Health Consultation

Analysis of Nitrate and Metals Sampling Data

EAST 67[TH] STREET GROUNDWATER PLUME

ODESSA, ECTOR COUNTY, TEXAS

EPA FACILITY ID: TXN000606614

MARCH 6, 2008

U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES
Public Health Service
Agency for Toxic Substances and Disease Registry
Division of Health Assessment and Consultation
Atlanta, Georgia 30333

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

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Prepared By:

Texas Department of State Health Services
Epidemiology & Disease Surveillance Unit
Health Assessment & Toxicology Group
Under a cooperative agreement with the
Agency for Toxic Substances and Disease Registry

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Purpose and Statement of Issues

The East 67th Street Groundwater Plume site is located north of the city limits of Odessa in Ector County, Texas [1]. A Public Health Assessment was prepared in response to groundwater contamination, which was initially detected in one of four Public Water Supply (PWS) wells that service the Devilla Mobile Home Park on East 67th Street [2, 3]. The groundwater contamination consisted of tetrachloroethene (also known as perchloroethene and/or PCE), trichloroethene (TCE), and their associated degradation products: *cis*-1,2-dichloroethene (DCE); 1,1-DCE; 1,2-dichloroethane (DCA); and 1,1-DCA. Note: a list of abbreviations is included as Appendix A.

Subsequent sampling was conducted by the United States Environmental Protection Agency (EPA) in January 2007. Sample results indicated nitrate concentrations above the Maximum Contaminant Level (MCL)[1] of 10 milligrams per liter (mg/L). Metals, including arsenic, lead, selenium, thallium, and vanadium, were elevated above their respective Health Assessment Comparison (HAC) values and warranted further review.

The Texas Department of State Health Services (DSHS) and the Agency for Toxic Substances and Disease Registry (ATSDR) reviewed the environmental information available for the site. We also evaluated the exposure pathways through which the public could contact nitrate and metals from the site. Residents who use water from the wells with high nitrates or metals would be exposed.

The Texas Commission on Environmental Quality (TCEQ) sent letters notifying residents within a half-mile radius of elevated nitrates, and DSHS incorporated a public health message to prevent exposure for sensitive populations. The purpose of this Health Consultation is to present the nitrate information (See Appendix B) and to evaluate the sampling data and determine if exposure to the levels of metals in the drinking water wells are harmful to human health.

Background

Site Description

The East 67th Street Groundwater Plume is located just north of the city limits of Odessa, Ector County, Texas. The drinking water for residential and commercial/industrial properties in the area is obtained from the Trinity Aquifer. Thirty-one drinking water wells, which are located within a one-mile radius of the center of the plume, were identified with known contamination [2]. Water wells in the area have a terminal depth of approximately 150 feet, with a screened interval from 70 to 150 feet below grade surface. The center of the plume is located at the intersection of East 67th Street and Stevenson Avenue [2, 3]. See Figure 1.

[1] The MCLs are enforceable standards determined by EPA that take into account technical feasibility and potential health risks.

Site History

PCE, TCE, and cis-1,2-DCE were detected in the PWS of Devilla Mobile Home Park in 2005 during regular monitoring by the TCEQ. Additional sampling was conducted, and filtration systems were installed to mitigate exposure to chlorinated solvents, where necessary. The East 67th Street Groundwater Plume site was proposed to the National Priority List (NPL) on September 27, 2006, based on chlorinated hydrocarbon contamination.

In January 2007, drinking water wells were sampled, and samples were analyzed for nitrates and metals. The nitrates and metals were analyzed so the EPA can characterize the saturated zone and determine the best remediation strategy. Based on the sample results, nitrates were above the MCL of 10 mg/L in 18 of the 30 wells sampled. Per Texas State House Bill 3030, residents within 0.5 miles of the sample area were notified of the nitrates and informed of ways to prevent exposure in sensitive populations. The close proximity of drinking water wells to private septic systems is the likely source of nitrate contamination [4]. Nitrate information is provided as Appendix B.

Methods

To assess the potential health risks that may be associated with the contaminants found on a site, we compare contaminant concentrations with their media specific health assessment comparison (HAC) values for non-cancer and cancer endpoints. These values are guidelines that specify levels of chemicals in specific environmental media (air, soil, and water) that are considered safe for human contact with respect to identified human endpoints. Non-cancer screening values are generally based on ATSDR's minimal risk levels (MRLs)[2] and EPA's reference doses (RfDs)[3]. Both are based on the assumption that there is an identifiable exposure threshold (both for the individual and for populations) below which there are no observable adverse effects. Thus, MRLs and RfDs are estimates of daily exposures to contaminants that are unlikely to cause adverse non-cancer health effects even if exposure occurs for a lifetime. The HAC values used to evaluate cancer: the cancer risk evaluation guides (CREGs)[4], are based on EPA's chemical-specific cancer slope factors (CSFs)[5] and an estimated excess lifetime risk of developing cancer of one in one million persons exposed for a lifetime. The environmental media evaluation guides (EMEGs) are used as a screening tool to compare site specific soil, water, and/or air concentrations. The EMEGs are derived from the chemical's toxicity and default exposure criteria.

[2] An MRL is a contaminant specific exposure dose below those which might cause adverse health effects in the people most sensitive to such chemical-induced effects. MRLs generally are based on the most sensitive chemical-induced end point considered to be of relevance to humans.

[3] An RfD is an estimate (with a level of uncertainty from 10 to 1,000 times below the level of harmful effects) of a daily human exposure (including sensitive groups) that is likely to be without appreciable risk of deleterious effects during a lifetime.

[4] A CREG is the concentration of a chemical in specific media (air, soil, or water) corresponding to an excess estimated lifetime cancer risk of one in one million (1 in 1,000,000) persons exposed for a lifetime.

[5] A CSF is the upper 95th percentile confidence limit of the slope of the dose-response curve and is expressed in unit of measure of $(mg/kg\text{-}day)^{-1}$.

Exceeding either a non-cancer or a cancer screening value does not necessarily mean that the contaminant will cause harm; rather it suggests that potential exposure to the contaminant warrants further consideration.

In 1974, the U.S. Congress passed the Safe Drinking Water Act which required that EPA determine safe levels of chemicals in public drinking water. EPA has set the MCL for nitrate at 10 mg/L. The MCLs are enforceable standards that take into account technical feasibility and potential health risks [5].

Sample data were provided by the EPA. DSHS did not review the analytical packet to determine laboratory adherence to Quality Assurance/Quality Control (QA/QC) protocols. Rather, DSHS is depending on EPA's review of QA/QC protocols to be accurate and committed to good common practice.

Environmental Sampling

In January 2007, 46 private drinking water wells and four PWS wells were sampled for metals: 30 of the wells were sampled for nitrates. Information pertaining to nitrates is available in Appendix B. Several parameters, including arsenic, selenium, and thallium were analyzed at method detection limits (MDLs) well above their respective MCLs of 2 micrograms per liter (µg/L), 50 µg/L, and 10 µg/L, respectively. Thallium and selenium were identified above the MDLs. Because of the high metal concentrations, some samples were reanalyzed for thallium and selenium at lower MDLs to determine the extent of elevated metals, and all samples were reanalyzed for arsenic [6]. Other metals, including lead and vanadium, were detected above their respective screening values.

Discussion

Arsenic, lead, thallium, and vanadium exceeded one or more screening value. Concentrations of these metals were further evaluated to determine if adverse health effects are anticipated.

<u>Arsenic</u>

Arsenic was detected in 35% of the sampled wells, and detected concentrations ranged from 7.3 µg/L to 14.3 µg/L. An average of 5.6 µg/L was calculated by using half of the method detection limit (MDL, 6.8 µg/L) in place of non-detected values, which biases the average high to be more conservative. These concentrations exceed the CREG (0.02 µg/L), the children's chronic EMEG (3 µg/L), and in some cases the adult chronic EMEG (10 µg/L).

To further evaluate the possible adverse health effects caused from ingesting water with arsenic, an exposure dose was estimated. The maximum observed concentration (14.3 µg/L) and the following default parameters were used to calculate the dose: intake rate of water for adults, two liters of water per day (L/day); intake rate of water for children, 1 L/day; availability factor, 1;

exposure frequency, 1 to reflect daily exposure; adult body weight, 70 kg; and child body weight 16 kg.

The estimated doses for adults (0.00041 mg/kg/day) and for children (0.00089 mg/kg/day) were less than the established MRL (0.005 mg/kg/day) for acute (0 to 14 days) exposure but slightly higher than the MRL (0.0003 mg/kg/day) for chronic (more than 365 days) exposure. Estimated dose parameters and results are shown on Table 1.

The MRL for chronic exposure is based on a study conducted in Taiwan where farmers were exposed to high levels of arsenic in their well water. The most common adverse health effect is the formation of dermal lesions (thickened and darkened patches of skin) [7]. A No Observed Adverse Effect Level (NOAEL) of 0.0008 mg/kg/day was established by the study data, and the chronic MRL is based on this value with a safety factor of three for human variability. The worst case scenario was used to estimate exposure, by assuming that residents would be exposed to the highest observed arsenic concentration at the site (14.3 µg/L) and by using default body weights for adults and children that very conservative (equal to 154 and 35 pounds, respectively). Residents are more likely to be exposed to less than the estimated average concentration of 5.6 µg/L, at which concentration the estimated exposure doses are well below the NOAEL. Because the residents are not likely to be exposed to the maximum concentration and because the default body weights are very conservative, it is unlikely that non-cancerous health effects will occur.

An estimated lifetime cancer risk was calculated using the maximum arsenic concentration, a residency of 30 years, and default body weight. Based on the calculations, the consumption of water with the highest known arsenic concentration would result in “low increased risk” of cancer. Again, this is the worst case scenario. Residents are more likely to be exposed to the average arsenic concentration identified in onsite wells. When exposed to the average arsenic concentration, the estimated lifetime cancer risk indicates “no apparent increased risk” of cancer from arsenic exposure. Additionally, the residency time of 30 years is over-estimated. The high metals data are likely due to changes in the groundwater caused by changes in chemical electrical potentials from breakdown of the chlorinated solvent plume [8]. Estimated lifetime cancer risk calculations showing the average exposure and over-estimated exposure period are shown on Table 2.

Lead

One of the sampled wells had a lead concentration (39.2 µg/L) above the US EPA action level of 15 µg/L. The action level is intended to evaluate public water supply systems. If 10% of homes in a public water supply system have lead levels above the action level, then a preventative action, such as decreasing the corrosivity of the water is required. The action level does not apply to private systems. There are no other health-based screening levels.

Estimated blood lead levels indicate no significant increases when the highest lead concentration and standard slope factors for children (0.03 µg lead/dL blood per µg lead/L water), adult females (0.03 µg lead/dL blood per µg lead/L water), and adult males (0.06 µg lead/dL blood per µg lead/L water) were used in calculations (Table 3). The Centers for Disease Control and Prevention (CDC) has determined that a child’s blood lead level above 10 µg lead/dL blood is considered elevated [9]. Based on the estimated increase in blood lead levels, the lead in water should not result in adverse health effects for children or adults.

Thallium

Thallium was detected in three wells at concentrations of 112 µg/L, 132 µg/L, and 107 µg/L. These values and the MDL used (100 µg/L) exceed the MCL of 2 µg/L and the EPA's lifetime health advisory for drinking water (LTHA) of 0.5 µg/L. When nearby wells were sampled for thallium using a lower MDL of 1.2 µg/L, thallium was not detected in any of the nearby wells.

In pure form, thallium is a bluish-white metal, and it is most commonly used to manufacture electronic devices, switches and closures [10]. Historically, thallium was a common component of rat poison, before it was banned in 1977. Since 1984, all thallium in the United States has been imported. The most likely sources of thallium include smelters, power plants, and cement factories. Thallium is easily taken up by plant roots.

When thallium enters the body, it goes to the kidneys and liver. Approximately half of the thallium ingested will be excreted in urine, and to a lesser extent, feces within three days.

No data is available to determine the health effects of low doses (less than 1 g) of thallium exposure over a long period of time. High doses of thallium over a short period of time can result in adverse health effects on the liver, nervous system, lungs, heart, and kidneys [10]. This is based on observations after a single dose of thallium (estimated 54-110 mg/kg). A NOAEL of 0.2 mg/kg/ day for 90 days was observed in rats. During this exposure period, no respiratory, gastrointestinal, liver, kidney, or cardiovascular effects were observed. Hair loss has been reported in humans exposed to high concentrations of thallium for short periods of time, and exposure greater than or equal to 1.2 mg/kg/day for 15 weeks resulted in hair loss in rats. There are no reliable data to determine the effects of thallium on unborn babies. However, thallium does cross the placenta, and studies in pregnant rats indicated that 0.08 mg/kg/day was the LOAEL with respect to affecting the learning ability in offspring. No dose-response curve was noticed at higher concentrations, and no structural changes were observed. Reproductive effects, including change in testicular morphology and function, were observed in rats given 0.7 mg/kg/day thallium for 60 days.

There is no information for long term exposure to thallium in humans or animals. Exposure doses were calculated for children and adults, using default parameters for water intake (1 L/day and 2 L/day, respectively), weight (16 kg and 70 kg, respectively), and the highest thallium concentration (132 µg/L). Exposure doses were estimated at 0.0083 mg/kg/day and 0.0038 mg/kg/day (Table 4). Based on the information from animal studies, the estimated exposure doses at the "worst case scenario" concentration are unlikely to cause adverse health effects for short term or intermediate exposure periods. The health effects of thallium over long periods of time are not known.

Vanadium

Vanadium was detected in 42% (21) of the sampled wells, and detected concentrations ranged from 32.6 µg/L to 81.5 µg/L with an average of 28.4 µg/L when half of the detection limit is used in place of non-detected values. The detected concentrations exceed the children's health based screening level for intermediate (more than 14, but less than 365 days) exposure of 30 µg/L. The adult intermediate EMEG is 100 µg/L.

Using the maximum concentration, an exposure dose was estimated using the following default parameters: intake rate of water for adults, two liters of water per day (L/day); intake rate of water for children, 1 L/day; availability factor, 1; exposure frequency, 1 to reflect daily exposure; adult body weight, 70 kg; and child body weight 16 kg. The estimated dose for adults (0.00233 mg/kg/day) was less than the established MRL for intermediate exposure (0.003 mg/kg/day). The estimated dose for children (0.00509 mg/kg/day) was slightly higher than the MRL. No chronic MRL was available for comparison. Estimated dose parameters and results are shown on Table 5.

Vanadium is a naturally occurring white to grey metal [11]. The intermediate MRL is based on studies in mice which received vanadium as sodium metavanadate ($NaVO_3$) in drinking water for three months, and 0.3 mg/kg/day was the LOAEL at which renal effects (hemorrhagic foci) were observed. The MRL incorporates uncertainty factors of 10 for animal to human extrapolation and 10 for human variability. The estimated dose was only slightly above the MRL, and two orders of magnitude were considered when deriving the MRL. Based on this information, it is unlikely that adverse health effects will occur for intermediate exposure. No information is available to determine the adverse health effects of chronic vanadium exposure.

Child Health Considerations

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

Default parameters for children, including body weight and water intake rate, were used to calculate estimated exposure doses.

Conclusions

The available data indicate that arsenic, lead, thallium, and vanadium were detected in private drinking water wells at concentrations above their respective screening values. It is unlikely that arsenic and lead found in the well water will cause adverse health effects. However, the health effects of chronic thallium and vanadium exposure are not known. Based on this information, the metals identified in the drinking water wells pose an **indeterminate public health hazard**.

Recommendations

Because the future exposure to drinking water poses an **indeterminate public health hazard**, obtaining an alternative water source would be most protective of public health. The US EPA is currently working with the City of Odessa to provide water to the residents to prevent exposure to contaminants associated with the chlorinated solvent plume also affecting the area.

Public Health Action Plan

DSHS and TCEQ sent a letter dated April 17, 2007 to residents that notified them of the nitrates identified in private drinking water wells in the area. In the letter, residents were informed of the possible health effects and of ways to prevent exposure.

To prevent long-term exposure to contaminants for all residents, DSHS will contact the US EPA to insure that all residents have connected to the alternative water source.

Authors, Technical Advisors

Michelle N. Bost, MS, CHMM
Environmental Specialist
DSHS Health Assessment & Toxicology Program

Susan Prosperie, MS, RS
Manager
DSHS Exposure Assessment & Surveillance Group

John Villanacci, PhD, NREMT-I
Principal Investigator/Director
DSHS Environmental & Injury Epidemiology and Toxicology Branch

Jennifer Lyke
Regional Representative
ATSDR Region 6 - Dallas, Texas

George Pettigrew, PE
Senior Regional Representative
ATSDR Region 6 - Dallas, Texas

Jeffrey Kellam
Environmental Health Scientist
Division of Health Assessment and Consultation
Superfund and Program Assessment Branch
ATSDR - Atlanta, Georgia

Certification

This health consultation for the East 67th Street Groundwater Plume site located in Odessa, Ector County, Texas was prepared by the Texas Department of State Health Services (DSHS) under a cooperative agreement with the Agency for Toxic Substances and Disease Registry (ATSDR) in accordance with approved methodologies and procedures existing at the time this health consultation was initiated. Editorial review was completed by the Cooperative Agreement partner.

Technical Project Officer, CAT, CAPEB, DHAC, ATSDR

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

Team Lead, CAT, CAPEB, ATSDR

References

1. http://maps.odessa-tx.gov
2. Texas Commission on Environmental Quality. Hazard Ranking System Documentation Record. East 67th Street Ground Water Plume. September 2006.
3. Texas Commission on Environmental Quality. Screening Site Inspection Report. East 67th Street Ground Water Plume. February 2006.
4. Texas Department of State Health Services. Record of Communication. Phone conversation with Vincent Malott, US EPA Remedial Project Manager, February 1, 2007.
5. US Environmental Protection Agency. Groundwater and drinking water. Available from URL: http://www.epa.gov/safewater/mcl.html
6. Texas Department of State Health Services. Record of Communication. Email from Vincent Malott, US EPA Remedial Project Manager, July 2, 2007.
7. Agency for Toxic Substances and Disease Registry. Toxicological Profile for Arsenic. Atlanta: ATSDR, September 2000.
8. Texas Department of State Health Services. Record of Communication. Email from Vincent Malott, US EPA Remedial Project Manager, April 26, 2007.
9. Agency for Toxic Substances and Disease Registry. Toxicological Profile for Lead. ATSDR. September 2005.
10. Agency for Toxic Substances and Disease Registry. Toxicological Profile for Thallium. ATSDR. July 1992.
11. Agency for Toxic Substances and Disease Registry. Toxicological Profile for Vanadium. ATSDR. July 1992.

Tables and Figure

Table 1: Estimated Arsenic Exposure Doses

ATSDR Chronic Oral MRL:	0.0003	mg/kg/day
	adults	**children**
Dose=C*IR*AF*EF/BW (mg/kg/day)	0.00041	0.00089
C=contaminant concentration (mg/L)	0.0143	0.0143
IR=intake rate of water (L/day)	2	1
AF=bioavailability factor (%, assumed 100% or 1)	1	1
EF=exposure factor (unitless)	1	1
BW=body weight (kg)	70	16

Table 2: Estimated Lifetime Cancer Risk due to Arsenic Exposure

ER=estimated theoretical risk=CSF*dose	**3.69E-05**
dose=C*IR*EF/BW	0.0000684
C=average contaminant concentration (mg/L)	0.00560
IR=intake rate of water (L/day)	2
EF=exposure factor (unitless)	0.42739726
years of residence	30
days per week	7
weeks per year	52
years in a lifetime	70
days in a year	365
BW=body weight (kg)	70
CSF=cancer slope factor (mg/kg/d)-1	0.54

Table 3: Estimated Annual Blood Lead Level Increases

Sample Type	Lead Maximum Concentration (µg/L)	Annual Exposure Factor	Blood Slope Factor (µg/dL/µg/L)	Blood Lead Increase (µg/dL)
1 day a week				
water (child)	39.6	0.14	0.03	0.166
water (adult M)	39.6	0.14	0.06	0.333
water (adult F)	39.6	0.14	0.03	0.166
2 days a week				
water (child)	39.6	0.28	0.03	0.333
water (adult M)	39.6	0.28	0.06	0.665
water (adult F)	39.6	0.28	0.03	0.333
3 days a week				
water (child)	39.6	0.43	0.03	0.511
water (adult M)	39.6	0.43	0.06	1.022
water (adult F)	39.6	0.43	0.03	0.511
4 days a week				
water (child)	39.6	0.57	0.03	0.677
water (adult M)	39.6	0.57	0.06	1.354
water (adult F)	39.6	0.57	0.03	0.677
5 days a week				
water (child)	39.6	0.71	0.03	0.843
water (adult M)	39.6	0.71	0.06	1.687
water (adult F)	39.6	0.71	0.03	0.843
6 days a week				
water (child)	39.6	0.85	0.03	1.010
water (adult M)	39.6	0.85	0.06	2.020
water (adult F)	39.6	0.85	0.03	1.010
7 days a week				
water (child)	39.6	1	0.03	1.188
water (adult M)	39.6	1	0.06	2.376
water (adult F)	39.6	1	0.03	1.188

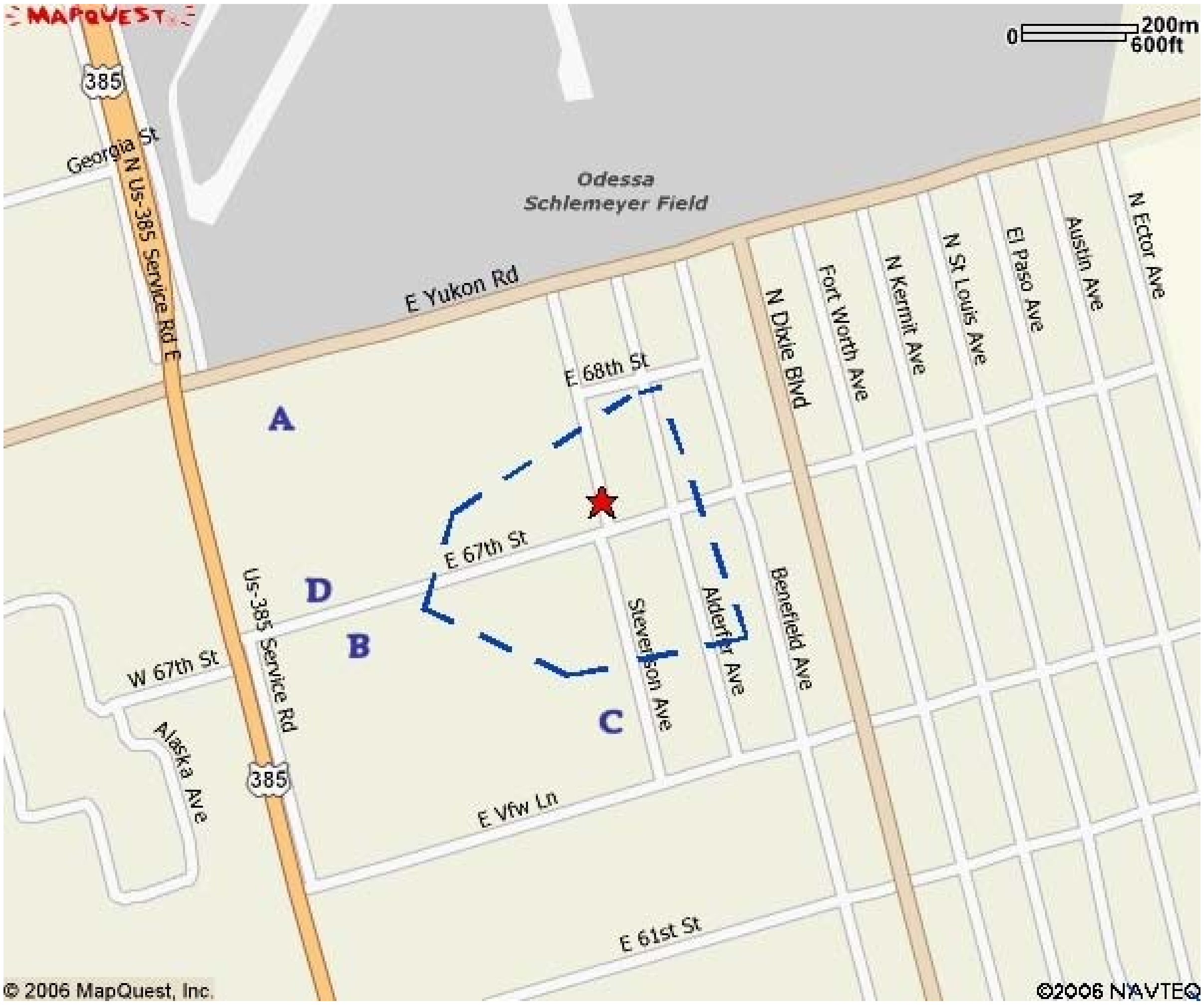
Table 4: Estimated Thallium Exposure Doses

	adults	children
Dose=C*IR*AF*EF/BW (mg/kg/day)	0.0038	0.0083
C=contaminant concentration (mg/L)	0.132	0.132
IR=intake rate of water (L/day)	2	1
AF=bioavailability factor (%, assumed 100% or 1)	1	1
EF=exposure factor (unitless)	1	1
BW=body weight (kg)	70	16

Table 5: Estimated Vanadium Exposure Doses

ATSDR Intermediate Oral MRL:	0.003	mg/kg/day
Dose=C*IR*AF*EF/BW (mg/kg/day)		
	adults	**children**
Average Dose	0.00081	0.00178
Highest Possible Dose	0.00233	0.00509
C_a=average contaminant concentration (mg/L)	0.0284	0.0284
C_{max}=maximum contaminant concentration (mg/L)	0.0815	0.0815
IR=intake rate of water (L/day)	2	1
AF=bioavailability factor (%, assumed 100% or 1)	1	1
EF=exposure factor (unitless)	1	1
BW=body weight (kg)	70	16

Figure 1 – Estimated Chlorinated Solvent Plume Location



Adapted from TCEQ Hazard Ranking System Documentation Record, 2006.

Legend:

★ - estimated center of the plume at East 67th Street and Stevenson Avenue

╱ - estimated plume boundaries, as determined by TCEQ

A – general location of Cotton Pipe

B – general location of CASE-Permian

C – general location of Devilla Mobile Home Park

D – general location of Brenntag

Figure 2 – Estimated Nitrate Plume

Appendices

Appendix A – Abbreviations

ATSDR	Agency for Toxic Substances and Disease Registry
bgs	Below grade surface
CERCLA	Comprehensive Environmental Response, Compensation and Liability Act (1980)
CREG	Cancer Risk Evaluation Guide
DCA	1,1- and/or 1,2-dichloroethane
DCE	*cis*- and/or *trans*-1,2-dichloroethene, *cis*- and/or *trans*-1,2-dichloroethylene
DHHS	Department of Health and Human Services
DSHS	Department of State Health Services
EMEG	Environmental Media Evaluation Guide
EPA	Environmental Protection Agency
g	Gram
HAC	Health Assessment Comparison value
HRS	Hazard Ranking System
IARC	International Agency for Research on Cancer
IRIS	Integrated Risk Information System
kg/day	Kilograms per day
L/day	Liters per day
LOAEL	Lowest Observed Adverse Effect Level
MCL	Maximum Contaminant Level
MDL	Method Detection Limit, used for laboratory analysis
µg/L	Micrograms of substance per liter
mg/L	Milligrams of substance per liter
mg/kg/day	Milligrams of substance per kilogram of body weight per day
MRL	Minimal Risk Level
ND	The analyte was not detected above the method detection limit
NOAEL	No Observed Adverse Effect Level
NPL	National Priorities List
NTP	National Toxicology Program
PCB	Polychlorinated biphenyls
PCE	Tetrachloroethene, perchloroethene
PHA	Public Health Assessment
ppb	Parts per billion
ppm	Parts per million
PVC	Polyvinyl chloride
PWS	Public Water System
QA/QC	Quality Assurance/Quality Control
RfD	Reference Dose
RMEG	Reference Dose Media Evaluation Guide
SSDAT	Superfund Site Discovery and Assessment Team
SARA	Superfund Amendments and Reauthorization Act (1986)
TCA	Trichloroethane
TCE	Trichloroethene, trichloroethylene
TCEQ	Texas Commission on Environmental Quality
VOC	Volatile Organic Compound

Appendix B – Nitrates

Nitrate (NO_3^-) and nitrite (NO_2^-) are naturally occurring inorganic ions that are a part of the global nitrogen cycle. They are the byproduct of waste breakdown when bacteria cause waste matter to decompose [12]. In addition, nitrates are commonly found as active ingredients in fertilizers, gunpowder, and in cured meats as a preservative. The decomposition of fertilizers and human or animal waste result in nitrate formation. Nitrates are highly water soluble and travel quickly from soil into groundwater.

Humans are exposed to nitrates in the diet from preserved meats, from vegetables (such as spinach, cauliflower, broccoli, and collard greens), and/or from ingestion of drinking water contaminated with nitrates. On average, vegetables account for greater than 70% of human nitrate intake [12].

When ingested, nitrate is distributed throughout the body before being absorbed into the large intestine. In the large intestine, microorganisms convert the nitrate to nitrite, which is then reabsorbed by the body via the bloodstream. Blood contains hemoglobin, which binds oxygen, and carries it throughout the body. Nitrite oxidizes the ferrous iron (Fe^{2+}) in hemoglobin to create ferric iron (Fe^{3+}). Ferric iron does not bind oxygen [12]. This condition is called acquired methemoglobinemia.

The liver converts nitrates to nitrite and other metabolites which are excreted in urine. Approximately 70% of the ingested nitrate leaves the body in urine within 24 hours [12]. Approximately 25% of the ingested nitrate is excreted in saliva [12]. Ingested nitrate that is not converted to nitrite can be eliminated without causing harmful effects [13].

Infants younger than four months are at the greatest risk of adverse health effects from ingestion of nitrate for several reasons. The pH of the gut in infants is higher than that of children and adults. The higher pH increases the numbers of bacteria which convert nitrate to nitrite. Hemoglobin in young infants partially consists of fetal hemoglobin, which is more easily oxidized by nitrites; additionally, infants younger than six months have low amounts of enzymes needed to convert methemoglobin back to hemoglobin [12, 14]. Most cases of methemoglobinemia occur in infants, as a result of preparing formula with contaminated well water [13].

Increased methemoglobin has been observed in pregnant women. At or near 30 weeks gestation, pregnant women might be more sensitive to induction of methemoglobin [12]. Some studies have associated high nitrate concentrations in drinking water with miscarriages [15]. Nitrate is not passed from the mother to infant in breast milk when drinking water contains 100 ppm or less nitrate [13, 16].

The effects of nitrate on blood result in additional effects on the body. Severe methemoglobin can result in respiratory effects such as shortness of breath (difficult or painful breathing) and rapid breathing, as well as respiratory tract irritation. The most common effect of methemoglobinemia is a chocolate-brown or slate-grey central cyanosis, commonly referred to as "blue baby syndrome" [12].

The RfD for nitrate is based on early clinical signs of methemoglobinemia in infants (0 to 3 months), who consume formula [17]. It is also based on 0.64 L/day water used to prepare formula for an infant weighing 4 kg or 8.8 pounds.

Nitrates were detected above the MCL in both the private and public drinking wells. The highest nitrate concentration at the site was 30.2 mg/L, based on the January 2007 sampling data. The site average was 15.4 mg/L. Exposure doses were estimated to determine at what nitrate concentrations adverse health effects were not anticipated. Infant weights in the 50th percentile were used in estimating the exposure doses for infants, and default values were used to estimate exposure doses in small children and adults.

Because the nitrate groundwater contamination was widespread (and to comply with Texas House Bill HB 3030), TCEQ sent letters to area residents to let them know that nitrate concentrations were elevated. DSHS included the following public health message in the letter:

> Based on current literature and estimates of exposure, bottled water should be used to mix infant formula and foods, and as a precaution children should drink bottled water if the nitrate concentration in drinking water is above 25 ppm (or mg/L). This is based on a 35-pound child drinking one liter of nitrate-contaminated water per day. Pregnant women should exercise caution and limit drinking water with an excess of 10 ppm nitrate.

Additional References

12. Agency for Toxic Substances and Disease Registry. Case Studies in Environmental Medicine. Revised January 2001.

13. Greer, Frank R., Michael Shannon, the Committee on Nutrition, and the Committee on Health. Infant Methemoglobinemia: The Role of Dietary Nitrate in Food and Water. Pediatrics, September 2005. Vol. 116(3): 784-786.

14. Knobeloch, Lynda, Barbara Salna, Adam Hogan, Jeffrey Postel, and Henry Anderson. Blue babies and nitrate-contaminated well water. Environmental Health Perspectives. July 2000. Vol. 108(7): 675-678.

15. Morbidity and Mortality Weekly Report. Spontaneous abortions possibly related to ingestion of nitrate-contaminated well water – LaGrange County, Indiana, 1991-1994. July 5, 1995. Vol 45(26): 569-572.

16. Dusdieker, L. B., P. J. Stumbo, B. C. Kross, and C. I. Dungy. Does increased nitrate ingestion elevate nitrate levels in human milk? Archives of Pediatrics and Adolescent Medicine. March 1996. Vol. 150(3): 311-314.

17. Integrated Risk Information System. Information specific to nitrate, available at URL: http://www.epa.gov/iris/subst/0076.htm