Health Consultation

PALESTINE FOUNDRY INVESTIGATION
PALESTINE, ANDERSON COUNTY, TEXAS

EPA FACILITY ID: TXN000605670

JULY 21, 2005

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

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

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

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HEALTH CONSULTATION

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Prepared by:

Texas Department of State Health Services
Under a Cooperative Agreement with the
Agency for Toxic Substances and Disease Registry

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Acronyms and Abbreviations

ATSDR	Agency for Toxic Substances and Disease Registry
CERCLA	Comprehensive Environmental Response, Compensation and Liability Act of 1980
CREG	Carcinogenic Risk Evaluation Guide
DSHS	Texas Department of State Health Services
EMEG	Environmental Media Evaluation Guide
EPA	Environmental Protection Agency
HAC Value	Health Assessment Comparison Value
HOD	Health Outcome Data
LD_{50}	Lethal dose, 50 percent killed
LOAEL	Lowest Observable Adverse Effects Level
MCL	Maximum Contaminant Level
mg/kg	milligrams per kilogram
mg/L	milligrams per liter
MRL	Minimal Risk Level
MRLi	Minimal Risk Level intermediate duration exposure
NOAEL	No Observable Adverse Effects Level
ppb	Parts per billion
ppm	Parts per million
QA/QC	Quality Assurance/Quality Control
RfD	Reference Dose
SARA	Superfund Amendments and Reauthorization Act of 1986
TDH	Texas Department of Health
µg/kg/day	Microgram per kilogram per day
µg/dL	Micrograms per deciliter

Executive Summary

The Texas Department of State Health Services (DSHS) and the Agency for Toxic Substances and Disease Registry (ATSDR) were asked by the U. S. Environmental Protection Agency (EPA) to determine the public health significance of arsenic, lead, and vanadium levels found in soil samples collected from residential yards and playgrounds in Palestine, Texas. Specifically, EPA asked DSHS and ATSDR to determine whether the contaminants found in the soil pose a public health threat. Additionally, EPA asked DSHS and ATSDR to review available blood lead data for children and cancer incidence for the area.

Sites that could cause adverse health effects as result of long-term exposures (more than 1 year) to hazardous substances are designated a public health hazard. We have concluded that the site poses a public health hazard because children could be exposed to doses of lead that could result in slight increases in blood lead under some exposure scenarios.

Based on the environmental sampling data and current health outcome data we concluded:

- Assuming that the levels of lead found in these soil samples are representative of concentrations to which children and adults could be exposed, children could be exposed to doses of lead that could result in slight increases in blood lead under some exposure scenarios. Lead levels that exceed 500 mg/kg in surface soil may pose a health hazard to children.

- Compared to the state as a whole, a greater percentage of the children from Palestine that have been tested have blood lead levels greater than or equal to 10 µg/dL. Interpreting the blood lead data for this area is difficult because of the small number of tests, the multifaceted individual behavior of children, the multiple pathways of exposure, and the multiple source nature of lead exposure. It is not likely that the lead in the soil is the sole cause of the elevated blood lead levels found in these children, but for some of the children the lead in the soil could be a contributing factor.

- It is unlikely that the maximum arsenic and vanadium levels in the soil pose a public health hazard to children, including those exhibiting pica behavior (eating unusual amounts of non-food items) based on the 0-1 inch surface soil sampling results and plausible exposure scenarios. Further, chronic ingestion of soil with the maximum levels of arsenic representative of those from the areas sampled would not pose a significant cancer risk.

- The analysis of incidence and mortality data for zip code 75801 (Palestine) showed cancers to be within normal ranges for both males and females. The cancers examined included: prostate, liver and intrahepatic bile duct, kidney and renal pelvis, bladder, and lung and bronchus.

Purpose

The Texas Department of State Health Services (DSHS) and the Agency for Toxic Substances and Disease Registry (ATSDR) were asked by the U. S. Environmental Protection Agency (EPA) to determine the public health significance of arsenic, lead, and vanadium levels found in soil samples collected from residential yards and playgrounds in Palestine, Texas. Specifically, EPA asked DSHS and ATSDR to determine whether the contaminants found in the soil pose a public health threat. Additionally, EPA asked DSHS and ATSDR to review available blood lead data for children in the area and cancer incidence (Note: A listing of Acronyms and Abbreviations follows the Table of Contents).

Background

Site Description

The area in question, a mixed industrial/residential area, is located in the southeastern portion of Palestine, Anderson County, Texas. Although the exact source and extent of the contamination have not yet been determined, historically, two facilities operated in the area: the Palestine Light, Heat, and Power Company and the former George M. Dilley and Son, Founders and Machinists Shop (also known as the Palestine Foundry). Both properties are accessible to children as well as adults. The Palestine Light, Heat and Power Company site is a one-acre site that was formerly a town gas operation. The site consists of several waste piles (railroad ties, concrete blocks, and rock piles) from the adjacent railroad system on the east side of the property. The Palestine Foundry site is north of the Palestine Light, Heat and Power Company and east of the railroad tracks on South May Street. It was the first large industry in Palestine and operated from 1873 to 1949. Historically, several buildings and a smoke stack may have existed on the property; only two buildings currently remain [1]. Across S. May Street from the Palestine Foundry are residential homes. The fence along the road does not prevent access to the foundry property. The foundry office building is dilapidated and thereby poses a physical hazard. The area did not have evidence of trespassing.

Community Health Concerns

To begin gathering community health concerns, the community involvement liaison contacted the DSHS Regional staff and representatives for the city of Palestine. In February 2005 DSHS staff accompanied EPA on door-to-door visits in the neighborhoods around the two former facilities. DSHS did not identify community health concerns related to the site.

Discussion

Introduction

The environmental data that DSHS used in this discussion include soil-sampling data collected by EPA in September 2004 [1]. EPA collected 83 composite samples; 71 were collected from residential yards and playgrounds. Twelve were collected along railroad right-of-ways. The samples evaluated were collected at the surface (0–1 inch). To assess the public health significance of areas that we believe children were more likely to frequent, the DSHS focused on results of metals analyses of soil in residential yards and playgrounds. We relied on the information provided to DSHS as having had adequate quality assurance/quality control (QA/QC) with regard to data collection, chain-of-custody, laboratory procedures, and data reporting. In assessing the potential public health significance of these sample results, we used

conservative assumptions to determine theoretical public health risks. The estimates used in this assessment do not apply to any specific individual or group of individuals.

Child Health Considerations

We recognize that the unique vulnerabilities of children demand special attention. Windows of vulnerability (critical periods) exist during development, particularly during early gestation, but also throughout pregnancy, infancy, childhood and adolescence periods when toxicants may permanently impair or alter structure and function [2]. Unique childhood vulnerabilities may be present because, at birth, many organs and body systems (including the lungs and the immune, endocrine, reproductive, and nervous systems) have not yet achieved structural or functional maturity. These organ systems continue to develop throughout childhood and adolescence. Children may exhibit differences in absorption, metabolism, storage, and excretion of toxicants, resulting in higher biologically effective doses to target tissues. Depending on the affected media and because of behavior patterns specific to children, they may be more exposed to contaminants than are adults. In an effort to account for children's unique vulnerabilities, and in accordance with ATSDR's Child Health Initiative [3] and EPA's National Agenda to Protect Children's Health from Environmental Threats [4], we used the potential exposure of children to the contaminants found in the soil as a guide in assessing the potential public health risks associated with this site.

Surface Soil Contaminant Evaluation

To assess the potential health risks associated with exposure to the contaminants found in the soil, we compared the concentrations measured to health-based screening values. These screening values represent contaminant specific levels in the soil that are considered safe for human contact with respect to identified health endpoints. Screening values are used to determine which contaminants need further evaluation. There are screening values for non-cancer and cancer health effects.

Non-cancer screening values generally are based on ATSDR's minimal risk levels (MRLs). ATSDR develops MRLs for each route of exposure: skin absorption, ingestion, and inhalation. MRLs are developed for various lengths of exposure: acute (less than 14 days), intermediate (15 to 364 days), and chronic (greater than 365 days). ATSDR presents these MRLs in chemical-specific toxicological profiles. These profiles provide information on health effects, environmental transport, human exposure, and regulatory status. When an ATSDR MRL is not available, we use the EPA's Reference Dose (RfD). RfDs are estimates of daily human exposure that are unlikely to cause non-cancer adverse health effects over a lifetime of exposure. MRLs and RfDs 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 health effects.

When chemical compounds have been classified as human carcinogens, probable human carcinogens, or possible human carcinogens, we use cancer-screening values to determine if they warrant a closer look. Cancer screening values are based on EPA's chemical specific cancer slope factors (CSF) and an estimated excess lifetime cancer risk of one-in-one-million persons exposed for a lifetime.

Exceeding either a non-cancer or a cancer screening value does not necessarily mean that the contaminant will cause harm; however, it does suggest that potential exposure to the contaminant warrants further consideration. We review and integrate relevant toxicological information with plausible exposure scenarios. When possible, for non-cancer endpoints, often we compare estimated exposures to known effect levels in humans or to documented No Observed Adverse Effect Levels (NOAEL) and/or Lowest Observed Adverse Effect Levels (LOAEL) in animals. We look at the weight-of-evidence to determine the public health significance of the contaminants that exceed the screening values.

Arsenic

Non–Carcinogenic Effects of Arsenic

The non-cancer screening values that we used for arsenic in soil [20 milligrams per kilogram (mg/kg) for children and 200 mg/kg for adults] are based on the EPA oral reference dose (RfD) for arsenic of 0.3 microgram per kilogram per day (µg/kg/day) [5]. The RfD was derived by dividing the identified NOAEL of 0.8 µg/kg/day (obtained from human epidemiologic studies) by an uncertainty factor of three to account for the lack of data on reproductive toxicity and to account for some uncertainty as to whether the NOAEL accounts for all sensitive individuals. There is not a clear consensus among scientists regarding the oral RfD. Arguments for various values within a factor of 2-3 of the recommended RfD value have been made. The LOAEL associated with these epidemiologic studies was 14 µg/kg/day, where exposure to arsenic above this level resulted in hyperpigmentation of the skin, keratosis (patches of hardened skin), and possible vascular complications [5-7]. We used standard assumptions for body weight (70 kg adult; 15 kg child) and soil ingestion (100 mg/day for an adult; 200 mg/day for a child; 5,000 mg/day for a pica child) to calculate the screening value. Screening values calculated using child exposure scenarios also are conservative (health protective) with respect to protecting adults.

The arsenic concentrations in all 83 surface soil (0-1 inches) samples collected ranged from 2.0 milligrams per kilogram (mg/kg) to 82.8 mg/kg with an average concentration of 32.4 mg/kg (Figure 1). Seventy-one of the 83 samples collected were from residential yards and playgrounds. The arsenic concentrations in these samples also ranged from 2.0-82.8 and had a similar average (32.3 mg/kg). Approximately 73 percent of the samples exceeded the non-cancer screening value for children of 20 mg/kg. Arsenic levels did not exceed adult screening values. Twenty-eight percent of the samples exceeded 40 mg/kg, a concentration two times greater than the non-cancer screening value for children. Assuming that the concentrations of arsenic found in these soil samples are representative of the concentrations to which a person could be exposed, a 15 kg child ingesting 200 mg regularly of soil with the average arsenic concentrations measured in soil from this area (82.8 mg/kg) could be exposed to arsenic at levels above the NOAEL (0.8 µg/kg/day), but it is less likely that they would be exposed to levels above the LOAEL for serious effects (50 µg/kg/day). Since by definition neither the NOAEL nor the LOAEL represent a sharp dividing line between “safe” and “unsafe” exposures, we assume that the public health significance of the arsenic increases as the ratio of the RfD (based on the NOAEL) to the estimated site-specific exposure dose decreases. We refer to this ratio as the margin of exposure (MOE). Under a variety of exposure conditions to the levels of arsenic measured in the soil samples collected, both children and adults could be subjected to exposure scenarios where the MOE would be less than 10. The significance of this is that the exposure dose is at a ‘safe’ level but it is nearing a level that we are less confident about (Tables 1–8).

Short-Term or Sporadic Pica Behavior

Soil pica behavior (ingestion of more than 1.0 gram[1] of soil per day) may occur in a sizable portion of children throughout the year [8]. While any individual child may only exhibit pica behavior infrequently, the behavior is not limited to a small subset of the population. It has been estimated that approximately 62% of children will ingest >1.0 gram of soil on 1-2 days/year. Additionally, 42% of children will ingest >5 grams of soil and 33% will ingest >10 grams of soil on 1-2 days per year. For some contaminants, periodic pica episodes potentially could result in acute intoxication [8]. To explore the potential public health significance of acute exposure to arsenic due to pica behavior at this site, we considered the scenario of a 15 kg child sporadically ingesting 5,000 mg of soil per day. At a soil arsenic concentration of 82.8 mg/kg and a bioavailability factor of 100%, the daily dose of absorbed arsenic during the pica events could be 27.6 µg/kg/day – below the acute LOAEL for serious effects (50 µg/kg/day) reported by Mizuta [9]. Assuming 100% bioavailability, a child regularly exhibiting pica behavior would have to ingest over 9,000 mg of soil per day to receive a dose approaching the acute LOAEL. Effects associated with this acute LOAEL include nausea, vomiting, diarrhea, occult blood in feces, gastric and duodenal juice, and abnormal electrocardiogram [9]. This level of exposure is considered unlikely to occur.

Carcinogenic Effects of Arsenic

The carcinogenic screening value for arsenic of 0.5 mg/kg is based on EPA's cancer slope factor (CSF) for skin cancer and an estimated excess lifetime cancer risk of 1 additional cancer in 1 million people exposed for 70 years. Arsenic was detected at concentrations above its carcinogenic screening value in virtually all the soil samples; however, the levels of arsenic normally found in the environment also exceed this screening value [5]. Nonetheless, people who regularly ingest soil from some of these areas could have some theoretical excess increased lifetime risk for developing cancer. Cancer risk estimates for this area range from 3.0×10^{-7} (assuming 100% bioavailability, ingestion of 50 mg/day of the average soil arsenic concentration of 32.4 mg/kg one day per week for 30 years) to 7.6×10^{-5} (assuming 100% bioavailability, ingestion of 100 mg/day of the maximum soil arsenic concentration of 82.8 mg/kg everyday for 30 years) (Tables 9-10). Qualitatively, depending on specific exposure scenarios, we interpret these estimates to indicate that chronic ingestion of soil from these areas would not be likely to significantly affect the risk of developing cancer.

Public Health Implications of Exposure to Arsenic

There is considerable controversy about assessing potential risks associated with exposure to arsenic. Both the RfD and the CSF are based on human ecological studies that have recognized uncertainties with respect to the assignation of exposure. Such studies find it difficult to avoid errors in assigning people to specific exposure groups. The studies on which the RfD and the CSF are based also involved exposure to arsenic in drinking water. The ability of the body to absorb arsenic in water is likely higher than the ability of the body to absorb arsenic in soil. In our primary analysis, we assumed that the relative bioavailability of arsenic in the soil was 100%. Studies conducted for EPA at various Superfund sites have found the bioavailability of the arsenic in the soil to be less than 100 percent. Assuming 100 % absorption is conservative with respect to protecting public health, and to some unknown degree, over estimates risk.

[1] 1 gram is equal to 1,000 milligrams (about the same size as a pack of artificial sweetener)

Recently, EPA contracted with the College of Veterinary Medicine at the University of Missouri, Columbia (CVMUM) to assess the relative bioavailability of arsenic in soil from a site in El Paso, Texas. Using a juvenile swine model CVMUM reported the relative bioavailability of arsenic in the soil from that area to be 40% [10]. To explore what effect such a factor might have on the potential public health risks, we applied this factor to the soil results from Palestine, Texas. Under some conditions of exposure, both children and adults could still be subjected to exposure scenarios where the MOE with respect to the RfD would be less than 10 (Tables 11-14). Using this bioavailability factor we found that under a variety of exposure scenarios children could be exposed to arsenic concentrations approaching the NOAEL (Tables 15-18).

While it is unlikely that we could attribute any specific health outcome for any one individual to exposure to arsenic, based on these data, under some conditions the concentrations of arsenic in the soil from this area could be considered unacceptable with respect to noncancer health effects.

The mechanisms through which arsenic causes cancer are not known; however, arsenic is not believed to act directly with DNA. Since the studies used to derive the CSF are based on exposure doses much higher than those likely to be encountered at this site, it is questionable whether it is appropriate to assume linearity for the dose-response assessment for arsenic at low doses. The actual dose-response curve at low doses may be sublinear which would mean that the risk estimates in this consultation overestimate the actual risks. Qualitatively, the cancer risk estimates that we derived for potential exposures at this site range from a no apparent increased lifetime risk to an insignificant increased lifetime risk of developing cancer. These cancer risk estimates are unremarkable. Liver, bladder, kidney, and lung cancer all have been associated with exposure to arsenic. While there are certainly both cases of- and deaths due to these types of cancer in Palestine, Texas, it would be impossible to determine if any one of these cancers was caused by exposure to arsenic. The incidence and mortality of these cancer types are similar to what would be expected based on state rates.

Lead

The lead concentrations in all 83 surface soil (0–1 inches) samples and the subset of 71 residential yard and playground samples ranged from 11.2 milligrams lead per kilogram soil (mg/kg) to 1,170 mg/kg. The average concentrations of lead for all of the 83 surface soil samples and for the subset of residential yard and playground surface soil samples were 197 mg/kg and 209 mg/kg respectively. Approximately 9 % of the samples collected between 0 and 1 inch in depth exceeded EPA's action level of 500 mg/kg. Assuming that the levels of lead found in these soil samples are representative of concentrations to which children and adults could be exposed, children could be exposed to lead doses that could result in slight increases in blood lead level under some exposure scenarios.

Non-carcinogenic Effects of Lead

Although no threshold level for adverse health effects has been established, evidence suggests that adverse effects occur at blood lead levels at least as low as 10µg/dL. The Centers for Disease Control and Prevention (CDC) has determined that a blood lead level greater than or equal to 10µg/dL in children indicates excessive lead absorption and constitutes the grounds for intervention. The 10µg/dL level is based on observations of enzymatic abnormalities in the red

blood cells at blood levels below 25 µg/dL and observations of neurologic and cognitive dysfunction in children with blood lead levels between 10 and 15µg/dL [11, 12].

In general, soil lead will have the greatest impact on the blood lead levels of preschool age children. These children are more likely to play in dirt and to place their hands and other contaminated objects in their mouths. They are better at absorbing lead through the gastrointestinal tract than adults, and they are more likely to exhibit the types of nutritional deficiencies that facilitate the absorption of lead. The predicted 95th percentile blood lead level for children that is associated with a soil lead concentration of between 400 to 500 mg/kg is approximately 10 µg/dL. In other words, a child regularly exposed to soil lead levels greater than 400 to 500 mg/kg should have no more than a 5% chance of having a blood lead level greater than 10 µg/dL as a result of that exposure. Fitting a lognormal distribution to the available data we estimate that approximately 9 % of the surface soil samples from the area could exceed 500 mg/kg (Figure 2).

The DSHS Childhood Lead Poisoning Prevention Program compared the percentage of children with elevated blood lead levels residing in Palestine, Texas (for the years 1996 to 2003) to the percentage of children with elevated blood lead levels for Texas as a whole (Appendix B). Tests of children under the age of 6 years were included in the comparison. For the eight-year period, 48 of the 534 venous blood samples (9%) from children residing in Palestine were greater than or equal to 10 µg/dL. For the same eight-year period, 16,850 of the 530,010 venous blood samples (3.2 %) from Texas children were greater than or equal to 10 µg/dL. Due to the small number of children tested in Palestine caution should be used in drawing conclusions from these data.

Carcinogenic Effects of Lead

Lead has not been shown to be carcinogenic in humans; however, high doses of lead have been found to produce kidney tumors in laboratory studies of rats and mice. The extremely high cumulative doses of lead used in animal studies are difficult to extrapolate to low-level exposure in humans, and do not provide a sufficient basis for quantitative risk assessment. Based on animal data, EPA currently classifies lead as a B2 carcinogen (probable human carcinogen) [12].

Public Health Implications of Exposure to Lead

In the absence of site specific information regarding the bioavailability of lead in the soil and other potential sources of exposure to lead, in Texas a soil lead level greater than 500 mg/kg generally is regarded as requiring further attention. This is based on the increased probability that a child regularly exposed to soil at this level could have an elevated blood lead level as defined by the CDC. Lead has not been identified as a human carcinogen therefore exposure to lead in soil poses no carcinogenic risk.

Assuming that these data were randomly collected, approximately 9 percent of soil samples taken from this area would be expected to be greater than 500 mg/kg. Assuming that each sample is representative of a yard, 9 percent of the yards might have elevated soil lead levels. Soil lead levels greater than 500 mg/kg in yards from homes with small children could be considered unacceptable. Because elevated blood lead levels are a consequence of exposure to lead, data for Palestine from the Childhood Lead Poisoning Prevention Program were evaluated. Approximately 9 percent of the children tested over the past 8 years had elevated blood lead levels. Statewide, only 3.2 percent of the children tested have elevated levels. Caution should be

used in drawing conclusions from these data. Because of the multi-source nature of exposure to lead, as well as the small number of children tested from Palestine during any given year, and the method of data collection, these data, should not be used to draw conclusions with respect to associations between blood lead levels and the lead in the residential and parks soil.

Vanadium

ATSDR has developed an intermediate oral MRL for vanadium of 0.003 mg/kg/day. This translates to an intermediate non-cancer screening value of 200 mg/kg for a child and 2,000 mg/kg for an adult. EPA has proposed a provisional chronic oral RfD of 0.001 mg/kg/day [14] based on human data from which a NOAEL for systemic effects was identified, and animal data from which a critical effect of kidney toxicity was identified [13, 14]. Because EPA's provisional RfD is more protective (with respect to public health) than ATSDR's MRL, we used the RfD to calculate appropriate non-cancer screening values for the vanadium in the soil. To calculate the screening values we assumed that a 15 kilogram child consumes 200 mg of the contaminated soil per day and a 70 kilogram adult consumes 100 mg of the contaminated soil per day. Based on these standard assumptions and a 350 day per year exposure scenario, the screening values for vanadium in surface soil are 78 mg/kg for children and 730 mg/kg for adults.

The vanadium concentrations in all of the surface soil samples collected (n=83) ranged from 9.2 mg/kg to 253 mg/kg with an average concentration of 76 mg/kg (Figure 3). The average vanadium concentration in the residential and playground surface soils samples (n=71 of the 83) was slightly higher than that of all of the samples combined at 92.7 mg/kg. Approximately 55 percent of the 71 residential yard and playground samples exceeded the non-cancer screening value for children while vanadium concentrations were approximately 2 ½ times less than the vanadium screening value for adults (730 mg/kg). Assuming that the average concentration of vanadium found in these soil samples are representative of the concentrations to which people could be exposed, children could be exposed to vanadium at doses that approach those of the EPA's provisional RfD and result in margins of exposure (MOEs) of less than 10 (Tables 19-22).

Carcinogenic Effects of Exposure to Vanadium

No human studies are available on the carcinogenicity of vanadium. However, no increase in tumor frequency was noted in rats and mice chronically exposed to 0.5 to 4.1 mg-vanadium/kg-body weight as vanadyl sulfate in drinking water [15]. Currently, vanadium is not classified as a human carcinogen.

Public Health Implications of Exposure to Vanadium

Determining the public health implications of vanadium in soil is difficult. The toxic effects of vanadium are greater when vanadium is inhaled as compared to when it is taken orally. Protein and other trace elements in the diet may have an affect on its toxicity and the toxic effects also may vary by species. Humans who have taken relatively large doses for up to five months only reported minor complaints at the higher doses; whereas, in animals numerous effects such as weight loss, dehydration, depressed growth, cardiac irregularities, and loss of renal function have been reported. Whether vanadium is essential to the diet is controversial. There is in vivo evidence that vanadium may be needed for normal iodine and/or thyroid function and other

evidence that it may have some effect on glucose metabolism. Although a variety of inconsistent deficiency symptoms have been reported in animals, no specific function for vanadium has been identified for humans.

We used EPA's provisional reference dose to assess potential public health implications of vanadium on children. Assuming a relative bioavailability factor of 100%, under a variety of exposure scenarios it is possible that children could be exposed to doses approaching the RfD; however when you consider plausible exposure scenarios, exposure to the soil poses no public health hazard.

Health Outcome Data Evaluation

Analysis of Cancer Incidence and Mortality Data

The Cancer Epidemiology and Surveillance (CES) Branch of the Texas Department of State Health Services examined the occurrence of cancer in zip code 75801, Palestine, Texas. The CES evaluated 1995–2000 incidence data (the best available data) and 1993–2002 mortality data for cancers of the liver and intrahepatic bile duct, kidney and renal pelvis, bladder, prostate, and lung and bronchus (cancer sites with possible associations with exposure to arsenic) [Appendix C]. Incidence data are the best indicator of the occurrence of cancer in an area because they show how many cancers were diagnosed each year and are considered complete (more than 95%) statewide through 2001. Incidence data for 2001 in Palestine, Texas cannot be used at this time because completeness estimates are currently less than 90% for the area. From 1995 to 2000 there were 119 cases of prostate cancer, 31 cases of kidney and renal pelvis cancer, 56 cases of bladder cancer, 12 cases of liver and intrahepatic bile duct cancer, and 159 cases of lung and bronchus cancer. For the years 1993 to 2002 the number of deaths due to prostate, kidney and renal pelvis, bladder, liver and intrahepatic bile duct, and lung and bronchus cancer was 41, 21, 19, 20, and 237, respectively. Compared to what would be expected based on state rates the analysis of incidence and mortality data showed cancers of the prostate, liver and intrahepatic bile duct, kidney and renal pelvis, bladder, and lung and bronchus incidence and mortality to be within normal ranges for both males and females (Appendix C).

Conclusions

1. Assuming that the levels of lead found in these soil samples are representative of concentrations to which children and adults could be exposed, children could be exposed to doses of lead that could result in slight increases in blood lead level under some exposure scenarios. Lead levels that exceed 500 mg/kg in surface soil may pose a health hazard to children.

2. Compared to the state as a whole, a greater percentage of the children from Palestine that have been tested have blood lead levels greater than or equal to 10 µg/dL. Interpreting the blood lead data for this area is difficult because of the small number of tests, the multifaceted individual behavior of children, the multiple pathways of exposure, and the multiple source nature of lead exposure. It is not likely that the lead in the soil is the sole cause of the elevated blood lead levels found in these children, but for some of the children the lead in the soil could be a contributing factor.

3. Based on the 0-1 inch surface soil sampling results and plausible exposure scenarios, it is unlikely that the maximum arsenic and vanadium levels in the soil pose a public health hazard to children including those exhibiting pica behavior (eating unusual amounts of non-food items). Further, chronic ingestion of soil with maximum levels of arsenic measured at 0-1 inch would not pose a significant cancer risk.

4. The analysis of incidence and mortality data for zip code 75801 (Palestine) showed cancers to be within normal ranges for both males and females. The cancers examined included: prostate, liver and intrahepatic bile duct, kidney and renal pelvis, bladder, and lung and bronchus.

Based on the environmental sampling data and current health outcome data, we have concluded that this site poses a public health hazard because children could be exposed to doses of lead that could result in slight increases in blood lead under some exposure scenarios.

Recommendations

1. As a general precaution, children below 6 years of age should have blood lead testing.

2. Provide education on ways that residents can reduce their exposure to elevated levels of contaminants.

3. Access to areas with lead above the 500 ppm should be restricted or the soil should be removed and replaced.

Public Health Action Plan

Actions Completed

1. EPA notified residents of the soil test results for their individual properties.

2. EPA collected soil samples at several childcare facilities.

3. DSHS EIET Branch provided local physicians with educational material regarding environmental exposure and recommending childhood blood lead testing according to CDC guidelines.

Actions Planned

1. DSHS EIET Branch will evaluate additional soil sampling data particularly that of daycare or other childcare facilities as results become available.

2. DSHS Cancer Epidemiology and Surveillance Branch will update the analysis for zip code 75801, Palestine, Texas when the 2001 incidence data for the area are considered more than 90% complete.

3. DSHS EIET Branch will provide education to residents who have elevated levels of contaminants in their soil on ways to reduce their exposure.

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Certification

This Palestine Foundry, Anderson County, Texas, Public Health Assessment was prepared by the Texas Department of State Health Services (DSHS) under a cooperative agreement with the federal Agency for Toxic Substances and Disease Registry (ATSDR). It was completed in accordance with approved methodologies and procedures existing at the time the health assessment was initiated. Editorial review was completed by the Cooperative Agreement partner.

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The Division of Health Assessment and Consultation (DHAC), ATSDR, has reviewed this health consultation and concurs with its findings.

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Appendix A – Figures

Figure 1
Palestine, Texas Soil Arsenic Concentrations (0-1 inches)

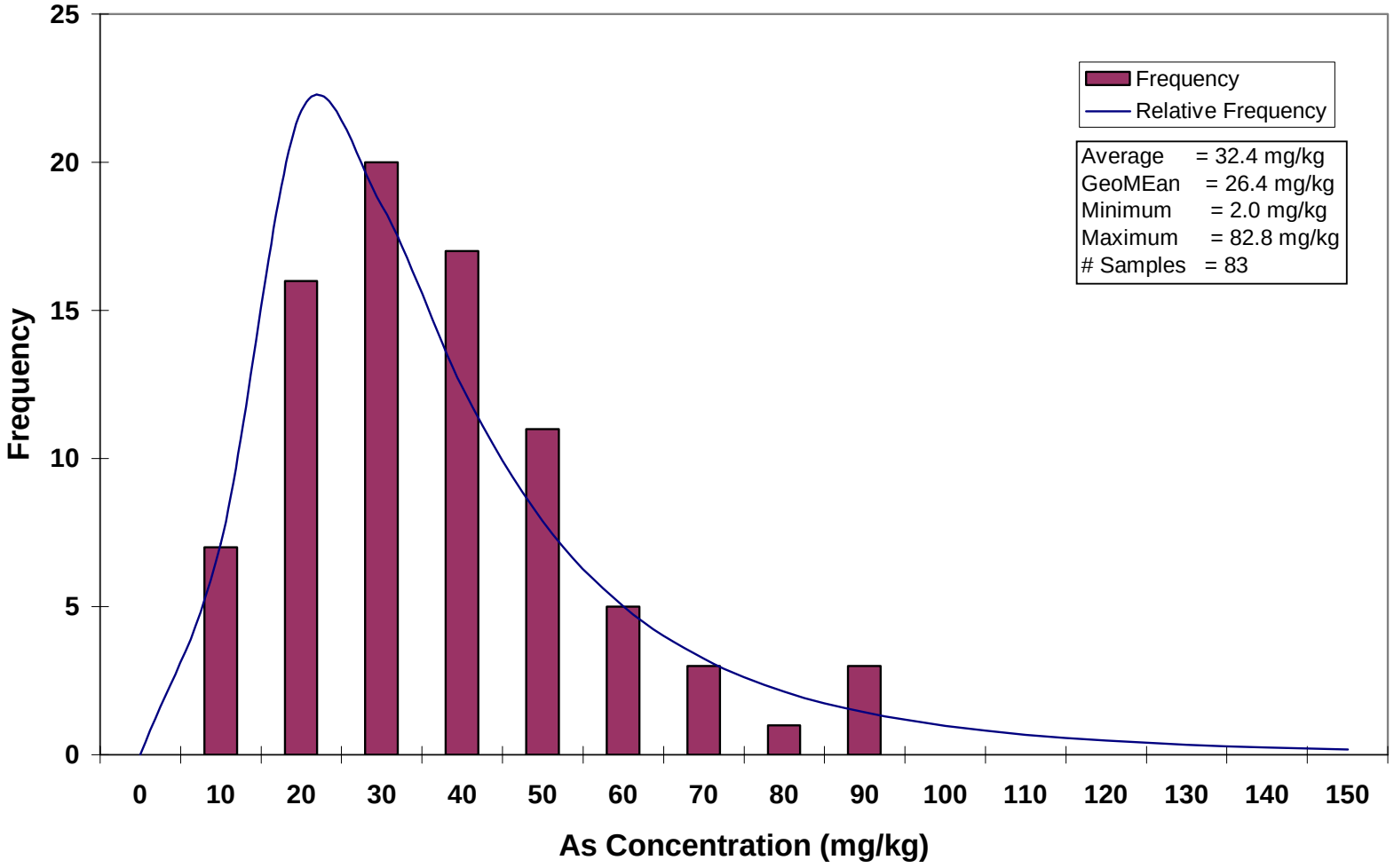


Figure 2
Palestine, Texas Soil Lead Concentrations (0-1 inches)

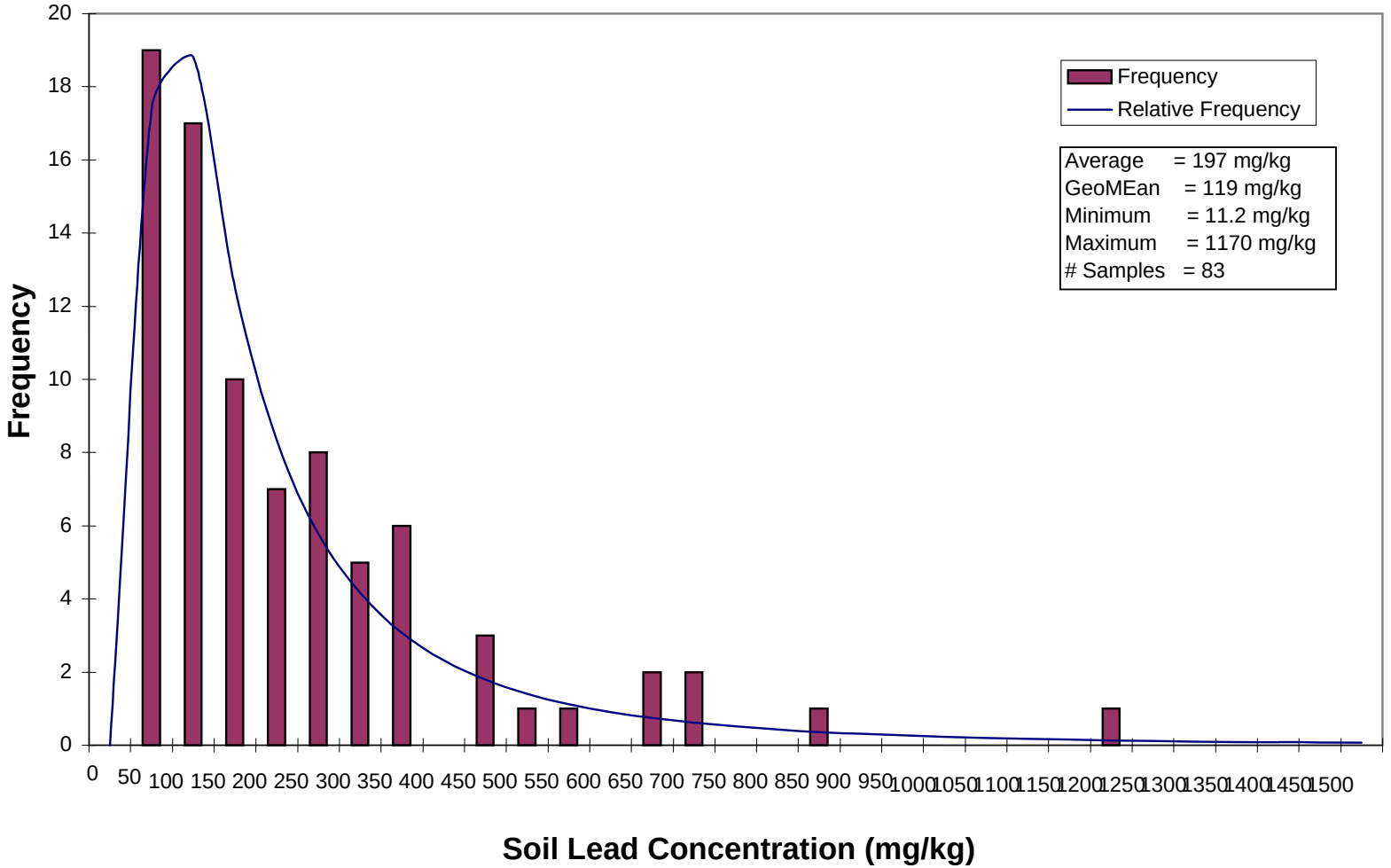
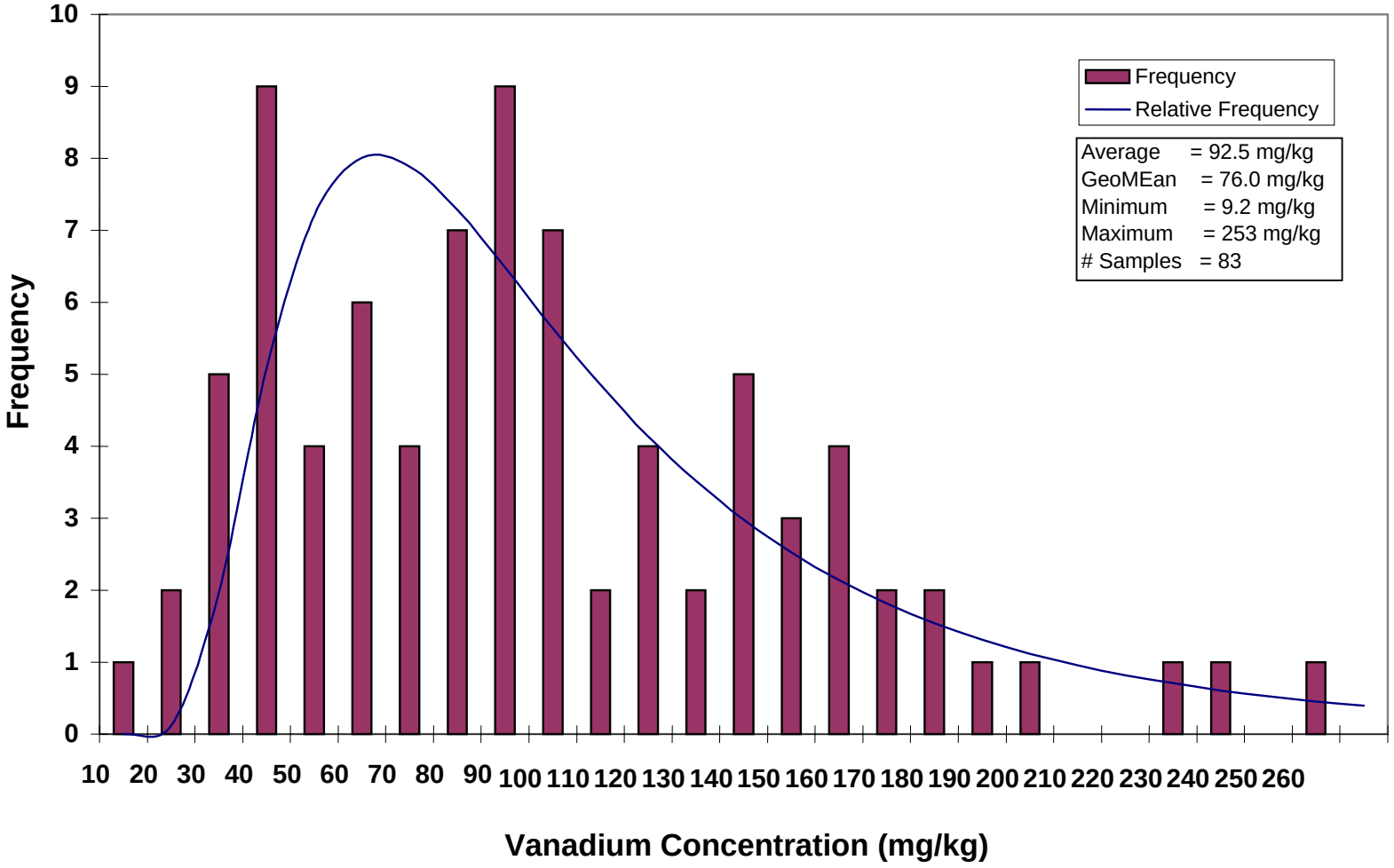


Figure 3
Palestine, Texas Soil Vanadium Concentrations (0-1 inches)



Appendix B – Exposure Estimates and Related Information

Table 1. Estimated Exposure Dose and Margin of Exposure to Arsenic Reference Dose Various Exposure Scenarios for Children (Assumes 100 percent Bioavailability)							
Soil Ingestion Rate (mg/day)	**Days per week of exposure at MAXIMUM reported concentration**						
	1	**2**	**3**	**4**	**5**	**6**	**7**
Exposure Dose (micrograms per kilogram body weight per day)							
50	0.04	0.08	0.12	0.16	0.20	0.24	0.28
75	0.06	0.12	0.18	0.24	0.30	0.35	0.41
100	0.08	0.16	0.24	0.32	0.39	0.47	0.55
125	0.10	0.20	0.30	0.39	0.49	0.59	0.69
150	0.12	0.24	0.25	0.47	0.59	0.71	0.83
175	0.14	0.28	0.41	0.55	0.69	0.83	0.97
200	0.16	0.32	0.47	0.63	0.79	0.95	1.10
Margin of exposure (Reference Dose/Estimated Exposure Dose)							
50	7.6	3.8	2.5	1.9	1.5	1.3	1.1
75	5.1	2.5	1.7	1.3	1.0	0.9	0.7
100	3.8	1.9	1.3	1.0	0.8	0.6	0.5
125	3.0	1.5	1.0	0.8	0.6	0.5	0.4
150	2.5	1.3	0.9	0.6	0.5	0.4	0.4
175	2.2	1.1	0.7	0.5	0.4	0.4	0.3
200	1.9	1.0	0.6	0.5	0.4	0.3	0.3

Shaded areas represent MOEs less than 10. Maximum reported concentration = 82.8 mg/kg

Table 2. Estimated Exposure Dose and Margin of Exposure to Arsenic Reference Dose Various Exposure Scenarios for Adults (Assumes 100 percent Bioavailability)							
Soil Ingestion Rate (mg/day)	**Days per week of exposure at MAXIMUM reported concentration**						
	1	**2**	**3**	**4**	**5**	**6**	**7**
Exposure Dose (micrograms per kilogram body weight per day)							
50	0.01	0.02	0.03	0.03	0.04	0.05	0.06
75	0.01	0.03	0.04	0.05	0.06	0.08	0.09
100	0.02	0.03	0.05	0.07	0.08	0.10	0.12
Margin of exposure (Reference Dose/Estimated Exposure Dose)							
50	35.5	17.8	11.8	8.9	7.1	5.9	5.0
75	23.7	11.8	7.9	5.9	4.7	4.0	3.4
100	17.8	8.9	5.9	4.4	3.6	3.0	2.5

Shaded areas represent MOEs less than 10. Maximum reported concentration = 82.8 mg/kg

Table 3. Estimated Exposure Dose and Margin of Exposure to Arsenic Reference Dose Various Exposure Scenarios for Children (Assumes 100 percent Bioavailability)							
Soil Ingestion Rate (mg/day)	**Days per week of exposure at AVERAGE reported concentration**						
	1	**2**	**3**	**4**	**5**	**6**	**7**
Exposure Dose (micrograms per kilogram body weight per day)							
50	0.02	0.03	0.05	0.06	0.08	0.09	0.11
75	0.02	0.05	0.07	0.09	0.12	0.14	0.16
100	0.03	0.06	0.09	0.12	0.15	0.19	0.22
125	0.04	0.08	0.12	0.15	0.19	0.23	0.27
150	0.05	0.09	0.14	0.19	0.23	0.28	0.32
175	0.05	0.11	0.16	0.22	0.27	0.32	0.38
200	0.06	0.12	0.19	0.25	0.31	0.37	0.43
Margin of exposure (Reference Dose/Estimated Exposure Dose)							
50	19.4	9.7	6.5	4.5	3.9	3.2	2.3
75	13.0	6.5	4.3	3.2	2.6	2.2	1.9
100	9.7	4.9	3.2	2.4	1.9	1.6	1.4
125	7.8	3.9	2.6	1.9	1.6	1.3	1.1
150	6.5	3.2	2.2	1.6	1.3	1.1	1.0
175	5.6	2.8	1.9	1.4	1.1	1.0	0.8
200	4.9	2.4	1.6	1.2	1.0	0.9	0.7

Shaded areas represent MOEs less than 10. Average concentration = 32.4 mg/kg

Table 4. Estimated Exposure Dose and Margin of Exposure to Arsenic Reference Dose Various Exposure Scenarios for Adults (Assumes 100 percent Bioavailability)							
Soil Ingestion Rate (mg/day)	**Days per week of exposure at AVERAGE reported concentration**						
	1	**2**	**3**	**4**	**5**	**6**	**7**
Exposure Dose (micrograms per kilogram body weight per day)							
50	0.00	0.01	0.01	0.01	0.02	0.02	0.02
75	0.00	0.01	0.01	0.02	0.02	0.03	0.03
100	0.01	0.01	0.02	0.03	0.03	0.04	0.05
Margin of exposure (Reference Dose/Estimated Exposure Dose)							
50	90.7	45.4	30.3	22.7	18.2	15.1	13.0
75	60.5	30.3	20.2	15.1	12.1	10.1	8.6
100	45.4	22.7	15.1	11.3	9.1	7.6	6.5

Shaded areas represent MOEs less than 10. Average concentration = 32.4 mg/kg

Table 5. Estimated Exposure Dose and Margin of Exposure to Arsenic NOAEL Various Exposure Scenarios for Children (Assumes 100 percent Bioavailability)							
Soil Ingestion Rate (mg/day)	**Days per week of exposure at MAXIMUM reported concentration**						
	1	**2**	**3**	**4**	**5**	**6**	**7**
Exposure Dose (micrograms per kilogram body weight per day)							
50	0.04	0.08	0.12	0.16	0.20	0.24	0.28
75	0.06	0.12	0.18	0.24	0.30	0.35	0.41
100	0.08	0.16	0.24	0.32	0.39	0.47	0.55
125	0.10	0.20	0.30	0.39	0.49	0.59	0.69
150	0.12	0.24	0.35	0.47	0.59	0.71	0.83
175	0.14	0.28	0.41	0.55	0.69	0.83	0.97
200	0.16	0.32	0.47	0.63	0.79	0.95	1.10
Margin of exposure (NOAEL/Estimated Exposure Dose)							
50	20.3	10.1	6.8	5.1	4.1	3.4	2.9
75	13.5	6.8	4.5	3.4	2.7	2.3	1.9
100	10.1	5.1	3.4	2.5	2.0	1.7	1.5
125	8.1	4.1	2.7	2.0	1.6	1.4	1.2
150	6.8	3.4	2.3	1.7	1.4	1.1	1.0
175	5.8	2.9	1.9	1.5	1.2	1.0	0.8
200	5.1	2.5	1.7	1.3	1.0	0.9	0.7

Shaded areas represent MOEs less than 10. Maximum reported concentration = 82.8 mg/kg

Table 6. Estimated Exposure Dose and Margin of Exposure to Arsenic NOAEL Various Exposure Scenarios for Adults (Assumes 100 percent Bioavailability)							
Soil Ingestion Rate (mg/day)	**Days per week of exposure at MAXIMUM reported concentration**						
	1	**2**	**3**	**4**	**5**	**6**	**7**
Exposure Dose (micrograms per kilogram body weight per day)							
50	0.01	0.02	0.03	0.03	0.04	0.05	0.06
75	0.01	0.03	0.04	0.05	0.06	0.08	0.09
100	0.02	0.03	0.05	0.07	0.08	0.10	0.12
Margin of exposure (NOAEL/Estimated Exposure Dose)							
50	94.7	47.3	31.6	23.7	18.9	15.8	13.5
75	63.1	31.6	21.0	15.8	12.6	10.5	9.0
100	47.3	23.7	15.8	11.8	9.5	7.9	6.8

Shaded areas represent MOEs less than 10. Maximum reported concentration = 82.8 mg/kg

Table 7. Estimated Exposure Dose and Margin of Exposure to Arsenic NOAEL Various Exposure Scenarios for Children (Assumes 100 percent Bioavailability)							
Soil Ingestion Rate (mg/day)	**Days per week of exposure at AVERAGE reported concentration**						
	1	**2**	**3**	**4**	**5**	**6**	**7**
Exposure Dose (micrograms per kilogram body weight per day)							
50	0.02	0.03	0.05	0.06	0.08	0.09	0.11
75	0.02	0.05	0.07	0.09	0.12	0.14	0.16
100	0.03	0.06	0.09	0.12	0.15	0.19	0.22
125	0.04	0.08	0.12	0.15	0.19	0.23	0.27
150	0.05	0.09	0.14	0.19	0.23	0.28	0.32
175	0.05	0.11	0.16	0.22	0.27	0.32	0.38
200	0.06	0.12	0.19	0.25	0.31	0.37	0.43
Margin of exposure (NOAEL/Estimated Exposure Dose)							
50	51.9	25.9	17.3	13.0	10.4	8.6	7.4
75	34.6	17.3	11.5	8.6	6.9	5.8	4.9
100	25.9	13.0	8.6	6.5	5.2	4.3	3.7
125	20.7	10.4	6.9	5.2	4.2	3.5	3.0
150	17.3	8.6	5.8	4.3	3.5	2.9	2.5
175	14.8	7.4	4.9	3.7	3.0	2.5	2.2
200	13.0	6.5	4.3	3.2	2.6	2.2	1.9

Shaded areas represent MOEs less than 10. Average concentration = 32.4 mg/kg

Table 8. Estimated Exposure Dose and Margin of Exposure to Arsenic NOAEL Various Exposure Scenarios for Adults (Assumes 100 percent Bioavailability)							
Soil Ingestion Rate (mg/day)	**Days per week of exposure at AVERAGE reported concentration**						
	1	**2**	**3**	**4**	**5**	**6**	**7**
Exposure Dose (micrograms per kilogram body weight per day)							
50	0.00	0.01	0.01	0.01	0.02	0.02	0.02
75	0.00	0.01	0.01	0.02	0.02	0.03	0.03
100	0.01	0.01	0.02	0.03	0.03	0.04	0.05
Margin of exposure (NOAEL/Estimated Exposure Dose)							
50	242	121	81	60	48	40	35
75	161	81	54	40	32	27	23
100	121	60	40	30	24	20	17

Shaded areas represent MOEs less than 10. Average concentration = 32.4 mg/kg

Table 9. Excess Lifetime Cancer Risk Estimates for Arsenic							
Soil Ingestion Rate (mg/day)	**Days per week of exposure at the MAXIMUM reported concentration**						
	1	**2**	**3**	**4**	**5**	**6**	**7**
Excess Lifetime Cancer Risk Estimates assuming 100% bioavailability							
50	7.7×10^{-7}	3.1×10^{-6}	7.0×10^{-6}	1.2×10^{-5}	1.9×10^{-5}	2.8×10^{-5}	3.8×10^{-5}
75	1.2×10^{-6}	4.6×10^{-6}	1.0×10^{-5}	1.9×10^{-5}	2.9×10^{-5}	4.2×10^{-5}	5.7×10^{-5}
100	1.6×10^{-6}	6.2×10^{-6}	1.4×10^{-5}	2.5×10^{-5}	3.9×10^{-5}	5.6×10^{-5}	7.6×10^{-5}
Excess Lifetime Cancer Risk Estimates assuming 40% bioavailability							
50	3.1×10^{-7}	1.2×10^{-6}	2.8×10^{-6}	5.0×10^{-6}	7.7×10^{-6}	1.1×10^{-5}	1.5×10^{-5}
75	4.6×10^{-7}	1.8×10^{-6}	4.2×10^{-6}	7.4×10^{-6}	1.2×10^{-5}	1.7×10^{-5}	2.3×10^{-5}
100	6.2×10^{-7}	2.5×10^{-6}	5.6×10^{-6}	9.9×10^{-6}	1.6×10^{-5}	2.2×10^{-5}	3.0×10^{-5}

Maximum reported concentration = 82.8 mg/kg

Table 10. Excess Lifetime Cancer Risk Estimates for Arsenic							
Soil Ingestion Rate (mg/day)	**Days per week of exposure at the AVERAGE reported concentration**						
	1	**2**	**3**	**4**	**5**	**6**	**7**
Excess Lifetime Cancer Risk Estimates assuming 100% bioavailability							
50	3.0×10^{-7}	1.2×10^{-6}	2.7×10^{-6}	4.8×10^{-6}	7.6×10^{-6}	1.1×10^{-5}	1.5×10^{-5}
75	4.5×10^{-7}	1.8×10^{-6}	4.1×10^{-6}	7.3×10^{-6}	1.1×10^{-5}	1.6×10^{-5}	2.2×10^{-5}
100	6.1×10^{-7}	2.4×10^{-6}	5.5×10^{-6}	9.7×10^{-6}	1.5×10^{-5}	2.2×10^{-5}	3.0×10^{-5}
Excess Lifetime Cancer Risk Estimates assuming 40% bioavailability							
50	1.2×10^{-7}	4.8×10^{-7}	1.1×10^{-6}	1.9×10^{-6}	3.0×10^{-6}	4.4×10^{-6}	5.9×10^{-6}
75	1.8×10^{-7}	7.3×10^{-7}	1.6×10^{-6}	2.9×10^{-6}	4.5×10^{-6}	6.5×10^{-6}	8.9×10^{-6}
100	2.4×10^{-7}	9.7×10^{-7}	2.2×10^{-6}	3.9×10^{-6}	6.1×10^{-6}	8.7×10^{-6}	1.2×10^{-5}

Average reported concentration = 32.4 mg/kg

Table 11. Estimated Exposure Dose and Margin of Exposure to Arsenic Reference Dose Various Exposure Scenarios for Children (Assumes 40 percent Bioavailability)							
Soil Ingestion Rate (mg/day)	**Days per week of exposure at MAXIMUM reported concentration**						
	1	**2**	**3**	**4**	**5**	**6**	**7**
Exposure Dose (micrograms per kilogram body weight per day)							
50	0.02	0.03	0.05	0.06	0.08	0.09	0.11
75	0.02	0.05	0.07	0.09	0.12	0.14	0.17
100	0.03	0.06	0.09	0.13	0.16	0.19	0.22
125	0.04	0.08	0.12	0.16	0.20	0.24	0.28
150	0.05	0.09	0.14	0.19	0.24	0.28	0.33
175	0.06	0.11	0.17	0.22	0.28	0.33	0.39
200	0.06	0.13	0.19	0.25	0.32	0.38	0.44
Margin of exposure (Reference Dose/Estimated Exposure Dose)							
50	19.0	9.5	6.3	4.8	3.8	3.2	2.7
75	12.7	6.3	4.2	3.2	2.5	2.1	1.8
100	9.5	4.8	3.2	2.4	1.9	1.6	1.4
125	7.6	3.8	2.5	1.9	1.5	1.3	1.1
150	6.3	3.2	2.1	1.6	1.3	1.1	0.9
175	5.4	2.7	1.8	1.4	1.1	1.0	0.8
200	4.8	2.4	1.6	1.2	1.0	0.8	0.7

Shaded areas represent MOEs less than 10. Maximum reported concentration = 82.8 mg/kg

Table 12. Estimated Exposure Dose and Margin of Exposure to Arsenic Reference Dose Various Exposure Scenarios for Adults (Assumes 40 percent Bioavailability)							
Soil Ingestion Rate (mg/day)	**Days per week of exposure at MAXIMUM reported concentration**						
	1	**2**	**3**	**4**	**5**	**6**	**7**
Exposure Dose (micrograms per kilogram body weight per day)							
50	0.00	0.01	0.01	0.01	0.02	0.02	0.02
75	0.01	0.01	0.02	0.02	0.03	0.03	0.04
100	0.01	0.01	0.02	0.03	0.03	0.04	0.05
Margin of exposure (Reference Dose/Estimated Exposure Dose)							
50	88.8	44.4	29.6	22.2	17.8	14.8	12.7
75	59.2	29.6	19.7	14.8	11.8	9.9	8.5
100	44.4	22.2	14.8	11.1	8.9	7.4	6.3

Shaded areas represent MOEs less than 10. Maximum reported concentration = 82.8 mg/kg

Table 13. Estimated Exposure Dose and Margin of Exposure to Arsenic Reference Dose Various Exposure Scenarios for Children (Assumes 40 percent Bioavailability)							
Soil Ingestion Rate (mg/day)	**Days per week of exposure at AVERAGE reported concentration**						
	1	**2**	**3**	**4**	**5**	**6**	**7**
Exposure Dose (micrograms per kilogram body weight per day)							
50	0.01	0.01	0.02	0.02	0.03	0.04	0.04
75	0.01	0.02	0.03	0.04	0.05	0.06	0.06
100	0.01	0.02	0.04	0.05	0.06	0.07	0.09
125	0.02	0.03	0.05	0.06	0.08	0.09	0.11
150	0.02	0.04	0.06	0.07	0.09	0.11	0.13
175	0.02	0.04	0.06	0.09	0.11	0.13	0.15
200	0.02	0.05	0.07	0.10	0.12	0.15	0.17
Margin of exposure (Reference Dose/Estimated Exposure Dose)							
50	48.6	24.3	16.2	12.2	9.7	8.1	6.9
75	32.4	16.2	10.8	8.1	6.5	5.4	4.6
100	24.3	12.2	8.1	6.1	4.9	4.1	3.5
125	19.4	9.7	6.5	4.9	3.9	3.2	2.8
150	16.2	8.1	5.4	4.1	3.2	2.7	2.3
175	13.9	6.9	4.6	3.5	2.8	2.3	2.0
200	12.2	6.1	4.1	3.0	2.4	2.0	1.7

Shaded areas represent MOEs less than 10. Average concentration = 32.4 mg/kg

Table 14. Estimated Exposure Dose and Margin of Exposure to Arsenic Reference Dose Various Exposure Scenarios for Adults (Assumes 40 percent Bioavailability)							
Soil Ingestion Rate (mg/day)	**Days per week of exposure at AVERAGE reported concentration**						
	1	**2**	**3**	**4**	**5**	**6**	**7**
Exposure Dose (micrograms per kilogram body weight per day)							
50	0.00	0.00	0.00	0.01	0.01	0.01	0.01
75	0.00	0.00	0.01	0.01	0.01	0.01	0.01
100	0.00	0.01	0.01	0.01	0.01	0.02	0.02
Margin of exposure (Reference Dose/Estimated Exposure Dose)							
50	227	113	76	57	45	38	32
75	151	76	50	38	30	25	22
100	113	57	38	28	23	19	16

Shaded areas represent MOEs less than 10. Average concentration = 32.4 mg/kg

Table 15. Estimated Exposure Dose and Margin of Exposure to Arsenic NOAEL Various Exposure Scenarios for Children (Assumes 40 percent Bioavailability)							
Soil Ingestion Rate (mg/day)	**Days per week of exposure at MAXIMUM reported concentration**						
	1	**2**	**3**	**4**	**5**	**6**	**7**
Exposure Dose (micrograms per kilogram body weight per day)							
50	0.02	0.03	0.05	0.06	0.08	0.09	0.11
75	0.02	0.05	0.07	0.09	0.12	0.14	0.17
100	0.03	0.06	0.09	0.13	0.16	0.19	0.22
125	0.04	0.08	0.12	0.16	0.20	0.24	0.28
150	0.05	0.09	0.14	0.19	0.24	0.28	0.33
175	0.06	0.11	0.17	0.22	0.28	0.33	0.39
200	0.06	0.13	0.19	0.25	0.32	0.38	0.44
Margin of exposure (NOAEL/Estimated Exposure Dose)							
50	50.7	25.4	16.9	12.7	10.1	8.5	7.3
75	33.8	16.9	11.3	9.5	6.7	5.6	4.8
100	25.4	12.7	8.5	6.3	5.1	4.2	3.6
125	20.3	10.1	6.7	5.1	4.1	3.4	2.9
150	16.9	8.5	5.6	4.2	3.4	2.8	2.4
175	14.5	7.3	4.8	3.6	2.9	2.4	2.1
200	12.7	6.3	4.2	3.2	2.5	2.1	1.8

Shaded areas represent MOEs less than 10. Maximum reported concentration = 82.8 mg/kg

Table 16. Estimated Exposure Dose and Margin of Exposure to Arsenic NOAEL Various Exposure Scenarios for Adults (Assumes 40 percent Bioavailability)							
Soil Ingestion Rate (mg/day)	**Days per week of exposure at MAXIMUM reported concentration**						
	1	**2**	**3**	**4**	**5**	**6**	**7**
Exposure Dose (micrograms per kilogram body weight per day)							
50	0.00	0.01	0.01	0.01	0.02	0.02	0.02
75	0.01	0.01	0.02	0.02	0.03	0.03	0.04
100	0.01	0.01	0.02	0.03	0.03	0.04	0.05
Margin of exposure (NOAEL/Estimated Exposure Dose)							
50	238	119	79	59	48	40	34
75	159	79	53	40	32	26	23
100	119	59	40	30	24	20	17

Shaded areas represent MOEs less than 10. Maximum reported concentration = 82.8 mg/kg

Table 17. Estimated Exposure Dose and Margin of Exposure to Arsenic NOAEL Various Exposure Scenarios for Children (Assumes 40 percent Bioavailability)							
Soil Ingestion Rate (mg/day)	**Days per week of exposure at AVERAGE reported concentration**						
	1	**2**	**3**	**4**	**5**	**6**	**7**
Exposure Dose (micrograms per kilogram body weight per day)							
50	0.01	0.01	0.02	0.02	0.03	0.04	0.04
75	0.01	0.02	0.03	0.04	0.05	0.06	0.06
100	0.01	0.02	0.04	0.05	0.06	0.07	0.09
125	0.02	0.03	0.05	0.06	0.08	0.09	0.11
150	0.02	0.04	0.06	0.07	0.09	0.11	0.13
175	0.02	0.04	0.06	0.09	0.11	0.13	0.15
200	0.02	0.05	0.07	0.10	0.12	0.15	0.17
Margin of exposure (NOAEL/Estimated Exposure Dose)							
50	129.6	64.8	43.2	32.4	25.9	21,6	18.5
75	86.4	43.2	28.8	21.6	17.3	14.4	12.4
100	64.8	32.4	21.6	16.2	13.0	10.8	9.3
125	51.9	25.9	17.3	13.0	10.4	8.6	7.4
150	43.2	21.6	14.4	10.8	8.6	7.2	6.2
175	37.0	18.5	12.4	9.3	7.4	6.2	5.3
200	32.4	16.2	10.8	8.1	6.5	5.4	4.6

Shaded areas represent MOEs less than 10. Average concentration = 32.4 mg/kg

Table 18. Estimated Exposure Dose and Margin of Exposure to Arsenic NOAEL Various Exposure Scenarios for Adults (Assumes 40 percent Bioavailability)							
Soil Ingestion Rate (mg/day)	**Days per week of exposure at AVERAGE reported concentration**						
	1	**2**	**3**	**4**	**5**	**6**	**7**
Exposure Dose (micrograms per kilogram body weight per day)							
50	0.00	0.00	0.00	0.01	0.01	0.01	0.01
75	0.00	0.00	0.01	0.01	0.01	0.01	0.01
100	0.00	0.01	0.01	0.01	0.01	0.02	0.02
Margin of exposure (NOAEL/Estimated Exposure Dose)							
50	605	302	202	151	121	101	86
75	403	202	134	101	81	67	58
100	302	151	101	76	60	50	43

Shaded areas represent MOEs less than 10. Average concentration = 32.4 mg/kg

Table 19. Estimated Exposure Dose and Margin of Exposure to Vanadium Reference Dose Various Exposure Scenarios for Children							
Soil Ingestion Rate (mg/day)	**Days per week of exposure at MAXIMUM reported concentration**						
	1	**2**	**3**	**4**	**5**	**6**	**7**
Exposure Dose (micrograms per kilogram body weight per day)							
50	0.120	0.241	0.361	0.482	0.602	0.723	0.843
75	0.181	0.361	0.542	0.723	0.904	1.084	1.265
100	0.241	0.482	0.723	0.964	1.205	1.446	1.687
125	0.301	0.602	0.904	1.205	1.506	1.807	2.108
150	0.361	0.723	1.084	1.446	1.807	2.169	2.530
175	0.422	0.843	1.265	1.687	2.108	2.530	2.952
200	0.482	0.964	1.446	1.928	2.410	2.891	3.373
Margin of Exposure (Reference Dose / Estimated Exposure Dose)							
50	8.3	4.2	2.8	2.1	1.7	1.4	1.2
75	5.5	2.8	1.8	1.4	1.1	0.9	0.8
100	4.2	2.1	1.4	1.0	0.8	0.7	0.6
125	3.3	1.7	1.1	0.8	0.7	0.6	0.5
150	2.8	1.4	0.9	0.7	0.6	0.5	0.4
175	2.4	1.2	0.8	0.6	0.5	0.4	0.3
200	2.1	1.0	0.7	0.5	0.4	0.4	0.3

Shaded areas represent MOEs less than 10. Maximum concentration = 253 mg/kg
EPA's provisional RfD (0.001 mg/kg/day)

Table 20. Estimated Exposure Dose and Margin of Exposure to Vanadium Reference Dose Various Exposure Scenarios for Adults							
Soil Ingestion Rate (mg/day)	**Days per week of exposure at MAXIMUM reported concentration**						
	1	**2**	**3**	**4**	**5**	**6**	**7**
Exposure Dose (micrograms per kilogram body weight per day)							
50	0.026	0.052	0.077	0.103	0.129	0.155	0.181
75	0.039	0.077	0.116	0.155	0.194	0.232	0.271
100	0.052	0.103	0.155	0.207	0.258	0.310	0.361
Margin of Exposure (Reference Dose / Estimated Exposure Dose)							
50	38.7	19.4	12.9	9.7	7.7	6.5	5.5
75	25.8	12.9	8.6	6.5	5.2	4.3	3.7
100	19.4	9.7	6.5	4.8	3.9	3.2	2.8

Shaded areas represent MOEs less than 10. Maximum concentration = 253 mg/kg
EPA's provisional RfD (0.001 mg/kg/day)

Table 21. Estimated Exposure Dose and Margin of Exposure to Vanadium Reference Dose Various Exposure Scenarios for Children							
Soil Ingestion Rate (mg/day)	**Days per week of exposure at AVERAGE reported concentration**						
	1	**2**	**3**	**4**	**5**	**6**	**7**
Exposure Dose (micrograms per kilogram body weight per day)							
50	0.044	0.088	0.132	0.176	0.220	0.264	0.308
75	0.066	0.132	0.198	0.264	0.330	0.396	0.463
100	0.088	0.176	0.264	0.352	0.440	0.529	0.617
125	0.110	0.220	0.330	0.440	0.551	0.661	0.771
150	0.132	0.264	0.396	0.529	0.661	0.793	0.925
175	0.154	0.308	0.463	0.617	0.771	0.925	1.079
200	0.176	0.352	0.529	0.705	0.881	1.057	1.233
Margin of Exposure (Reference Dose /Estimated Exposure Dose)							
50	22.7	11.4	7.6	5.7	4.5	3.8	3.2
75	15.1	7.6	5.1	3.8	3.0	2.5	1.2
100	11.4	5.7	3.8	2.8	2.3	1.9	1.6
125	9.1	4.5	3.0	2.3	1.8	1.5	1.3
150	7.6	3.8	2.5	1.9	1.5	1.3	1.1
175	6.5	3.2	2.2	1.6	1.3	1.1	0.9
200	5.7	2.8	1.9	1.4	1.1	1.0	0.8

Shaded areas represent MOEs less than 10. Average concentration = 92.5 mg/kg
EPA's provisional RfD (0.001 mg/kg/day)

Table 22. Estimated Exposure Dose and Margin of Exposure to Vanadium Reference Dose Various Exposure Scenarios for Adults							
Soil Ingestion Rate (mg/day)	**Days per week of exposure at AVERAGE reported concentration**						
	1	**2**	**3**	**4**	**5**	**6**	**7**
Exposure Dose (micrograms per kilogram body weight per day)							
50	0.009	0.019	0.028	0.038	0.047	0.057	0.066
75	0.014	0.028	0.042	0.057	0.071	0.085	0.099
100	0.019	0.038	0.057	0.076	0.094	0.113	0.132
Margin of Exposure (Reference Dose /Estimated Exposure Dose)							
50	105.9	53.0	35.3	26.5	21.2	17.7	15.1
75	70.6	35.3	23.5	17.7	14.1	11.8	10.1
100	53.0	26.5	17.7	13.2	10.6	8.8	7.6

Shaded areas represent MOEs less than 10. Maximum concentration = 92.5 mg/kg
EPA's provisional RfD (0.001 mg/kg/day)

Appendix C – Blood Lead Report

TEXAS DEPARTMENT OF STATE HEALTH SERVICES

Texas Childhood Lead Poisoning Prevention Program

Blood Lead Levels from Venous Blood Samples for Tested Unduplicated Children under Age 6 Residing in Palestine, Texas and Texas Overall from 1996 – 2003

1996

Blood Lead Levels	Palestine, Texas				Texas Overall			
	Venous Results[a]		Results All Sample Types [b]		Venous Results[a]		Results All Sample Types [b]	
	No.	Percent	No.	Percent	No.	Percent	No.	Percent
Not Elevated (Pb<10 µg/dL)	97	85.1%	243	83%	84,533	95.4%	215,690	94.2%
Elevated (Pb>=10 µg/dL	17	14.9%	50	17%	4,061	4.6%	13,272	5.8%
Total Tests	114	100.0%	293	100.0%	88,594	100.0%	228,962	100.0%
% Total Venous	39%				39%			

1997

Blood Lead Levels	Palestine, Texas				Texas Overall			
	Venous Results[a]		Results All Sample Types [b]		Venous Results[a]		Results All Sample Types [b]	
	No.	Percent	No.	Percent	No.	Percent	No.	Percent
Not Elevated (Pb<10 µg/dL)	92	91.1%	192	89%	77,443	96.1%	216,637	95%
Elevated (Pb>=10 µg/dL	9	8.9%	24	11%	3,126	3.9%	11,391	5%
Total Tests	101	100.0%	216	100.0%	80,569	100.0%	228,028	100.0%
% Total Venous	47%				35%			

1998

Blood Lead Levels	Palestine, Texas				Texas Overall			
	Venous Results[a]		Results All Sample Types [b]		Venous Results[a]		Results All Sample Types [b]	
	No.	Percent	No.	Percent	No.	Percent	No.	Percent
Not Elevated (Pb<10 µg/dL)	93	93%	184	90%	70,643	96.4%	205,357	95.2%
Elevated (Pb>=10 µg/dL	7	7%	21	10%	2,676	3.6 %	10,269	4.8%
Total Tests	100	100.0%	205	100.0%	73,319	100.0%	215,626	100.0%
% Total Venous	49%				34%			

1999

Blood Lead Levels	Palestine, Texas				Texas Overall			
	Venous Results[a]		Results All Sample Types [b]		Venous Results[a]		Results All Sample Types [b]	
	No.	Percent	No.	Percent	No.	Percent	No.	Percent
Not Elevated (Pb<10 µg/dL)	56	93.3%	157	92.9%	62,809	97.2%	183,663	96.5%
Elevated (Pb>=10 µg/dL	<5	6.7%	12	7.1%	1,805	2.8%	6,636	3.5%
Total Tests	60	100.0%	169	100.0%	64,614	100.0%	190,299	100.0%
% Total Venous	36%				34%			

2000

Blood Lead Levels	Palestine, Texas				Texas Overall			
	Venous Results[a]		Results All Sample Types [b]		Venous Results[a]		Results All Sample Types [b]	
	No.	Percent	No.	Percent	No.	Percent	No.	Percent
Not Elevated (Pb<10 µg/dL)	41	95.3%	132	92.3%	57,497	97.2%	177,351	96.9%
Elevated (Pb>=10 µg/dL	<5	4.7%	11	7.7%	1,673	2.8%	5,755	3.1%
Total Tests	43	100.0%	143	100.0%	59,170	100.0%	183,106	100.0%
% Total Venous	30%				32%			

2001

Blood Lead Levels	Palestine, Texas				Texas Overall			
	Venous Results[a]		Results All Sample Types [b]		Venous Results[a]		Results All Sample Types [b]	
	No.	Percent	No.	Percent	No.	Percent	No.	Percent
Not Elevated (Pb<10 µg/dL)	57	92%	157	91.8%	56,344	98.1%	180,110	97.4%
Elevated (Pb>=10 µg/dL	5	8%	14	8.2%	1,120	1.9%	4,836	2.6%
Total Tests	62	100.0%	171	100.0%	57,464	100.0%	184,946	100.0%
% Total Venous	36%				31%			

2002

Blood Lead Levels	Palestine, Texas				Texas Overall			
	Venous Results[a]		Results All Sample Types [b]		Venous Results[a]		Results All Sample Types [b]	
	No.	Percent	No.	Percent	No.	Percent	No.	Percent
Not Elevated (Pb<10 µg/dL)	23	95.8%	124	96.1%	62,765	98.1%	202,364	97.2%
Elevated (Pb>=10 µg/dL	<5	4.2%	5	3.9%	1,245	1.9%	5,907	2.8%
Total Tests	24	100.0%	129	100.0%	64,010	100.0%	208,271	100.0%
% Total Venous	19%				31%			

2003

Blood Lead Levels	Palestine, Texas				Texas Overall			
	Venous Results[a]		Results All Sample Types [b]		Venous Results[a]		Results All Sample Types [b]	
	No.	Percent	No.	Percent	No.	Percent	No.	Percent
Not Elevated (Pb<10 µg/dL)	27	90.0%	131	89.7%	71,126	98.4%	242,831	98.1%
Elevated (Pb>=10 µg/dL	<5	10.0%	15	10.3%	1,144	1.6%	4,696	1.9%
Total Tests	30	100.0%	146	100.0%	72,270	100.0%	247,527	100.0%
% Total Venous	21%				29%			

- Results are based on the date the blood specimen was taken.

[a] Venous samples results are considered conclusive. Venous results are based on the first venous test for each child.

[b] All sample types includes venous, capillary, and unknown. Capillary samples may produce false negatives if the child's hands are not properly cleaned prior to testing. Results are based on the first test for each child.

Appendix D – Cancer Epidemiology Surveillance Report

Summary of Investigation Into the Occurrence of Cancer

Zip Code 75801, Palestine, Anderson County, Texas

1993-2002

November 10, 2004

Background:

Concern about a possible excess of cancer prompted the Cancer Epidemiology and Surveillance (CES) Branch of the Texas Department of State Health Services to examine the occurrence of cancer in zip codes 75801, Palestine, Texas. Local residents were concerned that arsenic may be causing cancer among residents. The CES evaluated 1995–2000 incidence data (the best available data) and 1993–2002 mortality data for cancers of the liver and intrahepatic bile duct, kidney and renal pelvis, bladder, prostate, and lung and bronchus. The scientific literature has shown an association between arsenic and these cancer sites. Incidence data are the best indicator of the occurrence of cancer in an area because they show how many cancers were diagnosed each year and are considered complete (more than 95%) statewide through 2001. However incidence data for 2001 in Palestine, Texas cannot be used at this time because completeness estimates are currently less than 90% for Public Health Region 4 (PHR 4). Cancer mortality data are used as a supplemental measure and are complete for the entire state through 2002. The rest of this report examines the investigative methods the CES 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 CES to investigate the possibility of excess cancer in a community. The cancer cluster investigation is not used to prove that cancer was caused by environmental or other risk factors because it is extremely difficult to determine exactly what causes a particular cancer in a particular individual. Individuals are often exposed to many cancer-causing agents over their lifetime and not everyone who is exposed will get cancer. Instead, the cancer cluster investigation is specifically intended to answer the question “Is there an excess of cancer in the area or population of concern?” Two other questions that must be answered before an environmental hazard can be possibly associated with the cancer are: 1) Is there evidence of exposure to hazardous substances, and 2) Can the exposure and the cancer type be linked by a statistical association? To answer these questions, the CES often works with the Environmental & Injury Epidemiology and Toxicology Branch, the Spatial Approaches to Health Outcomes program within the Texas Department of State Health Services, and other state and federal agencies.

The CES follows guidelines recommended by the Centers for Disease Control and Prevention for investigating cancer clusters.[1] To determine if a true excess of cancer is occurring and if further study is warranted at the time of the initial investigation, biologic and epidemiologic evidence is 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 indicate a cluster needing further study. Because excesses of cancer may occur by chance alone, the role of chance is also considered in the statistical analysis.

If further study is indicated, the CES will determine the feasibility of conducting an epidemiologic study examining the cancer and the exposure. If the epidemiologic study is feasible, the final step is to perform an etiologic investigation to see if the cancer can be related to the exposure. 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 and deaths 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 or die from cancer. This is important because peoples' 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 1–2 present the number of observed cases and deaths for males and females, the number of "expected" cases and deaths, the standardized incidence ratio (SIR) or standardized mortality ratio (SMR), and the corresponding 99% confidence interval. The standardized incidence or mortality ratio (SIR, SMR) is simply the number of observed cases or deaths compared to the number of "expected" cases or deaths. When the SIR or SMR of a selected cancer is equal to 1.00, then the number of observed cases or deaths is equal to the expected number of cases or deaths, based on the incidence or mortality in the rest of the state. When the SIR or SMR is less than 1.00, fewer people developed or died of cancer than we would have expected. Conversely, an SIR or SMR greater than 1.00 indicates that more people developed or died of cancer than we would have expected. To determine if an SIR or SMR 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 the likelihood that the result occurred by chance into account. It also indicates the range in which we would expect the SIR or SMR 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 or SMR. As long as the 99% confidence interval contains 1.00, this indicates that the SIR or SMR is still within the range one might expect and, therefore, not statistically significant.

Results:

The analysis of incidence data for zip codes 75801, Palestine, Texas, from January 1, 1995–December 31, 2000, and mortality data from January 1, 1993–December 31, 2002, showed

cancers of the prostate, liver and intrahepatic bile duct, kidney and renal pelvis, bladder, and lung and bronchus incidence and mortality to be within normal ranges for both males and females. Analysis summaries are presented in Tables 1–2.

Discussion:
Like other studies, this cancer cluster investigation had limitations. The number of years of incidence data examined was limited to seven years and did not include data for the most recent years. Ten years of mortality data were examined as a supplemental measure and did include data for one more recent year. Also, cancer incidence data are based on residence at the time of diagnosis. It is possible that some residents who may have been exposed and developed cancer no longer lived in the area at the time of diagnosis so were not included in the data. However, it is also possible that people with no exposure may have moved into the area and then developed cancer because of other factors. These cases are included in the investigation.

This study was also limited in its power to detect a small effect from some environmental or other type of exposure. However, a large increased risk of cancer in an area could have been observed using this methodology.

Recommendations:
Based on the findings and the information discussed above, further study is not recommended at this time to determine whether the various cancers in zip code 75801, Palestine, Texas may be associated with exposure to arsenic. The CES will update this analysis for zip code 75801, Palestine, Texas when the 2001 incidence data for PHR 4 are considered more than 90% complete.

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. In Texas, as in the United States, cancer is the second leading cause of death, exceeded only by heart disease. 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, usually 20 to 40 years. Conditions that have prevailed for only the last 5 or 10 years are unlikely to be related to the current incidence of cancer in a community.

The chances of a person developing cancer as a result of exposure to an environmental contaminant are slight. According to Richard Doll and Richard Peto, renowned epidemiologists at the University of Oxford, pollution and occupational exposures are estimated to collectively cause 4–6% of all cancer deaths.[2] 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.[3] In contrast, the National Cancer Institute estimates that lifestyle factors such as tobacco use and diet cause 50 to 75 percent of cancer deaths.[4] Eating a healthy diet and refraining from tobacco are the best ways to prevent many kinds of cancer.

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.

Known Risk Factors for Cancers Examined in This Investigation:
The following is a brief discussion summarized from the National Cancer Institute and the American Cancer Society about cancer risk factors for the specific cancers studied in this investigation.[5,6]

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.

Lung 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. 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-napthylamine, 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 use of the herb Aristocholia Fangchi.

Kidney Cancer

Kidney cancer risk factors include smoking, obesity, diet, occupational exposure to heavy metals or organic solvents, misuse of certain pain medications over a long period of time, advanced kidney disease, and aging. Men have higher rates of kidney cancer.

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 have been associated in the literature with bile duct cancer include dioxin, nitrosamines, and polychlorinated biphenyls (PCBs).

For additional information about cancer, visit the "Resources" link on our web site at http://www.tdh.state.tx.us/tcr/.

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

References:

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4. National Cancer Institute. 2001 Cancer Progress Report. http://progressreport.cancer.gov/doc.
5. American Cancer Society web site. http://www.cancer.org.
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Table 1

Number of Observed and Expected Cancer Cases and Race Adjusted Standardized Incidence Ratios, Selected Cancers, Zip Code 75801, Palestine, TX, 1995–2000

Males				
Site	**Observed**	**Expected**	**SIR**	**99% CI**
Prostate	119	150.70	0.79	0.62 – 1.00
Kidney and Renal Pelvis	19	18.04	1.05	0.53 – 1.85
Bladder	42	28.58	1.47	0.95 – 2.16
Liver and Intrahepatic Bile Duct	<5	8.28	0.48	0.08 – 1.52
Lung and Bronchus	101	102.52	0.99	0.75 – 1.27
Females				
Site	**Observed**	**Expected**	**SIR**	**99% CI**
Kidney and Renal Pelvis	12	10.48	1.15	0.47 – 2.30
Bladder	14	9.92	1.41	0.63 – 2.71
Liver and Intrahepatic Bile Duct	8	3.72	2.15	0.69 – 4.99
Lung and Bronchus	58	67.21	0.86	0.60 – 1.20

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 1995–2000. 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.

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11/10/2004

Table 2

Number of Observed and Expected Cancer Deaths and Race Adjusted Standardized Mortality Ratios, Selected Cancers, Zip Code 75801, Palestine, TX, 1993–2002

Males				
Site	**Observed**	**Expected**	**SMR**	**99% CI**
Prostate	41	51.90	0.79	0.51 – 1.17
Kidney and Renal Pelvis	11	11.11	0.99	0.39 – 2.05
Bladder	12	10.04	1.20	0.49 – 2.40
Liver and Intrahepatic Bile Duct	13	13.14	0.99	0.42 – 1.94
Lung and Bronchus	143	144.22	0.99	0.79 – 1.23
Females				
Site	**Observed**	**Expected**	**SMR**	**99% CI**
Kidney and Renal Pelvis	10	6.54	1.53	0.57 – 3.27
Bladder	7	5.35	1.31	0.38 – 3.21
Liver and Intrahepatic Bile Duct	7	7.19	0.97	0.28 – 2.38
Lung and Bronchus	94	89.80	1.05	0.79 – 1.36

Note: The SMR (standardized mortality ratio) is defined as the number of observed deaths divided by the number of expected deaths. The latter is based on race-, sex-, and age-specific cancer mortality rates for Texas during the period 1993–2002. The SMR 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.

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