).



Like sodium and potassium, ingested lithium is readily and almost completely absorbed into the gastrointestinal tract and uniformly distributed in body water (VDH 1997; Lagerkvist 2002). Lithium is normally present in all organs and tissues (Schrauzer 2002). Excretion is chiefly through the kidneys, but some is eliminated in the feces (VDH 1997). The kidneys excrete unchanged over 95% of a single oral dose of lithium ion (Lagerkvist 2002). Between one-third and two-thirds of an administered dose is excreted during the initial 6–12 hour phase, followed by slow excretion over the next 10–14 days (Lagerkvist 2002).

Medicine has used lithium carbonate and lithium citrate (lithium salts) as a psychiatric drug for almost half a century to treat manic-depressive illness (bipolar disorder). A large body of clinical literature on lithium-induced toxicity includes a number of reviews and books on lithium pharmacokinetics (USEPA 2008; Lagerkvist 2002; Salocks 2003). Lithium is administered therapeutically in daily oral doses of 400–1800 mg/day as lithium carbonate (a carbonate salt) for the treatment of manic and endogenous depression (USEPA 2008; Salocks 2003; Gillman 2012). Lithium's dominant pharmacological activity is changes in neurotransmission, neuroendocrine function, and renal mechanisms (Lagerkvist 2002). But lithium has a low therapeutic index (i.e., ratio of dose associated with therapeutic efficacy to dose associated with adverse effects) that results in adverse effects at dose levels associated with therapeutic dose levels (USEPA 2008). Given that all therapeutic serum concentrations are associated with adverse effects, long-term treatment strategies for individual patients need to balance the beneficial effects of lithium therapy with the risks and severity of toxicity (USEPA 2008). Serum lithium concentrations are often monitored to maintain optimum dosage; serum lithium concentrations strongly correlate with symptoms of lithium poisoning (Lagerkvist 2002; Salocks 2003). Poisoning can occur in patients whose lithium dosage has increased or in persons whose renal function has decreased, resulting in an increase in serum lithium levels (Lagerkvist 2002). In most patients, disorders of water and electrolyte balance precede the lithium intoxication (Lagerkvist 2002). But ample evidence supports the view that at low levels, lithium causes no serious adverse health effects (VDH 1997).

Manic depressive illness (bipolar disorder) is treated with therapeutic lithium serum levels around 0.5 to 1.4 millimole/L (mmol/L) (or 3.5 to 9.7 mg/l), which are usually achieved with daily doses of about 400–1800 mg of lithium carbonate (Gillman 2012; USEPA 2008). Concentrations of 0.8–1.0 mmol/L, however, are generally accepted as providing optimal therapeutic effects (PDR 2006; Baldessarini 2001). Some of the common side effects at these therapeutic lithium levels are moderate nephrogenic diabetes insipidus (form of diabetes insipidus primarily due to pathology of the kidney), fine hand tremor, weight gain, increased thyroid-stimulating hormone (TSH) values, hypothyreosis, and diarrhea (Lagerkvist 2002; Salocks 2003). The most common adverse renal effect is nephrogenic diabetes insipidus (USEPA 2008). Studies have estimated that renal concentrating ability is impaired in at least 50% of patients undergoing lithium treatment, with polyuria (passing of an excessive quantity of urine) and polydipsia (excessive thirst) in approximately 20% of patients (Presne 2003; Gitlin 1999; McIntyre 2001). Another common neuropsychiatric side effect is hand tremor, which occurs in 25–50% of patients and diminishes over time (Lagerkvist 2002).

Lithium toxicity in humans might occur at serum levels of about 1.0 mmol/L (or 6.94 mg/l), but toxic effects typically appear more frequent when serum levels increase above 1.5 mmol/L (or 10.4 mg/l) (Gillman 2012; Salocks 2003). Mild toxicity can occur at serum levels from 1.5 to 2.0 mmol/L (or 10.4 mg/l to 13.8 mg/l) with signs of tremor, lethargy, irritability, muscle
weakness and slurred speech (Lagerkvist 2002). Lithium serum levels of 2.0 to 2.5 mmol/L (or 13.3 to 17.3 mg/l) is classified as moderate intoxication, with signs of disorientation, confusion, drowsiness, restlessness, unsteady gait, coarse tremor, dysarthria, and muscle fasciculations (Lagerkvist 2002). As toxicity develops, progressive central nervous system impairment occurs (Lagerkvist 2002). Lithium serum levels higher than 2.5 mmol/L are classified as severe intoxication, and lithium serum levels higher than 3.5 mmol/L (or 24.9mg/l) are potentially fatal (Lagerkvist 2002).

The extensive body of literature and data on the adverse effects associated with the long-term oral lithium therapy in the treatment of bipolor disorders lacks adequate dose-response data to identify a single critical effect for lithium exposure (USEPA 2008). But retrospective and prospective studies and findings in case reports provide consistent evidence that the kidney is a primary target organ for lithium in men and women; the reports also provide evidence that adverse renal effects occur over the range of desired therapeutic serum concentrations (0.6-1.4 mmol Li/L) (USEPA 2008). Due to its serious nature and its frequency of occurrence, lithiuminduced renal toxicity has been the subject of numerous clinical and animal studies. The most common adverse renal effect is nephrogenic diabetes insipidus, which reduces the capacity of the kidneys to preserve free water, resulting in impaired renal-concentrating ability and the production of excessively dilute urine (USEPA 2008). Clinically, this manifests as polyuria, with secondary thirst and volume depletion (USEPA 2008). Nephrogenic diabetes insipidus appears to be reversible early in treatment, but may be progressive during the first decade, leading to irreversible damage over time (Gitlin 1999). A small percentage of patients show progressive renal failure, indicated by a pronounced decrease in glomerular filtration rate and renal insufficiency, with little or no proteinuria (Markowitz et al. 2000). Severe decreases in glomerular filtration rates have resulted in the need for maintenance hemodiaylsis, typically after 10 or more years of lithium therapy (USEPA 2008).

The available animal data show that lithium produces adverse effects in several organs and systems at exposure levels that result in serum lithium concentrations in same range as those targeted for therapeutic use in humans (USEPA 2008). The lower bound of the concentration range for therapeutic serum lithium (0.6 mmol/L) is identified as the LOAEL for increased urine volume and decreased urine-concentrating ability (USEPA 2008). The available literature on clinical and animal studies does not identify a NOAEL for adverse effects associated with therapeutic lithium (USEPA 2008).

U.S. EPA selected this 0.6-mmol/L LOAEL as the derivation basis for oral lithium exposure's provisional, chronic RfD (USEPA 2008). Using the pharmacokinetic of lithium, a steady-state serum lithium concentration of 0.6 mmol/L (4.2 mg/L) in a 70-kg adult corresponds to a LOAEL dose of 2.1 mg/kg/day (USEPA 2008). The provisional chronic RfD for lithium of  $2 \times 10^{-3}$  mg/kg/day was derived by dividing the LOAEL of 2.1 mg/kg/day by an uncertainty factor of 1000. The uncertainty factor includes a factor of 10 to extrapolate from a LOAEL to a NOAEL, a factor of 10 to protect susceptible individuals, and a factor of 10 to account for database insufficiencies (USEPA 2008).

Lithium concentrations in serum of nonpatient populations—including exposed workers—are low: in the order of a 1000 times lower than the concentrations found in patients receiving lithium drugs (Lagerkvist 2002). For this reason, systemic adverse effects due to lithium (e.g., nephrogenic diabetes insipidus, fine hand tremor, weight gain, increased TSH values) are



unlikely to occur at occupational and background environmental exposure levels to lithium and lithium compounds (Lagerkvist 2002).

#### II.D.7.a. Comparison of Estimated Lithium Doses to Health Effect Levels

In Tables B1 and B3, all the estimated lithium exposure doses are below the LOAEL of 2.1 mg/kg/day for infants and adults drinking groundwater from Melton and Bethel Valley monitoring wells under the RME scenario. But in one Melton Valley well (OMW-1D) the mean lithium concentration for the RME and the CTE scenarios results in estimated infant exposure doses within an order of magnitude of the LOAEL (See Table 8). Under the RME scenario, the estimated exposure doses for an infant are within a factor of 4 of the LOAEL, and the estimated adult doses are within a factor of 16 of the LOAEL. Under a CTE scenario with average or typical water intake, the estimated exposure doses for an infant are within a factor of 9, and an adult exposure dose is within a factor of 40 of the LOAEL. This LOAEL is based on the lower bound of the therapeutic serum lithium range, which often results in nephrogenic diabetes insipidus, with increased urine volume and decreased urine-concentrating ability. These adverse renal effects typically occur within the first 2 years of receiving therapeutic doses of lithium and are reversible early in treatment. But the effects might be progressive during the first decade, leading to irreversible damage over time. Thus, an infant and adult chronically drinking groundwater from the Melton Valley well OMW-1D at a higher than average water intake rate, and even at an average water intake rate, could receive an exposure dose near the lower bound of that desired therapeutic lithium dose range known to cause nephrogenic diabetes insipidus—an adverse renal effect.

And using the RME scenario, the estimated lithium exposure doses are within two orders of magnitude of the LOAEL for an infant in six Melton Valley wells (OMW-1A, OMW-1AA, OMW-1B, OMW-1C, OMW-2C, OMW-2D) and for an adult in three Melton Valley wells (OMW-1B, OMW-2C, OMW-2D). Using the CTE scenario, the estimated lithium exposure dose for an infant ingesting groundwater from Melton Valley monitoring wells OMW-1B, OMW-2C, and OMW-2D is within 50 times the LOAEL (see Table 8). Chronic ingestion of groundwater from three monitoring wells (OMW-1B, OMW-2C, OMW-2D) by an infant or an adult might result in nephrogenic diabetes insipidus; because the estimated exposure doses are within two orders of magnitude of the LOAEL and a NOAEL has not been derived for lithium. Nevertheless, none of the off-site DOE monitoring wells in Melton Valley are used for home purposes, and no one is known currently to ingest any Melton Valley groundwater containing lithium at public health hazard levels. ATSDR considers the levels of lithium in Melton Valley groundwater to be a *potential*, as opposed to an *actual*, public health hazard.

Again as previously stated, most Melton Valley area residents have chosen to stop using their private well and connect their residences to the Watts Bar Utility District public water supply. Still, some Melton Valley residents with private wells continue to use the groundwater for home purposes. Such persons, especially those undergoing lithium treatment, need to be careful about drinking Melton Valley groundwater that contains lithium. The additional lithium in the groundwater will increase lithium dose levels and will increase the risk of nephrogenic diabetes insipidus. Also, the source of lithium and the vertical and lateral extent of the lithium contaminant plume are unknown, and ATSDR does not have any results of chemical or

radiological analysis of groundwater from some of these private wells still used for home purposes.

For Bethel Valley, all the estimated infant and adult lithium exposure doses in Table B3 above the lithium RfD are more than three orders of magnitude lower than the lithium LOAEL of 2.1 mg/kg/day. Therefore, chronic ingestion of lithium at the reported levels in off-site Bethel Valley groundwater is not expected to cause harmful health effects.

Melton Valley		Mean	Exposure Dose (mg/kg/day)		
Well Station		Concentration (ppb)	Infant	Adult	
	RME scenario	186.0	2.6E-02	6.6E-03	
	CTE scenario	100.0	1.2E-02	2.7E-03	
OMW-1AA	RME scenario	272 F	3.8E-02	9.7E-03	
	CTE scenario	272.0	1.8E-02	4.0E-03	
	RME scenario	705.0	1.0E-01	2.5E-02	
UIVIVV-ID	CTE scenario		4.5E-02	1.0E-02	
	RME scenario	305.8	4.3E-02	1.0E-02	
UNIW-IC	CTE scenario		1.9E-02	4.5E-03	
OMW-1D	RME scenario	3584.4	5.1E-01	1.3E-01	
	CTE scenario		2.3E-01	5.2E-02	
OMW-2C	RME scenario	1223.0	1.7E-01	4.3E-02	
	CTE scenario		7.9E-02	1.8E-02	
OMW-2D	RME scenario	839.7	1.2E-01	2.9E-02	
	CTE scenario		5.4E-02	1.2E-02	

 Table 8. Estimated Exposure Doses for Lithium In Melton Valley Groundwater

 $ppb - part per billion = micrograms per liter (\mu g/L)$ 

mg/kg/day - milligram per kilogram per day

RME scenario – higher than average water intake rates, high end of the exposure distribution (approximately 95<sup>th</sup> percentile)

CTE scenario – average or typical water intake rates

OMW- Off-site Monitoring Well

## II.D.8. Manganese

Table B1 in Appendix B shows the estimated manganese exposure doses for an infant drinking groundwater from the two Melton Valley wells (OMW-3B, OMW-3C) that exceed the ATSDR noncancer screening guideline for manganese. As such, ATSDR further examined the health effect of manganese from the ingesting groundwater from these two monitoring wells. The concentrations of manganese detected in groundwater from the other Melton Valley wells and all the Bethel Valley wells are not at levels constituting a health hazard (i.e., the manganese concentrations are below the ATSDR groundwater comparison value) and we won't further discuss them.

Manganese is a natural constituent of many rock types and comprises about 0.1 percent of the earth's crust. Manganese routinely appears at low levels in groundwater, drinking water, and soil, routinely appears naturally in most foods, and might even be added to some foods. It's an essential trace element the body needs to break down amino acids and produce energy (ATSDR 2012c). The primary exposure to manganese is eating food or manganese-containing nutritional supplements. Vegetarians who consume foods rich in manganese such as grains, beans, and nuts,



as well as heavy tea drinkers, might have a higher intake of manganese than would the average person (ATSDR 2012c). Drinking water containing manganese or swimming or bathing in water containing manganese could result in exposure to low levels. When ingested, the body absorbs only 3 to 5 percent of manganese, with most of the ingested manganese excreted in feces. (Davidson et al. 1988; Mena et al. 1969). Typically, people have small amounts of manganese in their bodies. Under normal circumstances, the body regulates the amount so it has neither too much nor too little (USEPA 1984b). If someone consumes large amounts of manganese, large amounts are excreted.

Because of uncertainties associated with drinking water studies of children, ATSDR is unable to derive a chronic oral MRL for manganese (ATSDR 2012c). But because of the prevalence of manganese at hazardous waste sites and the fact that manganese is an essential nutrient, ATSDR uses an interim guidance value of  $1.6 \times 10^{-1}$  mg/kg/day (ATSDR 2012c). The interim guidance value is based on the Tolerable Upper Intake Level for adults of 11 mg/day established by the U.S. Food and Nutrition Board/ Institute of Medicine (FNB/IOM 2001) based on a NOAEL for Western diets (ATSDR 2012c).

II.D.8.a. Comparison of Estimated Manganese Doses to Health Effect Levels

The data in Table 3B show that the maximum estimated infant manganese exposure dose of 5.7  $\times 10^{-2}$  mg/kg/day, based as it is on the RME scenario and 100 percent absorption, is 2.8 times less than the interim guidance value for manganese. Therefore, considering

- Only a small percent of manganese is typically absorbed,
- The homeostatic mechanism regulates the amount manganese in the body, and
- The estimated dose is below the guidance value,

ATSDR concludes no harmful health effects will result from chronic ingestion of manganese at the reported levels in off-site Melton Valley groundwater.

### II.D.9. Strontium

The data in Table B1 in Appendix B show that the estimated strontium exposure doses for an infant drinking groundwater from two Melton Valley wells (OMW-1D, OMW-2D) exceeded the ATSDR noncancer screening guideline for strontium. As such, ATSDR further examined the health effect of ingesting strontium in the groundwater from these two monitoring wells. The strontium concentrations detected in groundwater from the other Melton Valley wells and all the Bethel Valley wells were not at levels constituting a health hazard (i.e., the strontium concentrations were below the ATSDR groundwater comparison value) and we won't further discuss them.

Strontium is a naturally occurring element found usually as a constituent of rocks, soil, dust, coal, and oil (ATSDR 2004). Naturally occurring strontium is not radioactive and is known either as stable strontium or simply strontium. Strontium can form a variety of stable compounds in soil, can dissolve in water, and can move deeper in the soil to underground water (ATSDR 2004). Strontium compounds are in ceramics and glass products, pyrotechnics, paint pigments, fluorescent lights, and medicines (ATSDR 2004). Strontium can also appear as several radioactive isotopes; the most common of which is <sup>90</sup>Sr (ATSDR 2004). <sup>90</sup>Sr forms in nuclear

reactors or during explosion of nuclear weapons. As it decays, radioactive strontium generates beta particles.

Stable strontium dissolved in water originates from strontium in rocks and soil that water runs over and through (ATSDR 2004). Some strontium is suspended in water. Typically, the amount of strontium measured in drinking water in different parts of the United States is less than 1 ppm (ATSDR 2004). Strontium is present in nearly all fresh waters, with concentrations ranging between 0.5 and 1.5 ppm and with higher levels occurring in the vicinity of celestite-rich limestone deposits (ATSDR 2004).

Strontium is found nearly everywhere, albeit in small amounts. Exposure to low levels of strontium occurs by breathing air, eating food, drinking water, or accidentally eating soil or dust that contains strontium (ATSDR 2004). Food and drinking water are the largest sources of exposure to strontium (ATSDR 2004). Because of the nature of strontium, some of it gets into fish, vegetables, and livestock. But for humans, the greatest percentage of dietary strontium comes from grain, leafy vegetables, and dairy products. The total estimated daily exposure to stable strontium is approximately 3.3 mg/day ( $4.6 \times 10^{-2}$  mg/kg/day): 2 mg/day from drinking water and 1.3 mg/day from the diet (ATSDR 2004).

When someone ingests strontium, only a small portion leaves the intestines and enters the bloodstream (ATSDR 2004). Studies in animals suggest that infants might absorb more strontium from the intestines than do adults (ATSDR 2004). Studies conducted in infants and children indicate that approximately 15–30 percent of dietary strontium is absorbed, similar to estimates in adults (ATSDR 2004). Most of the ingested strontium is eliminated through feces during the first day or so after exposure (ATSDR 2004). Once strontium enters the bloodstream it's distributed throughout the body; a large portion will accumulate in bone and will be stored in the bone for a long time (i.e., a period of years) (ATSDR 2004). Because bones in young animals are actively growing, young animals are more sensitive than are adult animals to excessive strontium intake (Storey1962).

The scientific literature indicates that data are sparse for harmful health effects of stable strontium in humans; but the data do indicate a possibility of skeletal effects (ATSDR 2004). Rachitic bone (abnormalities of bone structure and bone mineralization) is a characteristic of chronic strontium exposure (USEPA 1996). Numerous animal studies report adverse effects on skeletal development in juveniles following ingestion of excess stable strontium (ATSDR 2004). The U.S. EPA RfD ( $6.0 \times 10^{-1}$  mg/kg/day) is based on a young rat study with a NOAEL of 190 mg/kg/day strontium (USEPA 1996). U.S. EPA divided the NOAEL by an uncertainty factor of 300 to account for species-to-species extrapolation, incomplete database, and sensitive subpopulations (USEPA 1996). Also in the young rat study, a LOAEL of 380 mg/kg/day was derived based on inhibition of bone calcification (Storey1962).

### II.D.9.a. Comparison of Estimated Strontium Doses to Health Effect Levels

Data in Table B1 from monitoring well OMW-2D shows the maximum estimated strontium exposure doses for infants  $(9.4 \times 10^{-1} \text{ mg/kg/day})$ . This exposure dose assumes that 100 percent gastrointestinal absorption is 200 times less than the NOAEL (190 mg/kg/day) and 400 times less than the LOAEL (380 mg/kg/day) associated with inhibition of bone calcification. Further, a more realistic exposure scenario assumes 30 percent gastrointestinal absorption of strontium following an infant ingesting OMW-2D groundwater would result in an estimated strontium



exposure dose of  $2.8 \times 10^{-1}$  mg/kg/day. This dose is less than the U.S. EPA RfD, 670 times less than the NOAEL, and 1350 times less than the LOAEL. Therefore, a review of the estimated strontium exposure doses shows that infants who ingest off-site Melton Valley groundwater are not at risk of harmful effects from strontium. At the reported levels in off-site Melton Valley groundwater, ATSDR does not anticipate adverse health effects from strontium exposure.

### II.D.10. Thallium

In Table B1 in Appendix B, the estimated thallium exposure doses for an infant and adult drinking groundwater from four Melton Valley wells (OMW-1A, OMW-1C, OMW-1D, OMW-2C) exceeded the ATSDR noncancer screening guideline for thallium. As such, ATSDR further examined the health effect of ingesting thallium in the groundwater from four monitoring wells. The concentrations of thallium detected in groundwater from the other Melton Valley wells and all the Bethel Valley wells were not at health hazard levels (i.e., the thallium concentrations were below the ATSDR groundwater comparison value) and we won't further discuss them.

Pure thallium is a bluish-white metal found in trace amounts in the earth's crust. In its pure form, thallium is odorless and tasteless (ATSDR 1992b). Thallium is a byproduct from smelting other metals and has not been produced in the United States since 1984 (ATSDR 1992b). In the past, thallium was used as a depilatory agent and as a treatment for ringworm, venereal diseases, TB, and malaria (ATSDR 1992b). The United States banned its use as a pesticide in 1972 (ATSDR 1992b). Currently, thallium compounds are used in the semiconductor industry, in the manufacture of optic lenses and low-melting temperature glass, low-temperature thermometers, alloys, electronic devices, mercury lamps, fireworks, and imitation gems, and clinically as an imaging agent in the diagnosis of certain tumors (ATSDR 1992b).

Exposure to thallium occurs from the air, water, and food. Most exposures occur from eating food, such as thallium-contaminated homegrown fruits and green vegetables (ATSDR 1992b). On average, a person takes in about 2 micrograms of thallium per gram of food daily (ATSDR 1992b). Thallium enters food because plants easily take it up through the roots. Cigarette smoking is also a thallium source—people who smoke have twice as much thallium in their bodies as do nonsmokers (ATSDR 1992b). When someone swallows thallium, most of it is absorbed and rapidly goes to various parts of the body, especially the kidney and liver (ATSDR 1992b). Thallium leaves the body slowly. Most of the thallium leaves the body in urine and to a lesser extent in feces (ATSDR 1992b). About half the thallium leaves the body within 3 days (ATSDR 1992b).

A review of the scientific literature shows that thallium salts cause a wide spectrum of adverse effects in humans and animals (USEPA 2009). Alopecia is an effect characteristic of thallium exposure (USEPA 2009). Alopecia (loss of hair) generally occurs within 2 weeks of exposure and is reversible when thallium exposure ceases (USEPA 2009). But available human studies of thallium toxicity in humans do not provide useful information on establishing a dose-response associated with oral exposure to smaller amounts of thallium for longer periods (USEPA 2009).

The noncancer screening guideline for thallium in Table B1 is a provisional peer-reviewed toxicity value (PPRTV) derived by U.S. EPA's Superfund Health Risk Technical Support Center. The PPRTV is based on a NOAEL of  $4.0 \times 10^{-2}$  mg/kg-day from an animal study (USEPA 2009). In this study, 10 % of rats had hair follicle atrophy and alopecia at a LOAEL of  $2.0 \times 10^{-1}$  mg/kg/day; this result is consistent with thallium toxicity in both animals and humans

and U.S. EPA so characterized it as possibly treatment-related (USEPA 2009). U.S. EPA divided this dose by an uncertainty factor of 3,000 to account for humans (who are more sensitive than are rats to thallium), for some humans being more sensitive than others, and for a lack of reproductive and chronic toxicity data (USEPA 2009).

II.D.10.a. Comparison of Estimated Thallium Doses to Health Effect Levels

In Table B1, the estimated thallium exposure doses for an infant and an adult drinking Melton Valley groundwater from four monitoring wells are more than 100 times less than the NOAEL  $(4.0 \times 10^{-2} \text{ mg/kg-day})$ . These doses are more than 500 times less than the LOAEL  $(2.0 \times 10^{-1} \text{ mg/kg/day})$  associated with hair follicle atrophy and alopecia in 10 % of rats. So infants and adults drinking Melton Valley groundwater are not at risk of harmful effects from thallium. At the reported thallium levels in off-site Melton Valley groundwater ATSDR does not expect adverse health effects from exposure.

### II.D.11. Fluoride

In the Melton Valley Table B1,12 wells have estimated fluoride exposure doses for an infant that exceed the fluoride screening guideline. Six wells have estimated fluoride exposure doses for an adult that exceed the fluoride screening guideline. Elevated concentrations of fluoride were consistently detected in the groundwater samples from these Melton Valley wells (10 DOE monitoring wells and two former residential wells) over the monitoring period. The maximum concentration of 6,420 ppb fluoride was detected in Melton Valley groundwater from monitoring well OMW-2B, with a mean fluoride concentration of 6,100 ppb. None of these Melton Valley wells, however are a current drinking water sources. In Bethel Valley Table B3, three residential wells have estimated fluoride exposure doses for an infant, and one residential well has estimated fluoride exposure doses for an adult above the fluoride screening guideline. Only one of these Bethel Valley residential wells (RMW-97) is currently used for home purposes.

As such, ATSDR further examined the potential health effects from fluoride in the groundwater from these Melton Valley and Bethel Valley wells. The fluoride levels detected in the groundwater from the other Melton Valley and Bethel Valley wells are not at levels constituting a health hazard (i.e., the concentrations of fluoride in groundwater are below the comparison value) and we won't further discuss them.

Fluorine is a naturally occurring, widely distributed element in a reactive gas form; it combines with metals to make fluorides such as sodium fluoride and calcium fluoride, both white solids (ATSDR 2003). The concentration of fluorides in soils is usually between 200 and 300 ppm (ATSDR 2003). Levels might be higher in areas with fluoride-containing mineral deposits. Higher levels might also occur where phosphate fertilizers are used, where coal-fired power plants or fluoride-releasing industries are located, or in the vicinity of hazardous waste sites (ATSDR 2003). Sodium fluoride dissolves easily in water, but calcium fluoride does not. Levels of fluorides in surface water average about 200 ppb; levels in well water generally range from 20 to 1,500 ppb but often exceed 1,500 ppb in parts of the southwest United States (ATSDR 2003). Many communities fluoridate their water supplies. The recommended fluoride level is around 1,000 ppb. In the United States, approximately 15,000 water systems serving about 162 million persons are fluoridated in the optimal range of 700 to 1,200 ppb, either occurring naturally or through adjustment (ATSDR 2003).



Contaminated air, food, drinking water, soil, and dental products are some of the ways the general population might be exposed to fluorides (ATSDR 2003). People living in communities with fluoridated water or high levels of naturally occurring fluoride might be exposed to higher fluoride levels. The average daily fluoride intake by adults from food and water is estimated as 1 mg in a community with less than 700 ppb fluoride in the water, and about 2.7 mg if the water is fluoridated in the optimal range of 700 to 1,200 ppb fluoride (ATSDR 2003). Dental products used in the home such as toothpastes, rinses, and topically applied gels contain high fluoride concentrations (range 230–12,300 ppm) but are not intended for ingestion (ATSDR 2003). Toothpastes contain 900–1,100 ppm fluoride, most often as sodium fluoride levels. Most of the fluoride in food or water that's swallowed enters the bloodstream quickly through the digestive tract (ATSDR 2003). After entering the body, about half of the fluoride leaves quickly in urine—usually, unless large amounts are ingested within a 24-hour period (ATSDR 2003). Most of the fluoride ion that stays in the body is stored in bones and teeth.

The scientific literature indicates that a small amount of fluoride added to toothpaste and drinking water helps prevent tooth decay, but high fluoride levels can harm health (ATSDR 2003). At fluoride levels five times higher than levels typically found in fluoridated water, fluoride can result in denser bones (ATSDR 2003). If exposure is high enough, however, these same bones can become more fragile and brittle in adults, and the risk of breakage might increase (ATSDR 2003). Skeletal fluorosis can be caused by eating, drinking, or breathing very large amounts of fluorides over many years. This disease only occurs after long-term exposures and can cause denser bones, joint pain, and a limited range of joint movement (ATSDR 2003). Skeletal fluorosis is extremely rare in the United States; it's occurred in some people consuming greater than 30 times the amount of fluoride typically found in fluoridated water (ATSDR 2003).

Excessive fluoride exposure during the time teeth are forming can cause visible changes in teeth—a condition known as dental fluorosis. This condition only develops while the teeth are forming in the jaw and before they erupt into the mouth (i.e., usually younger than 8 years of age) (ATSDR 2003). After the teeth have developed and erupted, they cannot become fluorosed. Most enamel fluorosis seen today is of the mildest form, characterized by a few, almost-invisible white spots on the teeth (ATSDR 2003). In moderate cases large white spots appear on the teeth (mottled teeth), and some brown spots (ATSDR 2003). In severe cases, the teeth are pitted, are fragile, and can sometimes break. The appearance of affected teeth is not identical for all children exposed to the same level of fluoride in the drinking water.

Most of the epidemiologic studies of people living in areas with fluoridated water or naturally high levels of fluoride in drinking water have not found an association between fluoride and cancer risk (ATSDR 2003). Two animal cancer studies were inconclusive (ATSDR 2003). The National Research Council (NRC) Subcommittee on Health Effects of Ingesting Fluoride reviewed more than 50 epidemiological human studies and animal studies that evaluated the relationship between fluoride concentrations in drinking water and cancer (NRC 2006). The NRC Subcommittee concluded that the weight of evidence does not support an association between fluoride exposure and increased cancer risk in humans (NRC 2006). The International Agency for Research on Cancer (IARC) has determined that the carcinogenicity of fluoride to humans is not classifiable (ATSDR 2003).

The scientific literature includes a number of studies on the possible association between exposure to fluoridated water and the risk of increased bone fractures, particularly hip fractures (ATSDR 2003). A meta-analysis of these data, as well as other clinical studies, found a significant correlation between exposure to high levels of fluoride and an increased relative risk of nonvertebral fractures (ATSDR 2003). ATSDR selected the Li et al. (2001) study as the basis for the chronic-duration oral MRL of 0.05 mg/kg/day for fluoride (ATSDR 2003). Li and colleagues examined communities with higher levels of naturally occurring fluoride in the water. They found increases in the incidence of hip fractures in residents exposed to 4 ppm fluoride and higher compared with the incidence rates in communities with approximately 1 ppm fluoride in the water (Li et al. 2001). Li and colleagues identified a NOAEL of  $1.5 \times 10^{-1}$  mg/kg/dav and LOAEL of  $2.5 \times 10^{-1}$  mg/kg/day (Li et al. 2001). Note the narrow gap of only 0.1 mg/kg/day between the dose with no adverse effect (NOAEL) and the dose with an effect level (LOAEL). ATSDR calculated the MRL by dividing the NOAEL of  $1.5 \times 10^{-1}$  mg/kg/day by an uncertainty factor of 3 to account for human variability (ATSDR 2003). This MRL is only five times less than the adverse effect level (LOAEL), again because of the narrow gap between the NOAEL and LOAEL. Also, ATSDR used a partial uncertainty factor, given that the most sensitive subpopulation-elderly men and women-was examined to derive the NOAEL and LOAEL.

### II.D.11.a. Comparison of Estimated Fluoride Doses to Health Effect Levels

In Melton Valley, we used an RME scenario (i.e., higher than average water intake rates, 95<sup>th</sup> percentile) and the mean fluoride concentrations in groundwater to estimate chronic infant exposure doses. We found seven Melton Valley wells exceeded the NOAEL and six Melton Valley wells exceeded the LOAEL (See Table 9). Groundwater in monitoring wells OMW-1B and OMW-2B contained estimated infant fluoride exposure doses of  $8.4 \times 10^{-1}$  mg/kg/day and  $8.7 \times 10^{-1}$  mg/kg/day—more than 5 times greater than the NOAEL and 3.4 times greater than the LOAEL. The infant exposure doses for the other four Melton Valley wells ranged from 1 and 3.2 times higher than the LOAEL. In six Melton Valley monitoring wells, the estimated adult and child (i.e., 6 to 11 years of age) exposure doses were within a factor of 5 of the LOAEL. For two of these wells (OMW-1B, OMW-2B) the estimated child exposure doses of  $2.3 \times 10^{-1}$  mg/kg/day and  $2.4 \times 10^{-1}$  mg/kg/day were basically equivalent to the LOAEL.

Using the CTE scenario (average or typical water intake rates) and the mean fluoride concentrations in Melton Valley, the chronic infant exposure doses were higher than the LOAEL for fluoride in two wells (OMW-1B, OMW-2B) and within a factor of 2 of the LOAEL in four other wells (See Table 9). Also, all the estimated adult and child exposure doses in Table 9 for the CTE scenario were within one order of magnitude of the LOAEL for fluoride. The estimated adult and child exposure doses for OMW-1B, and OMW-2B were within a factor of 3 of the LOAEL. For OMW-1C and OMW-2C the doses were within a factor of 5 of the LOAEL. Thus again using the CTE scenario, estimated infant chronic exposure doses for fluoride were higher than the LOAEL. The adult and child exposure doses were within a factor 2 of the LOAEL. That means chronic (more than 1 year) ingestion of fluoride at the reported levels in the off-site Melton Valley groundwater by an infant, child, or adult could result in exposure doses associated with denser bones—bones often more brittle or fragile that would increase the risk of breakage. None of the DOE monitoring wells in Melton Valley were used for home purposes. That means as of the time of this health consultation no one was known to be drinking excessively



fluoridated Melton Valley groundwater. ATSDRconsiders the reported levels of fluoride in the Melton Valley groundwater a *potential* rather than an *actual* public health hazard.

Most residents in the Melton Valley area chose to stop using their private wells and had their residences connected to the Watts Bar Utility District water supply. But the source of the fluoride and the vertical and lateral extent of the fluoride contaminant plume remain unknown, and at the time of this health consultation a few Melton Valley residents with private wells continued to use groundwater for home purposes. Moreover, ATSDR does not have any results of chemical or radiological analysis of groundwater from some of these private wells used for home purposes and we are unable to determine the health implications of using the groundwater from these private wells.

Melton Valley Well Station		Mean	Exposure Dose (mg/kg/day)		
		Concentration (ppb)	Infant	Child	Adult
OMW-1B	RME scenario	5892	8.4E-01	2.3E-01	2.1E-01
	CTE scenario	JU72	3.8E-01	8.9E-02	8.7E-02
OMW-1C	RME scenario	3345	4.8E-01	1.3E-01	1.2E-01
	CTE scenario		2.2E-01	5.0E-02	4.9E-02
OMW-1D	RME scenario	1796	2.6E-01	7.0E-02	6.4E-02
	CTE scenario		1.2E-01	2.7E-02	2.6E-02
OMW-2B	RME scenario	6100	8.7E-01	2.4E-01	2.2E-01
	CTE scenario		3.9E-01	9.2E-02	9.0E-02
OMW-2C	RME scenario	3190	4.5E-01	1.2E-01	1.1E-01
	CTE scenario		2.5E-01	5.9E-02	5.8E-02
OMW-3C	RME scenario	2660	3.8E-01	1.0E-01	9.4E-02
	CTE scenario		1.7E-01	4.0E-02	3.9E-02
OMW-4C	RME scenario	1483	2.1E-01	5.0E-02	5.3E-02
	CTE scenario		9.5E-01	2.2E-02	2.1E-02

Table 9. Estimated Ex	posure Doses for	Fluoride In Meltor	n Valley	v Groundwater
	pood. c = 00000 . 0.			

ppb – part per billion = micrograms per liter ( $\mu$ g/L)

mg/kg/day - milligram per kilogram per day

RME scenario – higher than average water intake rates, high end of the exposure distribution (approximately 95<sup>th</sup> percentile)

CTE scenario – average or typical water intake rates

OMW – Off-site Monitoring Well

In Table B3 for Bethel Valley, the RME scenario shows that the estimated, chronic fluoride exposure doses for an infant were greater than the NOAEL in two wells (RWA-97, RWA-104) and, for RWA-104, 1.4 times greater than the LOAEL. Two other wells (RWA-102, RWA-97) were 3.2 and 1.3 times less than the LOAEL, respectively. But researchers collected only one groundwater sample from most of the former Bethel Valley residential wells, including RWA-104 and RWA-102. Researchers collected 10 groundwater samples from residential well RWA-97, which is currently used for home purposes, including drinking water. The fluoride concentrations were consistently detected in RWA-97 groundwater ranging from 1,300 ppb to 1,450 ppb, with a mean concentration of 1,350 ppb.

Under a CTE scenario with average or typical intake of Bethel Valley groundwater from RWA-97, which at the time of this health consultation was used for home purposes, the estimated fluoride exposure dose  $(8.7 \times 10^{-2} \text{ mg/kg/day})$  was 1.7 times below the NOAEL for an infant. Again using the CTE scenario for RWA-97, the estimated fluoride exposure doses for an adult  $(1.9 \times 10^{-2} \text{ mg/kg/day})$  and child  $(2.0 \times 10^{-2} \text{ mg/kg/day})$  were also less than the NOAEL. Thus with the CTE-scenario estimated exposure doses for chronic fluoride under the NOAEL, the risk of skeletal fluorosis (i.e., often more brittle or fragile, denser bones with increased breakage risk) is low. Ingestion of groundwater from residential well RWA-97 is not expected to result in health effects. But this residential well continues as a water supply for home use, including drinking water. Without continued monitoring of RWA-97, ATSDR cannot ensure that this conclusion will remain valid, especially if fluoride concentrations or other constituents increase over time.

Under the CTE scenario, the estimated fluoride exposure dose for RWA-104 is  $1.5 \times 10^{-1}$  mg/kg/day for an infant. That's equivalent to the NOAEL and 1.6 times below the LOAEL. The adult and child doses ( $3.5 \times 10^{-2}$  mg/kg/day) are below the NOAEL and 7 times less than the LOAEL. Thus, with estimated chronic fluoride exposure doses for an infant

- More than the NOAEL for the RME and CTE scenarios,
- More than the LOAEL under the RME scenario, and
- Only 1.6 times less than the LOAEL based on the CTE scenario,

from chronic ingestion of Bethel Valley groundwater from RWA-104 by an infant could result in exposure doses associated with skeletal fluorosis (i.e., denser bones that are often more brittle or fragile, with increased risk of bone fractures in elderly men and women). But again, former residential well RWA-104 is not currently used for home purposes and no one drinks the groundwater from this well. Therefore, ATSDR considers chronic ingestion of fluoride levels reported in RWA-104 groundwater a *potential* public health hazard.

As stated, the source and the vertical and lateral extent of the fluoride contaminant plumes are unknown, and a few Bethel Valley residents with private wells continue to use the groundwater for domestic purposes. ATSDR does not have any results of chemical or radiological analysis of groundwater from some of these private wells used for domestic purposes and we are unable to determine the health implications of using the groundwater from these private wells.

## II.D.12. Volatile Organic Compounds (VOCs) in Bethel Valley Groundwater

We have only a limited analysis of volatile organic compounds (VOCs) in Bethel Valley groundwater. Bethel Valley well RWA-104 is a 610-foot deep well initially drilled as a residential well but was ultimately found unusable because of the water quality. (TDEC 2011). Three groundwater samples from RWA-104 were analyzed for the full spectrum of VOCs. Only a few groundwater samples from approximately 10 other Bethel Valley wells were analyzed, and then only for a limited number of VOCs.

As shown in Table B3 in Appendix B, researchers found three VOCs (benzene, bromodichloromethane, and chloroform) in the RWA-104 groundwater samples with mean concentration levels resulting in estimated infant and adult exposure doses above the noncancer screening guideline. In Table B4, only one VOC (chloroform) was detected at a mean concentration resulting in estimated excess cancer risk that exceeded the cancer screening guideline. ATSDR further examined the effect levels reported in the scientific literature and



more fully evaluated potential combined exposure to these three RWA-104 VOCs from ingestion of, inhalation of, and dermal contact with groundwater.

In Table B3 and in Table B4, the estimated exposure doses and excess cancer risk for an infant and adult ingesting the other VOCs detected in groundwater from well RWA-104 were below the noncancer and cancer screening guidelines and do not constitute a health hazard. We won't further discuss these VOCs and ATSDR does not have enough VOC data from the other Bethel Valley wells to make a public health evaluation.

ATSDR evaluates home use of groundwater with volatile organic compounds (VOCs) using a cumulative exposure dose from ingestion, inhalation, and dermal contact. In addition to the oral exposure dose from drinking water, for volatile organic compounds a person might also absorb these compounds directly from contaminated water through the skin contact (dermal dose) and from breathing the gaseous compound that escapes into the air (inhalation dose). Studies have shown that exposure to volatile organic compounds from exposure routes other than direct ingestion might be as large as the exposure from ingestion alone. The inhalation exposure dose due to volatization during a shower might equal the ingestion exposure dose (Wan 1990). Fifty to 90% of volatile organic compounds in water might volatize during showering, laundering, and other activities (Moya 1999; Giardino 1996). Similarly, the dermal exposure dose has been estimated to equal 30% of the ingested dose (Maine DEP 1992). Given the results of these studies, combined VOC exposure doses include an inhalation exposure dose that's equal to 70% of the ingestion dose, and a dermal contact exposure dose that's 30% of the ingestion dose. These secondary exposures to the VOCs in drinking water essentially represent a doubling of the ingestion dose. Note that the cumulative exposure dose from ingestion, inhalation, and dermal contact with groundwater from the offsite residential well RWA-104 were estimated assuming that exposure occurred in a residential setting; that is, that exposure was continuous (24 hours per day, 365 days per year).

### II.D.13. Benzene

Benzene is a colorless, flammable liquid that evaporates quickly into air and dissolves slightly in water. People can begin to smell the sweet odor of benzene in air at approximately 60 ppm (ATSDR 2007d). Most people can begin to taste benzene in water at 0.5–4.5 ppm (ATSDR 2007d).

Benzene has both industrial and natural sources and appears in air, water, and soil. Made mostly from petroleum, benzene is one of the top 20 chemicals produced in the United States (ATSDR 2007d). Bbenzene is used to make other chemicals, such as styrene (for Styrofoam and other plastics), cumene (for various resins), and cyclohexane (for nylon and synthetic fibers) (ATSDR 2007d). The manufacture of some types of rubbers, lubricants, dyes, detergents, drugs, and pesticides also involves benzene. Gas emissions from volcanoes and forest fires are natural sources that also contribute to benzene in the environment (ATSDR 2007d). Benzene is present in crude oil and gasoline and cigarette smoke.

Everyone encounters a small amount of benzene every day, whether in the outdoor environment, in the workplace, or in the home. Exposure is mainly through breathing air that contains benzene. Major exposure sources are tobacco smoke, automobile service stations, exhaust from motor vehicles, and industrial emissions (ATSDR 2007d). Vapors (or gases) from products such as glues, paints, furniture wax, and detergents are also benzene exposure sources (ATSDR 2007d).

The level of benzene exposure through food, beverages, or drinking water is not as high as through air. Drinking water typically contains less than 0.1 ppb benzene (ATSDR 2007d). Some bottled water, liquor, and food might also contain some benzene. Leakage from underground gasoline storage tanks or from landfills and hazardous waste sites that contain benzene can result in benzene contamination of well water (ATSDR 2007d). People with benzene-contaminated tap water can be exposed from drinking the water or from eating foods prepared with the water. Exposure can also result from breathing in benzene while showering, bathing, or cooking with benzene-contaminated water (ATSDR 2007d).

When a person is exposed to benzene in food or drink, most of the benzene passes through the lining of the gastrointestinal tract and enters the bloodstream (ATSDR 2007d). Once in the bloodstream, benzene travels throughout the body and can temporarily reside in the bone marrow and fat (ATSDR 2007d). In the liver and bone marrow, benzene converts to metabolites. These metabolites cause some of the harmful effects of benzene exposure (ATSDR 2007d). But most benzene metabolites leave the body in the urine within 48 hours after exposure (ATSDR 2007d).

The scientific literature indicates the prevalent noncancer systemic effect resulting from relatively low levels of benzene exposure is a depressed number of blood cell types (hematotoxicity) that manifest as anemia (decrease red blood cells or low concentration of hemoglobin), leukopenia (decrease white blood cells (leukocytes)), or thrombocytopenia (decrease platelets) in humans and in animals (ATSDR 2007d). ATSDR developed a benchmark dose limit (BDL) of  $1.4 \times 10^{-2}$  mg/kg/day from a chronic benzene inhalation study of 250 workers (approximately two-thirds female) exposed to benzene at two shoe manufacturing facilities in Tianjin, China that demonstrated the lowest LOAEL for a hematological endpoint (B cell count) (ATSDR 2007d). ATSDR derived the chronic oral MRL of  $5.0 \times 10^{-4}$  mg/kg/day by dividing the BDL by an uncertainty factor of 30 for route-to-route extrapolation (i.e., inhalation to oral) (ATSDR 2007d).

### II.D.13.a. Comparison of Estimated Benzene Doses to Health Effect Levels

Researchers detected benzene in all three groundwater samples from Bethel Valley well RWA-104. The mean level was 18.9 ppb, and the range was 0.31 to 56.1 ppb. Using the RME scenario, the estimated cumulative chronic exposure doses (ingestion plus inhalation plus derma contact) for an infant  $(5.4 \times 10^{-3} \text{ mg/kg/day})$  and adult  $(1.3 \times 10^{-3} \text{ mg/kg/day})$  were 2.5 and 10 times less than the BDL of  $1.4 \times 10^{-2} \text{ mg/kg/day}$ , respectively. Using the mean benzene level and a CTE scenario, the estimated cumulative infant exposure dose of  $2.4 \times 10^{-3} \text{ mg/kg/day}$  was 6 times less than the BDL; the estimated cumulative adult exposure dose of  $5.4 \times 10^{-4} \text{ mg/kg/day}$  was 26 times less than the BDL. Thus with the estimated exposure doses within an order of magnitude of the BDL, chronic infant and adult ingestion of benzene in the groundwater from Melton Well RWA-104 could result in hematotoxicity. But residential well RWA-104, despite containing benzene at levels of health concern, has never been used for home purposes and no one has ever been exposed to the benzene in the groundwater. The benzene levels in the groundwater from RWA-104 is a *potential* public health hazard.

In studies of exposed workers, benzene's carcinogenicity is well documented (ATSDR 2007d). Epidemiological studies and case reports provide clear evidence of a causal relationship between 1) occupational exposure to benzene and benzene-containing solvents, and 2) the occurrence of acute myelogenous leukemia (AML)(ATSDR 2007d). Although the epidemiological studies are



generally limited, a consistent excess leukemia risk across studies indicates that benzene is the causal factor (ATSDR 2007d). Table B4 shows the excess cancer risk for an infant  $(1.5 \times 10^{-6})$  and adult  $(3.7 \times 10^{-6})$  based on the RME scenario and on the mean concentration level of benzene detected in the groundwater from RWA-104. Both risks are well below the cancer screening guideline of 1 in 10,000  $(1 \times 10^{-4})$  and are not at levels constituting an excess cancer risk. Ingestion of benzene in the groundwater is not expected to result in an excess cancer risk.

The source of the benzene and the vertical and lateral extent of the benzene contaminant plume are unknown, and, at the time of this health consultation, a few Bethel Valley residents with private wells continued to use the groundwater for home purposes. Moreover, ATSDR does not have any results of chemical or radiological analysis of groundwater from some of these private wells currently used for home purposes.

### II.D.14. Bromodichloromethane

Bromodichloromethane (BDCM) is a colorless, heavy, nonburnable liquid usually found evaporated in air or dissolved in water (ATSDR 1989). Most BDCM in the environment is a byproduct when chlorine is added to drinking water to kill disease-causing organisms (ATSDR 1989). Chemical manufacturers also make small amounts of BDCM for use in laboratories or in other chemicals.

The most likely means of exposure to BDCM for most people is by drinking chlorinated water (ATSDR 1989). Usually the levels in drinking water are between 1 and 10 ppb (ATSDR 1989). BDCM is also found in some food and beverages such as ice cream or soft drinks made with chlorinated water, but this is probably not a major exposure source (ATSDR 1989). BDCM has been found in chlorinated swimming pools, where exposure might occur by breathing the vapors or by skin absorption (ATSDR 1989).

Studies in animals show that almost all BDCM swallowed in water or food will enter the body by moving from the stomach or intestines into the blood (ATSDR 1989). BDCM removal is fairly rapid (about 95% in 8 hours) by breathing it out through the lungs and, in smaller amounts, eliminating it in urine and feces (ATSDR 1989). BDCM does not usually build up in the body (ATSDR 1989).

The scientific literature indicates in animals, the main effect of eating or drinking large amounts of BDCM is injury to the liver and kidneys (ATSDR 1989). Long-term studies in animals reveal that the kidney is also susceptible to injury by BDCM at dose levels similar to those that affect the liver (ATSDR 1989). In rats, cytomegaly (i.e., abnormal enlargement of a cell or group of cells) in the kidney was noted following chronic exposure to 25 mg/kg/day BDCM (ATSDR 1989). This LOAEL of 25 mg/kg/day has been selected as the most appropriate value for calculation of the chronic MRL for BDCM (ATSDR 1989). The ATSDR chronic oral MRL of  $2.0 \times 10^{-2}$  mg/kg/day was derived from the LOAEL by dividing by an uncertainty factor of 1000 (10 for using a LOAEL, 10 for extrapolating from animal to human, and 10 for human variability) (ATSDR 1989).

II.D.14.a. Comparison of Estimated Bromodichloromethane Doses to Health Effect Levels

As shown in Appendix B - Table B3, BDCM was detected at a concentration of 193.31 ppb in only one groundwater sample collected from Bethel Valley RWA-104. The estimated cumulative

chronic BDCM exposure dose (ingestion plus inhalation plus derma contact) for an infant ( $5.4 \times 10^{-2}$  mg/kg/day) with a RME scenario is 462 times less than the 25-mg/kg/day LOAEL. Using the mean BDCM level and a CTE exposure scenario, the estimated cumulative infant exposure dose of  $2.4 \times 10^{-2}$  mg/kg/day is 1042 times less than the LOAEL for BDCM. Therefore, with the estimated exposure doses more than two orders of magnitude below than the LOAEL, chronic ingestion of BDCM in the groundwater from Bethel Valley well RWA-104 is not expected to cause harmful health effects.

### II.D.15. Chloroform

Chloroform is a colorless liquid with a pleasant, nonirritating odor and a slightly sweet taste (ATSDR 1997). Chloroform is also known as trichloromethane. Most of the chloroform found in the environment comes from industry (ATSDR 1997). Nearly all the chloroform made in the United States today is used to make other chemicals.

Chloroform enters the environment from chemical manufacturers and paper mills (ATSDR 1997). It's also found in wastewater from sewage treatment plants and drinking water to which chlorine has been added (ATSDR 1997). Chlorine is added to most drinking water and many wastewaters to destroy bacteria. Small amounts of chloroform form as an unwanted product while adding chlorine to water (ATSDR 1997). Chloroform enters the environment in many ways, so small amounts are likely to appear almost anywhere (ATSDR 1997).

Chloroform also dissolves easily in water, but does not do a good job sticking to soil (ATSDR 1997). This means it can travel down through soil to groundwater, where it can enter a water supply (ATSDR 1997). Chloroform lasts for a long time in both the air and in groundwater (ATSDR 1997). Chloroform does not appear to build up in great amounts in plants and animals, but some foods might contain chloroform in small amounts (ATSDR 1997).

People are exposed to small amounts of chloroform in drinking water and in beverages such as soft drinks made using chloroformed water (ATSDR 1997). People are most likely to be exposed to chloroform by drinking water and breathing chloroformed indoor or outdoor air. The amount of chloroform normally expected to be in the air ranges from 0.02 to 0.05 parts of chloroform per billion parts (ppb) of air and from 2 to 44 ppb in treated drinking water (ATSDR 1997). The concentration of chloroform in surface water and untreated groundwater is estimated as 0.1 ppb (ATSDR 1997).

Blood carries chloroform to all parts of the body, such as the fat, liver, and kidneys (ATSDR 1997). Although chloroform usually collects in body fat, it will eventually leave the body once exposure has ceased (ATSDR 1997). Some of the chloroform that enters the body is broken down into other chemicals (ATSDR 1997). These metabolites can attach to other chemicals inside the cells of the body and, if they collect in high enough amounts, might cause harmful effects. Some of the metabolites also leave the body in the air breathed out, and a small amount of the metabolites leave the body in the urine and feces (ATSDR 1997).

The scientific literature indicates the liver is a primary target of chloroform toxicity in humans, but some evidence that suggests the damage might be reversible (ATSDR 1997). In chronic-duration exposure studies, liver effects have been observed in rats, mice, and dogs after oral exposure to chloroform (ATSDR 1997). The lowest oral dose administered to animals in chronic studies was 15 mg/kg/day, which increased serum glutamic pyruvic transaminase (SGPT) in dogs after 2 years of exposure (Heywood et al. 1979). Serum glutamic pyruvic transaminase is



an enzyme normally present in the liver and released into blood when the liver is damaged (ATSDR 1997). We used the 15-mg/kg/day LOAEL to derive a chronic oral MRL of  $1.0 \times 10^{-2}$  mg/kg/day by dividing the LOAEL by an uncertainty factor of 1000 (10 for using a LOAEL, 10 for extrapolating from animal to human, and 10 for human variability)(ATSDR 1997). The acute oral MRL  $3.0 \times 10^{-1}$  mg/kg/day was based on a NOAEL of 26 mg/kg/day in the drinking water for 4 days for hepatic effects in mice (ATSDR 1997). To derive the acute oral MRL, we divided the acute NOAEL of 26.4 mg/kg/day by an uncertainty factor of 100 (10 for extrapolation from animals to humans and 10 for human variability) to arrive at the 0.3-mg/kg/day MRL (ATSDR 1997).

II.D.15.a. Comparison of Estimated Chloroform Doses to Health Effect Levels

Researchers detected chloroform in three groundwater samples collected from residential well RWA-104. The three chloroform concentrations were 0.45 ppb, 3.52 ppb, and 6,000 ppb, with a mean of 2001.3 ppb. As shown in Table 10, the RME-scenario estimated cumulative chronic chloroform exposure doses (ingestion plus inhalation plus dermal contact) for an infant and an adult is 27 times and 107 times less, respectively, than the 15-mg/kg/day chronic LOAEL. Using the mean chloroform level and a CTE exposure scenario, the estimated cumulative infant exposure dose is 57 times less than the LOAEL, and the estimated adult exposure dose is 258 times less than the LOAEL. Given the estimated infant exposure doses for the RME and CTE scenarios are within two orders of magnitude of the LOAEL, chronic ingestion of groundwater from Bethel Valley well RWA-104 by an infant might cause harmful health effects. But due to the large variation in chloroform concentration over an extended period, an infant is unlikely to ingest a higher than average amount of groundwater containing the mean 2001.3 ppb chloroform concentration. Thus chronic exposure to chloroform in the groundwater from RWA-104 is not expected to cause harmful health effects.

Duration of	Chloroform		Cumulative Exposure Dose (mg/kg/day)		
Exposure	Concentrati	on (ppb)	Infant	Adult	
Chronic	2001.3	RME scenario	5.6E-01	1.4E-01	
(> 1 year)	(mean)	CTE scenario	2.6E-01	5.8E-02	
Acute	6000	RME scenario	1.8	4.2E-01	
(< 14 days)	(max.)	CTE scenario	7.8E-01	1.8	

Table 10. Estimated Cumulative Exposure Doses for Chloroform in Bethel Valley Well RWA-104Groundwater

ppb – part per billion = micrograms per liter ( $\mu$ g/L)

mg/kg/day - milligram per kilogram per day

Reasonable Maximum Exposure (RME) scenario – higher than average water intake rates, high end of the exposure distribution (approximately 95<sup>th</sup> percentile)

Central Tendency Exposure (CTE) scenario - average or typical water intake rates

Cumulative Exposure Dose includes exposure from ingestion of chloroform in groundwater, direct skin contact with chloroform in groundwater, and inhalation of gaseous chloroform that escaped the groundwater into the household air

Given the large variation in the three chloroform concentrations in the groundwater over time, ATSDR evaluated an acute (less than 14 days) exposure to 6,000 ppb chloroform using the RME and CTE scenarios. The estimated cumulative chloroform exposure doses in Table 10 indicate

that all the estimated acute exposure doses for an infant and an adult were more than one order of magnitude below the 26.4-mg/kg/day NOAEL. With the estimated exposure doses more than one order of magnitude below the NOAEL, we do not expect acute ingestion of chloroform from Bethel Valley RWA-104 groundwater to cause harmful health effects.

U.S. EPA has determined that chloroform is a probable human carcinogen (ATSDR 1997, USEPA 2001). Several chronic, oral exposure animal studies found chloroform was carcinogenic (ATSDR 1997). Cancer of the liver and kidneys developed in rats and mice that over long periods ate food or drank water containing large amounts of chloroform (ATSDR 1997). Results of epidemiologic studies of people who drank chlorinated water showed a possible link between the chloroform in chlorinated water and the occurrence of cancer of the colon and urinary bladder (ATSDR 1997). Under the USEPA Proposed Guidelines for Carcinogen Risk Assessment, chloroform is likely to be carcinogenic to humans by all routes of exposure under high-exposure conditions that lead to cytotoxicity and regenerative hyperplasia in susceptible tissues (USEPA 2001). Chloroform is not likely to be carcinogenic to humans by any route of exposure under exposure conditions that do not cause cytotoxicity and cell regeneration (USEPA 2001). Therefore, if chloroform exposure is not likely to cause non-cancer health effects, it is unlikely to cause cancer.

In Table B4 in Appendix B, the estimated excess cancer risks for a child and adult ingesting chloroform in groundwater from Bethel Valley well RWA-104 are slightly higher than cancer screening guidelines of 1 in 10,000 ( $1 \times 10^{-4}$ ). Using the CTE scenario with 1) the average or typical water intake, 2) the mean chloroform concentration, and 3) the estimated cumulative exposure doses, the estimated excess cancer risks for a child and adult ingesting (plus inhalation and dermal contact) chloroform in groundwater are 4.4E-05 and 2.6E-05, respectively. With these estimated cancer risks for average water intake less than the cancer screening guidelines, chloroform in the Bethel Valley groundwater is not at excess cancer-risk levels.

### II.D.16. Volatile Organic Compounds (VOCs) in Melton Valley Groundwater

In Table B1, Appendix B, two VOCs (cis 1,2-Dichloroethene and Trichloroethylene) were detected in only one out of five groundwater samples collected from one Melton Valley well (OMW-1B) at concentration levels that resulted in estimated infant and adult exposure doses above the noncancer screening guideline. Unlike Bethel Valley, in Melton Valley the full spectrum of VOCs were analyzed in groundwater samples collected from each Melton Valley DOE monitoring well and from former residential wells used as monitoring wells. Between 3 and 7 groundwater samples were collected from each well. Cis 1,2-Dichloroethene was detected in 2 out of 45 (4 percent) groundwater samples collected from Melton Valley. Trichloroethylene was detected in 3 out of 100 (3 percent) groundwater samples collected in one groundwater sample from OMW-1B, the concentrations of these two VOCs detected in the other groundwater samples were less than 1 ppb. As such, ATSDR further examined the health effects from ingesting these two VOCs in groundwater from OMW-1B. DOE's Melton Valley monitoring well OMW-1B was screened at depth of 280 to 360 feet below the ground surface (TDEC 2011).

The estimated exposure doses for the other three VOCs (Benzene, Bromodichloromethane, Vinyl Chloride) detected in groundwater from other Melton Valley wells were below the noncancer screening guideline and did not constitute a health hazard (Appendix B -Table B1). The



estimated excess cancer risks for an infant and an adult ingesting trichloroethylene and other VOCs in Melton Valley groundwater from all the wells were less than the cancer screening guidelines (Appendix B- Table B2). The levels of trichloroethylene and other VOCs detected in the Melton Valley groundwater were not at levels constituting an excess cancer risk. We won't further discuss any cancer risk from these VOCs.

#### II.D.17. Cis 1,2-Dichloroethene

1,2-Dichloroethene is a highly flammable, colorless liquid with a sharp, harsh odor (ATSDR 1996). 1,2-dichloroethene is usually in two forms: one is called cis-1,2-dichloroethene, and the other is called trans-1,2-dichloroethene (ATSDR 1996). Sometimes both forms are present as a mixture. 1,2-Dichloroethene is most often used to produce solvents and in chemical mixtures (ATSDR 1996). 1,2-Dichloroethene enters the environment through industrial activity. This chemical has been found in air, water, and soil (ATSDR 1996). 1,2-Dichloroethene is released to the environment from chemical factories that make or use it, from landfills and hazardous waste sites containing this chemical, from chemical spills, from burning of objects made of vinyl, and from breakdown of other chlorinated chemicals (ATSDR 1996).

1,2-Dichloroethene below soil surfaces in landfills or hazardous waste sites may dissolve in water, seep deeper into the soil, and possibly contaminate groundwater (ATSDR 1996). Some 1,2-dichloroethene may escape as a vapor (ATSDR 1996). Once in groundwater, 1,2-dichloroethene needs about 13-48 weeks for half a given amount to break down (half-life in water) (ATSDR 1996). Small amounts of 1,2-dichloroethene found in landfills over time might break down into vinyl chloride, which is believed to be a more hazardous chemical, but the possibility is minimal (ATSDR 1996).

People can be exposed to 1,2-dichloroethene by breathing contaminated air or by drinking contaminated tap water. If home tap water is contaminated, people could also be breathing 1,2-dichloroethene vapors while cooking, bathing, or washing dishes (ATSDR 1996). People who live in cities or suburbs are more likely to be exposed than are people living in rural areas (ATSDR 1996).

1,2-Dichloroethene can enter the body through lungs when breathing contaminated air and through the stomach and intestines when eating or drinking contaminated food or water (ATSDR 1996). When 1,2-dichloroethene enters the body, the blood and other tissues absorb it. The liver breaks it down into other compounds (ATSDR 1996).

The scientific literature consensus is that increased relative kidney weight in male and female rats is the critical effect from oral exposure to cis 1,2-Dichloroethene (McCauley et al. 1995, 1990). The cis 1,2-Dichloroethene RfD of  $2 \times 10^{-3}$  mg/kg/day is based on a benchmark dose limit (BDL<sub>10</sub>) of 5.1 mg/kg/day, which in turn is based on a 10 percent change in relative kidney weight of male rats compared with the control (USEPA 2010). The RfD is derived by dividing the BDL<sub>10</sub> of 5.1 mg/kg/day by an uncertainty factor of 3000 (factors of 10 for potentially sensitive human subpopulations, 10 for the variability in extrapolating from laboratory animals to humans, 10 for extrapolating from a subchronic exposure duration to estimate chronic exposure conditions, and 3 to account for database deficiencies)(USEPA 2010).

II.D.17.a. Comparison of Estimated cis 1,2-Dichloroethene Doses to Health Effect Levels

As shown in Table B1 in Appendix B, cis 1,2-Dichloroethene was detected at a concentration of 80.8 ppb in only one groundwater sample collected from well OMW-1B. Using this cis 1,2-Dichloroethene concentration and a CTE exposure scenario, the estimated cumulative infant exposure dose (i.e., ingestion plus inhalation plus dermal contact) of  $1.0 \times 10^{-2}$  mg/kg/day is 510 times less than the BDL<sub>10</sub>. The estimated cumulative adult exposure dose of  $2.4 \times 10^{-3}$  mg/kg/day is 2,125 times less than the BDL<sub>10</sub>. Thus only one groundwater sample in Melton Valley (4 percent of all groundwater samples) contained elevated levels of cis 1,2-Dichloroethene. With the estimated cumulative exposure doses more than two orders of magnitude below the LOAEL, chronic ingestion of cis 1,2-Dichloroethene in the groundwater from Melton Valley is not expected to cause harmful health effects.

## II.D.18. Trichloroethylene (TCE)

Trichloroethylene, also known as TCE, is a nonflammable, colorless liquid with a somewhat sweet odor and a sweet, burning taste (ATSDR 2007e). TCE is used mainly as a solvent to remove grease from metal parts (ATSDR 2007e). TCE can also be found in some household products, including typewriter correction fluid, paint removers, adhesives, and spot removers (ATSDR 2007e).

The largest source of TCE in the environment is evaporation from factories that use it to remove grease from metals (ATSDR 2007e). It can also enter the air and water when disposed of at chemical waste sites. It evaporates easily, but can stay in the soil and in groundwater. Once TCE is in water, much will evaporate into the air. In groundwater the breakdown is much slower because of the much slower evaporation rate (ATSDR 2007e). Very little TCE breaks down in the soil; it can pass through the soil into underground water (ATSDR 2007e).

TCE is in the outdoor air at levels far less than 1 ppm (ATSDR 2007e). Some of the water supplies in the United States have TCE (ATSDR 2007e). Monitoring studies found average levels in surface water ranging from 0.1 to 1 ppb of water and an average level of 7 ppb in groundwater (ATSDR 2007e). In the United States, about 400,000 workers are routinely exposed to TCE (ATSDR 2007e). People living near hazardous waste sites might be exposed to it in the air or in their drinking water, or in the water used for bathing or cooking.

TCE enters the body when a person breathes air or drinks water containing it. If a person drinks TCE, most of it will be absorbed into the blood (ATSDR 2007e). Once in the blood, the liver changes much of the TCE into other chemicals (ATSDR 2007e). The majority of these breakdown products leave the body in the urine within a day (ATSDR 2007e). People also quickly breathe out much of the TCE that's in their bloodstreams (ATSDR 2007e). Some of the TCE or its breakdown products can be stored in body fat for a brief period and thus might build up in the body if exposure continues (ATSDR 2007e).

The scientific literature indicates adverse noncancer effects associated with oral TCE exposure include decreased body weight, liver and kidney effects, and neurological, immunological, reproductive, and developmental effects (USEPA 2011b). U.S. EPA has developed RfDs for the more sensitive endpoints within each type of TCE toxicity (USEPA 2011b). Multiple RfDs for effects from oral studies are in the relatively narrow range of  $3.0 \times 10^{-4}$  to  $8.0 \times 10^{-4}$  mg/kg/day (USEPA 2011b). The two lowest TCE RfDs of  $8.0 \times 10^{-4}$  mg/kg/day are for increased kidney



weight in rats and  $5.0 \times 10^{-4}$  mg/kg/day for both heart malformations in rats and decreased thymus weights in mice (USEPA 2011b). A third TCE RfD of  $3.0 \times 10^{-4}$  mg/kg/day for increased toxic nephropathy in rats is from an inhalation study (USEPA 2011b). ATSDR used the TCE RfD of  $5.0 \times 10^{-4}$  mg/kg/day. It reflects the midpoint among the similar RfDs for the critical effects at  $4.0 \times 10^{-4}$  mg/kg/day for developmental immunotoxicity in mice. Also,  $5.0 \times 10^{-3}$  mg/kg/day for both heart malformations in rats and decreased thymus weights in mice is within 25 percent of each of the other RfDs (USEPA 2011).

This TCE RfD is based on oral studies of the critical effects of heart malformations (rats), adult immunological effects (mice), and developmental immunotoxicity (mice) (USEPA 2011). U.S. EPA took the  $5.1 \times 10^{-3}$  mg/kg/day RfD from a physiologically based pharmacokinetic (PBPK) model of TCE metabolism in rats and humans and applied it to the animal study that observed an increased rate of heart defects in newborn rats born to mothers exposed to TCE in drinking water. U.S. EPA obtained a 99th percentile human equivalent dose (HED99) of  $5.1 \times 10^{-3}$  mg/kg/day. The HED99 is the dose derived from animal studies that takes into account the physiologic and pharmacokinetic differences in animal models and humans. A HED99 of  $5.1 \times 10^{-3}$  mg/kg/day TCE derived for a 1 percent response rate of fetal heart malformation in humans maybe consistent with the critical effects of heart malformations in rats.

#### II.D.18.a. Comparison of Estimated TCE Doses to Health Effect Levels

As shown in Table B1 in Appendix B, TCE was detected at a concentration of 81.1 ppb in only one groundwater sample from well OMW-1B. Using this TCE concentration level and a CTE exposure scenario, the estimated cumulative infant exposure dose (ingestion plus inhalation plus dermal contact) of  $1.0 \times 10^{-2}$  mg/kg/day is higher than the HED<sub>99</sub> of  $5.1 \times 10^{-3}$  mg/kg/day. But the estimated adult exposure dose of  $2.4 \times 10^{-3}$  mg/kg/day is 2 times lower than the HED<sub>99</sub>. Thus the one groundwater sample in Melton Valley containing an elevated level of TCE results in estimated exposure doses that correspond to a 1 percent response rate for fetal heart malformations in humans. Therefore, chronic ingestion of TCE at the concentration detected in groundwater from monitoring well OMW-1B could cause harmful health effects and is a *potential* public health hazard. Once again, however, groundwater from this monitoring well is not used for home purposes, and only 1 percent of the Melton Valley groundwater sample (1 out of 100) contained an elevated TCE level.

### **III. Conclusions**

ATSDR has evaluated groundwater samples collected in 2010 and 2011 from off-site residential wells and from off-site DOE monitoring wells in Melton Valley and Bethel Valley, across the Clinch River and downgradient from the DOE Oak Ridge Reservation. ATSDR concludes

- 1. Based on the results of the off-site groundwater sampling, ATSDR knew of no one exposed at public health hazard levels to chemicals and radionuclides in off-site groundwater in Melton and Bethel Valleys.
- 2. In Melton Valley, groundwater samples from seven off-site DOE monitoring wells contained concentrations of lead, lithium, fluoride, and trichloroethylene at levels that could cause harmful health effects with chronic (long-term, more than 1 year) ingestion of groundwater, dermal contact with groundwater, or inhalation of gaseous chemicals that escape into the air. Chronic ingestion, dermal contact, and inhalation of chemicals in the

groundwater from these wells could be a public health hazard, but no one was or is exposed to the groundwater from these monitoring wells. For information on each chemical, the specific off-site monitoring wells, and potential health effects, see Table 5, Figure 1, and the discussion of the specific chemical in this consultation's Public Health Implications section. Potential health impacts from combined action of chemical mixtures are not evaluated because there is no known exposure to chemicals at public health hazard levels.

- 3. In Bethel Valley, groundwater samples from one off-site, former residential well now used only for monitoring contained concentrations of fluoride and benzene at levels that could cause harmful health effects with chronic (long-term, more than 1 year) ingestion of groundwater, dermal contact with groundwater, or inhalation of gaseous chemicals that escape into the air. Such chronic ingestion, dermal contact, and inhalation of these chemicals in the groundwater from this private well would be a public health hazard. But this former residential well was not and is not used for domestic purposes; no one is exposed to the chemicals in the groundwater sampled in this well. For information on each chemical, the specific off-site private well, and potential health effects, see Table 6, Figure 1, and the discussion of the specific chemical in this consultation's Public Health Implications section. Potential health impacts from combined action of chemical mixtures are not evaluated because there is no known exposure to chemicals at public health hazard levels.
- 4. ATSDR cannot conclude whether chemicals and radionuclides in Melton Valley and Bethel Valley groundwater could harm the health of some residents currently using groundwater from off-site private wells for home purposes. The groundwater from some of these off-site private wells has not been sampled and analyzed for chemical or for radioactive contaminants. Without analytical results from continued monitoring of the groundwater from all off-site private residential wells used for home purposes, ATSDR cannot determine whether groundwater in these wells contains contaminants at public health hazard levels.
- 5. ATSDR cannot adequately characterize the public health hazard of exposure to chemicals in groundwater of private wells sampled in Bethel Valley. Too few samples were collected, and therefore too few samples were analyzed for metals and volatile organic chemicals. Thus ATSDR cannot characterize adequately exposure to chemicals in the groundwater over an extended period.
- 6. People undergoing lithium treatment need to be cautious about drinking any Melton Valley and Bethel Valley groundwater that contains lithium. The additional lithium in the groundwater can increase their lithium dose levels and increase their risk of nephrogenic diabetes insipidus (form of diabetes insipidus primarily due to pathology of the kidney) and other lithium intoxication side effects.

## **IV. Recommendations**

1. Conduct a comprehensive well use survey and inventory of all off-site private wells in Melton Valley and Bethel Valley. The survey area should include an area from the Clinch River toward the west and downgradient of the DOE site for at least 1 mile



2. Monitor the groundwater in private wells within the Melton and Bethel Valley survey area that are used for domestic purposes. If elevated levels of chemicals are found in the groundwater or residents do not want to monitor the groundwater they should find an alternative source of water for domestic use, such as connecting to the Watts Bar Utility District. Groundwater monitoring should include analysis for metals, volatile organic chemicals, gross beta, and gross alpha. Quarterly monitoring will allow adequate characterization of exposure over an extended period and the temporal and spatial (vertical and lateral) extent of contaminat plumes in the off-site groundwater.

## V. Public Health Action Plan

The public health action plan for Oak Ridge Reservation off-site groundwater describes actions ATSDR has taken and will take. This plan identifies public health hazards found in the public health consultation and provides action items designed to mitigate and to prevent harmful human health effects resulting from exposure to chemicals and radionuclides in the groundwater off-site from ORR.

#### V.A. Completed public health actions:

- 1. ATSDR completed a public health assessment in 2006 on the DOE Oak Ridge Reservation entitled *Evaluation of Potential Exposures to Contaminated Off-Site Groundwater from the Oak Ridge Reservation*
- 2. ATSDR reviewed and evaluated the 2010 and 2012 off-site groundwater data collected by TDEC and the DOE from off-site residential wells and off-site DOE monitoring wells in areas across the Clinch River from the DOE Oak Ridge Reservation
- 3. ATSDR prepared this health consultation to address public health issues related to the detected levels of contaminants in the off-site groundwater samples

### V.B. Scheduled public health actions:

- 1. Completion of the ATSDR health consultation
- 2. ATSDR is available to provide technical assistance upon request to review
  - a. Work plans for future off-site groundwater monitoring and recommendations to protect public health
  - b. Off-site groundwater sampling data and recommendations to protect public health for local residents monitoring their private wells or for DOE, TDEC, or U.S. EPA conducting follow-up environmental investigations
- 3. ATSDR is available to assist in addressing health concerns upon request by
  - a. Providing fact sheets on the potential for toxicological effects from chemicals, and
  - b. Collaborating with local physicians and medical facilities to help medical professionals interpret the potential for health effects from exposure to contaminants, should any such exposures occur.

## VII. Authors, Technical Advisors

Jack E. Hanley, M.P.H. Environmental Health Scientist Central Branch Division of Community Health Investigations Agency for Toxic Substances and Disease Registry

Carol Connell Health Physicist Central Branch Division of Community Health Investigations Agency for Toxic Substances and Disease Registry

Barbara Anderson, P.E., MSEnvE Environmental Health Scientist Science Support Branch Division of Community Health Investigations Agency for Toxic Substances and Disease Registry

### **Independent Reviewers**

Paul Charp, Ph.D. Senior Health Physicist Eastern Branch Division of Community Health Investigations Agency for Toxic Substances and Disease Registry

Trent D. LeCoultre, MSEH, REHS LCDR, US Public Health Service Environmental Health Scientist Central Branch Division of Community Health Investigations Agency for Toxic Substances and Disease Registry

Karl V. Markiewicz, Ph.D. Senior Toxicologist Eastern Branch Division of Community Health Investigations Agency for Toxic Substances and Disease Registry



## VIII. References

[ACCLPP] Advisory Committee on Childhood Lead Poisoning Prevention (ACCLPP) of the Centers for Disease Control and Prevention (CDC). 2012. Low Level Lead Exposure Harms Children: A Renewed Call for Primary Prevention. Atlanta: ACCLPP; January 2012. Available from: <u>http://www.cdc.gov/nceh/lead/ACCLPP/Final\_Document\_030712.pdf</u>.

[ATSDR] Agency for Toxic Substances and Disease Registry. 1989. Toxicological profile for bromodichloromethane. Atlanta: US Department of Health and Human Services; December.

[ATSDR] Agency for Toxic Substances and Disease Registry. 1992a. Toxicological profile for antimony and compounds. Atlanta: US Department of Health and Human Services; September.

[ATSDR] Agency for Toxic Substances and Disease Registry. 1992b. Toxicological profile for thallium. Atlanta: US Department of Health and Human Services; June.

[ATSDR] Agency for Toxic Substances and Disease Registry. 1992c. Toxicological profile for cis-1,2-dichloroethene. Atlanta: US Department of Health and Human Services; August.

[ATSDR] Agency for Toxic Substances and Disease Registry. 1997. Toxicological profile for chloroform. Atlanta: US Department of Health and Human Services; September.

[ATSDR] Agency for Toxic Substances and Disease Registry. 2003. Toxicological profile for fluorides, hydrogen Fluoride, and fluorine. Atlanta: US Department of Health and Human Services; September.

[ATSDR] Agency for Toxic Substances and Disease Registry. 2004. Toxicological profile for strontium. Atlanta: US Department of Health and Human Services; April.

[ATSDR] Agency for Toxic Substances and Disease Registry. 2006. Public Health Assessment, Evaluation of Potential Exposures to Contaminated Off-Site Groundwater from the Oak Ridge Reservation, Oak Ridge Reservation. Atlanta: US Department of Health and Human Services. July 2006.

[ATSDR] Agency for Toxic Substances and Disease Registry. 2007a. Toxicological profile for arsenic. Atlanta: US Department of Health and Human Services; August.

[ATSDR] Agency for Toxic Substances and Disease Registry. 2007b. Toxicological profile for barium. Atlanta: US Department of Health and Human Services; August.

[ATSDR] Agency for Toxic Substances and Disease Registry. 2007c. Toxicological profile for lead. Atlanta: US Department of Health and Human Services; August.

[ATSDR] Agency for Toxic Substances and Disease Registry. 2007d. Toxicological profile for benzene. Atlanta: US Department of Health and Human Services; August.

[ATSDR] Agency for Toxic Substances and Disease Registry. 2007e. Toxicological profile for Tricholoethylene. Atlanta: US Department of Health and Human Services; September.

[ATSDR] Agency for Toxic Substances and Disease Registry. 2012a. Toxicological profile for cadmium. Atlanta: US Department of Health and Human Services; September.

[ATSDR] Agency for Toxic Substances and Disease Registry. 2012b. Toxicological profile for chromium. Atlanta: US Department of Health and Human Services; September.

[ATSDR] Agency for Toxic Substances and Disease Registry. 2012c. Toxicological profile for manganese. Atlanta: US Department of Health and Human Services; September.

Baldessarini, R.J. and F.I. Tarazi. 2001. Drugs and the treatment of psychiatric disorders. In: Goodman and Gilman's The Pharmacological Basis of Therpeutics. J.G. Hardman, L.E. Limbird and A.G. Gilman, Ed. McGraw-Hill Companies, Inc., Medical Publishing Division, New York. Cited in US Environmental Protection Agency. 2008. Provisional Peer Reviewed Toxicity Values for Lithium (CASRN 7439-93-2).Cincinnati: Superfund Health Risk Technical Support Center. June.

[CDPH] Chromiuim-6 in drinking water: Sampling results. California Department of Public Health. http://ww2.cdph.ca.gov/certlic/drinkingwater/pages/chromium6sampling.aspx. August 21, 2008. Cited in: Agency for Toxic Substances and Disease Registry. 2012. Toxicological profile for chromium. Atlanta: US Department of Health and Human Services; September.

Davidsson L, Cederblad A, Hagebo E, et al. 1988. Intrinsic and extrinsic labeling for studies of manganese absorption in humans. J Nutr 118:1517–1524. Cited in: Agency for Toxic Substances and Disease Registry. 2012. Toxicological profile for manganese. Atlanta: US Department of Health and Human Services; September.

[FNB/IOM] Food and Nutrition Board, Institute of Medicine. 2008. Manganese. Dietary reference intakes for vitamin A, vitamin K, arsenic, boron, chromium, copper, iodine, iron, manganese, molybdenum, nickel, silicon, vanadium, and zinc. A Report of the Panel on Micronutrients, subcommittees on upper reference levels of nutrients and of interpretation and uses of dietary reference intakes, and the standing committee on the scientific evaluation of dietary reference intakes. Washington, DC: Food and Nutrition Board. Institute of Medicine. National Academy Press, 394-419. April. Cited in: Agency for Toxic Substances and Disease Registry. 2012. Toxicological profile for manganese. Atlanta: US Department of Health and Human Services; September.

Gillman K. 2012. Lithium in the Environment, Diet and Body. Queensland, Australia: PsychoTropical Research; October. Available from: <u>http://www.psychotropical.com/index.php/mood-stabilisers/210-lithium-in-the-environment-diet-and-body</u>.



Giardino NJ and Andelman JB. 1996. Characterization of the emissions of trichloroethylene, chloroform, and 1,2 dibromo-3-chloropropane in a full-size, experimental shower. J Exposure Anal Environ Epidemiol 6 413-423.

Gitlin M. 1999. Lithium and the kidney: an updated review. Drug Saf. 20(3)231-243. Cited in US Environmental Protection Agency. 2008. Provisional Peer Reviewed Toxicity Values for Lithium (CASRN 7439-93-2).Cincinnati: Superfund Health Risk Technical Support Center. June.

Hardman, J.G., Limbird, L.E., Molinoff, P.B., Ruddon, R.W., and Gilman, A.G. 1996. Goodman and gilman's the pharmacological basis of therapeutics. Ninth Edition. McGraw-Hill Companies, Inc.; New York, NY. Cited in: Salocks, C. Kaley, K. 2003. Technical support document: toxicology clandestine drug labs: methamphetamine, volume 1, number 4, lithium. Sacramento: California Environmental Protection Agency, Office of Environmental Health Hazard Assessment; September.

Helsel DR. 2012. Statistics for censored environmental data using minitab and r, 2<sup>nd</sup> Edition. John Wiley & Sons, Inc., Hoboken, New Jersey.

[IOM] Institute of Medicine. 2001. Chromium. Dietary reference intakes for vitamin A, vitamin K, arsenic, boron, chromium, copper, iodine, iron, manganese, molybdenum, nickel, silicon, vanadium, and zinc. A Report of the Panel on Micronutrients, subcommittees on upper reference levels of nutrients and of interpretation and uses of dietary reference intakes, and the standing committee on the scientific evaluation of dietary reference intakes. Washington, DC: Food and Nutrition Board. Institute of Medicine. National Academy Press, 197-223. Cited in: Agency for Toxic Substances and Disease Registry. 2012. Toxicological profile for chromium. Atlanta: US Department of Health and Human Services; September.

[IARC] International Agency for Research on Cancer. 1982. Inorganic fluorides used in drinking-water and dental preparations. In: IARC Monographs on the evaluation of carcinogenic risk of chemicals to humans. Vol. 27: Some aromatic amines, anthraquinones and nitroso compounds, and inorganic fluorides used in drinking-water and dental preparations. Lyon, France: World Health Organization, International Agency for Research on Cancer, 237-303. Cited in: Agency for Toxic Substances and Disease Registry. 2003. Toxicological profile for Fluorides, Hydrogen Fluoride, and Fluorin. Atlanta: US Department of Health and Human Services; September.

Johnson P, Goldberg S, Mays M, Dawson B. 2003. Threshold of trichloroethylene contamination in maternal drinking waters affecting fetal heart development in the rat. Environ Health Perspect, 111, 289-292. Cited in: [USEPA 2011b] US Environmental Protection Agency. 2011. Integrated risk information system. Trichloroethylene (CASRN 79-01-6). Available from: http://www.USEPA.gov/IRIS/subst/0199.htm

Kjellstrom T, Borg K, Lind B. 1978. Cadmium in feces as an estimator of daily cadmium intake in Sweden. Environ Res 15:242–251. Cited in: Agency for Toxic Substances and Disease

Registry. 2012. Toxicological profile for cadmium. Atlanta: US Department of Health and Human Services; September.

Lagerkvist BJ, Lindell B. 2002. The Nordic Expert Group for Criteria Documentation of Health Risks from Chemicals - 131. Lithium and lithium compounds. Stockholm, Sweden: National Institute for Working Life, Nordic Council of Ministers.

Li Y, Liang CK, Slemenda CW, et al. 2001. Effect of long term exposure to fluoride in drinking water on risks of bone fractures. J Bone Miner Res 16(5):932-939. Cited in: Agency for Toxic Substances and Disease Registry. 2003. Toxicological profile for Fluorides, Hydrogen Fluoride, and Fluorin. Atlanta: US Department of Health and Human Services; September.

Maine DEP/DHS. 1992. Draft Guidance Manual for Human Health Assessments at Hazardous Waste Substance Sites. State of Maine Department of Environmental Protection and Department of Health Services, Augusta, ME, Sept. 1, 1992.

Markowitz GS, Radhakrishnan J, Kambham N, Valeri AM, Hines WH, D'Agati VD, 2000. Lithium Nephrotoxicity: A progressive combined glomerular and tubulointerstitial nephrophy. Journal of the American Society of Nephrology 11: 1439–1448.

McCauley PT, Robinson M, Daniel FB, et al. 1990. The effects of subacute and subchronic oral exposure to cis-1,2-dichloroethylene in rats. Health Effects Research Laboratory, US Environmental Protection Agency, Cincinnati, OH and Toxic Hazards Division, Air Force Aerospace Medical Research Laboratory, Wright-Patterson Air Force Base, OH; unpublished report. Cited in: [USEPA 2010] US Environmental Protection Agency. 2010. Integrated risk information system. cis-1,2-Dichloroethylene (CASRN 156-59-2). Available from: http://www.USEPA.gov/iris/subst/0418.htm.

McCauley PT, Robinson M, Daniel FB. et al. 1995. The effects of subacute and subchronic oral exposure to cis-1,2-dichloroethylene in Sprague-Dawley rats. Drug Chem Toxicol 18:171–184. Cited in: [USEPA 2010] US Environmental Protection Agency. 2010. Integrated risk information system. cis-1,2-Dichloroethylene (CASRN 156-59-2). Available from: <u>http://www.USEPA.gov/iris/subst/0418.htm</u>.

McIntyre RS, Mancini DA, Parikh S, Kennedy SH, 2001. Lithium revisited. Can. J. Psychiatry. 46:322-327. Cited in US Environmental Protection Agency. 2008. Provisional Peer Reviewed Toxicity Values for Lithium (CASRN 7439-93-2).Cincinnati: Superfund Health Risk Technical Support Center. June.

Mena I, Horiuchi K, Burke K, et al. 1969. Chronic manganese poisoning: individual susceptibility and absorption of iron. Neurology 19:1000–1006. Cited in: Agency for Toxic Substances and Disease Registry. 2012. Toxicological profile for manganese. Atlanta: US Department of Health and Human Services; September.

Moya J, Howard-Reed C, and Corsi RL. 1999. Volatization of chemicals from tap water to indoor air from contaminated water used for showering. Environ Sci Technol 33:2321-2327.



[NRC] National Research Council. 2006. Fluoride in drinking water: A scientific review of USEPA's standards. Committee on Fluoride in Drinking Water, Board on Environmental Studies and Toxicology, Division on Earth and Life Studies. Washington, DC: National Academies of Sciences.

[NTP] National Toxicology Program. 1994. Toxicology and carcinogenesis studies of barium chloride dihydrate -(CAS No. 10326-27-9) in F344/N rats and B6C3F1 mice. National Toxicology Program. TR432. Cited in: Agency for Toxic Substances and Disease Registry. 2007. Toxicological profile for barium. Atlanta: US Department of Health and Human Services; August.

[NTP] National Toxicology Program. 2008a. NTP technical report on the toxicology and carcinogenesis studies of sodium dichromate dihydrate (CAS No. 7789-12-0) in F344/N rats and B6C3F1 mice (drinking water studies). Washington, DC: National Toxicology Program. NTP TR 546. http://ntp.niehs.nih.gov/files/546\_web\_FINAL.pdf. August 13, 2008. Cited in: Agency for Toxic Substances and Disease Registry. 2012. Toxicological profile for chromium. Atlanta: US Department of Health and Human Services; September.

[PDR] Physician's Desk Reference. 2006. 60th ed. L. Murray, Ed. Montvale, NJ: Thompson PDR. Cited in US Environmental Protection Agency. 2008. Provisional Peer Reviewed Toxicity Values for Lithium (CASRN 7439-93-2).Cincinnati: Superfund Health Risk Technical Support Center. June.

Presne C, Fakhouri F, Noël L H. 2003. Lithium-induced nephropathy: rate of progression and prognostic factors. Kidney Int. 64:585-592. Cited in US Environmental Protection Agency. 2008. Provisional Peer Reviewed Toxicity Values for Lithium (CASRN 7439-93-2).Cincinnati: Superfund Health Risk Technical Support Center. June.

Salocks, C. Kaley, K. 2003. Technical support document: toxicology clandestine drug labs: methamphetamine, volume 1, number 4, lithium. Sacramento: California Environmental Protection Agency, Office of Environmental Health Hazard Assessment; September.

Schrauzer, G. 2002. Lithium: occurrence, dietary intakes, nutritional essentiality. Journal of American College of Nutrition 21 (1): 14-21.

[Storey 1961] Storey, E. 1961. Strontium "rickets" bone calcium and strontium changes. Austral. Ann. Med. 10: 213-222. Cited in: US Environmental Protection Agency. 2012. Integrated risk information system. Strontium (CASRN 7440-24-6).

Symons, J. 2001. Plain talk about drinking water, 4<sup>th</sup> Edition. Denver: American Water Works Association.

Stokinger HE. 1981. Lithium. Patty's Industrial Hygiene and Toxicology. Volume 2A. Third Revised Edition. Clayton GD and Clayton FE eds. John Wiley and Sons; New York, NY. Cited in: Salocks C. Kaley K. 2003. Technical support document: toxicology clandestine drug labs: methamphetamine, volume 1, number 4, lithium. Sacramento: California Environmental Protection Agency, Office of Environmental Health Hazard Assessment; September.

Storey E. 1962. Intermittent bone changes and multiple cartilage defects in chronic strontium rickets in rats. J Bone Jt Surg Am 44B(1):194-208. Cited in: Agency for Toxic Substances and Disease Registry. 2004. Toxicological profile for strontium. Atlanta: US Department of Health and Human Services; April.

[TDEC] Tennessee Department of Environment and Conservation. 2011. DOE Oversight Division. Environmental Monitoring Report January through December 2010 - Groundwater Monitoing for the Oak Ridge Reservation and its Environs. Authors: Sebastian JE, Davies G, Worthington CE. Nashville, TN: Tennessee Department of Environment and Conservation. April 2011.

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(3):453–63. Cited in: Agency for Toxic Substances and Disease Registry. 2007. Toxicological profile for arsenic. Atlanta: US Department of Health and Human Services; September.

Tseng WP. 1977. Effects and dose-response relationship of skin and blackfoot disease with arsenic. Environmental Health Perspectives 19:109-19. Cited in: Agency for Toxic Substances and Disease Registry. 2007. Toxicological profile for arsenic. Atlanta: US Department of Health and Human Services; September.

USEPA]. Health assessment document for chromium. 1984a. Research Triangle Park, NC: Environmental Assessment and Criteria Office, US Environmental Protection Agency. EPA600883014F. Cited in: Agency for Toxic Substances and Disease Registry. 2012. Toxicological profile for chromium. Atlanta: US Department of Health and Human Services; September.

[USEPA] US Environmental Protection Agency. 1984b. Health assessment document for manganese. Final draft. Cincinnati, OH: US Environmental Protection Agency, Office of Research and Development. USEPA-600/8-83-013F. Cited in: Agency for Toxic Substances and Disease Registry. 2012. Toxicological profile for manganese. Atlanta: US Department of Health and Human Services; September.

[USEPA] US Environmental Protection Agency. 1985. Drinking water criteria document on cadmium. Office of Drinking Water, Washington, DC. (Final draft). Cited in: US Environmental Protection Agency. 2012. Integrated risk information system. Cadmium (CASRN 7440-43-9).

[USEPA] US Environmental Protection Agency. 1994. Integrated risk information system. Cadmium (CASRN 7440-43-9). Available from <u>http://www.USEPA.gov/iris/subst/0141.htm</u>.

JUSEPA] US Environomental Protection Agency. 1996. Integrated risk information system. Strontium. (CASRN 7440-24-6). "Available from: <u>http://www.epa.gov/iris/subst/0550.htm</u>.

[USEPA] US Environmental Protection Agency. 1998a. Integrated risk information system. Chromium (CASRN 18540-29-9). Available from: <u>http://www.USEPA.gov/iris/subst/0144.htm</u>.



[USEPA] US Environmental Protection Agency. 1998b. Integrated risk information system. Strontium (CASRN 7440-24-6). Available from: <u>http://www.USEPA.gov/iris/subst/0144.htm</u>.

[USEPA] US Environmental Protection Agency. 2001. Integrated risk information system. Chloroform (CASRN 67-66-3). Available from: <u>http://www.epa.gov/iris/subst/0025.htm</u>.

[USEPA] US Environmental Protection Agency. 2004. Integrated risk information system. Lead and compounds (inorganic) (CASRN 7439-92-1). Available from: <u>http://www.USEPA.gov/iris/subst/0144.htm</u>.

[USEPA] US Environmental Protection Agency. 2008. Provisional Peer Reviewed Toxicity Values for Lithium (CASRN 7439-93-2).Cincinnati: Superfund Health Risk Technical Support Center. June.

[USEPA]. US Environmental Protection Agency. 2009. Thallium and Compounds (CAS No. 7440-28-0) In support of summery information on the integrated risk information System (IRIS). USEPA/635/R-08/001F. September. Cited in: US Environmental Protection Agency. 2012. Integrated risk information system. Thallium (CASRN 7440-28-0).

[USEPA] US Environmental Protection Agency. 2010. Integrated risk information system. cis-1,2-Dichloroethylene (CASRN 156-59-2). Available from: http://www.USEPA.gov/iris/subst/0418.htm.

[USEPA] US Environmental Protection Agency. 2011a. Exposure factor handbook: 2011 Final. Washington DC: National Center for Environmental Assessment, USEPA/600/R-09/052F.

[USEPA] US Environmental Protection Agency. 2011b. Integrated risk information system. Trichloroethylene (CASRN 79-01-6). Available from: <u>http://www.USEPA.gov/IRIS/subst/0199.htm</u>

[USEPA] US Environmental Protection Agency. 2012. Drinking Water Contaminants. National Primary Drinking Water Regulations. Available from: http://water.USEPA.gov/drink/contaminants/index.cfm#Primary.

[VDH] Virginia Department of Health. 1997. Fact sheet on lithium. Richmond, Virginia: Virginia Department of Health, Bureau of Toxic Substances; February.

Wan JK, et al. 1990. Chloroform Exposure and the Risk Associated with Multiple Uses of Chlorinated Tap Water. Risk Analysis 1990; 10(4):581-585.

[WHO] World Health Organization. 2004. Guidelines for drinking-water quality. Vol. 1. Recommendations. 3rd ed. Geneva, Switzerland: World Health Organization. http://www.who.int/water\_sanitation\_health/dwq/gdwq3/en/. March 08, 2006. Cited in: Agency for Toxic Substances and Disease Registry. 2012. Toxicological profile for chromium. Atlanta: US Department of Health and Human Services; September.

## Appendix A. Chemical Screening Methodology

ATSDR evaluates all the groundwater sampling data using a two-step chemical screening process to identify chemicals that are of potential public health concern and require an in-depth evaluation of the contaminate concentration in the groundwater to determines the public health implications of exposure. For chemicals selected for further evaluation, ATSDR reviews available human studies as well as experimental animal studies to understand the disease-causing potential of a chemical and to compare site-specific exposure dose estimates with doses shown to cause health effects. This process enables ATSDR to weigh the available evidence in light of uncertainties and offer perspective on the plausibility of harmful health outcomes under site-specific conditions.

### I. Comparing Environmental Concentrations to Comparison Values

In the first step of the chemical screening process, ATSDR screened the groundwater chemical data by comparing the maximum concentration of each chemical detected in off-site groundwater against ATSDR's conservative (protective), chemical-specific, drinking water comparison value. The maximum concentrations are used at this step of the screening process as a conservative (protective) measure although it is known that people are exposed to a range of concentrations in the groundwater over time and not just to the maximum reported levels. ATSDR's cancer risk evaluation guides (CREGs), environmental media evaluation guides (EMEGs), and reference dose media evaluation guides (RMEGs) are conservative (protective), drinking water comparison values developed for screening environmental concentrations of chemicals in groundwater. Drinking water comparison values are concentrations developed by ATSDR for each chemical from available scientific literature concerning exposure and health effects. Because comparison values reflect concentrations that are many times lower than concentrations that have been observed to cause adverse health effects in studies on experimental animals or in human epidemiologic studies, comparison values are protective of public health in essentially all exposure situations. As a result, exposures to chemical concentrations detected at or less than (below) ATSDR's comparison values are not expected to cause health effects in people. Therefore, levels less than the drinking water comparison values do not pose a public health hazard and are not evaluated further.

### II. Comparing Estimated Exposure Doses to Screening Guideline Values

In the second step of the chemical screening process, ATSDR further evaluates the non-cancer and cancer health effects of each chemical identified in the first step with a maximum groundwater concentration above conservative (protective), chemical-specific, drinking water comparison values.

### II.A. Non-cancer Screening

For non-cancer health effects, ATSDR calculates chronic (1-year annual) exposure doses for an infant and adult and compares these estimated exposure doses to conservative (protective) chemical-specific health-based (non-cancer) guideline values, including ATSDR's minimal risk



levels (MRLs) and EPA's reference doses (RfDs). For each well station, the chronic exposure dose for each chemical is estimated using the mean groundwater concentration in each well and a reasonable maximum exposure (RME) scenario for an infant (birth to less than 1 year) and adult (age 21 to age 65) (See estimated chronic exposure doses in Appendix B – Table B1 and Table B3).

For each well, the mean groundwater concentration is calculated using applicable methods for the data set (Helsel 2012). ATSDR followed Helsel's recommended methods for estimation of summary statistics (Helsel 2012). The mean chemical concentration in the groundwater from each well is used because for each well, the concentration of chemicals in the groundwater well varies over time. Furthermore, all of the chemicals were not detected in all of the samples. Thus, it is more likely that people would be exposed to a range of chemical concentrations over time.

The RME refers to people who are at the high end of age-specific water ingestion rates (approximately the 95th percentile). ATSDR chose to use more conservative RME scenarios to estimate exposure doses that are higher than average, but are still within a realistic range of exposure. Use of the RME scenario is health-protective and results in a more conservative screening process because the exposure doses are calculated using the high-end exposure distribution. An infant who drinks formula prepared with contaminated groundwater may be at a higher risk because of the large volume of water consumed relative to body size.

Age Range (years)	Water Ingestion Rate 95th Percentile (mL/day)	Body Weight (kg)
Infant Birth to <1 year	1,113	7.8
Adult 21 to <65 year	2848	80

The following reasonable maximum exposure (RME) parameters are used to calculate the chronic exposure dose from groundwater:

Note: USEPA Exposure Factor Handbook: 2011 Exposure Frequency (EF) - 1

The following general equation is used to calculate chronic (1-year annual) exposure doses:

$$D = \frac{C \times IR \times EF \times AF}{BW}$$
Where:  

$$D = Exposure dose (mg/kg/day)$$

$$C = Mean concentration of chemical (mg/L)$$

$$IR = Intake rate (L/day)$$

$$EF = Exposure frequency, exposure events per year of exposure (unitless)$$

$$AF = Absorption Factor (bioavailability factor) (unitless)$$

$$BW = Body weight (kg)$$

The absorption faction (bioavailability factor) represents, as a percent, the total amount of a substance ingested that actually enters the bloodstream and is therefore possibly available to harm a person. Typically, the bioavailability factor is assumed to be 1 (100%) for screening purposes—that is, all of a substance to which a person is exposed is assumed to be absorbed. However, ATSDR considers the bioavailability factor when conducting a further, refined, indepth evaluation of exposures and substance toxicology to determine the public health implications of exposure

ATSDR's chemical-specific, health-based non-cancer guideline values (ATSDR's MRLs and EPA's RfDs) are estimated doses of daily human exposure to substances that are likely to be without appreciable risk of adverse (harmful) non-cancer health effects over a specified duration of exposure. MRLs and RfDs are derived for chemicals using the no-observable-adverse-effect-level (NOAEL)/lowest-observed-adverse-effect level (LOAEL)/uncertainty factor approach. A NOAEL is the highest tested dose of a substance that has been reported to have no harmful (adverse) health effects on people or animals in a study. A LOAEL is the lowest tested dose of a substance that has been reported to cause harmful (adverse) health effects in people or animals.

Health-based guideline values are derived when reliable and sufficient human or animal data exist for a given route of exposure to identify the most sensitive health effect considered to be of relevance to humans. Because of the uncertainty and the lack of precise toxicologic information on people who might be most sensitive (for example, infants, the elderly, or persons who are nutritionally or immunologically compromised) to the effects of hazardous substances, MRLs and RfDs have built-in safety factors, making the MRL and RfD doses considerably lower than doses at which health effects have been observed in studies on experimental animals or in human epidemiologic studies. Therefore, these screening guideline values are below doses that cause adverse health effects in people most sensitive to such effects. Consistent with the public health principle of prevention, ATSDR uses this conservative (protective) chemical screening approach to maximize human health protection and to address the uncertainty in toxicologic information.

These chemical-specific, health-based guideline values, which serve as screening levels, are used to identify chemicals for further consideration. It is important to note that MRLs and RfDs are not thresholds for health effects and are not intended to define cleanup or action levels. They are intended only to serve as a screening tool to help public health professionals decide what chemicals and pathways to evaluate further and to look at more closely. Estimated exposure doses that are less than (below) MRLs or RfDs pose no public health hazard and are not evaluated further. While exposure doses at or below the respective health-based guideline values can be considered safe, estimated exposure doses greater than (above) these screening guideline values do not automatically imply that adverse health effects will occur. Rather, it is an indication that ATSDR should conduct a more refined public health implication evaluation of the chemical by reviewing the health effect levels reported in the scientific literature and by more fully reviewing potential exposures.

### **II.B. Carcinogenic Health Effects**

To screen for cancer effects, ATSDR also evaluates the carcinogenic potential of chemicals identified in the first step with a maximum groundwater concentration above drinking water



comparison values. ATSDR calculates the potential excess cancer risk for children and adults by multiplying the estimated chronic exposure doses and the EPA cancer slope factors (CSFs) (See estimated excess cancer risk in Appendix B in Table B2 and Table B4). This calculation estimates the potential excess cancer risk expressed as the proportion of a population that might be affected by a carcinogen during a lifetime of exposure.

The estimated chronic exposure doses are based on the mean concentration of a chemical in each well and conservative RME lifetime exposure scenarios for children and adults. For each well ATSDR followed Helsel's recommended methods for estimation of the mean groundwater concentration (Helsel 2012). The conservative RME lifetime exposure scenario for children is from birth to age 21 (21 years of exposure averaged over a lifetime of 78 years) and for adults from age 21 to age 65 (44 years of exposure averaged over a lifetime of 78 years). The EPA's cancer slope factors (CSFs) are an estimate of the relative potency of carcinogens.

The following general equation is used to estimate cancer risk:

Cancer Risk = (Age-specific Dose x CSF) x <u>Age-specific years of exposure</u> Lifetime in years

The following table below list the age-specific parameters used in calculating the age-specific exposure dose and age-specific risk.

Age Group	RME Ingestion Rate	Body Weight	Duration
	(ml/day)	(kg)	(years)
Birth to <1 year	1,113	7.8	1
1 to <2 year	893	11.4	1
2 to <6 year	1,052	17.4	4
6 to <11 year	1,251	31.8	5
11 to <16 year	1,744	56.8	5
16 to <21 year	2,340	71.6	5
21 to <65 year	2,848	80	44
65 to<78 year	2,604	76.0	13

Note: USEPA Exposure Factor Handbook: 2011

Exposure Frequency (EF) – 1

Lifetime – 78 years

For example, an estimated cancer risk of 1 in a million  $(1 \times 10^{-6})$  predicts the probability of one additional cancer over the background cancer in a population of 1 million. Because conservative models are used to derive CSFs, the exposure doses associated with these potential risks are typically orders of magnitude lower than doses reported in the toxicologic literature to cause carcinogenic effects. As such, estimated cancer risk less than (below) 1 in 10,000 (less than 1 x 10<sup>-4</sup>) indicate that the toxicologic literature would support a finding of low or no apparent risk of cancer. An estimated cancer risk greater than (exceed) 1 in 10,000 (greater than 1 x 10-4); however, indicates that ATSDR should carefully review the scientific literature before making conclusions about potential cancer risks.

### III. Public Health Implication Evaluation

If the estimated exposure doses exceed the MRL or RfD in the chemical screening process or the estimated cancer risk is greater than  $1 \times 10^{-4}$ , ATSDR conducts a refined, in-depth public health implications evaluation on these potential contaminants of concern. This public health implications evaluation reviews available human studies as well as experimental animal studies to understand the disease-causing potential of a chemical and to compare estimated site-specific exposure doses with doses shown to cause health effects.

In this evaluation, ATSDR further analyzes site-specific exposure variables (such as exposure intake rates, duration, and frequency). The likelihood that adverse health outcomes will actually occur depends on site-specific exposure conditions, individual differences, and factors that affect the route, magnitude, and duration of actual exposure. ATSDR reviews the weight-of-evidence of toxicologic and epidemiologic data and health effects variables, including the form and bioavailability of the chemical to obtain information about the toxicity of the chemicals to more completely understand the public health implications of exposure. Weight-of-evidence refers to the extent to which the available scientific information supports the hypothesis that a substance causes an adverse effect in humans. This process enables ATSDR to weigh the available evidence in light of uncertainties and offer perspective on the plausibility of harmful health outcomes under site-specific conditions.

In the evaluation of each chemical, ATSDR considered multiple factors including chemical and physical properties, bioavailability, and the frequency and duration of the estimated exposures. ATSDR also considered characteristics of the exposed population—such as age, sex, genetics, lifestyle, nutritional status, and health status—which influence how individuals absorb, distribute, metabolize, and excrete contaminants. Where appropriate, these characteristics are included in the chemical-specific discussions. Also, where appropriate, ATSDR evaluated acute exposures (less than 14 days) and exposures to children (ages 6 to 11 years of age).

To evaluate chronic exposure to the contaminants of concern in the groundwater, ATSDR first uses the estimated exposure doses in Tables B1 and B3 and the excess cancer risks in Tables B2 and B4 of Appendix B. These estimated exposure doses for each well station are calculated using the mean concentration of the chemical in the groundwater of each well and the RME scenario for infants and adults. The RME scenario is a health-protective assumption and overestimates the average groundwater consumption, but is still within the realistic range of exposure.

Since this RME method overestimates the true average exposure dose values, ATSDR will, if appropriate, base its health evaluation on a more realistic, site-specific exposure using a central tendency exposure (CTE) scenario. The central tendency exposure scenario refers to individuals who have average or typical water intake rates. ATSDR tries to estimate realistic, site-specific exposure scenarios to enable comparisons to actual health effect levels reported in the scientific literature. For example, 1,183 ml/day for an adult (504 ml/day for an infant) was used as the water ingestion rate to evaluate a more realistic exposure instead of the 2,604 ml/day for adult (1,113 ml/day for an infant) used in the RME screening evaluation (EPA 2011).



ATSDR recognizes that developing fetuses, infants, and children can be more sensitive to exposures than adults. As a policy, unless data are available to suggest otherwise, ATSDR considers children to be more sensitive and vulnerable than adults. Thus, the health impact to children is considered first when evaluating exposures as is the potential adverse effects to a community. The health impacts to other groups within the community (such as the elderly, chronically ill, and people engaging in high-exposure practices) also receive special attention during the evaluation.

ATSDR uses existing scientific information, which can include the results of medical, toxicologic, and epidemiologic studies, and the data collected in disease registries to determine the likelihood of health effects that may result from exposures. The science of environmental health is still developing and sometimes scientific information on the health effects of certain substances is not available. In this case, this report suggests further public health actions that are needed.
## Appendix B. Tables

 Table B1. Estimated Chronic Exposure Doses Compared to Noncancer Health-based Screening Guideline Values for Chemicals in Off-Site

 Melton Valley Groundwater with Concentrations Higher than ATSDR Comparison Values

Substance Name	Well Station	Number of Samples Detected/Analyzed	Mean Screening Concentration (ppb) [method calculated]	Estimated Exposure Dose (mg/kg/day)		Non-cancer Screening Guideline (mg/kg/day)	-cancer sening deline kg/day)		s the nated tre Dose ed the cancer ening eline?
				Infant	Adult			Infant	Adult
			Melton Valle	ey .					
Metals									
Antimony	OMW-1D	9/9	8.8 [Std]	1.2E-03	3.1E-04	4.0E-04	RfD	Yes	No
Arsenic	OWM-1AA	1/6	1.87 [1]	2.6E-4	66E-5	3.0E-04	MRL	No	No
	OWM-1A	10/12	14.1 [KM]	2.0E-3	5.0E-4			Yes	Yes
	OWM-1B	12/12	13.2 [Std]	1.9E-3	4.7E-4			Yes	Yes
	OWM-1C	6/6	5.1 [Std]	7.2E-4	1.8E-4			Yes	No
	OMW-1D	18/18	16.2 [Std]	2.3E-03	5.7E-04			Yes	Yes
	OMW-2A	5/7	3.1 [KM]	4.5E-04	1.1E-04			Yes	No
	OMW-2B	6/6	8.1 [Std]	1.1E-03	2.9E-04			Yes	No
	OMW-2C	10/10	4.3 [Std}	6.3E-04	1.5E-04			Yes	No
	OMW-2D	8/12	15.5 [KM]	2.2E-03	5.5E-04			Yes	Yes
	OMW-3B	2/8	4.0 [1]	5.8E-04	1.4E-04			Yes	No
	OMW-3C	4/6	2.2 [KM]	3.2E-04	7.9E-05			Yes	No
	OMW-4A	2/8	3.7 [1]	5.3E-04	1.3E-04			Yes	No
	OMW-4B	2/6	3.4 [1]	4.9E-04	1.2E-04			Yes	No
	RWA-59	1/7	2.0 [1]	2.8E-04	7.1E-05			No	No
	RWA-94	1/1	0.97 [1]	1.4E-04	3.4E-05			No	No
Barium	OWM-2D	6/6	1754.7 [Std]	2.5E-01	6.2E-02	2.0E-01	MRL	Yes	No
Cadmium	OMW-1C	1/6	4.0 [1]	5.7E-04	1.4E-04	1.0E-04	MRL	Yes	Yes
	OMW-1D	1/9	15.8 [1]	2.2E-03	5.6E-04			Yes	Yes



Substance Name	Well Station	Number of Samples Detected/Analyzed	Mean Screening Concentration (ppb) [method calculated]	Estimated Exposure Dose (mg/kg/day)		posure g/day) Non-cancer Screening Guideline (mg/kg/day)		Does the Estimated Exposure Dose Exceed the Non-cancer Screening Guideline?	
				Infant	Adult			Infant	Adult
Chromium	OMW-1AA	6/6	39.1 [Std]	5.6E-03	1.4E-03	9.0E-04	MRL	Yes	Yes
	OMW-1B	3/6	6.4 [MLE]	9.1E-04	2.2E-04			Yes	No
	OMW-1C	1/6	6.9 [1]	9.9E-04	2.5E-04			Yes	No
	OMW-1D	3/9	6.6 [ROS]	9.4E-04	2.3E-04			Yes	No
	OMW-2C	10/10	47.2 [Std}	6.7E-03	1.7E-03			Yes	Yes
Lead	OMW-1C	1/6	23.1 [1]			15 ppb*	Action Level	Yes	Yes
	OMW-1D	8/9	12.7 [KM]					Yes	Yes
Lithium	OMW-1A	6/6	186.0 [Std]	2.6E-02	6.6E-03	2.0E-03	PPRTV	Yes	Yes
	OMW-1AA	6/6	272.5 [Std]	3.8E-02	9.7E-03			Yes	Yes
	OMW-1B	6/6	705.0 [Std]	1.0E-01	2.5E-02			Yes	Yes
	OMW-1C	6/6	305.8 [Std]	4.3E-02	1.0E-02			Yes	Yes
	OMW-1D	9/9	3584.4 [Std]	5.1E-01	1.3E-01			Yes	Yes
	OMW-2A	6/7	22.2 [KM]	3.2E-03	7.9E-04			Yes	No
	OMW-2AA	6/6	38.7 [Std]	5.5E-03	1.4E-03			Yes	No
	OMW-2B	6/6	82.5 [Std]	1.2E-02	2.9E-03			Yes	Yes
	OMW-2C	10/10	1223.0 [Std]	1.7E-01	4.3E-02			Yes	Yes
	OWM-2D	6/6	839.7 [Std]	1.2E-01	2.0E-02			Yes	Yes
	OMW-3A	6/6	13.1 [Std]	1.9E-03	4.7E-04			No	No
	OMW-3B	7/8	16.5 [KM]	2.3E-03	5.9E-04			Yes	No
	OMW-3C	6/6	40.5 [Std]	5.7E-03	1.4E-03			Yes	No
	OMW-4A	8/8	46.2 [Std]	6.6E-03	1.6E-03			Yes	No
	OMW-4B	5/6	35.2 [Std]	5.0E-03	1.2E-03			Yes	No
	OMW-4C	6/6	36.3 [Std]	5.2E-03	1.2E-03			Yes	No
	RWA-58	7/7	48.5 [Std]	6.9E-03	1.7E-03			Yes	No
	RWA-59	7/7	30.5 [Std]	4.3E-03	1.0E-03			Yes	No
	RWA-76	13/13	103.5 [Std]	1.5E-02	3.9E-03			Yes	Yes
	RWA-79	1/1	35.0 [1]	5.0E-03	1.2E-03			Yes	No

Substance Name	stance Name Well Number of Co Station Detected/Analyzed c		Mean Screening Concentration (ppb) [method calculated]	Estimated Exposure Dose (mg/kg/day)		xposure g/day) Non-cancer Screening Guideline (mg/kg/day)		Does the Estimated Exposure Dose Exceed the Non-cancer Screening Guideline?	
				Infant	Adult			Infant	Adult
	RWA-81	7/7	39.3 [Std]	5.6E-03	1.4E-04			Yes	No
	RWA-94	1/1	15 [1]	2.1E-03	5.3E-04			Yes	No
Manganese	OMW-3B	8/8	386.1 [Std]	5.5E-02	1.4E-02	5.0E-02	RfD	Yes	No
	OMW-3C	6/8	403.8 [Std]	5.7E-02	1.4E-02			Yes	No
Selenium	OWM-1D	9/14	11.4 [MLE]	1.6E-03	4.1E-04	5.0E-03	MRL	No	No
Strontium	OMW-1D	9/9	6310 [Std]	9.0E-01	2.2E-01	6.0E-01	RfD	Yes	No
	OMW-2D	6/6	6601 [Std]	9.4E-01	2.4E-01			Yes	No
Thallium	OMW-1A	2/6	0.6 [1]	8.0E-05	1.9E-05	1.0E-05	PPRTV	Yes	Yes
	OMW-1C	2/6	2.8 [1]	4.0E-04	9.9E-05			Yes	Yes
	OMW-1D	1/9	10.4 [1]	1.5E-04	3.7E-04			Yes	Yes
	OMW-2C	1/10	0.5 [1]	7.3E-05	1.8E-05			Yes	Yes
Uranium	OMW-1D	7/9	22.3 [KM]			30 ppb*	MCL	No	No
Inorganic Parameters									
Fluoride	OMW-1A	4/4	1050.7 [Std]	1.5E-01	3.7E-02	5.0E-02	MRL	Yes	No
	OMW-1AA	4/4	499 [Std]	7.1E-02	1.8E-02			Yes	No
	OMW-1B	4/4	5892 [Std]	8.4E-01	2.1E-01			Yes	Yes
	OMW-1C	4/4	3345 [Std]	4.8E-01	1.2E-01			Yes	Yes
	OMW-1D	6/6	1796 [Std]	2.6E-01	6.4E-02			Yes	Yes
	OMW-2B	4/4	6100 [Std]	8.7E-01	2.2E-01			Yes	Yes
	OMW-2C	6/6	3190 [Std]	4.5E-01	1.1E-02			Yes	No
	OMW-2D	4/4	885 [Std]	1.3E-01	3.1E-02			Yes	No
	OMW-3C	4/4	2660 [Std]	3.8E-01	9.4E-02			Yes	Yes
	OMW-4B	4/4	321 [Std]	4.6E-02	1.1E-02			No	No
	OMW-4C	4/4	1483 [Std]	2.1E-01	5.3E-02			Yes	Yes
	RWA-58	5/5	345 [Std]	4.9E-02	1.2E-02			No	No
	RWA-76	9/9	865 [Std]	1.2E-01	3.1E-02			Yes	No
	RWA-81	5/5	909 [Std]	1.3E-01	3.2E-02			Yes	No



Substance Name	Well Station	Number of Samples Detected/Analyzed	Mean Screening Concentration (ppb) [method calculated]	Estimated Exposure Dose (mg/kg/day) Infant Adult		Estimated Exposur Dose (mg/kg/day)		Estimated Exposure Dose (mg/kg/day)		3 Estimated Exposi Dose (mg/kg/da		Non-cancer Screening Guideline (mg/kg/day)	Source	Doe Estin Exposu Exce Non-o Scre Guida	s the nated re Dose ed the cancer ening eline?
									Adult						
Organics															
Benzene	OWM-1D	7/7	0.68 [Std]	9.8E-05	2.4E-05	5.0E-04	MRL	No	No						
	OWM-2D	3/4	1.9 [KM]	2.7E-04	6.8E-05			No	No						
Bromodichloromethane	OMW-4B	1/4	0.74 J [1]	1.0E-04	2.6E-05	2.0E-02	MRL	No	No						
1,2-Dichloroethene, cis	OMW-1B	1/5	80.8 [1]	1.1E-02	2.9E-03	2.0E-3	RfD	Yes	Yes						
Trichloroethylene	OMW-1B	1/5	81.1 [1]	1.1E-02	2.9E-03	5.0E-04	RfD	Yes	Yes						
Vinyl Chloride	OMW-1B	1/5	2.6 [2]	3.7E-04	9.4E-05	3.0E-03	MRL	No	No						

ppb – part per billion = micrograms per liter ( $\mu$ g/L)

mg/kg/day - milligram per kilogram per day

J - estimated quantitation

Methods used to calculate mean screening concentration. The mean screening concentrations are rounded.

Std - Standard methods for calculating a mean using detected values only for a data set.

KM - Kaplan-Meier nonparametric method for calculating mean of a data set with nondetects.

ROS - Regression on order statistics for data that includes nondetects.

MLE - Maximum likelihood estimation for data that includes nondetects.

1 - Mean not estimated because number of results is less than 3. Mean screening concentration based on maximum concentration detected.

MRL - minimal risk level (ATSDR)

RfD - reference dose (EPA)

MCL – Maximum Contaminant Level (EPA)

PPRTV - provisional peer review toxicity value derived by EPA Superfund Health Risk Technical Support Center

Bold indicates estimated exposure dose exceeds the non-cancer screening guideline.

\* - ATSDR used a groundwater concentration (ppb) as the Non-cancer Screening Guideline instead of a dose (mg/kg/day).

 Table B2. Estimated Excess Cancer Risk Compared to Cancer Screening Guideline for Chemicals in Off-Site Melton Valley Groundwater with

 Concentrations Higher than Comparison Values

Substance Name	Well Station	Mean Screening Concentrations (ppm)	U.S. EPA's Oral Cancer Slope Factor	Excess Ca	ncer Risk	Does the Estimated Cancer Risk Exceed the Cancer Screening Guideline (10 <sup>-4</sup> )?					
		[method calculated]	$(mg/kg/day)^{-1}$	Infant Adult Inj		Infant	Adult				
Melton Valley											
Inorganics											
Arsenic	OMW-1D (max mean)	16.2 J [Std]	1.5	1.3E-06	3.1E-06	No	No				
Organics	· · ·					·					
Benzene	OMW-1D	[Std]	5.5E-02	5.3E-08	1.3E07	No	No				
	OMW-2D	[KM]		1.3E-07	5.9E-08	No	No				
Bromodichloromethane	OMW-4B	0.68 0.74 [1]	6.2E-02	5.8E-08	1.4E-07	No	No				
Trichloroethene	OMW-1B	1.9 81.1 [1]	4.6E-02	6.3E-06	1.2E-05	No	No				
Vinyl Chloride	OMW-1B	2.63 [1]	1.4	2.1E-07	4.0E-07	No	No				

ppb – part per billion = micrograms per liter ( $\mu$ g/L)

mg/kg/day = milligram per kilogram per day

J - estimated quantitation

Methods used to calculate second-tier screening concentrations. The mean screening concentrations are rounded.

Std - Standard methods for calculating a mean using detected values only for a data set.

KM - Kaplan-Meier nonparametric method for calculating mean of a data set with nondetects.

1 - Mean not estimated because number of results is less than 3 samples. Second-tier screening concentration based on maximum concentration detected.

Risk was calculated by multiplying the estimated exposure dose for an infant and adult by EPA's oral cancer slope factor.

Cancer Screening Guideline  $(10^{-4})$  - Estimated cancer risk of 1 in 10,000, which means out of 10,000 people exposed one additional cancer might occur. Bold indicates estimated excess cancer risk exceeds the cancer screening guideline.



Table B3. Estimated Chronic Exposure Doses Compared to Noncancer Health-based Screening Guideline Values for Chemicals in Off-SiteGroundwater in Bethel Valley with Concentrations Higher than ATSDR Comparison Values

Substance Name	Well Station	Number of Samples Detected/Analyzed	Mean Screening Concentration (ppb) [method	Estimated Exposure Dose (mg/kg/day)		Non-cancer Screening Guideline (mathadary)	Source	Does the E Exposure Do the Non- Screening G	stimated se Exceed cancer fuideline?			
			calculated]	Infant	Adult	( <i>mg/kg/aay</i> )		Infant	Adult			
	Bethel Valley											
Metals												
Arsenic	RWA-22	1/1	1.3 J [1]	1.9E-04	4.6E-05	3.0E-04	MRL	No	No			
Chromium	RWA-104	1/1	29 [1]	4.1E-03	1.0E-03	9.0E-04	MRL	Yes	Yes			
Lithium	RWA-22	1/1	28 [1]	3.9E-03	9.9E04	2.0E0-3	PPRTV	Yes	No			
	RWA-97	10/10	49.6 [Std]	9.0E-03	1.8E-03			Yes	No			
	RWA-104	1/10	170 [1]	2.4E-0-2	6.0E-03			Yes	Yes			
Inorganic Parameters												
Fluoride	RWA-97	8/10	1350 [KM]	1.9E-01	4.8E-02	5.0E-02	MRL	Yes	No			
	RWA-102	1/1	530 [1]	7.6E-02	1.9E-02			Yes	No			
	RWA-104	1/1	2400 [1]	3.4E-01	8.5E-02			Yes	Yes			
Organics												
Benzene	RWA-104	3/3	18.91 [Std]	2.7E-03	6.7E-04	5.0E-04	MRL	Yes	Yes			
Bromodichloromethane	RWA-104	1/1	193 [1]	2.7E-02	6.9E-03	2.0E-02	MRL	Yes	No			
Bromoform	RWA-104	1/1	86.3 [1]	1.2E-02	3.1E-03	2.0E-02	MRL	No	No			
Carbon tetrachloride	RWA-104	1/1	5.19 <sub>[1]</sub>	7.4E-04	1.8E-04	4.0E-03	RfD	No	No			
Chloroform	RWA-104	3/3	2001.3 [Std]	2.8E-01	7.1E-02	1.0E-02	MRL	Yes	Yes			
Dibromochloromethane	RWA-104	1/1	99.7 <sub>[1]</sub>	1.4E-02	3.5E-03	9.0E-02	MRL	No	No			
1,2-Dichloroethane	RWA-104	1/1	1.73 [1]	2.5E-04	6.1E-05	6.0E-03	RfD	No	No			
1,1,2-Trichloroethane	RWA-104	1/1	5.43 [1]	7.7E-04	1.9E-04	4.0E-03	RfD	No	No			
1,2,3-Trichloropropane	RWA-104	1/1	1.47 [1]	2.1E-04	5.2E-05	4.0E-03	RfD	No	No			

 $ppb - part per billion = micrograms per liter (\mu g/L) mg/kg/day = millig$ 

mg/kg/day = milligram per kilogram per day

J - estimated quantitation

Methods used to calculate mean screening concentrations.

Std Standard methods for calculating a mean using detected values only for a data set.

KM Kaplan-Meier nonparametric method for calculating mean of a data set with nondetects.

1 Mean not estimated because number of results is less than 3. Mean screening concentration based on maximum concentration detected.

MRL = minimal risk level (ATSDR) RfD = reference dose (EPA)

PPRTV = provisional peer review toxicity value by EPA Superfund Health Risk Technical Support Center

Bold indicates estimated doses exceeds the non-cancer screening guideline.

Table B4. Estimated Excess Cancer Risk Compared to Cancer Screening Guideline for Chemicals in Off-Site Bethel Valley Groundwater withConcentrations Higher than ATSDR Comparison Values

Substance Name	Well Station	Mean Screening Concentrations (ppm)	EPA's Oral Cancer Slope Factor (mg/kg/day) <sup>-1</sup>	Excess Ca	uncer Risk	Does the Estimated Excess Cancer Risk Exceed the Cancer Screening Guideline $(10^{-4})$ ?					
		[methoa caiculatea]		Infant	Adult	Infant	Adult				
	Bethel Valley										
Inorganics											
Arsenic	OWM-104	1.3 J [1]	1.5	9.E-07	2.5E-07	No	No				
Organics											
Benzene	OWM-104	18.9 [Std]	5.5E-02	1.5E-06	3.7E-06	No	No				
Bromodichloromethane	OWM-104	193 [1]	6.2E-02	1.0E-05	3.7E-05	No	No				
Bromoform	OWM-104	86.3 [1]	7.9E-03	6.7E-06	1.7E-05	No	No				
Carbon Tetrachloride	OWM-104	5.19 [1]	7.0E-02	4.1E-07	1.0E-06	No	No				
Chloroform	OWM-104	2001.3 [Std]	3.1E-02	1.6E-04	3.9E-04	Yes	Yes				
Dibromochloromethane	OWM-104	99.7 [1]	8.4E-02	7.8E-06	1.9E-05	No	No				
1,2-Dichloroethane	OWM-104	1.73 [1]	9.1E-02	1.4E-07	3.3E-07	No	No				
1,1,2-Trichloroethane	OWM-104	5.43 [1]	5.7E-02	4.2E-07	1.1E-06	No	No				
1,2,3-Trichloropropane	OWM-104	1.47 [1]	3.0E+01	1.1E-07	2.8E-07	No	No				

ppb – part per billion = micrograms per liter ( $\mu$ g/L)

mg/kg/day = milligram per kilogram per day

J - estimated quantitation

Methods used to calculate second-tier screening concentrations. The mean screening concentrations are rounded.

Std - Standard methods for calculating a mean using detected values only for a data set.

1 - Mean not estimated because number of results is less than 3 samples. Second-tier screening concentration based on maximum concentration detected.

Risk was calculated by multiplying the estimated exposure dose for an infant and adult by EPA's oral cancer slope factor.

Cancer Screening Guideline  $(10^4)$  - Estimated cancer risk of 1 in 10,000, which means out of 10,000 people exposed one additional cancer might occur. Bold indicates estimated excess cancer risk exceeds the cancer screening guideline.

