



Public Health Assessment for

**BANDERA ROAD GROUNDWATER PLUME
LEON VALLEY, BEXAR COUNTY, TEXAS
EPA FACILITY ID: TXN000606565
SEPTEMBER 28, 2007**

**U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES
PUBLIC HEALTH SERVICE**

Agency for Toxic Substances and Disease Registry

THE ATSDR PUBLIC HEALTH ASSESSMENT: A NOTE OF EXPLANATION

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

In addition, this document has previously been provided to EPA and the affected states in an initial release, as required by CERCLA section 104 (i)(6)(H) for their information and review. The revised document was released for a 30-day public comment period. Subsequent to the public comment period, ATSDR addressed all public comments and revised or appended the document as appropriate. The public health assessment has now been reissued. This concludes the public health assessment process for this site, unless additional information is obtained by ATSDR which, in the agency's opinion, indicates a need to revise or append the conclusions previously issued.

Agency for Toxic Substances & Disease Registry Julie L. Gerberding, M.D., M.P.H., Administrator
Howard Frumkin, M.D., Dr.P.H., Director

Division of Health Assessment and Consultation..... William Cibulas, Jr., Ph.D., Director
Sharon Williams-Fleetwood, Ph.D., Deputy Director

Cooperative Agreement and Program Evaluation Branch Richard E. Gillig, M.C.P., Chief

Exposure Investigations and Site Assessment Branch Susan M. Moore, M.S., Chief

Health Promotion and Community Involvement Branch Susan J. Robinson, M.S., Chief

Site and Radiological Assessment Branch Sandra G. Isaacs, B.S., Chief

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PUBLIC HEALTH ASSESSMENT

BANDERA ROAD GROUNDWATER PLUME

LEON VALLEY, BEXAR COUNTY, TEXAS

EPA FACILITY ID: TXN000606565

Prepared by:

Texas Department of State Health Services
Epidemiology and Disease Surveillance Unit
Health Assessment and Toxicology Program
Under a Cooperative Agreement with the
Agency for Toxic Substances and Disease Registry

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 (EPA) and the individual states regulate the investigation and clean up of the sites.

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

Exposure: As the first step in the evaluation, ATSDR scientists review environmental data to see how much contamination is at a site, where it is, and how people might come into contact with it. Generally, ATSDR does not collect its own environmental sampling data; it reviews information provided by EPA, other governmental 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 potential health effects to children are considered first when evaluating the health threat to a community. The health effects 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 to determine the health effects that may result from exposures. That information can include the results of medical, toxicological, and epidemiological studies and data collected from disease registries. The science of environmental health is still developing, and sometimes scientific information on the health effects of certain substances is not available. When this is so, the report will suggest what further public health actions are needed.

Conclusions: The report presents conclusions about any public health threat posed by the site. When health threats have been determined for high-risk groups (such as the elderly, chronically ill, and people engaging in high risk practices), they will be summarized in the conclusion section of the report. Ways to stop or reduce exposure will then be recommended in the public health action plan.

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

Interactive Process: The health assessment is an interactive process. ATSDR solicits and evaluates information from numerous city, state, and federal agencies; the companies responsible for cleaning up the site; and the community. ATSDR then shares the conclusions with the community. Agencies are asked to respond to an early version of the report to make sure that the data they have provided is accurate and current. When informed of ATSDR's conclusions and recommendations, the agencies sometimes will begin to act on them before the final release of the report.

Community: ATSDR also needs to learn what people in the area know about the site and what concerns they may have about its affect on their health. Consequently, throughout the evaluation process, ATSDR actively gathers information and comments from the people who live or work near the site. Those include 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 written comments received from the public are responded to in the final version of the report.

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

Letters should be addressed as follows:

Attention: Chief, Program Evaluation, Records, and Information Services Branch, Agency for Toxic Substances and Disease Registry, 1600 Clifton Road (E60), Atlanta, Georgia 30333.

Table of Contents

| | |
|--|---|
| Summary | 3 |
| Purpose and Health Issues | 3 |
| Background..... | 3 |
| Site Description..... | 3 |
| Site History | 3 |
| Land and Natural Resource Use | 3 |
| Demographics | 3 |
| Site Visit..... | 3 |
| Community Health Concerns..... | 3 |
| Discussion | 3 |
| Introduction..... | 3 |
| Exposure Pathways | 3 |
| Determining Contaminants of Concern | 3 |
| Environmental Contamination and Public Health Implications | 3 |
| Response to Community Health Concerns | 3 |
| Health Outcome Data..... | 3 |
| Children’s Health Considerations | 3 |
| Conclusions..... | 3 |
| Recommendations..... | 3 |
| Public Health Action Plan..... | 3 |
| Actions Completed..... | 3 |
| Actions Planned | 3 |
| Authors, Technical Advisors, and Organizations | 3 |
| References..... | 3 |
| Certification | 3 |
| Appendix A: Acronyms and Abbreviations..... | 3 |
| Appendix B: Figure..... | 3 |
| Appendix C: Tables | 3 |
| Appendix D: Cancer Occurrence Investigation | 3 |
| Appendix E: Birth Defects Occurrence Investigation | 3 |

Summary

The Bandera Road Groundwater Plume site is located in Leon Valley, Bexar County, Texas, in northwestern San Antonio. Groundwater contamination was detected in private drinking water wells and consists of volatile organic compounds (VOCs), primarily tetrachloroethylene (PCE) and trichloroethylene (TCE), that exceed the regulatory standards (maximum contaminant level or MCL) established for public water systems. Other contaminants that exceeded health-based screening values included trihalomethanes and vinyl chloride. Groundwater from the plume area is used by residents and businesses for drinking and other household and business purposes.

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 and evaluated the exposure pathways through which the public could come into contact with contaminants from the site.

Data for air, soil, and surface water exposure pathways were not available for review; however, because contact with these media would be minimal, we do not expect them to pose a public health hazard.

We do not know how long residents using private wells in the Leon Valley area may have been exposed to contaminants in their drinking water or at what levels these exposures occurred. Based on lack of information, **past exposures to contaminants in private water wells pose an indeterminate public health hazard**. However, assuming past concentrations of PCE in the private wells are similar to current maximum concentrations, estimated exposure doses are well below levels that have been shown to cause adverse health effects in humans.

Elevated levels of PCE and TCE were first detected in private water wells in 2004. Granulated activated charcoal (GAC) water filtration systems were installed on five water wells used for drinking water in August 2004. Currently, drinking water wells with concentrations of PCE and TCE that exceed the MCL are equipped with filtration systems that reduce concentrations of contaminants to below detection. In the future, these residents will be connected to the public water supply system for drinking water. Based upon the current levels of trihalomethanes and vinyl chloride in the drinking water, if residents would be exposed to similar levels of these contaminants for 30 years, we would not expect to see non-cancerous or cancerous health effects due to exposure to these contaminants. Thus **current and future exposures to contaminants in private wells pose no apparent public health hazard**, provided the filtration systems are properly maintained while these wells are being used and residents do not use their private wells for drinking water after they are connected to the public water supply system.

From July 24, 2007 through August 24, 2007, the public was given the opportunity to make comments regarding the conclusions and recommendations of this health assessment document. No comments or concerns regarding this public health assessment document were received by DSHS.

As data become available, DSHS and ATSDR will re-evaluate the public health significance of this site, particularly if conditions change in the future.

Purpose and Health Issues

The Agency for Toxic Substances and Disease Registry (ATSDR) was established under the mandate of the Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA) of 1980. This act, also known as the “Superfund” law, authorized the U.S. Environmental Protection Agency (EPA) to conduct clean-up activities at hazardous waste sites. EPA was directed to compile a list of sites considered hazardous to public health. This list is termed the National Priorities List (NPL). The 1986 Superfund Amendments and Reauthorization Act (SARA) directed ATSDR to prepare a public health assessment (PHA) for each NPL site. (Note: Appendix A provides a listing of abbreviations and acronyms used in this report.)

In conducting the PHA, three types of information are used: environmental data, community health concerns, and health outcome data. The environmental data are reviewed to determine whether people in the community might be exposed to hazardous materials from the NPL facility. If people are being exposed to these chemicals, ATSDR will determine whether the exposure is at levels that might cause harm (see Table 1 for explanation of ATSDR’s conclusion categories). Community health concerns are collected to determine whether health concerns expressed by community members could be related to exposure to chemicals released from the facility. If the community raises concerns about specific diseases in the community, health outcome data (information from state and local databases or health care providers) can be used to address the community concerns. If ATSDR finds that harmful exposures existed, health outcome data also can be used to determine if illnesses are occurring and whether they could be associated with the hazardous chemicals released from the NPL facility.

In accordance with the Interagency Cooperative Agreement between ATSDR and the Texas Department of State Health Services (DSHS), this PHA was prepared for the Bandera Road Groundwater Plume site. This PHA presents conclusions about whether exposures are occurring and whether a health threat is present. In some cases, it is possible to determine whether exposures occurred in the past. However, a lack of appropriate historical data often makes it difficult to quantify past exposures. If a threat to public health exists, recommendations are made to stop or reduce the threat to public health.

Background

Site Description

The Bandera Road Groundwater Plume site is located in a residential/light commercial/industrial area in the northwestern part of San Antonio. Groundwater contamination consists of volatile organic compounds (VOCs), primarily tetrachloroethylene (also known as tetrachloroethene, perchloroethylene, or PCE) and trichloroethylene (also known as trichloroethene or TCE), which exceed the regulatory standards (maximum contaminant level or MCL) established for public water systems. The center of the groundwater plume lies between Poss Road and Grissom Road, approximately 590 feet southwest of Bandera Road in Leon Valley, Bexar County, Texas (Appendix B). The plume extends to Linklea Drive to the north, El Verde Road to the east, Shadow Mist Drive to the south, and Sawyer Road to the west.

The source of the groundwater contamination and the length of time residents were exposed to PCE and TCE in drinking water at levels that exceed the MCL have not been identified. There are approximately 50 potential sources of the contamination, including dry cleaners, automobile-related service facilities, and several light industrial sites in the area [1]. Ongoing investigations in the area may reveal the source of contamination.

Site History

The groundwater plume was discovered in 2004 during an investigation at a local shopping center by the Texas Commission on Environmental Quality (TCEQ) voluntary clean-up program. During this assessment, a drinking water well was identified with PCE at a level that exceeded the regulatory standard (MCL) of 5 micrograms/liter ($\mu\text{g/L}$) [2].

In June 2004, the TCEQ conducted a Site Screening Investigation under a grant program with the EPA Region 6 [1]. The initial environmental assessment involved a dry cleaner located near the intersection of Bandera Road and Grissom Road. The investigation included soil borings to evaluate an adjacent property, a water well survey, sampling of eight wells located within a ½-mile radius of the property, and a search for potential sources within a ½-mile radius of the property. Results of this assessment were inconclusive regarding the source(s) of the groundwater release [1].

The TCEQ has evaluated 33 private wells and nine public water systems located within a 2-mile radius of the center of the plume to determine the extent of groundwater contamination in this area. Based on this investigation, 11 private wells met the criteria for an observed release (contaminants detected above the laboratory method detection limit) of PCE and/or TCE, and six of these private wells had concentrations that exceeded the MCL. In August 2004, the TCEQ installed granulated activated charcoal (GAC) water filtration systems on five private wells that had levels of contamination that exceeded MCLs [1]. The owner of the sixth private well declined to have the GAC system installed because this well was being used for irrigation purposes only [3]. The filtration systems have been effective at reducing contamination to levels below detection [2].

The Bandera Road Groundwater Plume site was proposed to the NPL on September 27, 2006, and was added to the final list on March 7, 2007 [4]. Inclusion on the NPL allows federal funds and personnel to become available to further assess the nature and extent of the public health and environmental risks associated with the site. Currently, EPA is working with the City of Leon Valley and the San Antonio Water System to connect residents with affected private water wells to these public water systems [4].

Land and Natural Resource Use

The groundwater plume is located in Leon Valley, which is in the northwestern part of San Antonio. The land use is mainly residential and light commercial/industrial, with parks, playgrounds, schools, and day cares in the vicinity. One commercial business, a garden center, used a private well for irrigation of trees in the past, but this well is no longer in operation.

Groundwater in this area comes from the Edwards Aquifer, which provides drinking water to over a million people in south-central Texas. Minor geologic faults and fractured limestone in the investigation area allow for contamination to enter the Edwards Aquifer. According to TCEQ's Hazard Ranking System documentation record, an observed release to the Edwards Aquifer has been documented by chemical analysis [1].

Depth to static water level ranges from 120 to 212 feet below DSL (Datum Land Surface). Perched groundwater is not present in large quantities because of limestone in the area. Private water wells in the area are 280 to 500 feet below DSL and City of San Antonio public water supply wells are 780 to 824 feet below DSL. Both public and private drinking water wells are screened in the Edwards Aquifer [1].

Demographics

The 2000 United States Census reported a total population of 16,147 people living in 6,355 housing units within a 1-mile radius of the center of the plume (Appendix B). For this area, 12,136 people identified themselves as being white, 533 people identified themselves as being black, and 3,477 people identified themselves as some other race. Of this population, 7,701 people identified themselves as being of Hispanic or Latino origin. At the time of the census, there were 1,594 children under the age of six, and 3,680 women of child-bearing age (age 15 to 44) [5].

Six private wells have been identified with PCE and/or TCE contamination above the MCL; however, one of these wells was not used for drinking water. Based upon communication with these residents, 24 people may have been exposed to the affected groundwater in the past. The installation of filtration systems on five of these wells has eliminated exposure to these substances. How long (and at what level) contamination was present in these wells prior to the installation of the filtration systems is unknown.

Site Visit

DSHS personnel visited the area of the Bandera Road Groundwater Plume on July 27, 2006, September 19, 2006, November 2, 2006, and July 24, 2007. On July 27, 2006, EPA sponsored a meeting with DSHS personnel, United States Geological Service (USGS) representatives, and City of Leon Valley officials. The purpose of this meeting was to update the city officials on the status of listing the site on the NPL and to inform them of the superfund process and roles of agencies involved in the process. Following this meeting, EPA provided DSHS a tour of the plume area. On September 19, 2006, a DSHS representative attended a Leon Valley City Council Workshop. An EPA representative presented information to the city council about the history of the groundwater plume, actions to date, and the superfund process. The DSHS representative informed the city council of the PHA process. On November 2, 2006, the EPA conducted an availability session for the community. DSHS, TCEQ, and USGS representatives also were present. An EPA representative provided community members with information about the groundwater plume and the superfund process. A DSHS representative presented information about the PHA process and the health effects associated with PCE. DSHS representatives also talked with the community members individually about their health concerns and handed out fact sheets about PCE. Prior to this meeting, TCEQ representatives provided DSHS with a tour of the

plume area and pointed out the filtration systems on the wells. Public water supply wells for City of San Antonio and City of Leon Valley were observed within one mile of the plume boundary to the east and to the north. Another EPA availability session was conducted on July 24, 2007. EPA representatives updated the community on site activities. DSHS representatives announced the opening of the public comment period for the public health assessment and distributed copies of a site-specific fact sheet and the public health assessment.

Community Health Concerns

As part of the public health assessment process, DSHS and ATSDR try to learn what concerns people in the area might have about site-related effects on their health. Consequently, attempts were made to actively gather information and comments from people who live or work near the site.

To collect community health concerns related to the Bandera Road Groundwater Plume site, DSHS staff attended an EPA availability session on November 2, 2006. People attending this community meeting were generally concerned about the safety of their drinking water. Another concern was related to the safety of eating eggs and meat from ducks and chickens that drank contaminated water. EPA requested that DSHS address whether residents may continue to use private wells to water yards and gardens without a risk to human health.

Discussion

Introduction

The presence of chemical contaminants in the environment does not always result in exposure to or contact with the chemicals. Chemicals have the potential to cause adverse health effects only when people actually come into contact with them; therefore, exposure (the contact that people have with the contaminants) drives the PHA process.

Exposure does not always result in adverse health effects, so we must also evaluate whether the exposure could be sufficient to pose a hazard to people in the community. The factors that influence whether exposure to a contaminant or contaminants could or would result in adverse health effects include

- 1) the toxicological properties of the contaminant,
- 2) how much of the contaminant the individual is exposed to,
- 3) how often and/or how long the exposure occurs,
- 4) the manner in which the contaminant enters or contacts the body (breathing, eating, drinking, or skin/eye contact), and
- 5) the number of contaminants to which an individual is exposed (combinations of contaminants).

Once exposure occurs, characteristics such as age, sex, nutritional status, genetics, lifestyle, and health status of the exposed person influence how that person absorbs, distributes, metabolizes, and excretes the contaminant.

Exposure Pathways

People can be exposed to contaminants by breathing, eating, drinking, or coming into direct contact with a substance containing the contaminant. This section reviews available information to determine whether people in the community have been, currently are, or could in the future be exposed to contaminants associated with this site.

To determine whether people are exposed to site-related contaminants, investigators evaluate the environmental and human components leading to human exposure. This analysis consists of evaluating the five elements of an exposure pathway:

- 1) source of contamination,
- 2) transport through an environmental medium,
- 3) point of exposure,
- 4) route through which the contaminant can enter the body, and
- 5) a receptor population.

Exposure pathways can be complete, potential, or eliminated. For a person to be exposed to a contaminant, the exposure pathway must be complete. An **exposure pathway** is considered complete when all five elements in the pathway are present and exposure has occurred, is occurring, or will occur in the future. A **potential pathway** is missing at least one of the five elements, but could be complete in the future. An **eliminated pathway** is missing one or more elements and will never be completed.

Groundwater at the site is currently used for drinking water, food preparation, bathing, and for commercial/business purposes. Sampling data from June 2004 through April 2007 indicate that water from private wells contains PCE and TCE in excess of current drinking water standards. Contaminants, particularly VOCs that enter the home in potable water, present a situation in which residents could be exposed via multiple pathways. These include direct ingestion of the water, inhalation of the contaminant due to volatilization (when the contaminant becomes a gas and enters the air), and absorption of the contaminant through the skin during bathing. Thus, we would consider these all to be past completed exposure pathways (although we do not know how long residents may have been exposed to the contaminants in the past). Currently, filtration systems on the private drinking water wells are reducing contaminant concentrations to levels below analytical detection limits, and this pathway has been eliminated. Provided the filtration systems are maintained or residents are connected to and use the public water supply, this pathway will remain eliminated in the future. Contamination has not been detected in the Leon Valley and San Antonio public water supply wells utilizing the Edwards Aquifer in the same area; therefore, this is an eliminated pathway. However, a potential exposure pathway exists if contaminants migrate into the public water supply system in the future.

Data for ambient air, soil, biota, surface water, and sediment were not available for review and therefore we cannot assess these pathways. However, we do not expect exposure to these media at this site to be a significant exposure pathway. VOCs are not generally taken up and stored in biota and concentrations of VOCs in ambient air, soil, and surface water would be low due to evaporation and/or degradation. Additionally, the probability of regular inhalation, ingestion, or dermal contact with these substances in these media is low. Therefore we believe that the ambient air, soil, biota, surface water, and sediment pathways are eliminated and require no further

evaluation. When groundwater is contaminated with VOCs, there is the potential for these substances to migrate through the soil into overlying buildings. At this site, the depth to groundwater is greater than 100 feet. Therefore, because of the depth to groundwater, the vapor intrusion pathway is unlikely to be a pathway of concern.

Exposure pathways are summarized in Table 2. The following discussions incorporate only those pathways relevant and important to the site.

Determining Contaminants of Concern

When plausible potential exposure scenarios are identified, the potential public health significance of the exposure scenarios is assessed. This is done by comparing each contaminant detected with its media-specific health-based assessment comparison (HAC) value for non-cancer and cancer endpoints. These values are guidelines that specify levels of chemicals in specific environmental media (soil/sediment, air, and water) that are considered safe for human contact with respect to identified adverse health effects.

Non-cancer comparison values are also known as *environmental media evaluation guides* (EMEGs) or *reference dose media evaluation guides* (RMEGs). They are based on the ATSDR's minimal risk levels (MRLs)¹ or the EPA's reference doses (RfDs)². Both of these 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. Therefore, 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.

For contaminants that are considered to be known human carcinogens, probable human carcinogens, or possible human carcinogens, cancer risk evaluation guides (CREGs) are calculated using EPA's chemical-specific cancer slope factors (CSFs) and an estimated excess lifetime cancer risk of one-in-one million persons exposed for a lifetime.

Other comparison values used to evaluate drinking water data include MCLs and Lifetime Health Advisories (LTHAs). MCLs are regulatory standards for public water systems that are enforced by the EPA and TCEQ. MCLs are based on MCL Goals (MCLG), which are contaminant levels that would not cause any potential health problems [6]. For carcinogens, the MCLG is set at 0 µg/L [7]. The MCL is then set as close to the MCLG as possible, considering the ability of public water systems to detect and remove contaminants using suitable treatment technologies. Using an MCL as a screening level for a private water well ensures that people using those wells would be protected to the same extent as people using the public water system. LTHAs are non-enforceable drinking water standards set at levels that are not expected to cause any adverse non-carcinogenic effects for a lifetime of exposure [6].

¹ 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.

² An RfD is an estimate (with a level of uncertainty spanning perhaps an order of magnitude) of a daily exposure to the human population (including sensitive groups) that is likely to be without appreciable risk of deleterious effects during a lifetime.

Standard assumptions for body weight (70 kilogram or kg adult and 16 kg child) and water ingestion (2 liters/day or L/day for adults and 1 L/day for a child) were used to calculate both non-cancer and cancer HAC values.

The exposure assumptions used to establish these screening levels are conservative with respect to protecting public health; as a result, actual exposures are likely to be lower than those used to calculate the screening values. Exceeding a screening value does not mean that a contaminant represents a public health threat; rather, it suggests that the contaminant warrants further consideration. Assessing the public health significance of contaminants that exceed their respective screening levels involves reviewing and integrating relevant toxicological information with plausible exposures. We may estimate the magnitude of the public health significance by comparing the estimated exposures to identified “no observed” and “lowest observed” adverse effects levels (NOAELs and LOAELs) in animals and to known effect levels in humans, when available. We assess the public health significance of contaminants that exceed screening values by reviewing and integrating relevant toxicological information with reasonable maximum exposure scenarios.

Environmental Contamination and Public Health Implications

The environmental data used in this PHA were provided by TCEQ. A total of 224 drinking water samples were collected from private water wells and two public water supply system (providing water to a mobile home park and to the City of Leon Valley) from June 2004 through April 2007 and were analyzed for VOCs. Data from the following sampling events were analyzed in this PHA: June 2004, August 2004, December 2004, March 2005, November 2005, December 2005, May 2006, November 2006, and April 2007. For this PHA, DSHS relied on the information provided in the referenced documents and assumed adequate quality assurance/quality control procedures were followed with regard to data collection, chain-of-custody, laboratory procedures, and data reporting.

Drinking water data for the following contaminants of concern (contaminants that exceeded a non-cancer or a cancer HAC value) are presented in Table 3: PCE, TCE, trihalomethanes (bromodichloromethane or BDCM, bromoform, and dibromochloromethane), and vinyl chloride. The following sections will address the public health significance of these contaminants in the drinking water. EPA also included cis 1,2-dichloroethene as a potential contaminant of concern; however, no concentrations of this contaminant exceeded health-based screening values in the drinking water samples and thus this contaminant is not discussed.

Perchloroethylene

PCE is a man-made chemical that is commonly used in dry cleaning and metal-degreasing operations. Additionally, it is used as a building block for making other chemicals and is found in some household products such as water repellants, spot removers, adhesives, and wood cleaners [8].

PCE is a liquid at room temperature and evaporates easily into the air. Most PCE that gets into surface water and soil evaporates quickly into the air where it may persist for several months before being broken down into other chemicals. PCE in soil also travels into the groundwater where it may exist for several months. Under some conditions, PCE in groundwater is broken down into other compounds by bacteria, while under other conditions it sticks to soil. It does not build up in aquatic animals. Although PCE has been detected in fruits and vegetables, it is not clear if uptake of PCE occurred while the plants were growing or at some point after harvesting [8].

Exposure to PCE generally occurs by inhalation or ingestion. PCE is frequently found in the air because of evaporation from industrial or dry cleaning operations. Groundwater near these types of facilities or hazardous waste sites also may be contaminated with PCE. There is a potential for this contamination to end up in drinking water from private or public water wells. Occupational exposures also occur in those people that work with PCE. Regardless of the exposure route (breathing, eating, drinking, or touching), most PCE leaves the body from the lungs during exhalation. A small amount of PCE travels to the liver and is broken down to other compounds and excreted in urine within a few days [8].

PCE has been used as an anesthetic because it causes loss of consciousness. Single exposures involving inhalation of high concentrations of PCE in closed or poorly ventilated areas can cause dizziness, headache, sleepiness, confusion, nausea, difficulty in sleeping or walking, unconsciousness, or death. Repeated or long-term dermal exposures can cause skin irritation. Most of these types of exposures and health effects occur with accidental exposures to high concentrations of PCE in occupational (or hobby) environments. Some studies have shown that women who work in dry cleaning facilities may have more menstrual problems and spontaneous abortions than women who are not exposed; however, it is not known if these effects were caused by PCE because other possible causes were not considered [8].

Animal studies using inhalation and ingestion exposures have shown that PCE can cause liver and kidney damage or liver and kidney cancer. These studies were conducted using levels of PCE that are much higher than those most people are exposed to and the relevance of these studies to humans is unclear. The human health effects of breathing air or drinking water with low levels of PCE are not known [8].

The National Toxicology Program has categorized PCE as reasonably anticipated to be a human carcinogen based on sufficient evidence of carcinogenicity in animals. Inhalation of PCE has resulted in an increased incidence of liver tumors in male and female mice. Increased incidences of leukemia in male and female rats and rare kidney tumors in male rats also have been noted [9].

PCE in the body can be detected by breath and blood tests. PCE that is stored in fat is slowly released to the bloodstream and can be detected in the breath for weeks following heavy exposures. Breakdown products of PCE can be detected in blood and urine for several days after exposure; however, exposure to other chemicals can produce the same breakdown products and so these tests can not definitively show exposure to PCE [8].

Thirty-one drinking water samples had levels of PCE that exceeded the current regulatory standard (MCL of 5 µg/L), 28 samples exceeded the LTHA (10 µg/L), and three samples exceeded the child RMEG (100 µg/L) for PCE (Table 3). Two of the samples that exceeded the MCL were taken from a well without a filtration system and had PCE concentrations of 5.01 µg/L and 5.05 µg/L; however, water from this well is not used for drinking or other household purposes. The remaining samples that exceeded HAC values were collected prior to going through the filtration system, and PCE was not detected in post-filtration system samples. Because PCE in post-filtration samples did not exceed the non-cancerous screening value (RMEG), we do not expect non-cancerous health effects to occur. The estimated increased lifetime risk for cancer due to exposure to PCE in the drinking water could not be calculated because it is not known how long exposure may have occurred.

Trichloroethylene

TCE is a solvent that is used to remove grease from metal parts. It is found in some household products such as typewriter correction fluid, paint removers, adhesives, and spot removers [10]. It also is a breakdown product of PCE.

TCE is liquid at room temperature. TCE in surface water evaporates easily and is broken down within days to weeks. In groundwater, this process occurs much slower because of a slower evaporation rate. Very little TCE in soil gets broken down, and TCE in the soil can seep to groundwater. Although TCE can be found in some foods, this contamination is thought to originate from the use of contaminated water in food preparation. TCE does not build up in fish tissues [10].

People are exposed by breathing air or drinking or using water contaminated with TCE. These exposures generally occur in areas near factories that use TCE or near hazardous waste sites with TCE contamination. Workers at facilities that use TCE may be routinely exposed to the chemical. Half the TCE that is inhaled is removed from the body through exhaling, while the remaining portion gets into the bloodstream. TCE that is ingested also will travel to the blood. Once in the blood, TCE may be eliminated from the body or may be stored in body fat. TCE is eliminated by exhaling, or it travels to the liver where it is broken down into other compounds that are excreted in urine within a day. Breakdown products of TCE are stored in body fat [10].

TCE was once used as an anesthetic for surgery because inhalation of large amounts of TCE makes people dizzy or sleepy and could result in a loss of consciousness. Dermal exposure to concentrated solutions of TCE may result in skin rashes. Inhalation of moderate levels of TCE may result in headaches or dizziness, while exposure to higher concentrations may result in damage to facial nerves, liver and kidney damage, changes in heart beat, or possibly death [10].

Some studies have suggested that long term exposure to high levels of TCE in drinking water may result in an increase in adverse birth outcomes such as childhood leukemia, heart defects, a rare defect in the respiratory system, eye defects, neural tube defects, oral cleft palates, and hearing and speech impairments; however, the results of these studies are not conclusive [10].

The National Toxicology Program has classified TCE as reasonably anticipated to be a human carcinogen based on limited evidence on carcinogenicity in human studies. Experimental studies in animals have shown an increase incidence of malignant and/or a combination of malignant and benign tumors at multiple tissue sites in multiple species of animals after exposure to TCE via inhalation. In mice TCE induced tumors of the liver, lung, and blood. In rats exposure to TCE resulted in kidney cancer, interstitial-cell tumors of the testis, and possible leukemia [9].

Tests to determine if people have been exposed to TCE are available; however, they are not routinely performed in doctor's offices. TCE can be measured in the breath. Breath tests conducted soon after exposure can indicate if people have been exposed to a large or small amount of TCE. Breakdown products of TCE can be measured in the urine. Urine tests can produce the same results up to a week after exposure; however, other chemicals can produce the same breakdown products making exposure to TCE difficult to determine [10].

Of the drinking water samples collected prior to entering the filtration system, five samples had levels of TCE that exceeded the current regulatory standard (MCL of 5 $\mu\text{g/L}$) for TCE, the only comparison value that was available (Table 3). TCE was not detected in samples collected after the drinking water had passed through the filtration system. The estimated increased lifetime risk for cancer due to exposure to TCE in the drinking water could not be calculated because it is not known how long exposure may have occurred.

Trihalomethanes

Trihalomethanes are a group of disinfection byproducts that include BDCM, dibromochloromethane, chloroform, and bromoform [11]. These compounds are formed when chlorine (or other disinfectants used in drinking water) reacts with other naturally occurring organic and inorganic matter in water [12-13]. BDCM, bromoform, and dibromochloromethane, exceeded HAC values in drinking water samples and thus warrant further consideration.

People are exposed to trihalomethanes by drinking water that has been treated with chlorine; however, the health risks associated with exposure to disease-causing organisms (bacteria and viruses) in non-chlorinated drinking water are much greater than are the risks of adverse health effects from drinking chlorinated water. Other exposures may occur in swimming pools by breathing trihalomethanes that have evaporated from the water into the air or by uptake from the water through skin. Trihalomethanes in drinking water are usually at levels that are known to be without adverse health effects (between 1 and 10 $\mu\text{g/L}$). Once in the body, these compounds move into the blood and are removed from the body by being exhaled from the lungs. Trihalomethanes do not build up in the body, and following exposure the majority of these compounds are rapidly removed from the body (within 8 hours) [12-13].

Health effects associated with the ingestion of trihalomethanes include liver and kidney damage and slowing of normal brain activity (resulting in sleepiness or sedation). There is some evidence that ingestion of BDCM may affect the developing fetus [12]. Studies have shown exposure to bromoform and dibromochloromethane does not pose a high risk of affecting the chance of becoming pregnant or harming the unborn baby [13].

Long term exposure to these substances may cause liver and kidney cancer [12-13]. The National Toxicology Program has classified BDCM as reasonably anticipated to be a human carcinogen based upon sufficient evidence of carcinogenicity in animals. Ingestion of BDCM resulted in an increased incidence of kidney and large intestine tumors in male and female rats. There is no data available to assess the carcinogenic effects of long-term exposure to BDCM in humans [9]. The National Toxicology Program has not classified the carcinogenicity of bromoform and dibromochloromethane.

Tests of the blood, breath, and fat can determine if people have been exposed to trihalomethanes. However, special equipment is needed to perform these tests and they are not regularly performed in doctor's offices. Additionally, because these compounds are rapidly removed from the body, these tests are only effective in detecting recent exposures (within 1 or 2 days) [12-13].

Ten drinking water samples (seven samples from a well supplying a mobile home park³, one sample from an abandoned water well, one sample from a residential water well, and one sample from the City of Leon Valley⁴) had levels of BDCM that exceeded the CREG (0.6 µg/L) for this contaminant (Table 3). Twelve drinking water samples (nine samples from a well supplying a mobile home park, one sample from an abandoned water well, one sample from a residential water well, and one sample from the City of Leon Valley) had levels of dibromochloromethane that exceeded the CREG (0.4 µg/L) for this contaminant (Table 3). One drinking water sample from the City of Leon Valley had bromoform at a level that exceeded the CREG (4 µg/L) for this contaminant (Table 3).

Past exposures to trihalomethanes could not be quantified because it is not known how long exposure may have occurred in the past and at what levels of contamination. The risk for cancer due to exposure to trihalomethanes was calculated for adults using standard assumptions for body weight (70 kg adult), water intake rate (2 L/day adult), and the maximum concentration of each contaminant (6.95 µg/L for BDCM, 13.75 µg/L for dibromochloromethane, and 6.80 µg/L for bromoform). Based upon the current levels of trihalomethanes in the drinking water, if residents would be exposed to similar levels of these contaminants for 30 years, we would expect to see a no apparent increased lifetime risk for developing cancer.

Vinyl Chloride

Vinyl chloride is a breakdown product of synthetic chemicals such as PCE and TCE. Additionally, it is manufactured in the United States to make polyvinyl chloride (PVC) for a variety of plastic products such as pipes, wire and cable coatings, and packaging materials [14].

Vinyl chloride can exist as a liquid if it is kept under high pressure or at low temperatures. Under normal conditions, it exists as a gas. Vinyl chloride near the surface of water and soil evaporates

³ The well supplying the mobile home park is classified as a public water supply system with 39 connections providing drinking water to 117 people.

⁴ The City of Leon Valley public water supply system includes 2,478 connections and provides drinking water to 7,434 people. The drinking water sample was collected from one of two City of Leon Valley wells, the Grasshill Road well.

quickly. A limited amount of vinyl chloride can dissolve in water. It does not build up in plant or animal tissues [14].

The most likely exposure route for exposure to vinyl chloride is inhalation. Although vinyl chloride is not normally found in urban, suburban, or rural air, it has been detected in the air near plastics industries, hazardous wastes sites, and landfills. People may also be exposed to vinyl chloride by drinking water from contaminated wells. Once in the body, vinyl chloride quickly enters the blood. In the liver, vinyl chloride is changed into other substances that travel through the body in blood and are excreted in urine. Most vinyl chloride is eliminated from the body within 24 hours; however, some breakdown products produced by the liver take longer to eliminate [14].

Health effects associated with inhalation of vinyl chloride include dizziness, sleepiness, or a loss of consciousness. With fresh air, recovery is rapid. Inhalation of extremely high levels of vinyl chloride can damage the liver, lungs, kidneys, may damage the heart, and may prevent blood clotting. Long-term exposure to high levels of vinyl chloride in the air may result in changes in liver structure. People exposed to vinyl chloride in the workplace have reported nerve damage, immune reactions, problems with blood flow in the hands, damage to sperm and testes, irregular menstrual periods, and high blood pressure during pregnancy. Studies of women living near vinyl chloride plants did not show that exposure to vinyl chloride produced birth defects. Animal studies have shown that exposure to vinyl chloride may result in more miscarriages early in pregnancy, decreased birth weight, and a delay in skeletal development in fetuses.

The non-cancerous health effects associated with ingestion of vinyl chloride are unknown [14].

Studies have shown breathing air or drinking water with low levels of vinyl chloride may increase the risk of getting cancer; however, levels of vinyl chloride used in these studies were much higher than those found in ambient air and drinking water [14]. The National Toxicology Program has classified vinyl chloride as a known human carcinogen based on sufficient evidence of carcinogenicity in humans. Numerous epidemiological studies and case reports have shown an association between inhalation or ingestion of vinyl chloride and the development of a very rare tumor of the liver. Other studies have shown vinyl chloride to cause liver, brain, lung, lymphatic system, and hematopoietic system cancers [9].

Vinyl chloride can be measured in the breath, but the test must be done shortly after exposure and these tests do not give an indication of how much vinyl chloride a person was exposed to or the health effects associated with such exposures. These tests are not normally available at the doctor's office. Breakdown products of vinyl chloride can be detected in the urine, but exposure to other chemicals may produce the same breakdown products [14].

One drinking water sample from a residence with no filtration system had a concentration of vinyl chloride that exceeded the most conservative HAC value (CREG of 0.03 $\mu\text{g/L}$) for this contaminant. Past exposures to vinyl chloride could not be quantified because it is not known how long exposure may have occurred in the past and at what level of contamination. The risk for cancer due to exposure to vinyl chloride was calculated for adults using standard assumptions for body weight (70 kg adult), water intake rate (2 L/day adult), and the maximum concentration of

vinyl chloride (0.99 µg/L). Based upon the current level of vinyl chloride in the drinking water, if residents would be exposed to similar levels of this contaminants for 30 years, we would expect to see a no apparent increased lifetime risk for developing cancer.

Summary of Public Health Implications

We do not know how long residents using private wells in the Leon Valley area may have been exposed to contaminants in their drinking water and at what levels these exposures occurred. In the past, some residents using private wells for drinking water may have been exposed to PCE and TCE. Using drinking water data currently available allows us to provide an indication of what past exposures may have been. Further investigation in the area to identify sources of contamination or potential responsible parties and how long these potential sources were in existence could aid in better characterizing past exposures to these compounds.

In addition to exposure to these contaminants via ingestion, volatile compounds such as PCE and TCE can evaporate from water to air when water is used in the home, such as when water is used for taking a shower or washing dishes, resulting in additional exposure to these contaminants via the inhalation, as well as dermal, pathways.

PCE and TCE have shown the potential to have toxicological effects on the liver and kidney, and there is the possibility that exposure to a combination of these contaminants may result in additive effects on these target organs. Exposures to combinations of PCE and TCE have shown no evidence of a greater-than-additive effect on target organs, and, in some cases, there may be a less-than-additive effect [15]. Post-filtrations sampling results for both of these are well below levels known to cause harmful effects, so it is not likely that combined exposures to PCE and TCE would result in adverse health effects.

Information regarding past exposures (concentrations residents may have been exposed to and duration of exposure) is not available; therefore, we cannot assess the potential health implications associated with past exposures to contamination in drinking water wells. Based on lack of information, **past exposures to contaminants in private water wells pose an indeterminate public health hazard**. If a source or potentially responsible parties are identified, we may be able to characterize past exposures to these contaminants based upon how long potential sources have been in operation. However, assuming past concentrations of PCE in the private wells are similar to current maximum concentrations, estimated exposure doses are well below levels that have been shown to cause adverse health effects in humans.

Using the data that is currently available, there were several samples that exceeded their respective screening values for PCE and TCE, but the majority of these were pre-filtration samples; these contaminants were below the detection limit in the respective post-filtration samples. Two samples from a water well with no filtration system had PCE at levels that exceeded the MCL (5.01 µg/L and 5.05 µg/L); however, water from this well is not used for drinking or other household purposes. No other samples exceeded screening values for PCE and TCE, thus there is no indication that people are currently being exposed to elevated levels of these contaminants.

BDCM, bromoform, dibromochloromethane, and vinyl chloride exceeded screening values for cancerous health effects in several samples. Based upon the current levels of trihalomethanes and vinyl chloride in the drinking water, if residents would be exposed to similar levels of these contaminants for 30 years, we would not expect to see non-cancerous or cancerous health effects due to exposure to these contaminants.

Currently, drinking water wells with concentrations of PCE and TCE that exceed the MCL are equipped with filtration systems that reduce concentrations of contaminants to below detection. In the future, these residents will be connected to the public water supply system for drinking water. Thus **current and future exposures to contaminants in private wells pose no apparent public health hazard**, provided the filtration systems are properly maintained while these wells are being used and residents do not use their private wells for drinking water after they are connected to the public water supply system.

Response to Community Health Concerns

The community was concerned about the safety of their drinking water. Although we are not able to assess the potential health implications associated with past exposures to contaminants in the drinking water; currently, drinking the filtered water would not be expected to result in adverse health outcomes.

One family was concerned about the safety of eating eggs and meat from ducks and chickens that drank PCE-contaminated water. Regardless of the exposure route (ingestion of water for this scenario), most PCE is eliminated via exhalation. The small amount of PCE that remains in the body may be stored in fatty tissues (such as eggs). The amount of PCE that people may be exposed to via ingestion of eggs and meat from animals that consumed contaminated water would be much lower than the concentration of PCE in the water. For this family, the concentration of PCE in their water was well below the MCL, thus we do not expect to see health effects associated with eating eggs and meat from these animals.

The EPA asked DSHS to address the question of whether residents could continue to use private well water to irrigate lawns and gardens after being connected to the city water supply for drinking water and filtration systems are removed from private wells. Contaminants detected in groundwater in this area are volatile substances, meaning that when they are released into the atmosphere (for instance, in water from a sprinkler) they evaporate quickly. Volatile compounds move readily through plants and are released to the air above plants [16]. Although there is some uptake of these compounds into plants, the contaminants are lost due to volatilization and they do not accumulate in plants [17-18]. PCE and TCE in water used for irrigation evaporates quickly and plants do not accumulate these compounds. Because these compounds evaporate quickly when they are released into the air, exposure to PCE and TCE via inhalation is not expected to be a major exposure pathway. Based upon the current concentrations of PCE and TCE in private wells (pre-filtration samples), we do not expect there to be a problem with using this water to irrigate lawns and gardens.

Health Outcome Data

Health outcome data record certain health conditions that occur in populations. These data can provide information on the general health of communities living near a hazardous waste site. They also can provide information on patterns of specified health conditions. Some examples of health outcome databases are cancer registries, birth defects registries, and vital statistics. Information from local hospitals and other health care providers also can be used to investigate patterns of disease in a specific population. DSHS and ATSDR look at appropriate and available health outcome data when a completed exposure pathway or community concern exists.

Before filtration systems were installed on the affected wells, people may have been exposed to contaminants that have been associated with the development of cancer and/or birth defects. Consequently, the Texas Cancer Registry and Texas Birth Defects Registry conducted cluster investigations for these respective conditions. Both of these registries use methods that evaluate the occurrence of cancer or birth defects in a specified area and compare this rate to what would be expected to occur based upon state rates. Although it is not possible to determine if an individual's cancer or birth defect was caused by an environmental or other risk factor, these investigations are useful in determining if there is an excess of these health outcomes in an area or population (such as a community).

Incidence and prevalence are the most common ways to measure the occurrence of disease in a community. Incidence is the ratio of the number of new cases of a disease in that community for a specific time period (such as a year) relative to the total size of the population at risk during that time period [19]. Prevalence is the ratio of the total number of cases of a disease in a community at a specific time period relative to the number of individuals in the community at that time [19]. The Texas Cancer Registry uses incidence data to assess the occurrence of cancer in a community because it more accurately shows the number and types of cancer diagnosed each year. The population at risk for this calculation is the number of people living in the community [20]. For birth defects, because both live births and fetal deaths are reported, the population at risk consists of all conceptions that reach the gestational age at which the defect occurs (varies depending on the specific defect). It is not possible to know how many pregnancies reach the various gestational ages or why some early pregnancies end; therefore the Texas Birth Defects Registry uses prevalence data, using the total number of live births as an approximation of the population size [19].

The Texas Cancer Registry evaluated incidence of cancers in zip codes 78238 and 78240 for the years 1995 through 2003. Based upon state rates, the following cancers were found to be within expected ranges in both males and females for zip code 78238: prostate, breast, lung, colon and rectum, bladder, kidney and renal pelvis, liver and intrahepatic bile duct, non-Hodgkin's lymphoma, brain and other nervous system, selected leukemia subtypes, larynx, esophagus, cervix, Hodgkin's lymphoma, myeloma, testis, and stomach. For zip code 78240, the same cancers were found to be within expected ranges except lung cancer in females and liver intrahepatic bile duct cancer in males, which were found to be less than expected (see Appendix D) [20].

The Texas Birth Defects Registry evaluated the birth defect prevalence per 10,000 live births for 48 specific, routinely-reported birth defects and for infants and fetuses with any birth defect monitored by the registry. The Leon Valley prevalence for years 1997 through 2003 was

compared to the prevalence for Texas overall for years 1999 through 2003. Based on crude prevalence rates, the following four birth defects were found to be higher among deliveries to residents of Leon Valley than for all Texas residents: encephalocele (neural tube defect), ventricular septal defect (congenital heart defect), stenosis or atresia of the small intestine, and reduction defects of the lower limbs. Leon Valley has fewer younger mothers, more Hispanic mothers, and fewer African-American and non-Hispanic white mothers than Texas overall. After adjusting for maternal age and race/ethnicity, the prevalence of these four birth defects remained elevated compared to the state. However, these elevations were not statistically significant, meaning they could be due to chance alone (see Appendix E) [21]. Neural tube defects and congenital heart defects have both been associated with exposure to TCE in drinking water; however, the specific defects in these studies were not identified [10]. Other associations between the elevated birth defects and contaminants of concern at this site were not located.

Children's Health Considerations

In communities faced with air, water, or food contamination, children could be at greater risk than are adults from certain kinds of exposure to hazardous substances. A child's lower body weight and higher intake rate result in a greater dose of hazardous substance per unit of body weight. Sufficient exposure levels during critical growth stages can result in permanent damage to the developing body systems of children. Children are dependent on adults for access to housing, for access to medical care, and for risk identification. Consequently, adults need as much information as possible to make informed decisions regarding their children's health.

ATSDR and DSHS evaluated the likelihood for children living near the Bandera Road Groundwater Plume to be exposed to site contaminants at levels of health concern. Children are most likely to be exposed to the site contaminants via the ingestion of drinking water from private wells or the inhalation or dermal contact with water from these wells when it is used in the home. They may have been exposed to contaminants in the well water before the problem was recognized. However, if the children's water supply has a properly installed and maintained filtration system or if they are connected to the public water supply, no current pathway for exposure exists.

Conclusions

1. We do not know how long residents using private wells in the Leon Valley area may have been exposed to contaminants in their drinking water and at what levels these exposures occurred. Based on lack of information, **past exposures to contaminants in private water wells pose an indeterminate public health hazard**. However, assuming past concentrations of PCE in the private wells are similar to current maximum concentrations, estimated exposure doses are well below levels that have been shown to cause adverse health effects in humans.
2. Based upon the current levels of trihalomethanes and vinyl chloride in the drinking water, if residents would be exposed to similar levels of these contaminants for 30 years, we would not expect to see non-cancerous or cancerous health effects due to exposure to these contaminants.
3. Currently, drinking water wells with concentrations of PCE and TCE that exceed the MCL are equipped with filtration systems that reduce concentrations of contaminants to below detection. In the future, these residents will be connected to the public water supply system for drinking water. Thus **current and future exposures to contaminants in private wells pose no apparent public health hazard**, provided the filtration systems are properly maintained while these wells are being used and residents do not use their private wells for drinking water after they are connected to the public water supply system.
4. The Texas Cancer Registry evaluated incidence of cancers in zip codes 78238 and 78240 for the years 1995 through 2003. Based upon state rates, no cancers were found to be elevated in males or females.
5. The Texas Birth Defects Registry evaluated the birth defect prevalence per 10,000 live births for 48 specific, routinely-reported birth defects and for infants and fetuses with any birth defect monitored by the registry. The Leon Valley prevalence for years 1997 through 2003 was compared to the prevalence for Texas overall for years 1999 through 2003. Based on crude prevalence rates, encephalocele, ventricular septal defect, stenosis or atresia of the small intestine, and reduction defects of the lower limbs were elevated. After adjusting for maternal age and race/ethnicity, the prevalence of these four birth defects remained elevated compared to the state. However, these elevations were not statistically significant, meaning they could be due to chance alone.

Recommendations

1. The EPA and the TCEQ should continue to monitor and maintain private well filtration systems to ensure proper operation until a safe alternative drinking water source can be provided.
2. The EPA and the TCEQ should continue to identify and sample water wells (private and public) in the Bandera Road area that are being used for drinking and other household purposes.
3. The EPA and the TCEQ should attempt to determine the source of the groundwater contamination or potentially responsible parties. Understanding where the contamination originated and how long potential sources have been in operation will help characterize past exposures.
4. The DSHS and the ATSDR should review any additional environmental sampling results as they become available to ensure there are no current exposures to contaminants.

Public Health Action Plan

Actions Completed

1. The groundwater plume was discovered in 2004 during assessment activities at a local shopping center under the TCEQ voluntary clean-up program. TCEQ initiated a Site Screening Investigation to determine the extent of contamination.
2. A total of 224 drinking water samples were collected from private water wells and two public water systems (providing water to a mobile home park and to the City of Leon Valley) from June 2004 through April 2007 and were analyzed for VOCs.
3. The TCEQ installed GAC water filtration systems on five private wells that had levels of contamination that exceeded MCLs. The owner of the sixth private well declined to have the GAC system installed. The filtration systems have been effective at reducing contamination to levels below the detection limit.
4. The EPA conducted availability sessions for the community on November 2, 2006 and July 24, 2007 to provide community members with information about the groundwater plume and the superfund process.
5. For private residences with drinking water wells that have concentrations that exceed MCLs, the EPA has offered to replace the existing water supply with connection to the Leon Valley or San Antonio water supply system. As of July 12, 2007, all residents with drinking water wells in which contaminants above MCLs were detected have accepted EPA's offer for hook-ups to the public water supply. Two of these connections have already occurred and efforts are being pursued to hook up the remaining locations to a local public water supply.
6. This document was made available to EPA and TCEQ for technical review.
7. From July 24, 2007 through August 24, 2007, the public was given the opportunity to make comments regarding the conclusions and recommendations of this health assessment document. No comments or concerns regarding this public health assessment document were received by DSHS.

Actions Planned

1. The contaminant filtration systems, installed by the TCEQ on the private wells, will be removed after the owners are connected to a public water supply.
2. DSHS will send letters explaining potential health effects from ingesting contaminated drinking water to homeowners/residents that refuse to connect with a public water supply.

Authors, Technical Advisors, and Organizations

Report Prepared by

Carrie M. Bradford, MS, PhD
Toxicologist
DSHS Health Assessment & Toxicology Program

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

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

Jennifer Lyke
Regional Representative
ATSDR Region 6

George Pettigrew, PE
Senior Regional Representative
ATSDR Region 6

Jeff Kellam
Environmental Health Scientist
Division of Health Assessment and Consultation
Cooperative Agreement Program Evaluation Branch

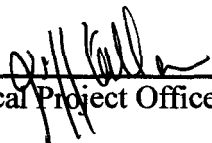
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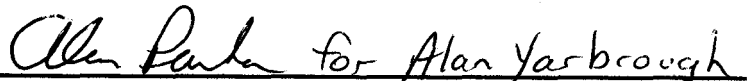
Certification

This public health assessment for the Bandera Road Groundwater Plume, Leon Valley, Bexar County, Texas was prepared by the Texas Department of State Health Services under a cooperative agreement with the Agency for Toxic Substances and Disease Registry (ATSDR). It is in accordance with approved methods and procedures existing when the time the public 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 consultation and concurs with its findings.



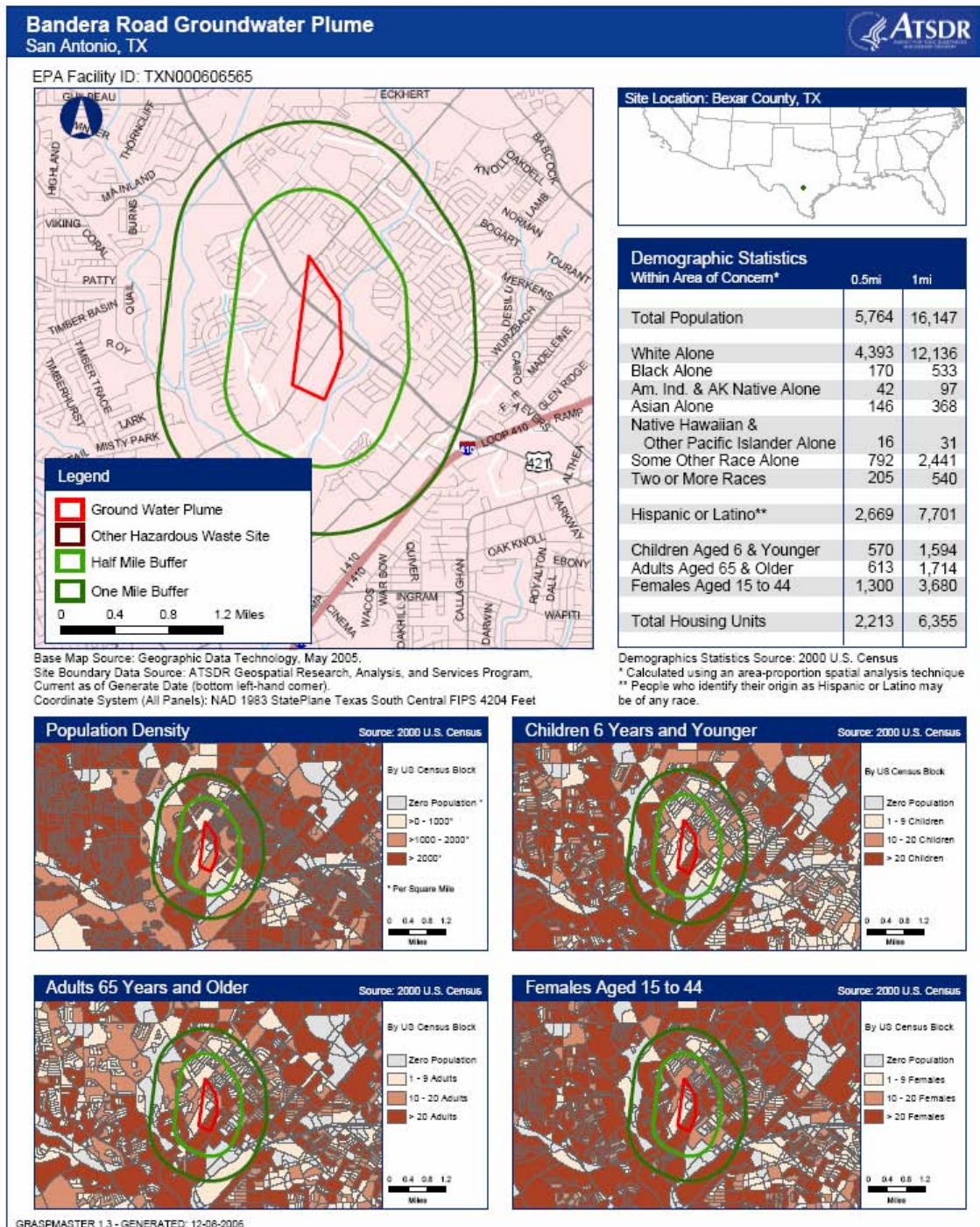
Team Lead, CAT, CAPEB, DHAC, ATSDR

Appendix A: Acronyms and Abbreviations

| | |
|-----------|--|
| ATSDR | Agency for Toxic Substances and Disease Registry |
| BDCM | bromodichloromethane |
| CERCLA | Comprehensive Environmental Response, Compensation and Liability Act of 1980 |
| CREGs | Cancer Risk Evaluation Guides |
| CSF | Cancer Slope Factor |
| DSHS | Texas Department of State Health Services |
| DSL | Datum Land Surface |
| EMEGs | Environmental Media Evaluation Guides |
| EPA | Environmental Protection Agency |
| GAC | granulated activated charcoal |
| HAC | Health-Based Assessment Comparison |
| kg | kilogram |
| L/day | liter per day |
| LOAELs | Lowest Observable Adverse Effect Levels |
| LTHAs | Lifetime Health Advisories |
| MCLs | Maximum Contaminant Levels |
| MCLG | Maximum Contaminant Level Goal |
| mg/kg/day | milligram per kilogram per day |
| MRLs | Minimal Risk Levels |
| NOAELs | No Observable Adverse Effect Levels |
| NPL | National Priorities List |
| PCE | perchloroethylene, tetrachloroethylene |
| PHA | Public Health Assessment |
| PVC | polyvinyl chloride |
| RfD | Reference Dose |
| RMEGs | Reference Dose Media Evaluation Guides |
| SARA | Superfund Amendments and Reauthorization Act of 1986 |
| TCE | trichloroethylene |
| TCEQ | Texas Commission on Environmental Quality |
| µg/L | microgram per liter |
| USGS | United States Geological Service |
| VOCs | volatile organic compounds |

Appendix B: Figure

Figure. Site location and demographic statistics for the Bandera Road Groundwater Plume [5].



Appendix C: Tables

Table 1. ATSDR Conclusion Categories.

| CATEGORY A. URGENT PUBLIC HEALTH HAZARD¹ | CATEGORY B. PUBLIC HEALTH HAZARD¹ | CATEGORY C. INDETERMINATE PUBLIC HEALTH HAZARD | CATEGORY D. NO APPARENT PUBLIC HEALTH HAZARD¹ | CATEGORY E. NO PUBLIC HEALTH HAZARD |
|--|---|--|--|---|
| <p>This category is used for sites where short-term exposures (<1 yr) to hazardous substances or conditions could result in adverse health effects that require rapid intervention.</p> <p>Criteria: Evaluation of available information² indicates that site-specific conditions or likely exposures have had, are having, or are likely to have in the future, an adverse impact on human health and requires immediate action or intervention. Such site-specific conditions or exposures might include the presence of serious physical or safety hazards, such as open mine shafts, poorly stored or maintained flammable/explosive substances, or medical devices which, upon rupture, could release radioactive materials.</p> | <p>This category is used for sites that pose a public health hazard due to the existence of long-term exposures(>1 yr) to hazardous substances or conditions that could result in adverse health effects.</p> <p>Criteria: Evaluation of available relevant information² suggests that, under site-specific conditions of exposure, long-term exposures to site-specific contaminants (including radionuclides) have had, are having, or are likely to have in the future, an adverse impact on human health that requires one or more public health interventions. Such site-specific exposures might include the presence of serious physical hazards, such as open mine shafts, poorly stored or maintained flammable/explosive substances, or medical devices, which, upon rupture, could release radioactive materials.</p> | <p>This category is used for sites in which critical data are <i>insufficient</i> with regard to extent of exposure and/or toxicologic properties at estimated exposure levels.</p> <p>Criteria: The health assessor must determine, using professional judgment, the criticality of such data and the likelihood that the data can be obtained and will be obtained in a timely manner. Where some data are available, even limited data, the health assessor is encouraged to the extent possible to select other hazard categories and to support their decision with clear narrative that explains the limits of the data and the rationale for the decision.</p> | <p>This category is used for sites where human exposure to contaminated media might be occurring, might have occurred in the past, and/or might occur in the future, but the exposure is not expected to cause any adverse health effects.</p> <p>Criteria: Evaluation of available information² indicates that, under site-specific conditions of exposure, exposures to site-specific contaminants in the past, present, or future are not likely to result in any adverse impact on human health.</p> | <p>This category is used for sites that, because of the absence of exposure, do NOT pose a public health hazard.</p> <p>Criteria: Sufficient evidence indicates that no human exposures to contaminated media have occurred, none are now occurring, and none are likely to occur in the future.</p> |

¹ Each of these designations represents a professional judgment made on the basis of critical data that ATSDR regards as sufficient to support a decision. It does not imply, however, that the available data are necessarily complete; in some cases, additional data may be required to confirm or further support the decision.

² Examples include environmental and demographic data; health outcome data; community health concerns information; toxicologic, medical, and epidemiologic data.

Table 2. Exposure Pathway Evaluation of the Bandera Road Groundwater Plume NPL site.

| Pathway Name | Contaminants of Concern | EXPOSURE PATHWAY ELEMENTS | | | | | Time | Comments |
|--------------|-------------------------|-------------------------------------|-----------------|------------------------------|--|--|-------------------------------|---|
| | | | Transport Media | Point of Exposure | Route of Exposure | Exposed Population | | |
| Groundwater | VOCs Source | chemical release (location unknown) | groundwater | private drinking water wells | ingestion inhalation ¹ dermal | residents employees at local businesses | past present future | complete – contamination was detected in private wells used for household purposes eliminated - with properly installed, operating, and maintained filtration systems or an alternative water supply (when city water becomes available) |
| Groundwater | VOCs | chemical release (location unknown) | groundwater | public water supply wells | ingestion inhalation ¹ dermal | residents employees at local businesses | past present future | eliminated – contamination has not been detected in the public water supply potential – migration of the groundwater plume may result in contamination in the public water supply |

¹ inhalation of contaminants in soil, groundwater, and surface water occurs when those substances volatilize (changing to a gas) from these media, for example, when taking a shower and using groundwater from a private well.

Table 2. Continued.

| Pathway Name | Contaminants of Concern | EXPOSURE PATHWAY ELEMENTS | | | | | Time | Comments |
|---------------|-------------------------|-------------------------------------|-----------------|--------------------|--|--|---------------------------|--|
| | | | Transport Media | Point of Exposure | Route of Exposure | Exposed Population | | |
| ambient air | no data Source | chemical release (location unknown) | air | ambient air | inhalation | residents employees at local businesses | past present future | No data available to evaluate pathway; however, not considered to be a significant pathway |
| soil | no data | chemical release (location unknown) | soil | residential yards | ingestion inhalation ¹ dermal | residents employees at local businesses | past present future | No data available to evaluate pathway; however, not considered to be a significant pathway |
| biota | no data | chemical release (location unknown) | food | locally grown food | ingestion | residents employees at local businesses | past present future | No data available to evaluate pathway; however, not considered to be a significant pathway |
| surface water | no data | chemical release (location unknown) | surface water | recreational use | ingestion inhalation ¹ dermal | residents employees at local businesses | past present future | No data available to evaluate pathway; however, not considered to be a significant pathway |
| sediment | no data | chemical release (location unknown) | sediment | recreational use | ingestion dermal | residents employees at local businesses | past present future | No data available to evaluate pathway; however, not considered to be a significant pathway |

¹ inhalation of contaminants in soil, groundwater, and surface water occurs when those substances volatilize (changing to a gas) from these media, for example, when taking a shower and using groundwater from a private well.

Table 3. Contaminants that exceeded HAC values in drinking water samples (224 samples). All other compounds were below the detection limit or, if detected, below the HAC value. Data from the following sampling events were analyzed in this PHA: June 2004, August 2004, December 2004, March 2005, November 2005, December 2005, May 2006, November 2006, and April 2007.

| Contaminant | HAC Value (µg/L) | Concentration Range (µg/L) | | Number of Samples that Exceed HAC Value |
|----------------------|------------------|----------------------------|-------------------------------|---|
| | | Prior to Filtration | After Filtration ^a | |
| PCE | 5 (MCL) | ND-115.2 | ND-5.05 | 31 |
| | 10 (LTHA) | | | 28 |
| | 100 (child RMEG) | | | 3 |
| TCE | 5 (MCL) | ND-7.53 | ND-3.81 | 5 |
| BDCM | 0.6 (CREG) | ND-6.95 | NA | 10 |
| Bromoform | 4 (CREG) | ND-6.80 | NA | 1 |
| Dibromochloromethane | 0.4 (CREG) | ND-13.75 | NA | 12 |
| Vinyl Chloride | 0.03 (CREG) | ND-0.99 | NA | 1 |

^a For PCE and TCE, also includes samples from wells that do not have a filtration system.

ND – Not Detected

NA – Not Applicable. No filtration system in place on wells in which these contaminants were detected.

Appendix D: Cancer Occurrence Investigation

Summary of Investigation into the Occurrence of Cancer Zip Codes 78238 and 78240, Leon Valley and San Antonio Bexar County, Texas 1995–2003 January 3, 2007

Background: Concern about a possible excess of cancer prompted the Texas Cancer Registry (TCR) of the Texas Department of State Health Services (DSHS) to examine the occurrence of cancer in zip codes 78238 and 78240, Leon Valley and San Antonio, Texas. Local citizens were concerned that tetrachloroethene, trichloroethene, dichloroethene, and vinyl chloride from the Bandera Road Groundwater Plume superfund site may be causing cancer. The TCR evaluated 1995–2003 incidence data for cancers of the prostate, breast, lung and bronchus, colon and rectum, bladder, kidney and renal pelvis, liver and intrahepatic bile duct, non-Hodgkin’s lymphoma, brain and other nervous system (brain/CNS), selected leukemia subtypes, larynx, esophagus, cervix, Hodgkin’s lymphoma, myeloma, testis, and stomach. The scientific literature has found the above cancer sites to be associated with exposure to tetrachloroethene, trichloroethene, dichloroethene, or vinyl chloride. Incidence data are the best indicator of the occurrence of cancer in an area because they more accurately show the number and types of cancer diagnosed each year than mortality data. Compared with previous investigations that included mortality data as a supplemental measure, the TCR now solely uses incidence data for assessment of possible cancer clusters. This is due to the improved timeliness, quality, and availability of incidence data which now also meet national standards for high data quality. The rest of this report examines the investigative methods the TCR used, the results of the investigation, recommendations, and general information on cancer risk factors.

Methodology: According to the National Cancer Institute, a cancer cluster is a greater than expected number of cancers among people who live or work in the same area and who develop or die from the same cancer within a short time of each other. The cancer cluster investigation is the primary tool used by the TCR to investigate the possibility of excess cancer in a community. The cancer cluster investigation cannot determine that cancer was associated with or caused by environmental or other risk factors. Instead, the cancer cluster investigation is specifically intended to address the question “Is there an excess of cancer in the area or population of concern?”

The TCR follows guidelines recommended by the Centers for Disease Control and Prevention for investigating cancer clusters¹ and often works with the DSHS Environmental and Injury Epidemiology and Toxicology Branch, as well as other state and federal agencies. In order to determine if an excess of cancer is occurring and if further study is recommended, biologic and epidemiologic evidence are considered. Such evidence may include documented exposures; the toxicity of the exposures; plausible routes by which exposures can reach people (ingesting, touching, breathing); the actual amount of exposure to the people which can lead to absorption in

the body; the time from exposure to development of cancer; the statistical significance of the findings; the magnitude of the effect observed; risk factors; and the consistency of the findings over time. The occurrence of rare cancers or unlikely cancers in certain age groups may also indicate a cluster needing further study. Because excesses of cancer may occur by chance alone, the role of chance is considered in the statistical analysis.

If further study is indicated, the TCR will determine the feasibility of conducting an epidemiologic study. If the epidemiologic study is feasible, the final step is to recommend and/or perform an etiologic investigation to see if the cancer(s) can be related to the exposure of concern. Very few cancer cluster investigations in the United States proceed to this stage.

To determine whether a statistically significant excess of cancer existed in the geographic areas of concern, the number of observed cases was compared to what would be "expected" based on the state cancer rates. Calculating the expected number(s) of cancer cases takes into consideration the race, sex, and ages of people who are diagnosed with cancer. This is important because a person's race, sex, and age all impact cancer rates. If we are trying to determine if there is more or less cancer in a community compared to the rest of the state, we must make sure that the difference in cancer rates is not simply due to one of these factors.

The attached Tables 1–4 present the number of observed cases for males and females, the number of "expected" cases, the standardized incidence ratio (SIR), and the corresponding 99% confidence interval. The standardized incidence ratio (SIR) is simply the number of observed cases compared to the number of "expected" cases. When the SIR of a selected cancer is equal to 1.00, then the number of observed cases is equal to the expected number of cases, based on the incidence in the rest of the state. When the SIR is less than 1.00, fewer people developed cancer than we would have expected. Conversely, an SIR greater than 1.00 indicates that more people developed cancer than we would have expected. To determine if an SIR greater than 1.00 or less than 1.00 is statistically significant or outside the variation likely to be due to chance, confidence intervals are also calculated.

A 99% confidence interval is used for statistical significance and takes into account the likelihood that the result occurred by chance. It also indicates the range in which we would expect the SIR to fall 99% of the time. If the confidence interval contains a range that includes 1.00, no statistically significant excess of cancer is indicated. The confidence intervals are particularly important when trying to interpret small numbers of cases. If only one or two cases are expected for a particular cancer, then the report of three or four observed cases will result in a very large SIR. As long as the 99% confidence interval contains 1.00, this indicates that the SIR is still within the range one might expect and, therefore, not statistically significant.

Results: The analysis of incidence data for zip code 78238, Leon Valley, Texas, from January 1, 1995–December 31, 2003, found cancers of the prostate, breast, lung, colon and rectum, bladder, kidney and renal pelvis, liver and intrahepatic bile duct, non-Hodgkin's lymphoma, brain/CNS, selected leukemia subtypes, larynx, esophagus, cervix, Hodgkin's lymphoma, myeloma, testis, and stomach to be within expected ranges in both males and females. Analysis summaries are presented in Tables 1–2.

During the same time period, analysis of incidence data for zip code 78240, San Antonio, Texas, found cancers of the prostate, breast, colon and rectum, bladder, kidney and renal pelvis, non-Hodgkin's lymphoma, brain/CNS, selected leukemia subtypes, larynx, esophagus, cervix, Hodgkin's lymphoma, myeloma, testis, and stomach to be within expected ranges in both males and females. Lung cancer in females and liver and intrahepatic bile duct cancer in males were found to be statistically significantly less than expected. Analysis summaries are presented in Table 3–4.

Discussion: Like other studies, this cancer cluster investigation had limitations. The incidence data did not include data for the most recent years. Also, cancer incidence data are based on residence at the time of diagnosis. It is possible that some residents who developed cancer no longer lived in the area at the time of diagnosis, so were not included in the analyses. However, it is also possible that people may have moved into the area and then developed cancer because of an exposure from a prior residential location or other factors. These cases are included in the investigation.

Recommendations: Based on the findings and the information discussed above, it is not recommended at this time to further examine the cancers in zip codes 78238 and 78240, Leon Valley and San Antonio, Texas. As new data or additional information become available, consideration will be given to updating or re-evaluating this investigation.

Information on Cancer and Cancer Risk Factors: Overall, the occurrence of cancer is common, with approximately two out of every five persons alive today predicted to develop some type of cancer in their lifetime.² In Texas, as in the United States, cancer is the leading cause of death for people under the age of 85.³ Also, cancer is not one disease, but many different diseases. Different types of cancer are generally thought to have different causes. If a person develops cancer, it is probably not due to one factor but to a combination of factors such as heredity; diet, tobacco use, and other lifestyle factors; infectious agents; chemical exposures; and radiation exposures. Although cancer may impact individuals of all ages, it primarily is a disease of older persons with over one-half of cancer cases and two-thirds of cancer deaths occurring in persons 65 and older. Finally, it takes time for cancer to develop, between 10–40 years can go by between the exposure to a carcinogen and a diagnosis of cancer.⁴

The chances of a person developing cancer as a result of exposure to an environmental contaminant are slight. Most experts agree that exposure to pollution, occupational, and industrial hazards account for fewer than 10% of cancer cases.⁵ The Harvard Center for Cancer Prevention estimates 5% of cancer deaths are due to occupational factors, 2% to environmental pollution and 2% to ionizing/ultraviolet radiation.⁶ In contrast, the National Cancer Institute estimates that lifestyle factors such as tobacco use and diet cause 50 to 75 percent of cancer deaths.⁷ Eating a healthy diet and refraining from tobacco are the best ways to prevent many kinds of cancer. It is estimated that one-third of all cancer deaths in this country could be prevented by eliminating the use of tobacco products. Additionally, about 25 to 30 percent of the cases of several major cancers are thought to be associated with obesity and physical inactivity.⁸

Known Risk Factors for Cancers Examined in This Investigation: The following is a brief discussion summarized from the American Cancer Society and the National Cancer Institute about cancer risk factors for the specific cancers studied in this investigation.^{9,10}

The occurrence of cancer may vary by race/ethnicity, gender, type of cancer, geographic location, population group, and a variety of other factors. Scientific studies have identified a number of factors for various cancers that may increase an individual's risk of developing a specific type of cancer. These factors are known as risk factors. Some risk factors we can do nothing about, but many are a matter of choice.

Breast Cancer: Simply being a woman is the main risk factor for developing breast cancer. Breast cancer can affect men, but this disease is about 100 times more common among women than men. White women are slightly more likely to develop breast cancer than are African-American women, but African Americans are more likely to die of this cancer because they are often diagnosed at an advanced stage when breast cancer is harder to treat and cure. Other risk factors for breast cancer include aging, presence of genetic markers such as the BRCA1 and BRCA2 genes, personal and family history of breast cancer, previous breast biopsies, previous breast irradiation, diethylstilbestrol therapy, oral contraceptive use, not having children, hormone replacement therapy, drinking alcohol, and obesity. Currently, research does not show a link between breast cancer risk and environmental pollutants such as the pesticide DDE (chemically related to DDT) and PCBs (polychlorinated biphenyls).

Prostate Cancer: Prostate cancer is the most common type of malignant cancer (other than skin) diagnosed in men, affecting an estimated one in five American men. Risk factors for prostate cancer include aging, a high fat diet, physical inactivity, and a family history of prostate cancer. African American men are at higher risk of acquiring prostate cancer and dying from it. Prostate cancer is most common in North America and northwestern Europe. It is less common in Asia, Africa, Central America, and South America.

Colon and Rectum Cancer: Researchers have identified several risk factors that increase a person's chance of developing colon cancer: family and personal history of colon cancer, hereditary conditions such as familial adenomatous polyposis, personal history of intestinal polyps and chronic inflammatory bowel disease, aging, a diet mostly from animal sources, physical inactivity, obesity, smoking, and heavy use of alcohol. People with diabetes have a 30%-40% increased chance of developing colon cancer. Recent research has found a genetic mutation leading to colorectal cancer in Jews of Eastern European descent (Ashkenazi Jews).

Lung and Bronchus Cancer: The greatest single risk factor for lung cancer is smoking. The American Cancer Society estimates that 87% of lung cancer is due to smoking. Several studies have shown that the lung cells of women have a genetic predisposition to develop cancer when they are exposed to tobacco smoke. Other risk factors include secondhand smoke, asbestos exposure, radon exposure, other carcinogenic agents in the workplace such as arsenic or vinyl chloride, marijuana smoking, recurring inflammation of the lungs, exposure to industrial grade talc, people with silicosis and berylliosis, personal and family history of lung cancer, and diet. In

some cities, air pollution may slightly increase the risk of lung cancer. This risk is far less than that caused by smoking.

Bladder Cancer: The greatest risk factor for bladder cancer is smoking. Men get bladder cancer at a rate four times that of women. Smokers are more than twice as likely to get bladder cancer as nonsmokers. Whites are two times more likely to develop bladder cancer than are African Americans. Other risk factors for bladder cancer include occupational exposure to aromatic amines such as benzidine and beta-naphthylamine, aging, chronic bladder inflammation, personal history of urothelial carcinomas, birth defects involving the bladder and umbilicus, infection with a certain parasite, high doses of certain chemotherapy drugs, and arsenic in your drinking water.

Kidney and Renal Pelvis Cancer: Kidney cancer risk factors include smoking, obesity, a sedentary lifestyle, occupational exposure to heavy metals or organic solvents, advanced kidney disease, family history, high blood pressure, certain medications, and aging. Men have higher rates of kidney cancer.

Hodgkin's Lymphoma: Some people who have reduced immune systems, for example, those with AIDS, and organ transplant patients, are at a higher risk of Hodgkin's lymphoma. Possible risk factors include being in young or late adulthood, being male, being infected with the Epstein-Barr virus, or having a first-degree relative with Hodgkin's lymphoma.

Non-Hodgkin's Lymphoma: Risk factors for non-Hodgkin's lymphoma include infection with *Helicobacter pylori*, human immunodeficiency virus (HIV), human T-cell leukemia/lymphoma virus (HTVL-1), or the Epstein-Barr virus and malaria. Other possible risk factors include aging, certain genetic diseases, radiation exposure, immuno-suppressant drugs after organ transplantation, benzene exposure, the drug Dilantin, exposure to certain pesticides, a diet high in meats or fat, or certain chemotherapy drugs.

Multiple Myeloma: The risk factors for multiple myeloma include aging, radiation exposure, family history, exposure from petroleum-related industry, obesity, or other plasma cell diseases. African Americans have higher rates of multiple myeloma.

Brain/CNS Cancer: The large majority of brain cancers are not associated with any risk factors. Most brain cancers simply happen for no apparent reason. A few risk factors associated with brain cancer are known and include radiation treatment, occupational exposure to vinyl chloride, immune system disorders, and family history of brain and spinal cord cancers. Exposure to aspartame (a sugar substitute) and exposure to electromagnetic fields from cellular telephones or high-tension wires have been suggested as risk factors, but most researchers agree that there is no convincing evidence to link these factors.

Liver and Intrahepatic Bile Duct Cancer: In contrast to many other types of cancer, the number of people who develop liver cancer and die from it is increasing. This cancer is about 10 times more common in developing countries. The risk factors for liver cancer include viral hepatitis, cirrhosis, long-term exposure to aflatoxin, exposure to vinyl chloride and thorium dioxide, older forms of birth control pills, anabolic steroids, arsenic in drinking water, tobacco use, bile duct

disease, ulcerative colitis, liver fluke infection, and aging. Chemicals that are associated with bile duct cancer include dioxin, nitrosamines, and polychlorinated biphenyls (PCBs).

Esophageal Cancer: Compared with women, men have a three-fold higher rate of esophageal cancer. African Americans are two times more likely to have esophageal cancer than whites. Other risk factors for esophageal cancer include aging, use of tobacco products, alcohol, obesity, gastric reflux, diets low in fruits and vegetables, lye ingestion, frequent drinking of very hot liquids, achalasia, tylosis, and esophageal webs.

Stomach Cancer: Stomach cancer is about twice as common in men as it is in women. Other risk factors for stomach cancer include *Helicobacter pylori* infection, diets high in smoked and salted foods, tobacco and alcohol abuse, previous stomach surgery, pernicious anemia, type A blood, familial cancer syndromes, aging, and stomach polyps. Japanese have a very high rate of stomach cancer when they live in Japan. If they move to the United States, the rate goes down after a number of years, but still remains higher than that of people born in the U.S.

Testicular Cancer: Risk factors for testicular cancer include cryptorchidism (undescended testicles), family history, multiple atypical nevi, HIV infection, cancer of the other testicle, and being non-Hispanic white. Most testicular cancers occur between the ages of 15 and 40.

Laryngeal Cancer: Risk factors for laryngeal and hypopharynx cancer include tobacco use, alcohol abuse, poor nutrition, infection with human papillomavirus, a weakened immune system, occupational exposure, gastroesophageal reflux disease, aging, and being male. Cancers of the larynx are about 50% more common among African Americans than among whites.

Cervical Cancer: Risk factors for cancers of the cervix include infections with the human papillomavirus, chlamydia, or HIV, early sexual activity, smoking, diets low in fruits and vegetables, low socioeconomic status, exposure to diethylstilbesterol (DES) in the womb, and family history of cancers of the cervix.

Acute Lymphocytic Leukemia (ALL): Possible risk factors for ALL include the following: being male, being white, being older than 70 years of age, past treatment with chemotherapy or radiation therapy, exposure to atomic bomb radiation, or having a certain genetic disorder such as Down syndrome.

Chronic Lymphocytic Leukemia (CLL): Possible risk factors for CLL include the following: being middle-aged or older, male, or white; a family history of CLL or cancer of the lymph system; having relatives who are Russian Jews or Eastern European Jews; or having exposure to herbicides or insecticides including Agent Orange, an herbicide used during the Vietnam War.

Acute Myeloid Leukemia (AML): Possible risk factors for AML include the following: being male; smoking, especially after age 60; having had treatment with chemotherapy or radiation therapy in the past; having had treatment for childhood ALL in the past; being exposed to atomic bomb radiation or the chemical benzene; or having a history of a blood disorder such as myelodysplastic syndrome.

Chronic Myeloid Leukemia (CML): Most people with CML have a gene mutation (change) called the Philadelphia chromosome. The Philadelphia chromosome is not passed from parent to child.

For additional information about cancer, visit the “Resources” link on our web site at <http://www.dshs.state.tx.us/tcr/>.

Questions or comments regarding this investigation may be directed to Ms. Brenda Mokry, Texas Cancer Registry, at 1-800-252-8059 or brenda.mokry@dshs.state.tx.us.

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Table 1

Number of Observed and Expected Cancer Cases and Race Adjusted Standardized Incidence Ratios, Selected Cancers, Zip Code 78238, Leon Valley, TX, 1995–2003

| Males | | | | |
|----------------------------------|----------|----------|------|--------------|
| Site | Observed | Expected | SIR | 99% CI |
| Breast | 0 | 0.94 | 0.00 | 0.00 – 5.63 |
| Prostate | 109 | 87.96 | 1.24 | 0.95 – 1.58 |
| Lung and Bronchus | 57 | 53.95 | 1.06 | 0.73 – 1.48 |
| Colon and Rectum | 44 | 36.28 | 1.21 | 0.79 – 1.77 |
| Bladder | 18 | 16.51 | 1.09 | 0.54 – 1.94 |
| Kidney and Renal Pelvis | 23 | 13.05 | 1.76 | 0.96 – 2.95 |
| Hodgkin's Lymphoma | 2 | 2.33 | 0.86 | 0.04 – 3.98 |
| Non-Hodgkin's Lymphoma | 15 | 13.76 | 1.09 | 0.50 – 2.05 |
| Liver and Intrahepatic Bile Duct | 14 | 8.00 | 1.75 | 0.78 – 3.35 |
| Brain/CNS | 3 | 5.52 | 0.54 | 0.06 – 1.99 |
| Esophagus | 3 | 4.41 | 0.68 | 0.08 – 2.49 |
| Larynx | 6 | 5.40 | 1.11 | 0.28 – 2.90 |
| Testis | 5 | 4.17 | 1.20 | 0.26 – 3.39 |
| Stomach | 12 | 7.56 | 1.59 | 0.65 – 3.19 |
| Myeloma | 6 | 4.28 | 1.40 | 0.36 – 3.66 |
| Acute Lymphocytic Leukemia | 2 | 1.85 | 1.08 | 0.06 – 5.01 |
| Chronic Lymphocytic Leukemia | 1 | 2.68 | 0.37 | 0.00 – 2.77 |
| Acute Myeloid Leukemia | 3 | 2.78 | 1.08 | 0.12 – 3.95 |
| Chronic Myeloid Leukemia | 1 | 1.43 | 0.70 | 0.00 – 5.21 |
| Aleukemic, Subleukemic, & NOS | 1 | 0.53 | 1.90 | 0.01 – 14.14 |

Note: The SIR (standardized incidence ratio) is defined as the number of observed cases divided by the number of expected cases. The latter is based on race-, sex-, and age-specific cancer incidence rates for Texas during the period 1995–2003. The SIR has been rounded to the second decimal place.

*Significantly higher than expected at the $p < 0.01$ level.

**Significantly lower than expected at the $p < 0.01$ level.

Table 2

Number of Observed and Expected Cancer Cases and Race Adjusted Standardized Incidence Ratios, Selected Cancers, Zip Code 78238, Leon Valley, TX, 1995–2003

| Females | | | | |
|----------------------------------|----------|----------|------|--------------|
| Site | Observed | Expected | SIR | 99% CI |
| Breast | 116 | 96.24 | 1.21 | 0.94 – 1.52 |
| Lung and Bronchus | 42 | 37.91 | 1.11 | 0.72 – 1.63 |
| Colon and Rectum | 41 | 33.35 | 1.23 | 0.79 – 1.82 |
| Bladder | 6 | 5.65 | 1.06 | 0.27 – 2.77 |
| Kidney and Renal Pelvis | 7 | 9.05 | 0.77 | 0.23 – 1.89 |
| Hodgkin's Lymphoma | 1 | 1.84 | 0.54 | 0.00 – 4.03 |
| Non-Hodgkin's Lymphoma | 15 | 13.15 | 1.14 | 0.52 – 2.14 |
| Liver and Intrahepatic Bile Duct | 6 | 3.82 | 1.57 | 0.40 – 4.10 |
| Brain/CNS | 3 | 4.80 | 0.63 | 0.07 – 2.29 |
| Esophagus | 1 | 1.35 | 0.74 | 0.00 – 5.50 |
| Larynx | 0 | 1.14 | 0.00 | 0.00 – 4.65 |
| Cervix | 9 | 11.58 | 0.78 | 0.27 – 1.73 |
| Stomach | 6 | 5.36 | 1.12 | 0.29 – 2.92 |
| Myeloma | 8 | 3.93 | 2.03 | 0.65 – 4.72 |
| Acute Lymphocytic Leukemia | 2 | 1.51 | 1.33 | 0.07 – 6.15 |
| Chronic Lymphocytic Leukemia | 1 | 1.98 | 0.50 | 0.00 – 3.75 |
| Acute Myeloid Leukemia | 3 | 2.39 | 1.25 | 0.14 – 4.59 |
| Chronic Myeloid Leukemia | 4 | 1.10 | 3.65 | 0.61 – 11.48 |
| Aleukemic, Subleukemic, & NOS | 0 | 0.55 | 0.00 | 0.00 – 9.62 |

Note: The SIR (standardized incidence ratio) is defined as the number of observed cases divided by the number of expected cases. The latter is based on race-, sex-, and age-specific cancer incidence rates for Texas during the period 1995–2003. The SIR has been rounded to the second decimal place.

*Significantly higher than expected at the $p < 0.01$ level.

**Significantly lower than expected at the $p < 0.01$ level.

Table 3
Number of Observed and Expected Cancer Cases and Race Adjusted Standardized Incidence Ratios, Selected Cancers, Zip Code 78240, San Antonio, TX, 1995–2003

| Males | | | | |
|--|----------|----------|--------|-------------|
| Site | Observed | Expected | SIR | 99% CI |
| Breast | 1 | 1.85 | 0.54 | 0.00 – 4.02 |
| Prostate | 189 | 161.41 | 1.17 | 0.96 – 1.41 |
| Lung and Bronchus | 83 | 104.35 | 0.80 | 0.59 – 1.05 |
| Colon and Rectum | 68 | 69.34 | 0.98 | 0.70 – 1.33 |
| Bladder | 24 | 34.01 | 0.71 | 0.39 – 1.17 |
| Kidney and Renal Pelvis | 21 | 23.38 | 0.90 | 0.47 – 1.54 |
| Hodgkin's Lymphoma | 5 | 5.14 | 0.97 | 0.21 – 2.75 |
| Non-Hodgkin's Lymphoma | 32 | 27.42 | 1.17 | 0.70 – 1.81 |
| Liver and Intrahepatic Bile Duct | 4 | 12.83 | 0.31** | 0.05 – 0.98 |
| Brain/CNS | 9 | 11.54 | 0.78 | 0.27 – 1.73 |
| Esophagus | 6 | 8.27 | 0.73 | 0.19 – 1.89 |
| Larynx | 10 | 9.68 | 1.03 | 0.38 – 2.21 |
| Testis | 5 | 10.75 | 0.47 | 0.10 – 1.32 |
| Stomach | 8 | 12.61 | 0.63 | 0.20 – 1.47 |
| Myeloma | 6 | 7.76 | 0.77 | 0.20 – 2.02 |
| Acute Lymphocytic Leukemia | 2 | 3.60 | 0.56 | 0.03 – 2.58 |
| Chronic Lymphocytic Leukemia | 3 | 5.58 | 0.54 | 0.06 – 1.97 |
| Acute Myeloid Leukemia | 4 | 5.63 | 0.71 | 0.12 – 2.24 |
| Chronic Myeloid Leukemia | 3 | 2.94 | 1.02 | 0.11 – 3.74 |
| Aleukemic, Subleukemic, & NOS | 0 | 1.24 | 0.00 | 0.00 – 4.27 |

Note: The SIR (standardized incidence ratio) is defined as the number of observed cases divided by the number of expected cases. The latter is based on race-, sex-, and age-specific cancer incidence rates for Texas during the period 1995–2003. The SIR has been rounded to the second decimal place.

*Significantly higher than expected at the $p < 0.01$ level.

**Significantly lower than expected at the $p < 0.01$ level.

Table 4

Number of Observed and Expected Cancer Cases and Race Adjusted Standardized Incidence Ratios, Selected Cancers, Zip Code 78240, San Antonio, TX, 1995–2003

| Females | | | | |
|----------------------------------|----------|----------|--------|-------------|
| Site | Observed | Expected | SIR | 99% CI |
| Breast | 198 | 204.03 | 0.97 | 0.80 – 1.16 |
| Lung and Bronchus | 49 | 82.67 | 0.59** | 0.40 – 0.85 |
| Colon and Rectum | 58 | 74.95 | 0.77 | 0.54 – 1.08 |
| Bladder | 8 | 13.15 | 0.61 | 0.20 – 1.41 |
| Kidney and Renal Pelvis | 11 | 17.32 | 0.64 | 0.25 – 1.32 |
| Hodgkin's Lymphoma | 4 | 4.49 | 0.89 | 0.15 – 2.81 |
| Non-Hodgkin's Lymphoma | 29 | 27.90 | 1.04 | 0.61 – 1.65 |
| Liver and Intrahepatic Bile Duct | 7 | 6.99 | 1.00 | 0.29 – 2.45 |
| Brain/CNS | 8 | 10.84 | 0.74 | 0.24 – 1.71 |
| Esophagus | 1 | 2.96 | 0.34 | 0.00 – 2.51 |
| Larynx | 0 | 2.39 | 0.00 | 0.00 – 2.22 |
| Cervix | 15 | 23.00 | 0.65 | 0.30 – 1.22 |
| Stomach | 9 | 9.88 | 0.91 | 0.32 – 2.03 |
| Myeloma | 4 | 7.93 | 0.50 | 0.08 – 1.59 |
| Acute Lymphocytic Leukemia | 2 | 2.92 | 0.69 | 0.04 – 3.18 |
| Chronic Lymphocytic Leukemia | 7 | 4.81 | 1.46 | 0.42 – 3.57 |
| Acute Myeloid Leukemia | 5 | 5.26 | 0.95 | 0.20 – 2.69 |
| Chronic Myeloid Leukemia | 2 | 2.45 | 0.82 | 0.04 – 3.79 |
| Aleukemic, Subleukemic, & NOS | 0 | 1.49 | 0.00 | 0.00 – 3.55 |

Note: The SIR (standardized incidence ratio) is defined as the number of observed cases divided by the number of expected cases. The latter is based on race-, sex-, and age-specific cancer incidence rates for Texas during the period 1995–2003. The SIR has been rounded to the second decimal place.

*Significantly higher than expected at the $p < 0.01$ level.

**Significantly lower than expected at the $p < 0.01$ level.

Prepared by:
 Brenda J. Mokry, Epidemiologist
 Texas Cancer Registry Branch
 Department of State Health Services
 01/03/2007

Appendix E: Birth Defects Occurrence Investigation

BIRTH DEFECT INVESTIGATION REPORT Birth Defects among Deliveries to Residents of Zip Codes 78238 & 78240 (Leon Valley), 1997-2003

Prepared January 25, 2007 by Mary Ethen, Epidemiologist
Birth Defects Epidemiology and Surveillance Branch
Texas Department of State Health Services

BACKGROUND

This analysis was requested by the Environmental and Injury Epidemiology and Toxicology Branch, Texas Department of State Health Services, for inclusion in a Health Outcomes Data Review their staff is preparing. The area of interest, defined as zip codes 78238 and 78240 in Leon Valley (which is in Bexar County), is a federal superfund site. A groundwater plume was detected in (June?) 2004. Contaminants of concern at the site are tetrachloroethene (PCE), trichloroethene (TCE), cis 1,2-dichloroethene (DCE), and vinyl chloride.

METHODS

Case Definition

The area of interest is in Bexar County. The Texas Birth Defects Registry began collecting information in this part of the state with deliveries in January 1997, and the most recent delivery year for which the registry has completed data collection is 2003. This time frame pre-dates the detection of the groundwater plume, however.

A case was defined as an infant or fetus...

- with any of 48 specific, routinely-reported birth defects, or with any birth defect monitored by the registry;
- born during 1997-2003;
- born to a mother whose residence address zip code at the time of delivery was 78238 or 78240.

Case Finding

The Texas Birth Defects Registry was searched to find cases meeting the case definition. The mother's residence address zip code at the time of delivery was based on information reported on the child's birth or fetal death certificate, when available. If a birth or fetal death certificate could not be found, the mother's residence zip code at the time of delivery was based on information in the Texas Birth Defects Registry that had been abstracted from hospital medical records.

Occurrence Evaluation

Crude Prevalence: Cases in the registry were used to calculate the birth prevalence per 10,000 live births for 48 specific, routinely-reported birth defects and for infants and fetuses with any birth defect monitored by the registry. The 95% confidence interval for each prevalence was calculated based on the Poisson distribution. In order to determine whether there was a statistically significant elevation in the occurrence of birth defects in Leon Valley during 1997-2003, the Leon Valley prevalences were compared to prevalences for Texas overall during 1999-2003. Prevalences were considered statistically significantly different if their 95% confidence intervals did not overlap. The time frame for the Texas comparison data was 1999-2003 because the Texas Birth Defects Registry was not statewide until 1999.

Adjusted Prevalence: The occurrence of many types of birth defects is known to vary between mothers of different age or racial/ethnic groups. For those birth defects that were statistically significantly elevated in Leon Valley based on crude prevalences, we calculated prevalences adjusted simultaneously for maternal age group and maternal racial/ethnic group. This adjustment accounts for differences in the maternal age and racial/ethnic composition of populations being compared, in this case, differences between Leon Valley during 1997-2003 and Texas during 1999-2003.

Using the direct method of standardization, birth defect prevalences for Leon Valley during 1997-2003 by maternal age and race/ethnic group were standardized (adjusted) to the maternal age and race/ethnic distribution of all Texas resident live births during 1999-2003. The resulting adjusted prevalences are the hypothetical prevalences that would have been observed in Leon Valley during 1997-2003 if it had the same maternal age and racial/ethnic distribution as Texas during 1999-2003. The DIRST module of Computer Programs for Epidemiologists¹, version 4.0, was used to calculate directly standardized prevalences and their associated 95% confidence intervals.

RESULTS

We examined the occurrence of 48 specific types of routinely-reported birth defects and any birth defect monitored by the registry among deliveries during 1997-2003 to women whose residence at the time of delivery was in Leon Valley (defined as zip code 78238 or 78240). Based on crude prevalences, four types of birth defects were statistically significantly higher among deliveries to residents of Leon Valley during 1997-2003 than for all Texas residents during 1999-2003 (Table 1). These defects were encephalocele (5.7 times the statewide prevalence), ventricular septal defect (VSD, 1.5 times the statewide prevalence), stenosis or atresia of the small intestine (3.1 times the statewide prevalence), and reduction defects of the lower limbs (3.4 times the statewide prevalence). No defects were statistically significantly lower in Leon Valley than the state as a whole.

Table 1. Crude and adjusted prevalence of 4 birth defects among Leon Valley resident deliveries, 1997-2003, compared to Texas, 1999-2003.

| Birth defect | Mother's residence at delivery | Time period | Type of prevalence | Prevalence ¹ (95% confidence interval) |
|--|--------------------------------|-------------|-----------------------|---|
| Encephalocele | Leon Valley ² | 1997-2003 | crude | 5.24 (1.43-13.42) |
| | | | adjusted ³ | 4.99 (0.10-9.88) |
| | Texas | 1999-2003 | comparison | 0.92 (0.78-1.06) |
| Ventricular septal defect | Leon Valley ² | 1997-2003 | crude | 66.85 (49.77-87.90) |
| | | | adjusted ³ | 62.54 (45.20-79.87) |
| | Texas | 1999-2003 | comparison | 45.24 (44.27-46.22) |
| Stenosis or atresia of the small intestine | Leon Valley ² | 1997-2003 | crude | 9.18 (3.69-18.91) |
| | | | adjusted ³ | 10.52 (2.38-18.66) |
| | Texas | 1999-2003 | comparison | 2.96 (2.71-3.20) |
| Reduction defects of the lower limbs | Leon Valley ² | 1997-2003 | crude | 6.55 (2.13-15.29) |
| | | | adjusted ³ | 10.59 (0.62-20.56) |
| | Texas | 1999-2003 | comparison | 1.93 (1.73-2.13) |

¹ Prevalence: cases per 10,000 live births.

² Residence address zip code 78238 or 78240.

³ Adjusted to the maternal age and race/ethnic distribution of Texas resident live births during 1999-2003.

After adjusting for maternal age and race/ethnicity, the prevalence of encephalocele among deliveries to Leon Valley residents decreased very slightly, from a crude prevalence 5.24 cases per 10,000 live births to 4.99 per 10,000 after adjustment (Table 1). The lower limit of the confidence interval decreased, however, such that the adjusted prevalence of encephalocele was not statistically significantly different from the statewide prevalence.

Similarly, the prevalence of ventricular septal defect decreased slightly, from 66.85 cases per 10,000 live births (crude) to 62.54 per 10,000 (adjusted), but the adjusted prevalence for VSD was not statistically significantly different from the statewide prevalence because the lower limit of the confidence interval decreased after adjustment.

The prevalence of stenosis or atresia of the small intestine increased after adjusting for maternal age and race/ethnic group, from 9.18 per 10,000 (crude) to 10.52 per 10,000 (adjusted), but the lower limit of the confidence interval dropped and the adjusted prevalence was not statistically significantly different from the prevalence for Texas overall.

The prevalence of reduction defects of the lower limbs also increased after adjustment, from 6.55 cases per 10,000 live births (crude) to 10.59 per 10,000 (adjusted). Again, the lower limit of the confidence interval declined and the adjusted prevalence was not statistically significantly elevated compared to the state.

DISCUSSION

We examined 48 specific types of birth defects and infants and fetuses with any monitored defects. At the 5% level of significance we would expect 2-3 defects to be significantly different in Leon Valley due to chance. Based on crude data, 4 types of birth defects were statistically significantly higher in Leon Valley than the state overall, and no defects examined were significantly lower in Leon Valley. The four defects that were elevated in Leon Valley based on crude prevalence data were as follows:

- Encephalocele: the protrusion of the brain substance through a defect in the skull;
- Ventricular septal defect: one or more openings in the wall between the right and left lower chambers of the heart, allowing mixing of oxygenated and unoxygenated blood;
- Stenosis or atresia of the small intestine: a narrowing or incomplete formation of the small intestine, obstructing movement of food through the digestive tract; and
- Reduction defects of the lower limbs: the congenital absence of a portion of the lower limb.

The live birth distribution for Leon Valley residents differs from that of the state for both maternal age group (Leon Valley has fewer younger mothers than Texas overall) and maternal race/ethnic group (Leon Valley has more Hispanic mothers, and fewer African American and non-Hispanic white mothers than Texas as a whole). Further, many birth defects are known to vary by maternal age or race/ethnicity or both, including at least 3 of the 4 defects that were elevated in Leon Valley based on crude prevalence data. To account for these facts, the data for Leon Valley were adjusted to the maternal age and race/ethnic distribution of Texas overall. This allows comparison of the Leon Valley data to Texas data, accounting for the differences by maternal age and race/ethnicity. A drawback of adjustment is that confidence intervals can become wider. For all 4 defects that were adjusted, the lower limits of the confidence intervals decreased after adjustment such that they were no longer statistically significantly different from the Texas prevalences.

The fact that the elevations observed in the crude prevalence data were no longer statistically significant after adjustment should be interpreted with caution, however. Adjusting for age and race/ethnicity did not substantially change the magnitude of the prevalence for 3 of the 4 birth defects that were elevated based on crude data, and for one defect (reduction defects of the lower limbs) the prevalence actually increased by 60% after adjustment. Because the prevalences did not substantially decrease with adjustment (only their confidence intervals extended lower), the elevations observed based on crude prevalence data cannot be attributed to differences in the maternal race/ethnic and age distribution of Leon Valley compared to Texas.

LIMITATIONS

The Texas Birth Defects Registry started collecting data in Bexar County with deliveries in 1997 and the most recent data available are for deliveries during 2003. The 1997-2003 data for Leon Valley presented in this report are for deliveries that occurred before the groundwater plume was detected in 2004, although it is possible the plume existed prior to its detection in 2004.

CONCLUSIONS

Four types of birth defects were statistically significantly elevated among residents of Leon Valley based on crude prevalence data: encephalocele, ventricular septal defect, stenosis or atresia of the small intestine, and reduction defects of the lower limbs. Adjusting for maternal age and race/ethnicity yielded adjusted prevalences that were no longer statistically significantly different from the statewide prevalences, but which were not substantially lower than the crude prevalences. The loss of significance after adjustment should therefore be interpreted with caution.

REFERENCE

1. Abramson JH, Gahlinger PM. Computer Programs for Epidemiologists: PEPI, version 4.0. Salt Lake City, Utah: Sagebrush Press, 2001.

For more information, contact Mary Ethen at the Birth Defects Epidemiology and Surveillance Branch at 512-458-7232, ext 2052, or email mary.ethen@dshs.state.tx.us, or visit our web site at <http://www.dshs.state.tx.us/birthdefects/>.