

# Health Consultation

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**PUBLIC COMMENT RELEASE**

**MIDLOTHIAN AREA AIR QUALITY PART 1:  
VOLATILE ORGANIC COMPOUNDS & METALS**

**MIDLOTHIAN, ELLIS COUNTY, TEXAS**

**DECEMBER 11, 2007**

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**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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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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VOLATILE ORGANIC COMPOUNDS & METALS

MIDLOTHIAN, ELLIS COUNTY, TEXAS

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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## Executive Summary

Residents of Midlothian, Texas, petitioned the Agency for Toxic Substances and Disease Registry (ATSDR) and the Texas Department of State Health Services (DSHS) to more fully characterize the emissions from multiple large industries in the area and evaluate potential health risks resulting from individual and aggregate chemical exposures.

In this initial health consultation we primarily address the various air contaminants identified from ambient air samples collected by the Texas Commission on Environmental Quality (TCEQ) in the Midlothian area (usually every 6 days) from May 1981 through March 2005. The 227 contaminants that we reviewed in this report include 119 volatile organic compounds (VOCs) and 108 metals and other inorganic substances present in particulate matter.

The ambient air data that were collected on an hourly basis for sulfur dioxide, hydrogen sulfide, nitric oxide, nitrogen dioxide, nitrogen oxides, ozone, and particulates under the U.S. Environmental Protection Agency's (EPA's) National Ambient Air Quality Standards (NAAQS) are being evaluated and will be presented in a separate health consultation.

### *Response to Petitioner and Community Health Concerns*

The 4 different lists (A, B, C, & D) of petitioner and community concerns are given in Appendix B. Each list contains from 4 to 8 individual (numbered) concerns. Responses to one or more of these concerns are addressed in the paragraphs below (identified by the list letter and concern number, e.g. C.3. refers to list C, concern number 3).

A.1. While it is true that "all the chemicals being released from cement kilns and steel mills have not been fully identified," this health consultation has evaluated 237 individual contaminants including 119 VOCs and 108 metals and other inorganic substances.

A.2. It is also true that "All the chemicals currently being incinerated and released have not been tested for carcinogenicity and endocrine disrupting potential." However, based on historical reviews of cancer incidence and/or mortality rates in Midlothian and Ellis County, no individual or aggregate cancer rates were significantly elevated with respect to the rest of the state.

A.4., C.3., & D.3. The community was concerned about the health effects of dioxins, metals, and mixtures of compounds. Air data for dioxins are not routinely collected in Texas; therefore it was not possible to evaluate the potential adverse health effects associated with these compounds. We evaluated available VOCs and metals air contaminant data with respect to its potential for causing adverse health effects in humans due to acute, intermediate, and/or chronic exposures. Only manganese exceeded its health based screening value for chronic inhalation exposures. However, based upon a review of the toxicological data, we would not expect to see adverse health effects due to either long-term or short-term exposure to manganese. Mixtures of compounds also were evaluated in this consultation. Long-term aggregate exposures to air contaminants in Midlothian are not expected to result in adverse non-cancer or cancer health effects.

A.5., A.7., & C.1. In this health consultation, DSHS has analyzed each and every individual air sampling result collected from all TCEQ sampling locations in the Midlothian area and has not relied on any TCEQ-summarized data. Also, DSHS has not relied on any of the TCEQ's effects

screening levels (ESLs) for determining potential health risks associated with exposures to airborne contaminants in Midlothian.

A.6. & D.4. The community was concerned that the potential for adverse health effects may be underestimated due to averaging of contaminant data over time. The initial screening of the air data involved comparing the maximum concentration for each contaminant to its most conservative health-based screening value. Contaminants whose maximum concentrations exceeded the most conservative health-based screening value were evaluated for acute, intermediate, and long-term exposures. None of the compounds examined (with the exception of benzene) had a single 24-hour measurement that exceeded its acute exposure guideline. The acute inhalation MRL for benzene was exceeded 3 isolated times in 13 years. Consequently, after reviewing all of the available data (which includes 94,932 individual 24-hour measurements), we find no evidence to suggest that adverse health effects would be anticipated as a result of any of the short-term or peak exposures to VOCs or Metals. The potential for adverse health effects due to exposure to EPA's NAAQS compounds will be evaluated in a future health consultation.

A.8., B.4., C.4., & D.1. The community was concerned about asthma, allergies, immune system deficiencies, and other health problems in adults as well as children. Data for these health problems are not routinely collected in Texas. Therefore, we were not able to systematically assess whether the levels of these conditions in Midlothian are different than in other areas of the state.

B.1., B.2., & D.2. Over the years, the Texas Cancer Registry and Texas Birth Defects Registry have conducted incidence, mortality, and prevalence investigations to determine if cancer and birth defect rates were higher or lower in the Midlothian area compared to the rest of the state (Appendix D). No statistically significant elevations of specific or total cancers were found. The prevalences for a few birth defects were higher than expected and for a few other birth defects were lower than expected based on state rates. These higher prevalence rates were not unique to Midlothian/Ellis County but were also observed throughout Health Service Region 3 (which includes 18 other counties primarily north and west of Ellis County). Because of the numerous factors involved, it is not possible to determine if these increases are due to environmental exposures or differences in reporting practices in this region compared with the rest of the state. Furthermore, it should be noted that only 3 of the 99 compounds with health based comparison values (i.e., ethylbenzene, 2-butanone, and methyl isobutyl ketone) listed "developmental effects" as the critical effect (i.e., the first observable physiological or adverse health effect occurring at the lowest exposure dose known to produce any effect at all). Hazard quotients for those 3 compounds were 0.000352, 0.0000653, and 0.00000793 respectively, levels that are far below levels that might be expected to result in an increased risk for birth defects.

B.3. It has been suggested that the Down syndrome cluster reported in Ellis, Hood, and Somervell Counties in 1991-1994 may have been related to a cesium-137 source melt that occurred at Chaparral Steel on September 16, 1993. This might seem plausible in that one of the risk factors for Down syndrome is exposure of the mother or the father to excessive radiation prior to conception of the child. However, the time line is not right for this to have been a possibility, because the non-disjunction of chromosome 21 that results in the manifestations of Down syndrome would have had to have occurred **prior** to the date of the cesium-137 source melt for 15 out of 18 of the reported Down syndrome cases (based on the estimated date of conception for each of the children with Down syndrome). Also, analysis of the wind rose patterns for Midlothian during a similar time period to the cluster (i.e., 1992-94), revealed that

the wind would have been blowing in the direction of one of the Down syndrome cases for less than 2% of the time during the 3-year period. Although the precise wind direction on the exact day of the source melt is not known, the predominant winds are out of the SSE during September, which would have been blowing toward none of the three Down syndrome cases whose estimated date of conception was after the cesium-137 source melt (two of these cases were from Granbury, which is approximately 44 miles west of Midlothian, and the other was from Palmer which is 21 miles ESE of Midlothian). And finally, although the exact quantity of radiation released is unknown, modeling of this release as though the entire source (approximately 89 millicuries of cesium-137) was vaporized and released into the air (and not caught in baghouse dust as most of it was), indicates that the additional radiation would not have been detectable above background radiation levels.

C.2. This concern turned out to be unfounded, in that all three CAMS monitoring locations have collected air sampling data on 97-99 of the 119 different VOCs, amounting to 60,396 individual contaminant measurements. The CAMS-94 location collected air sampling data on 52 metals or other inorganics present in PM<sub>2.5</sub> particulate matter amounting to 8,164 individual contaminant measurements, and the CAMS-302 location collected air sampling data on 24 metals or other inorganics present in PM<sub>10</sub> particulate matter, amounting to 4,344 individual contaminant measurements. Only the CAMS-52 location collected no air samples for metals or other inorganics present in particulate matter. The confusion may have arisen because the CAM sites only collect data for the NAAQS compounds on a continuous basis (i.e., 24 one-hour-average levels per day). The other contaminants (VOCs and metals) are collected noncontinuously as one 24-hour-average level collected once every 6 days.

C.4. & D.5. Health problems reported in domesticated animals and livestock were shared with veterinarians at Texas A&M University. While DSHS does not have animal-species-specific health-based comparison values to evaluate the risks for health effects in animals, many of the health-based comparison values used in our evaluation of human exposures are derived from animal studies and consequently, we would expect these human HAC values to be equally conservative in protecting animal health for most common domestic and farm animals.

### ***Past DSHS Health Data Reviews***

This health consultation summarizes a number of previously published investigations by the DSHS Cancer Registry and the DSHS Birth Defects Registry into cancer incidence, cancer mortality, and birth defect prevalence rates in Midlothian, Ellis County, and Health Service Region 3 compared with Texas (see Appendix D).

The DSHS Cancer Registry has conducted 4 cancer incidence and/or mortality investigations for Midlothian and/or Ellis County from November 1995 through May 2005. Prostate cancer mortality rates were significantly lower in Midlothian compared with Texas and prostate cancer incidence rates were significantly lower in Venus compared with Texas. None of the Midlothian or Ellis County cancer rates (including leukemia, colon, pancreas, lung, trachea, prostate, breast, brain, liver, bladder, uterus, non-Hodgkin's lymphoma, larynx, total childhood cancers, and total cancers) were reported to be significantly higher than the state as a whole.

Maternal age- and race/ethnicity-adjusted prevalence rates for total birth defects and for hypospadias/epispadias in Midlothian were significantly elevated with respect to Texas. Similarly adjusted prevalence rates for total birth defects and for craniosynostosis were significantly elevated in Ellis County with respect to Texas. Similarly adjusted prevalence rates

for total birth defects, craniosynostosis, microcephaly, hypospadias/epispadias, and obstructive genitourinary defects were significantly elevated in Health Service Region 3 with respect to Texas. Similarly adjusted prevalence rates for pyloric stenosis were significantly lower in Health Service Region 3 than in Texas as a whole.

### ***General Findings***

1. One hundred thirteen contaminants (47 VOCs and 66 metals or other inorganic compounds) had no levels exceeding the most conservative HAC value (or had no reported levels above the detection limit). No known health effects are associated with exposure to these contaminants at the concentrations measured in Midlothian; therefore, exposure to these contaminants would not be expected to result in adverse health effects.
2. Health based screening values were not available for 87 contaminants (59 VOCs and 28 metals or other inorganic compounds). Additional information is needed to determine the public health significance of these contaminants.
3. Thirteen VOCs had one or more measured level above the most protective health-based screening value. Three of the VOCs (1,1,2-trimethylbenzene; 1,3,5-trimethylbenzene; and m- and p- xylene) had one or more level above the most conservative contaminant-specific non-cancer screening value. Ten of the VOCs (benzene; 1,3-butadiene; carbon tetrachloride; chloroform; 1,2-dibromoethane; 1,2-dichloroethane; methylene chloride; 1,1,2,2-tetrachloroethane; 1,1,2-trichloroethane; and vinyl chloride) had one or more level above the most conservative contaminant-specific cancer screening value.
4. Fourteen metals or other inorganic compounds had one or more measured level above the most protective health-based screening value. Four of the metals or other inorganic compounds [chlorine (PM<sub>2.5</sub>), lead (TSP), manganese (TSP), and manganese (PM<sub>10</sub>)] had one or more level above the most conservative contaminant-specific non-cancer screening value. Ten metals [arsenic (PM<sub>10</sub>), arsenic (PM<sub>2.5</sub>), arsenic (TSP), beryllium (PM<sub>10</sub>), cadmium (PM<sub>10</sub>), cadmium (PM<sub>2.5</sub>), cadmium (TSP), chromium (PM<sub>10</sub>), chromium (PM<sub>2.5</sub>), and chromium (TSP)] had one or more level above the most conservative contaminant-specific cancer screening value.

### ***Background Comparisons***

1. Five out of 47 VOCs and 11 out of 66 metals or other inorganics that were below health-based screening levels nevertheless slightly exceeded average background levels (levels obtained from other areas in Texas and/or the US).
2. Sixteen out of 59 VOCs and 2 out of 28 metals or other inorganic compounds for which HAC values were not available had average levels slightly above average background.
3. All 13 VOCs having one or more level exceeding its minimum HAC value nevertheless had average levels that were below average background.
4. Seven out of 14 metals having one or more level exceeding its minimum HAC value [arsenic (PM<sub>10</sub>), beryllium (PM<sub>10</sub>), cadmium (PM<sub>10</sub>), chromium (PM<sub>10</sub>), lead (TSP), manganese (TSP), and manganese (PM<sub>10</sub>)] had average levels that also were above average background.

### ***Individual Contaminants – Non-Cancer Health Effects Evaluation***

Using reasonable maximum exposure scenarios, only manganese (both as PM<sub>10</sub> and as TSP) exceeded ATSDR's chronic inhalation MRL by a small margin. After an in-depth review of the toxicological information and the uncertainty factors used in deriving the chronic inhalation MRL, we concluded that it is highly unlikely that the manganese levels seen in Midlothian would result in any observable adverse health effects, even after long-term exposure.

### ***Individual Contaminants – Cancer Health Effects Evaluation***

Exposures Prior to 1982:

Based on ambient air samples collected prior to calendar year 1982, the estimated excess lifetime cancer risks associated with reasonable maximal exposure to arsenic (TSP), cadmium (TSP), and chromium (TSP) ranged from  $5.38 \times 10^{-5}$  (a total of 1 excess cancer in 18,597 people exposed for 70 years) to  $9.30 \times 10^{-5}$  (a total of 1 excess cancer in 10,748 people exposed for 70 years). If these exposures were to continue for 70 years, they would pose a low increased lifetime risk for cancer and would not be expected to result in measurable harmful health effects. Past exposures to these compounds (prior to 1982) therefore posed "no apparent public health hazard."

Exposures 1993 through 2005:

1. The estimated lifetime cancer risks associated with reasonable maximal exposure to arsenic (PM<sub>10</sub>), chromium (PM<sub>10</sub>), and chromium (PM<sub>2.5</sub>) ranged from  $1.68 \times 10^{-5}$  (a total of 1 excess cancer in 59,689 people exposed for 70 years) to  $6.8 \times 10^{-5}$  (a total of 1 excess cancer in 14,714 people exposed for 70 years). Based on available information, we have concluded that exposures to these contaminants pose a low increased lifetime risk for cancer and would not be expected to result in measurable harmful health effects.
2. The estimated lifetime cancer risks associated with reasonable maximal exposure to benzene, carbon tetrachloride, 1,2-dibromoethane, arsenic (PM<sub>2.5</sub>), beryllium (PM<sub>10</sub>), cadmium (PM<sub>10</sub>), and cadmium (PM<sub>2.5</sub>) ranged from  $1.2 \times 10^{-6}$  (a total of 1 excess cancer in 833,333 people exposed for 70 years) to  $9.66 \times 10^{-6}$  (a total of 1 excess cancer in 103,548 people exposed for 70 years). Based on available information we have concluded that exposures to these contaminants pose no apparent increased lifetime risk for cancer and would not be expected to result in measurable harmful health effects.
3. The estimated lifetime cancer risks associated with reasonable maximal exposure to 1,3-butadiene, chloroform, 1,2-dichloroethane, methylene chloride, 1,1,2,2-tetrachloroethane, 1,1,2-trichloroethane, and vinyl chloride ranged from  $5.06 \times 10^{-8}$  (a total of 1 excess cancer in 19,751,644 people exposed for 70 years) to  $8.47 \times 10^{-7}$  (a total of 1 excess cancer in 1,180,057 people exposed for 70 years). Based on available information we have concluded that exposures to these contaminants pose no increased lifetime risk for cancer and would not be expected to result in measurable harmful health effects.

### ***Aggregate Exposures – Non-Cancer Health Effects***

Only one critical non-cancer effect had a HI greater than or equal to 1.0 – the HI for CNS/neurological effects. Although several compounds contributed to CNS/neurological effects, manganese (PM<sub>10</sub>) contributed 96% of this result. The toxicological basis for the health-based criteria for manganese is based on a no-effects level that is over 2,100 times lower than the estimated reasonable maximal exposure estimates used in this analysis. Based on available

information, long-term aggregate exposures to air contaminants in Midlothian would not be likely to result in CNS/neurological effects, either under current or anticipated future conditions.

### ***Aggregate Exposures – Cancer Health Effects***

Total cancers had a cumulative risk for aggregate exposures that exceeded  $1 \times 10^{-4}$  (i.e., exceeded a total of 1 excess cancer in 10,000 people exposed for 70 years). However, this cancer risk estimate is based on the assumption that all chromium (PM<sub>10</sub>) present in the air is chromium(VI), an assumption that is inconsistent with information obtained from other areas of the state. Additional sampling is needed to determine the specific proportions of the major chromium oxidation states and to further refine the total cancer risk estimate.

### ***Overall Conclusions***

We found that the majority of the risks associated with exposure to the chemicals analyzed in this health consultation were low. However, we are classifying this site as an Indeterminate Public Health Hazard because further information is needed to fully characterize the extent of the public health hazard posed by air contaminants in Midlothian. This classification is based on the following facts:

1. Sixteen out of 59 VOCs and 2 out of 28 metals or other inorganic compounds for which health-based screening values were not available had average levels above average background (levels obtained from other areas in Texas and/or the U.S.). Additional information is needed to determine the public health significance of these contaminants.
2. While individual contaminants produced, at most, a low increased lifetime risk for cancer and no apparent public health hazard, under the aggregate exposure scenario, total excess lifetime cancer risk for all cancers combined could be interpreted as posing a public health hazard. However, this conclusion is based on the assumption that all the chromium detected in the air is of the most toxic form [i.e., chromium(VI)], an assumption that is inconsistent with information obtained from other areas of the state. The relative proportions of chromium(III) and chromium(VI) will need to be determined in order to accurately define the risk estimate for total cancer (all sites combined).
3. While this health consultation reviewed the majority of the contaminants measured in Midlothian air (119 VOCs and 108 metals and other inorganics), EPA's NAAQS compounds still need to be evaluated in a future consultation.
4. There are data gaps both in sampling locations and parameters of interest. No air data for the analysis of VOCs were collected prior to 1993. Air data for the analysis of metals and other inorganic compounds were collected at only one location from 1981 through 1984. No air data for these contaminants were collected prior to 1981 and none were collected between 1985 and 1992. For the time periods when air data does exist, data were collected from a limited number of monitoring stations and may not reflect conditions throughout the community. However, since the major monitoring locations were relatively close to one or more of the primary emission sources, we do not anticipate that air pollutant levels for much of the city would be too much higher than those observed.

### ***Recommendations***

We have made the following recommendations in response to these findings:

1. As resources allow, research the toxicology literature for contaminants measured in Midlothian air for which health-based screening values were not available, and determine the potential public health impact of exposures to these substances.
2. Collect additional ambient air samples from previously sampled locations to determine the specific distribution of chromium species and to refine the risk estimates for this contaminant.
3. Evaluate the levels of EPA's NAAQS compounds in the continuous air monitoring data.
4. Where possible identify and fill data gaps with additional data from TCEQ to identify any additional air contaminants that might need evaluation and/or sampling.

### ***Actions Completed***

1. Historically, the TCEQ has collected a vast amount of environmental data in Midlothian, Texas, including air monitoring samples, soil samples, vegetation samples, and others dating back to the early 1980's.
2. Earlier data were analyzed by the TCEQ using EPA methodology and TCEQ's screening levels [4, 10].
3. DSHS staff reviewed summarized monitoring data (1993 through 1995), attended numerous meetings with TCEQ staff and area residents, and distributed questionnaires to see if there were consistent reports of odors, or signs or symptoms of illnesses that might be related to environmental pollution.
4. The Texas Cancer Registry analyzed cancer morbidity and mortality data for Midlothian and Ellis County, looking for any significant increases in cancer rates in this area over the period 1993 through 2002.
5. The Texas Birth Defects Registry analyzed birth defect data for Midlothian, Ellis County, and Health Service Region 3, looking for any significant birth defect elevations during the period 1999 through 2003.
6. DSHS staff conducted site visits in 2005 to determine community concerns, as well as to gather information about the major industries in town. Data from the door-to-door survey (conducted in December 2005) and from mailers which were distributed to ascertain public health concerns were compiled and evaluated to determine additional community health concerns. These concerns were addressed in the "Response to Community Health Concerns" section of this document.
7. DSHS staff obtained detailed (not summarized) TCEQ air monitoring data from 1981 through 1984 and from January 1993 through March 2005 in an electronic format and created a database of monitoring results. With the completion of this health consultation, DSHS has analyzed this data for VOCs and metals or other inorganic compounds and compared these data to health-based screening levels published by ATSDR and EPA. A conservative exposure scenario was generated, and carcinogenic and non-carcinogenic risk estimates were calculated, assuming 70-year lifetime and/or chronic exposures at the reasonable maximal exposure levels seen in the Midlothian area.

### *Actions Under Way*

Currently, DSHS staff are analyzing the hourly NAAQS data (sulfur dioxide, hydrogen sulfide, nitric oxide, nitrogen dioxide, nitrogen oxides, ozone, and particulates) and preparing a health consultation to address these compounds.

### *Actions Planned*

1. DSHS and ATSDR will make this health consultation available to the public, local industries, the local government, and state and federal health/environmental agencies.
2. DSHS and ATSDR will continue to address the community's health concerns relating to air quality.
3. DSHS will discuss with ATSDR the possibility of researching the toxicology literature for contaminants measured in Midlothian air that were at levels above background and for which health-based screening values were not available.
4. DSHS will discuss with TCEQ the potential for determining the specific distribution of chromium species in Midlothian air.
5. DSHS will discuss with TCEQ the potential for identifying and filling data gaps and identifying any additional air contaminants that might need evaluation and/or sampling
6. DSHS will complete the analysis of the hourly NAAQS data.

## Background and Statement of Issues

In July 2005, a group of residents of Midlothian, Ellis County, Texas, submitted a petition to the Agency for Toxic Substances and Disease Registry (ATSDR)<sup>1</sup>. The petitioners requested that ATSDR address their concerns that emissions from nearby industries have been affecting their health. While the petitioners acknowledged the historic involvement of both the Texas Commission on Environmental Quality (TCEQ) and the Texas Department of State Health Services (DSHS), and their predecessor agencies<sup>2</sup>, they outlined continuing issues of concern (see Appendix B). Through a cooperative agreement between ATSDR and DSHS, the petition was accepted in late summer 2005. DSHS agreed to conduct a series of health consultations to address the concerns raised by the citizens.

The town of Midlothian is in Ellis County, 30 miles south of the Dallas/Fort Worth Metroplex. The town consists of commercial/retail buildings and residential properties. The city limits encompass 38 square miles of land [1]. Based on U.S. Census Bureau data for 2000, the population of Midlothian was 7,480; 2,333 of these individuals were under the age of 18 years [1]. While the U.S. Census Bureau has not published official population estimates since 2000, according to the Midlothian Economic Development Board [2], the 2007 population was approximately 13,800 with another 13,003 within the city's extra-territorial jurisdiction. According to the petition there are seven school campuses (6 primary/middle schools and 1 high school); approximately 5,800 children attend school in the city. The schools are clustered near the center of town, and many are located within two miles of one or more industries (see Appendix C, Figure 1).

Midlothian has a history of large scale industrial operations that include three cement plants and a steel mill. Based on a review of the TCEQ Point Source Emission Inventory [3], these industries (Ash Grove Cement Company, Holcim Ltd., TXI Midlothian Cement Plant, and Chaparral Steel Operations) are the top four emission sources in the county. Together they account for 33,018 tons of particulates (PM<sub>10</sub> and PM<sub>2.5</sub>), volatile organic compounds (VOCs), nitrogen oxides (NO<sub>x</sub>), sulfur dioxide, and carbon monoxide emissions per year. Ash Grove Cement Company and Holcim Ltd. are located on the north side of town while TXI Midlothian Cement Plant and Chaparral Steel Operations are located southwest of the city. Other smaller potential sources of industrial emissions include a compressed gas provider, an asphalt plant, dry cleaners, a plastic manufacturing facility, and a power plant [3]. A brief description of the cement manufacturing process and each of the four major facilities is provided below.

### Cement Manufacturing Process

The process of making cement begins by mining limestone from pits located adjacent to the cement plant. After mining, the limestone enters either a wet kiln or dry kiln process. Both processes are used in Midlothian. The wet kiln process requires that the limestone (combined

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<sup>1</sup> Note: Appendix A provides a listing of abbreviations and acronyms used in this report.

<sup>2</sup> Predecessor agencies for the TCEQ include the Texas Air Control Board (TACB) and the Texas Natural Resource Conservation Commission (TNRCC). The predecessor agency for DSHS was the Texas Department of Health (TDH).

with iron oxide, sand, and shale) be finely crushed, mixed with water to form a slurry, and introduced into the upper “cool” end of a rotating cylindrical kiln. Cylindrical kilns are typically 450 feet long and 12 feet in diameter with a 6 to 9 inch thick refractory brick lining. Fuel and air are pumped into the lower “hot” end of the kiln where they burn in a continuous jet of flame at temperatures up to 3,500 °F. The kiln is constructed on an incline, and the limestone slurry moves from the cool upper end to the lower, hot end by the rotating motion of the kiln and the pull of gravity. As the slurry moves closer to the heat source, all moisture evaporates, and numerous chemical reactions occur as the raw materials reach temperatures approaching 2,700 °F. The resulting semi-molten “clinker” is then discharged from the kiln to a cooling unit in golf-ball-sized chunks. After cooling, the clinker is finely ground together with gypsum to form the product known as Portland cement. The dry kiln process requires that the crushed limestone remain dry. The dry limestone is mixed with fuel (coal, tires, etc.) and heated before entering the kiln. The process time associated with manufacturing cement in dry kilns is much shorter than it is in wet kilns.

### **Ash Grove Cement Company**

The Ash Grove Cement Company (Ash Grove, f/k/a North Texas Cement and Gifford Hill) began operations with one wet kiln in 1966. Two additional wet kilns were added in 1969 and 1972 [4]. In 1974, the facility was permitted to use fuel oil to fire all three kilns (TACB Permit 838). The same permit was modified in 1987 to allow the use of Hazardous Waste Derived Fuel (HWDF). Individual permits were granted in 1977 to allow the use of coal in all three kilns. Reportedly, Ash Grove burned HWDF from 1986 to 1992 [4,5]. Use of HWDF was discontinued following a trial burn in 1992, which was conducted to determine whether the facility should be allowed to use HWDF under EPA and TACB rules [4]. In 1995, the facility was permitted to burn whole tires [4]. In October 2005, the facility was reportedly burning coal and tires as fuel.

### **Holcim Ltd.**

In 1987, the existing Holcim facility began operations as Holnam Texas L.P. (f/k/a Box Crow) with one dry kiln [4]. Based on conversations with a local community member the Holcim facility currently operates two dry kilns [6]. In 1998, Holcim installed the newer of the two dry kilns, and has since been exceeding the permitted NO<sub>x</sub> levels. Holcim reportedly applied to further increase NO<sub>x</sub> emissions. In August 2005, a settlement agreement between Blue Skies Alliance, Downwinders at Risk, and Holcim Inc. provided for selective non-catalytic reduction technology to be installed on the new kiln to decrease NO<sub>x</sub> emissions. The agreement also provided for the placement of an air monitor on the northeast corner of the Holcim property to monitor PM<sub>2.5</sub> [7].

According to TCEQ Flexible Permit No. 8996 and PSD-TX-454M3, Holcim can use the following as alternative fuels: a) rubber derived fuel, including tires, hoses, and off-specification rubber goods; b) non-hazardous oil-containing materials, including oil filter fluff, absorbents, rags, and grease; c) non-hazardous oil liquids, including oil, oil-water emulsions, and fuel oils; d) asphalt base composite roofing materials; e) wood chips; and f) activated compounds. Hazardous wasted derived fuel is not permitted.

## **TXI Midlothian Cement Plant**

Based on information obtained from TXI Midlothian Cement Plant (TXI), the largest of the three cement kilns, the TXI facility began operation in the southwest portion of Midlothian in 1960 with one wet cement kiln. Within twelve years, the plant operations increased to include four wet kilns. In 1987, TXI began using HWDF as an alternative fuel source. In 1995, TXI began incorporating slag, which includes chunks of unusable metal residue, from the nearby steel mill as a raw product to create a new cement product. A dry kiln went online in approximately 2002, allowing TXI to produce 2.8 million tons of cement per year. On September 25, 2006, the Executive Director of TCEQ signed a class II modification for TXI Operations, L.P., Industrial Hazardous Waste Permit # 50316. The modification authorized the installation and operation of tire-derived fuel systems on the four wet-process cement kilns [8,9].

## **Chaparral Steel Operations**

Chaparral Steel, a secondary steel mill, is located adjacent to the TXI facility. A secondary steel mill recycles metals to make steel. The facility, which was constructed in 1974, recycles automobiles to create steel beams and reinforcing bars. The operation consists of an automobile shredder, two arc furnaces, and three rolling mills. Air emissions are directed to one positive and two negative pressure bag houses [4]. The furnaces and mills are electric and gas-fired, respectively. At the time of the 1995 TNRCC evaluation, the non-metallic residue from the shredding process was disposed in a “fluff landfill” located in the TXI quarry.

## **Past Environmental Sampling and Data Reviews**

Air monitoring data were collected every six days for a variety of metals and other inorganic constituents of particulates in the Midlothian area sporadically from 1981 to 1984 in accordance with the national schedule. Samples were collected from the roof of the City Hall on North 8th Street and were analyzed for approximately 30 different parameters including total suspended particulates (TSP) adjusted for standard temperature and pressure (STP). No air data were available for the time period from January 1985 through December 1992.

In 1991, the TNRCC initiated an environmental monitoring program in and around Midlothian to evaluate soil, vegetation, slag, and stack emissions for 18 different metals and/or polychlorinated dibenzo-p-dioxins (PCDDs) and dibenzofurans (PCDFs). Of the 175 soil samples collected between 1991 and 1995, 1 sample exceeded the TNRCC’s soil screening level for lead (400 ppm), and 6 out of 140 soil samples exceeded the TNRCC’s soil screening level for arsenic (20 ppm). Measurements for all other soil metals were below their respective soil screening levels. Additional samples were collected in the vicinity of Chaparral Steel. Results from these samples show that 2 out of 22 soil samples collected just outside of the Chaparral property line exceeded the TNRCC’s soil screening level for lead (400 ppm), and 1 out of 22 soil samples exceeded the soil screening level for cadmium (40 ppm) [4, 10]. All other soil metals were below the TNRCC’s respective soil screening levels.

Among 60 soil samples tested, the Toxicity Equivalency Quotient (TEQ) for PCDDs and PCDFs ranged from 0.3-17.9 parts per trillion (ppt); all were below the ATSDR’s health-based soil guidance level of 50 ppt.

Slag (a by-product of steel production) samples were collected and analyzed for 13 different metals; none exceeded their respective soil screening levels.

As part of the Chaparral Steel special study, hay, wheat, and other vegetation samples were collected from the fields surrounding the steel mill. With the exception of aluminum, cadmium, and iron in samples collected in the field immediately south of Chaparral, all measured metal concentrations were below their respective maximum tolerable levels for cattle<sup>3</sup>.

Stack samples were collected from all three cement manufacturing facilities while they were burning different combinations of coal, HWDF, and/or tire-derived fuel. The total 2,3,7,8-Tetrachlorodibenzodioxin (TCDD) Toxicity Equivalency Quotient (TEQ) concentrations estimated for each of the test conditions were all below the TNRCC's screening levels.

Starting in 1993, the TNRCC began collecting air samples for VOCs, particulates, metals, and other inorganic compounds from various locations or Continuous Air Monitoring Stations (CAMS) around the city as follows (see Appendix E, Tables 1a & 1b and Appendix C, Figure 2):

- Tayman Drive (Site 0007):
  - PM<sub>10</sub> Total Particulates (0 to 10 µm), 1993 through 1996 (231 results)
  - Metals & Inorganic Compounds, None
  - VOCs (78 species), 1993 through 1997 (11,135 results)
  
- CAMS-94 (Site 0015):
  - PM<sub>10</sub> Total Particulates (0 to 10 µm), 1994 through 2004 (690 results)
  - PM<sub>2.5</sub> Fine Particulates (0 to 2.5 µm), 2002 through 2004 (157 results)
  - Metals & Inorganics in PM<sub>2.5</sub> (52 species), 2002 through 2004 (8,164 results)
  - VOCs (98 species), 1999 through 2005 (22,955 results)
  
- CAMS-52 (Site 0016):
  - PM<sub>10</sub> Total Particulates (0 to 10 µm), 1994 through 2004 (685 results)
  - Metals & Inorganic Compounds, None
  - VOCs (99 species), 1997 through 2004 (34,842 results)
  
- CAMS-302 (Site 0017):
  - PM<sub>10</sub> Total Particulates (0 to 10 µm), 1999 through 2004 (262 results)
  - Metals & Inorganics in PM<sub>10</sub> (24 species), 2001 through 2004 (4,344 results)
  - VOCs (97 species), 2004 through 2005 (2,599 results)

In 1996, the United States Environmental Protection Agency (EPA) conducted a cumulative risk assessment using air modeling data based upon estimated emissions for the industries in the area during 1985 and 1987 through 1990. In their report, no increased risk for developing cancer or potential for developing non-cancer health effects were identified above the EPA's regulatory standards for acceptable risk [11].

## Past DSHS and ATSDR Involvement and Data Reviews

Between 1992 and 1995 TDH and ATSDR periodically evaluated the air monitoring data collected in the Midlothian area and attended community meetings. The majority of samples

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<sup>3</sup> The National Academy of Science's Subcommittee on Mineral Toxicity has defined the maximum tolerable levels as "that dietary level that, when fed for a limited period, will not impair animal performance and should not produce unsafe residues in human food derived from the animal."

were below the screening levels considered to be health protective at that time [12]. Although no consistent pattern of symptoms or illnesses were noted among area residents, there were common complaints among the residents about sulfur odors and excessive dust.

At the request of various citizens groups, DSHS Birth Defects and Cancer Registries have analyzed data from Midlothian, Venus, Cedar Hill, Ellis County, and Health Service Region 3 to determine prevalence rates for various types of birth defects and the standardized incidence and mortality rates for various types of cancers in the aforementioned areas. Reports were written by the respective registries and summaries of those reports are presented in Appendix D.

## **Methods Used in this Consultation**

Because of the diversity of the health and environmental concerns and the volume of data available for the Midlothian area, several health consultations will be needed to address these concerns. In this consultation we reviewed available air monitoring data with respect to volatile organic compounds (VOCs), metals, and other inorganic compounds. Subsequent consultations are planned to address EPA's National Ambient Air Quality Standards (NAAQS) compounds and consideration of wind patterns and other weather data. Additional consultations may be added based on the results of these analyses.

## **Environmental Data**

We reviewed air monitoring data collected by the TCEQ in the Midlothian area from 1981 through 1984 and from January 1993 through March 2005. Air data were not available prior to 1981 or between January 1985 through December 1992. These data, collected every six days in accordance with the national schedule, include 119 VOCs collected from 4 different monitoring locations and 108 particulate and metal parameters collected from 13 different sampling locations (most data were collected from 6 locations) in and around Midlothian. Current sampling locations and historical sampling sites are shown in Appendix C, Figures 1 and 2. Monitoring site locations and the number of measurements made for VOCs and for metals/inorganic compounds at each site are shown in Appendix E, Tables 1a and 1b, respectively.

## **Quality Assurance/Quality Control**

We obtained detailed (not summarized) ambient air quality data that TCEQ collected in the Midlothian area from May 1981 through March 2005. In preparing this report, DSHS/ATSDR relied on the data provided to us by the TCEQ and assumed adequate quality assurance/quality control (QA/QC) procedures were followed with regard to data collection, chain of custody, laboratory procedures, and data reporting. For the purpose of analysis, concentrations reported as "ND" (or not detected) were assigned numerical values equal to ½ the detection limit for the compound.

## **Health-Based Assessment Comparison (HAC) Values**

Media-specific health-based assessment comparison (HAC) values for non-cancer health effects are generally based on ATSDR's minimal risk levels (MRLs), EPA's reference doses (RfDs), or

for air, EPA's reference concentrations (RfCs). MRLs, RfDs, and RfCs all are based on the assumption that there is an identifiable exposure dose for individuals including sensitive subpopulations, such as pregnant women, infants, children, the elderly, or the immunosuppressed, that is likely to be without appreciable risk for non-cancer health effects even if exposure occurs for a lifetime [13].

When a substance is listed as a carcinogen, the lowest available HAC value usually proves to be the cancer risk evaluation guide or CREG. CREGs are based on EPA's chemical specific cancer slope factor (CSF) and represent the concentration [for airborne contaminants, usually expressed as micrograms per cubic meter ( $\mu\text{g}/\text{m}^3$ )] that would result in a daily exposure dose [expressed as milligrams per kilogram per day ( $\text{mg}/\text{kg}/\text{day}$ )] and theoretical lifetime cancer risk level of one additional cancer case in one million people exposed (a risk of  $1 \times 10^{-6}$ ), assuming a 70 kg person breathes an average of 20 cubic meters ( $\text{m}^3$ ) of air per day over a 70 year lifetime [13].

In general, comparison values are derived for substances for which adequate toxicity data exist for the exposure route of interest. Comparison values may be available for up to three different exposure durations: acute (14 days or less), intermediate (15 to 365 days), and chronic (more than 365 days). Usually, HAC values based on long-term exposure guidelines are lower (more conservative) than HAC values based on short-term exposure guidelines. Thus, the initial screen usually involves comparing each discrete (i.e., short-term) contaminant level with a HAC value based on a long-term exposure guideline.

In some cases where conventional inhalation comparison values (i.e., inhalation MRLs or RfCs) were not available, we used the oral RfD to estimate a "Chronic Inhalation RfD" by converting the RfD dose (in  $\text{mg}/\text{kg}/\text{day}$ ) to an air concentration that would produce the same inhalation dose, assuming a 70 kg body weight and an inhalation daily volume of  $20 \text{ m}^3/\text{day}$ .

The list of substances lacking conventional inhalation comparison values was further shortened by identifying those substances which are known to be essential trace elements (i.e., calcium, chloride, chromium, copper, iodide, iron, manganese, magnesium, molybdenum, phosphorus, potassium, selenium, sodium, and zinc) and for which there are FDA Recommended Dietary Allowances (RDAs). For the essential trace elements, we calculated the air concentration that would produce an inhalation daily dose equal to 1% of the RDA (assuming a daily inhalation volume of  $20 \text{ m}^3/\text{day}$ ) and called this level the Chronic Inhalation RDA. In all cases (except manganese) where one of these elements already has another HAC value, the new "Chr Inh RDA" is more conservative than the preexisting HAC value. Therefore, we used the Chr Inh RDA only for those elements that had no other, more-conventional HAC value.

## Health-Based Screening

For the initial screening of contaminants, we compared the maximum measured air concentration for each contaminant with the minimum air HAC value for that contaminant. When all measured concentrations were below the lowest contaminant-specific HAC value, we concluded that there was no appreciable risk for adverse health effects from that contaminant and it was eliminated from further consideration. Exceeding a screening value did not mean that the contaminant represented a public health threat; rather it suggested that the contaminant warranted further consideration.

Thus, contaminants with one or more measured concentration greater than or equal to the minimum HAC value underwent further evaluation, in which long-term average exposure levels

were estimated, 95% upper confidence limits (95% UCLs) on the long-term averages were determined, and lifetime cancer risks and/or hazard quotients were calculated (see below for description of estimation of long-term exposure levels, and see Appendix F for method for determining 95% UCLs).

Further evaluation of contaminants that exceeded a screening value involved reviewing and integrating relevant toxicological information with the plausible exposures. This involved evaluating whether adverse health effects are possible or probable by comparing the estimated exposures to the reported No Observed Adverse Effect Levels (NOAELs) and/or Lowest Observed Adverse Effect Levels (LOAELs) in animals and/or humans (when available).

## Estimation of Long-Term Exposure Levels

Nearly all air samples collected for the measurement of VOCs, metals, and other inorganic substances have come from 4 primary sampling locations (sites 0007, 0015, 0016, and 0017). Site 0007 is approximately 1.2 miles northeast of Ash Grove and 1.6 miles northwest of Holcim. Sites 0015, 0016, and 0017 are approximately 1.6 miles south, 1.5 miles north, and 1.2 miles northwest of the TXI/Chaparral facilities respectively (see Appendix C, Figure 2 and Appendix E, Tables 1a & 1b). Some Midlothian neighborhoods are located within 1-1.5 miles of one of the major industrial facilities but most are farther away. Since emission levels tend to drop off with distance from the emission source, we expect the levels measured at the 4 primary sampling locations to be fairly representative of the upper range of levels to which the majority of the residents of Midlothian would be exposed. Of course individual exposure concentrations will vary from day-to-day due to changes in emission levels, wind speed and direction, and the movement of people around the city. Consequently, we have averaged the sample results from all monitoring sites together to give the best approximation of the average concentration to which Midlothian residents may have been exposed over extended periods of time.

EPA risk assessment guidance [14] recommends that two exposure point concentrations be used to estimate potential long-term exposures – one based on the arithmetic mean concentration and one based on the 95% upper confidence limit (UCL) of the arithmetic mean. The simple arithmetic mean is the sum of the daily contaminant levels divided by the total number of measurements for that specific contaminant. Using the 95% UCL of the arithmetic mean allows one to account for uncertainties that arise from limited sampling data. As sampling data become less limited (i.e., more samples) the uncertainties decrease and the 95% UCL approaches the true mean. While there are numerous methods that can be used to estimate the 95% UCL – the Land/H-statistic Method, Bootstrap re-sampling, Jackknife, etc. – we used a Monte Carlo simulation to sample repeatedly from the contaminant-specific frequency distributions of observed levels. A brief description of the Monte Carlo methodology used for this consultation can be found in Appendix F.

## Assessing Carcinogenic Risk

To estimate theoretical excess lifetime cancer risk associated with contaminants that exceeded their respective CREGs we multiplied EPA's contaminant-specific inhalation unit risk (IUR) with units of  $(\mu\text{g}/\text{m}^3)^{-1}$  by the 95% UCL of the estimated average daily exposure concentration (converted to  $\mu\text{g}/\text{m}^3$ ) for that contaminant. The IUR is the increase in the lifetime risk of an individual who is exposed to  $1 \mu\text{g}/\text{m}^3$  of the contaminant in air for 70 years. Cancer risk estimates represent the theoretical probability that any exposed individual may develop cancer as

a result of a given carcinogen exposure scenario. The reciprocal of the cancer risk estimate (i.e., 1 divided by the cancer risk estimate) gives the size of the exposed population necessary to expect to see 1 additional cancer case above the background rate if that population is followed for a 70-year “lifetime.” For example, a calculated cancer risk estimate of  $1 \times 10^{-6}$  implies that there is a theoretical probability of one additional cancer case over background rates in a population of 1 million people exposed continuously for a 70-year lifetime at the specified air concentration [13]. To put this in perspective, current US cancer statistics would indicate that approximately 4 out of 10 people will be diagnosed with cancer at some point in their lifetime. This translates to an expected “background” of 400,000 cancer cases occurring in a population of 1 million people followed throughout their lifetimes. Increasing that population’s risk for cancer by  $1 \times 10^{-6}$  brings the expected number of cases to 400,001. It should be noted that, because of the conservative models used to derive IURs, using the above approach provides a theoretical upper bound estimate of the excess risk; the true or actual excess risk is unknown and could be as low as zero [13].

Risk estimates for intermittent exposures and/or less-than-lifetime exposures can be calculated by multiplying the above theoretical cancer risk by an appropriate modifying factor. For example, if the exposure was 8 hours per day (instead of 24) and 260 days per year (instead of 365), the initial risk estimate would be multiplied by  $8/24$  and also by  $260/365$ . Some risk assessors have advocated using 30 years (the 95% UCL on the number of years a person is likely to live at any specific address) as the expected exposure duration and multiplied the lifetime risk estimate by a modifying factor of 0.429 (30 years/70 years). In this health consultation, we have taken a more conservative (health protective) approach and assumed that the duration of exposure was 70 years (no additional modifying factor); while a person may live at one specific address for only 30 years, they may well move to another address in the Midlothian area and still be in a similar exposure situation.

For this consultation, numerical cancer risk estimates have been assigned to public health hazard categories according to the following schedule: Cancer risks of  $9.99 \times 10^{-5}$  or lower were interpreted as “No Apparent Public Health Hazard” for lifetime exposures and cancer risks of  $1.00 \times 10^{-4}$  or higher were interpreted as posing a “Public Health Hazard” for lifetime exposures.

## Background Levels

Many contaminants found in the environment are common to most urban and suburban environments; thus, we also compared the contaminant concentrations found in Midlothian with those found in other urban and suburban areas. To do this we obtained “background” levels from a variety of sources. We obtained background levels for many of the contaminants from TCEQ monitoring results for the town of Kaufman, TX, a town of similar population size, no large industry, and which is only rarely down-wind from Midlothian. We also obtained background levels from the “39-city averages” quoted in the Hazardous Substance Data Base (HSDB), from multiple sites in the state of New York, and from levels described as “median urban levels” in the National Ambient Volatile Organic Compound (NAVOC) database and referenced in HSDB. Finally, we obtained many Texas-specific background levels through individual monitor data queries run against the EPA’s AirData database. In cases where more than one “background” level was found, we averaged all of the available background levels together to arrive at an overall background level. Contaminants for which background concentrations were available were evaluated by dividing the average concentration found in

Midlothian by the identified background level to obtain a “Background Quotient” (BQ). A BQ greater than 1.0 implies that the level in Midlothian is higher than average background; conversely, a BQ less than 1.0 implies that the level in Midlothian is lower than average background.

## **Evaluating Exposure to Chemical Mixtures**

While risk assessments often focus on identifying risks from single contaminant exposures, real-life situations such as the one in Midlothian involve the simultaneous exposure to multiple contaminants. Consequently, in addition to assessing the risks associated with exposure to individual contaminants, we also evaluated aggregate exposures from multiple contaminants for the Midlothian area, both for non-carcinogenic and for carcinogenic effects.

Simultaneous exposures to multiple chemicals may have additive effects (where the combined effect is equal to the sum of the effects of each agent alone), synergistic effects (where the combined effect is greater than the sum of the effects of each agent alone), or antagonistic effects (in which one substance interferes with the effects of another producing a less toxic effect), when compared to a single chemical exposure alone. In general, aggregate exposures to multiple chemicals at levels below their thresholds for minimal effects would, at most, be expected to produce a simple additive effect. Consequently, aggregate exposures to multiple chemicals were evaluated assuming an additive effect. It was also assumed that all compounds contributing to the exposure were elevated in unison and that people were exposed to all the chemicals at the same time.

### ***Chemical Mixtures and Non-Carcinogenic Effects***

To estimate the potential public health significance of simultaneous exposures to multiple chemicals, we tabulated all of the critical effects for each contaminant listed by the EPA on the Integrated Risk Information System (IRIS) database which were the basis for deriving the RfD or the RfC. We also tabulated all of the critical effects listed by the ATSDR in their Toxicological Profile series which were the basis for deriving their inhalation MRLs. The 95% UCL of the estimated average daily exposure dose was divided by the appropriate health-based value to calculate the 95% UCL on the Hazard Quotient (HQ) for a particular critical effect (e.g., CNS effects, developmental effects, liver toxicity, etc.). HQs from multiple contaminants known to produce critical effects of a similar nature or on the same organ system were summed to arrive at the Hazard Index (HI) for each critical effect as a result of exposure to the chemical mixture. Aggregate exposures with an HI less than 1.0 were considered to be without appreciable risk for adverse health effects. Aggregate exposures with an HI greater than 1.0 were subjected to further analysis to determine the potential public health significance.

### ***Chemical Mixtures and Carcinogenic Effects***

To estimate theoretical excess lifetime cancer risks associated with simultaneous exposures to multiple carcinogens, we tabulated all of the cancer critical effects for each contaminant listed by the EPA on the IRIS database which were the basis for deriving the IUR or the oral slope factor (if applicable). For each contaminant, the 95% UCL on the estimated average daily exposure was multiplied by the IUR to calculate the theoretical lifetime risk of developing certain types of cancer (e.g., lung, liver, kidney, etc.), assuming a continuous, 70-year exposure. Risks from exposures to multiple contaminants known to produce the same type of cancer were summed to

obtain an estimate of the total excess risk of developing that cancer as a result of exposure to the chemical mixture. Finally, all of the individual cancer risks were summed to obtain a cumulative cancer risk estimate. Aggregate exposures with a cumulative cancer risk estimate less than  $1 \times 10^{-4}$  were considered to be without appreciable risk for adverse health effects. Aggregate exposures with a cumulative cancer risk estimate greater than  $1 \times 10^{-4}$  were subjected to further analysis to determine the potential public health significance.

## **Child Health Considerations**

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

Health-based assessment comparison values such as the MRLs, RfDs, and RfCs used in this health consultation are all based on the assumption that there is an identifiable exposure dose for individuals including sensitive subpopulations (such as pregnant women, infants, children, the elderly, or the immunosuppressed) that is likely to be without appreciable risk for non-cancer health effects, even if exposure occurs for a lifetime. Each of these HAC values employs an uncertainty factor designed to account for human variability or sensitive subpopulations, including children. With regard to CREG values and potentially increased carcinogenic risks for children, only one of the carcinogens observed in Midlothian air (vinyl chloride) is listed by the EPA as having a mutagenic mode of action. Using the recommended additional age-dependent adjustment factors of 10 for exposures occurring between birth and 2.0 years, and 3 for exposures occurring between the ages of 2.0 and 6.0 years, we would anticipate a 31.3% higher lifetime risk than that calculated by conventional methods.

## **Results and Discussion**

### **Initial Screening Results**

We reviewed data for a total of 227 individual contaminants for this health consultation; this included 119 different VOCs and 108 different inorganic compounds (including metals). Sampling sites are shown in Appendix C, Figures 1 and 2 and site locations are described in Appendix E, Tables 1a and 1b.

Measured concentrations for 47 of the VOCs and 66 of the inorganic compounds were below the detection limit, or, if detected, below the most stringent health-based screening level. These contaminants (see Appendix E, Tables 2a and 2b, respectively) were eliminated from further consideration.

HAC values were not available for 59 of the VOCs and 28 of the inorganic compounds (see Appendix E, Tables 3a and 3b, respectively); however, 43 of the 59 VOCs and 26 of the 28 inorganic compounds were found at levels below the average background levels found in other areas of Texas and the United States. In the absence of health-based screening values, contaminant concentrations below normal background levels suggest that the risks posed by these contaminants in Midlothian are not different than the risks posed by these contaminants in other areas of Texas or the U.S. Conversely, the same cannot be said about the 16 VOCs or the 2 inorganic compounds found at levels above background. Additional information would be required to determine the public health significance of these compounds.

One or more measured concentration of the remaining 13 VOCs and 14 metals/inorganics exceeded the most stringent HAC value for that contaminant (see Appendix E, Tables 4a and 4b, respectively). The 95% UCL on the sample averages are compared with minimum non-cancer HAC values and the resultant hazard quotients and margins of safety for VOCs and metals/inorganics are summarized in Appendix E, Tables 5a and 5b, respectively. The 95% UCL on the sample averages (in  $\mu\text{g}/\text{m}^3$ ) and the contaminant-specific inhalation unit risk factors [in  $(\mu\text{g}/\text{m}^3)^{-1}$ ] are used to calculate cancer risk estimates for the carcinogenic VOCs and metals; these data are presented in Appendix E, Tables 6a and 6b, respectively. The public health significance of exposure to these compounds is discussed in the following sections on VOCs and metals/inorganics.

## Volatile Organic Compounds

We identified the following VOCs as having one or more measured concentration at or above the most stringent health-based screening level for the contaminant: benzene, 1,3-butadiene, carbon tetrachloride, chloroform, 1,2-dibromomethane, 1,2-dichloroethane, methylene chloride, 1,1,2,2-tetrachloroethane, 1,1,2-trichloroethane, 1,2,4-trimethylbenzene, 1,3,5-trimethylbenzene, vinyl chloride, and M+P-xylene (see Appendix E, Table 4a). Figures 3 through 15 (Appendix C) show individual maximum concentrations for each contaminant obtained in any 24-hour period plotted by sample date, the 95% UCL on the sample average, and the most conservative comparison value for each contaminant.

Below we discuss the various physical and chemical properties of each compound along with an evaluation that integrates the relevant toxicological information with plausible exposures. Wherever a plausible exposure exceeded the most stringent health-based comparison value, we compared the estimated exposures to known toxicological endpoints to determine whether adverse health effects are possible or probable.

### *Benzene*

Benzene is a highly flammable, colorless liquid with a sweet odor. Benzene evaporates into air very quickly and dissolves only slightly in water. It was first discovered and isolated from coal tar in the 1800s, but today is made mostly from petroleum. Benzene is used primarily to make other chemicals that are in turn used to make products such as Styrofoam®, plastics, resins, synthetic fibers, rubbers, lubricants, dyes, detergents, drugs, and pesticides. Benzene is present in crude oil, gasoline, and smoke from forest fires and cigarettes [15].

### Absorption, Distribution, & Elimination

Benzene can enter the body through the lungs, gastrointestinal tract, or through the skin. On exposure to high levels of benzene in air, about half of the benzene inhaled passes through the lining of the lungs and enters the bloodstream. When benzene is present in food or drink, most of the benzene ingested passes through the lining of the gastrointestinal tract and enters the bloodstream. During skin contact with benzene or benzene-containing products, a small amount will enter the body by passing through the skin and into the bloodstream. Once in the bloodstream, benzene travels throughout the body and can be temporarily stored in the bone marrow and fat. Benzene is converted to products, called metabolites, in the liver and bone marrow. Some of the harmful effects of benzene exposure are caused by these metabolites. Most of the metabolites of benzene leave the body in the urine within 48 hours after exposure [14].

### Adverse Health Effects

Very high levels of benzene in air (10,000,000 to 20,000,000 ppb) can result in death if a person is exposed for 5 to 10 minutes. Drowsiness, dizziness, rapid heart rate, headaches, tremors, confusion, and unconsciousness may result from exposure to lower levels (700,000 to 3,000,000 ppb). In most cases, people will stop feeling these effects when removed from the source of the exposures and provided with fresh air. Extended periods of exposure can reduce the production of red blood cells, which may cause anemia [16]. Reduction in other components in the blood can cause excessive bleeding. Some of these effects also may stop after stopping the exposure [14].

Occupational exposure to benzene may have resulted in decreased ovary size in some female workers who breathed high levels of benzene for many months. These women also had irregular menstrual periods. No information was available to determine if benzene caused the problems, and the concentration of benzene was undetermined. The effects of benzene exposure on the developing fetus and on male fertility are unknown [14].

Animal studies suggest benzene can cause low birth weight, bone marrow damage, and delayed bone formation in the fetus.

Benzene exposure can affect children in the same ways as adults. Benzene also has been shown to pass from the mother's blood to the fetus. It is not known if children are more susceptible to benzene poisoning than adults [14].

### Carcinogenicity

Long-term exposure to benzene can affect the immune system and cause cancer of the blood-forming organs. Exposure to benzene has been associated with a particular type of leukemia called acute myeloid leukemia (AML) [17,18,19,20,21]. The Department of Health and Human Services (DHHS) has determined that benzene is a known carcinogen. The International Agency for Research on Cancer (IARC) and the EPA have also determined that benzene is carcinogenic to humans [21].

### Health Assessment Comparison Values

The following HAC values have been established for inhalation exposures to benzene:

- CREG 0.0401 ppb (0.128  $\mu\text{g}/\text{m}^3$ )

- Chronic Inhalation RfC 9.39 ppb (30.0  $\mu\text{g}/\text{m}^3$ )
- Chronic Inhalation MRL 3.00 ppb (9.58  $\mu\text{g}/\text{m}^3$ )
- Acute Inhalation MRL 9.00 ppb (28.8  $\mu\text{g}/\text{m}^3$ )
- EPA's Inhalation Unit Risk  $2.2 \times 10^{-6}$  to  $7.8 \times 10^{-6}$  ( $\mu\text{g}/\text{m}^3$ )<sup>-1</sup>

### Results

Benzene was detected at quantifiable levels in 936 of the 952 ambient air samples. Benzene concentrations ranged from an assigned, non-detection value of 0.0005 ppb (one-half the detection limit) to 20.57 ppb, with an average concentration of 0.308 ppb. Through Monte Carlo analysis, the 95% UCL of the arithmetic mean for benzene was estimated to be 0.364 ppb (1.164  $\mu\text{g}/\text{m}^3$ ). The combined average background level of benzene found in 77 Texas sites in 2005, 14 New York State sites in 2003, and 4 New Jersey cities was calculated to be 0.434 ppb. The resulting BQ of 0.710 indicates that the average concentration of benzene found in Midlothian was 1.41 times lower than average background.

### Public Health Implications

*Non-carcinogenic Health Effects Evaluation:* The acute inhalation MRL is based on an animal study in which there was a depression in B- and T-lymphocytes in mice exposed to 10,200 ppb of benzene 6 hours per day for 6 days. Adjusting for 24 hour exposure, the LOAEL was multiplied by 6/24 to give a LOAEL<sub>HEC</sub> (human equivalent concentration) of 2,550 ppb. The acute inhalation MRL of 9 ppb was derived by dividing the LOAEL<sub>HEC</sub> of 2,550 ppb by an overall uncertainty factor of 300 (10 for use of a LOAEL, 3 for extrapolation from animals to humans, and 10 for human variability) [14].

In Midlothian, the acute inhalation MRL was exceeded 3 isolated times in 13 years and the highest observed value of 20.57 ppb is over 123 times lower than the LOAEL<sub>HEC</sub> upon which the acute inhalation MRL is based. Considering the size of the uncertainty factor used in deriving this HAC value and the rarity with which the MRL was exceeded, non-cancer health effects from acute exposure to benzene at the concentrations found in Midlothian are not likely.

The chronic inhalation MRL is based on benchmark dose (BMD) modeling of decreased B cell counts in workers exposed to low levels of benzene. Based on the study results, ATSDR identified a minimally adverse exposure level of 290 ppb. BMD modeling yielded a 95% lower confidence limit on the benchmark concentration (BMCL) of 100 ppb. The chronic inhalation MRL of 3.0 ppb was derived by first adjusting the BMCL for exposure frequency and duration [(100 ppb)  $\times$  (8 hours/24 hours)  $\times$  (6 days/7 days) = ~ 30 ppb] and then by applying an overall uncertainty factor of 10 for human variability [14].

In Midlothian, benzene levels exceeded the most conservative non-cancer screening value (the chronic inhalation MRL) in 7 out of 947 non-zero ambient air samples (0.74%). The highest observed value of 20.57 ppb is 14 times lower than the minimally adverse exposure level. The 95% UCL of the arithmetic mean for benzene of 0.364 ppb, which would be the most representative concentration for chronic exposure, is 8 times lower than the chronic inhalation MRL and 800 times lower than the minimally adverse exposure level. Based on the toxicological information used to derive the chronic inhalation MRL we would not expect to see adverse non-cancer health effects from either short-term or long-term exposure to benzene at the concentrations found in Midlothian.

*Carcinogenic Health Effects Evaluation:* In Midlothian, 926 out of 952 ambient air samples (97.3%) exceeded the CREG for benzene (0.0401 ppb), the most conservative HAC value for this contaminant. The 95% UCL of the arithmetic mean for benzene (0.364 ppb or 1.164  $\mu\text{g}/\text{m}^3$ ) exceeded the CREG for carcinogenic risk. We multiplied the 95% UCL, the most representative concentration for chronic exposure, by the more conservative IUR of  $7.8 \times 10^{-6} (\mu\text{g}/\text{m}^3)^{-1}$  to obtain an estimate of the cancer risk from lifetime exposure to benzene at the concentrations found in Midlothian. Based on these conservative assumptions, we calculated an increased lifetime risk of  $9.08 \times 10^{-6}$ . This means that if 110,170 people were exposed to the levels of benzene found in Midlothian every day for 70-years, theoretically, we would predict that one additional person might get cancer as a result of that exposure. Qualitatively, we would describe a risk of this magnitude as posing no apparent increased lifetime risk for cancer.

### ***1,3-Butadiene***

1,3-Butadiene is a colorless gas with a mild gasoline-like odor that often is found at low levels in urban air samples. The half-life of 1,3-butadiene is 2 hours during sunny conditions, and a few days during overcast (winter) conditions. Large quantities of 1,3-butadiene are produced from petroleum and it is used to make man-made rubber, predominantly to make tires. Small amounts of 1,3-butadiene are found in automobile exhaust, gasoline vapors, cigarette smoke, and the smoke of wood fires. The amount of 1,3-butadiene in the air may be much higher near polluted cities or near oil refineries, chemical manufacturing plants, and plastic and rubber factories where this chemical is made or used [22].

#### Absorption, Distribution, & Elimination

The distribution of 1,3-butadiene in several tissues in rats was measured following a 1-hour inhalation exposure to 129,000,000 ppb [23]. There was a high concentration of 1,3-butadiene in perinephric fat with lower levels in the brain, liver, septum, and kidney. These levels decreased with time; at 90 minutes following inhalation exposure, only trace levels of 1,3-butadiene could be found. Species differences in the distribution of inhaled 1,3-butadiene were studied in Sprague-Dawley rats and B6C3F1 mice [24,25]. The tissues from both species contained high concentrations of  $^{14}\text{C}$ -1,3-butadiene-derived radioactivity 1 hour post exposure. The mouse tissues contained up to seven times more of 1,3-butadiene and its metabolites in their tissues as compared to rats, while up to five times more was detected in their blood.

#### Adverse Health Effects

High levels of 1,3-butadiene exposure over a short period of time cause eye, nose, and throat irritation. The specific concentrations that result in these effects have not been determined. Accidental releases of pure 1,3-butadiene could result in a feeling of drunkenness or death; no such accidental releases have been reported so far [22].

Studies have shown that low levels of 1,3-butadiene exposure for long periods of time may increase the incidence of heart diseases, blood diseases, lung diseases, and even cancer in rubber industry workers. However, these workers also were exposed to other chemicals at the same time, and the other chemicals or a mixture of chemicals may have caused these effects. In addition, the effect of harmful habits like smoking was not considered in the evaluation of health risks of the rubber industry workers [22].

Laboratory animals that breathed high levels (250,000,000 ppb) of 1,3-butadiene for a short time (23 minutes) died. Extended exposures to lower concentrations of the chemical resulted in damage to nose tissues and to the organs responsible for blood cell production. Reproductive effects observed in animals include miscarriage and birth defects. Animals exposed to lower concentrations of 1,3-butadiene for more than one year suffered kidney disease and damage to the lung, liver, and reproductive organs [22].

### Carcinogenicity

There is sufficient evidence from epidemiologic studies in humans to conclude that 1,3-butadiene is carcinogenic to humans; an excess in cancers of the lymphohematopoietic system (leukemia and Non-Hodgkin's lymphoma) were observed in workers exposed to this chemical [26]. Rats and mice that breathed in small amounts of 1,3-butadiene for a long time period developed cancer in many organs [22,26].

### Health Assessment Comparison Values

The following HAC values have been established for inhalation exposures to 1,3-butadiene:

- CREG 0.0151 ppb (0.0333  $\mu\text{g}/\text{m}^3$ )
- Chronic Inhalation RfC 0.9 ppb (2.0  $\mu\text{g}/\text{m}^3$ )
- EPA's Inhalation Unit Risk  $3.0 \times 10^{-5}$  ( $\mu\text{g}/\text{m}^3$ )<sup>-1</sup>

### Results

1,3-Butadiene was detected at quantifiable levels in 79 of the 952 ambient air samples. 1,3-Butadiene concentrations ranged from an assigned, non-detection value of 0.0005 ppb (one-half the detection limit) to 0.340 ppb, with an average concentration of 0.00561 ppb. Through Monte Carlo analysis, the 95% UCL of the arithmetic mean for 1,3-butadiene was estimated to be 0.00703 ppb (0.0155  $\mu\text{g}/\text{m}^3$ ). The combined average background level of 1,3-butadiene found in 78 Texas sites in 2005, 14 New York State sites in 2003, and 4 New Jersey cities was calculated to be 0.157 ppb. The resulting BQ of 0.0359 indicates that the average concentration of 1,3-butadiene found in Midlothian air was 27.9 times lower than average background.

### Public Health Implications

*Non-Carcinogenic Health Effects Evaluation:* The chronic inhalation RfC for 1,3-butadiene is based on a 2-year study of mice exposed to various concentrations of 1,3-butadiene. Mice exposed to the lowest experimental dose of 6,250 ppb for a lifetime exhibited significant increases in the incidence of ovarian atrophy. BMD modeling of the study data yielded a BMCL of 880 ppb. The chronic inhalation RfC of 0.9 ppb was derived by dividing the BMCL (adjusted for exposure frequency and duration) by an overall uncertainty factor of 1,000 (10 for effect-level extrapolation, 10 for intraspecies variability, 3 for interspecies extrapolation, and 3 for database deficiencies) [26].

None of the measured 1,3-butadiene levels exceeded the chronic inhalation RfC, the most conservative non-cancer HAC value for this substance. The maximum recorded concentration in Midlothian (0.34 ppb) is 2.6 times lower than the chronic inhalation RfC and 2,500 times lower than the BMCL. The 95% UCL of the arithmetic mean concentration (0.00703 ppb) is over 125,000 times lower than the BMCL. Based on the toxicological information used to derive the

RfC we would not expect to see any signs or symptoms of adverse non-cancer health effects from either short-term or long-term exposure to 1,3-butadiene at the concentrations found in Midlothian.

*Carcinogenic Health Effects Evaluation:* In Midlothian, 66 of the 952 ambient air samples (6.9%) exceeded the CREG for 1,3-butadiene (0.0151 ppb), the most conservative HAC value for this contaminant. The 95% UCL of the arithmetic mean of the observed 1,3-butadiene levels was selected as the most representative concentration for estimating chronic lifetime exposures. Thus, we multiplied the IUR for 1,3-butadiene by the 95% UCL (converted to  $\mu\text{g}/\text{m}^3$ ) to obtain an estimate of the cancer risk from lifetime exposure to 1,3-butadiene at the concentrations found in Midlothian. Based on this conservative estimate, we calculated an increased lifetime risk of  $4.66 \times 10^{-7}$ . This means that if 2,144,299 people were exposed to the levels of 1,3-butadiene found in Midlothian every day for 70-years, theoretically, we would predict that one additional person might get cancer as a result of that exposure. Qualitatively, we would describe a risk of this magnitude as posing no increased lifetime risk for cancer.

### ***Carbon Tetrachloride***

Carbon tetrachloride is a clear liquid that quickly forms a gas and is used to make refrigeration fluid and propellants for aerosol cans. It does not occur naturally and its manufacture is being phased out. Historically it was widely used as a cleaning fluid by industry and dry cleaning operations, as well as by households for spot removal and degreasing. It was also used in fire extinguishers and as a fumigating pesticide. The majority of these historical uses were discontinued in the mid-1960s. In 1986, the use of carbon tetrachloride as a pesticide was discontinued [27].

#### Absorption, Distribution, & Elimination

Approximately 30 to 40% of the carbon tetrachloride that is inhaled enters the body, where it can temporarily accumulate in fat or enter the kidneys, liver, brain, lungs or skeletal muscle. Most of the carbon tetrachloride leaves the body quickly through expired air but some may be converted into metabolites. Carbon tetrachloride that is stored in fat may stay in the body longer [27].

#### Adverse Health Effects

Information about health effects of carbon tetrachloride in humans comes from cases of accidental, short term exposure to high levels of the chemical. The health effects from long term exposure to low levels of carbon tetrachloride are unknown [27].

Carbon tetrachloride can cause liver and/or kidney damage. In severe cases, parts of the liver may be destroyed, resulting in decreased liver function. Reduced kidney function can result in fluid retention in the body and waste products in the blood. Kidney failure was the primary cause of death from exposure to high concentrations of carbon tetrachloride. If damage to the kidneys and liver is not too severe, the effects will stop within days or weeks after the exposure stops [27].

The nervous system, including the brain, also is affected by exposure to high levels of carbon tetrachloride. Immediate effects include a feeling of drunkenness, headache, and sleepiness; these effects may be accompanied by nausea and vomiting and usually disappear within 1 to 2 days after the exposure stops. Stupor, coma, and permanent cell damage can occur in severe cases. Other less common effects on body tissue also can occur [27].

No studies are available to show the effects of breathing carbon tetrachloride on the human fetus. Some information suggests that carbon tetrachloride may be passed to a baby through breast milk, but the health effects are expected to be low. There is no information to determine how carbon tetrachloride is taken up or eliminated in children, although the processes are likely to be similar [27].

### Carcinogenicity

Although there have been three case reports of liver tumors developing in humans after carbon tetrachloride exposure, available information on this chemical's ability to cause cancer in humans is considered to be inadequate. Carbon tetrachloride has been shown to produce hepatocellular carcinomas in rats, mice, and hamsters (all animal species evaluated to date). Based on the animal data EPA has classified carbon tetrachloride as a probable human carcinogen [28].

### Health Assessment Comparison Values

The following HAC values have been established for inhalation exposures to carbon tetrachloride:

- CREG 0.0106 ppb (0.0667  $\mu\text{g}/\text{m}^3$ )
- Chronic Inhalation MRL 30.0 ppb (189  $\mu\text{g}/\text{m}^3$ )
- EPA's Inhalation Unit Risk  $1.5 \times 10^{-5} (\mu\text{g}/\text{m}^3)^{-1}$

### Results

Carbon tetrachloride was detected at quantifiable levels in 711 (7.46%) of the 952 ambient air samples. Carbon tetrachloride concentrations ranged from an assigned, non-detection value of 0.0005 ppb (one-half the detection limit) to 4.27 ppb, with an average concentration of 0.0907 ppb. Through Monte Carlo analysis, the 95% UCL of the arithmetic mean for carbon tetrachloride was estimated to be 0.102 ppb (0.644  $\mu\text{g}/\text{m}^3$ ). The combined average background level of carbon tetrachloride found in 67 Texas sites in 2005, 14 New York State sites in 2003, and 4 New Jersey cities was calculated to be 0.0952 ppb. The resulting BQ of 0.952 indicates that the average concentration of carbon tetrachloride found in Midlothian was slightly below average background.

### Public Health Implications

*Non-Carcinogenic Health Effects Evaluation:* The chronic inhalation MRL is based on inhalation toxicity studies in rats and mice. The critical effects observed at the LOAEL (25,000 ppb) included increased liver weight, elevated serum enzymes, and liver pathology. A NOAEL of 5,000 ppb was identified for these studies. The human equivalent concentrations were estimated to be 4,500 ppb and 900 ppb for the LOAEL and NOAEL, respectively. The chronic inhalation MRL (30 ppb) was derived by dividing the NOAEL by an overall uncertainty factor of 30 (3 for interspecies extrapolation and 10 for human variability) [27].

None of the carbon tetrachloride measurements exceeded the chronic inhalation MRL. The 95% UCL of the arithmetic mean for carbon tetrachloride, the value most representative of chronic exposure, was almost 300 times lower than the chronic inhalation MRL. Based on this

information we would not expect to see adverse non-cancer health effects from either short-term or long-term exposure to carbon tetrachloride at the concentrations found in Midlothian.

*Carcinogenic Health Effects Evaluation:* In Midlothian, 711 of the 952 ambient air samples (7.47%) exceeded the CREG for carbon tetrachloride (0.0106 ppb), the most conservative HAC value for this contaminant. The 95% UCL of the arithmetic mean of the observed carbon tetrachloride levels was selected as the most representative concentration for estimating chronic lifetime exposures. Thus, we multiplied the IUR for carbon tetrachloride by the 95% UCL (converted to  $\mu\text{g}/\text{m}^3$ ) to obtain an estimate of the cancer risk from lifetime exposure to carbon tetrachloride at the concentrations found in Midlothian. Based on this conservative estimate, we calculated an increased lifetime risk of  $9.66 \times 10^{-6}$ . This means that if 103,548 people were exposed to the levels of carbon tetrachloride found in Midlothian every day for 70-years, theoretically, we would predict that one additional person might get cancer as a result of that exposure. Qualitatively, we would describe a risk of this magnitude as posing no apparent increased lifetime risk for cancer.

### ***Chloroform***

Chloroform is a colorless liquid with a pleasant odor and a slightly sweet taste. It is used to make other chemicals. In the past, chloroform was used as an inhaled anesthetic for surgery. Chemical companies and paper mills are common sources of chloroform [29].

#### Absorption, Distribution, & Elimination

Based on human and animal studies, chloroform can easily enter the body through inhalation and is carried by the blood to the fat, liver, and kidneys. Because chloroform is volatile, it leaves the body shortly after the exposure stops. Chloroform may be converted to metabolites in the body, which may have other health effects in high amounts. Chloroform and some metabolites leave the body in expired air, while other metabolites leave the body in the urine and stool [29].

#### Adverse Health Effects

Breathing high concentrations of chloroform affects the central nervous system (brain), liver, and kidneys. Short-term exposures to high concentrations (approximately 900,000 ppb) have been shown to cause fatigue, dizziness, and headache in exposed workers. Long term exposure to chloroform may cause liver and kidney damage [29].

Reproductive problems and birth defects in humans are not known. However, rats and mice that breathed air containing elevated levels of chloroform (30,000 to 300,000 ppb) had miscarriages. Abnormal sperm were found in mice that breathed air containing elevated levels (400,000 ppb) of chloroform for a few days. Birth defects were observed in rats and mice when chloroform contaminated air was breathed during pregnancy [29].

#### Carcinogenicity

At high doses, chloroform has been reported to cause cancer in animals with significant increases in the incidence of liver tumors in male and female mice and significant increases in the incidence of kidney tumors in male rats and mice [30]. When examining the biology of the tumor production, the occurrence of tumors was species-, strain-, and gender-specific, and had only been observed when the dose was sufficiently high to cause cytotoxicity and regenerative

cell proliferation in the target organ. Based on adequate information on animals EPA has classified chloroform as a probable human carcinogen [30].

### Health Assessment Comparison Values

The following HAC values have been established for inhalation exposures to chloroform:

- CREG 0.00890 ppb (0.0435  $\mu\text{g}/\text{m}^3$ )
- Chronic inhalation MRL 20 ppb (97.6  $\mu\text{g}/\text{m}^3$ )
- Acute inhalation MRL 100 ppb (488  $\mu\text{g}/\text{m}^3$ )
- EPA's Inhalation Unit Risk  $2.3 \times 10^{-5}$  ( $\mu\text{g}/\text{m}^3$ )<sup>-1</sup>

### Results

Chloroform was detected at quantifiable levels in 210 (22.1%) of 952 ambient air samples. Chloroform concentrations ranged from an assigned, non-detection value of 0.0005 ppb (one-half the detection limit) to 0.260 ppb, with an average concentration of 0.00567 ppb. Through Monte Carlo analysis, the 95% UCL of the arithmetic mean for chloroform was estimated to be 0.00657 ppb (0.0321  $\mu\text{g}/\text{m}^3$ ). The combined average background level of chloroform found in 67 Texas sites in 2005, 14 New York State sites in 2003, and 2 New Jersey cities was calculated to be 0.034 ppb. The resulting BQ of 0.167 indicates that the average concentration found in Midlothian was 6.0 times lower than average background.

### Public Health Implications

*Non-Carcinogenic Health Effects Evaluation:* The chronic inhalation MRL is based on liver damage observed in workers exposed to chloroform for 1 to 4 years. The critical effect observed at the LOAEL (2,000 ppb) was hepatomegaly (enlarged liver). The chronic inhalation MRL of 20 ppb was derived by dividing the LOAEL by an overall uncertainty factor of 100 (10 for the use of a LOAEL and 10 for human variability) [29].

None of the chloroform measurements exceeded either the acute inhalation MRL (100 ppb) or the chronic inhalation MRL for chloroform (20 ppb), the most conservative non-cancer HAC value for this contaminant. Thus, we would not expect either acute or chronic adverse non-cancer health effects to result from either short-term or long-term exposure to chloroform at the concentrations found in Midlothian.

*Carcinogenic Health Effects Evaluation:* In Midlothian, 210 (22.1%) of the 952 ambient air samples exceeded the CREG for chloroform (0.00890 ppb), the most conservative HAC value for this contaminant. The 95% UCL of the arithmetic mean of the observed chloroform levels was selected as the most representative concentration for estimating chronic lifetime exposures. Thus, we multiplied the IUR for chloroform by the 95% UCL (converted to  $\mu\text{g}/\text{m}^3$ ) to obtain an estimate of the cancer risk from lifetime exposure to chloroform at the concentrations found in Midlothian. Based on this conservative estimate, we calculated an increased lifetime risk of  $7.38 \times 10^{-7}$ . This means that if 1,354,716 people were exposed to the levels of chloroform found in Midlothian every day for 70-years, theoretically, we would predict that one additional person might get cancer as a result of that exposure. Qualitatively, we would describe a risk of this magnitude as posing no increased lifetime risk for cancer.

## ***1,2-Dibromoethane***

1,2-Dibromoethane is a colorless liquid with a mild, sweet odor. It evaporates easily and can dissolve in water. 1,2-Dibromoethane stays in groundwater and in soil for a long time but breaks down quickly in the air. It is mostly man-made, although small amounts are created in the ocean. Historically, it was used to kill insects that affect crops and to protect grass on golf courses, as well as to kill fruit flies on tropical fruits. For a number of years, 1,2-dibromoethane was added to leaded gasoline to improve fuel efficiency. Most uses for this compound were banned by EPA in 1984 [31].

### Absorption, Distribution, & Elimination

Regardless of how 1,2-dibromoethane enters the body, it rapidly enters the bloodstream where it is taken up by the liver and kidneys. Once in these organs, 1,2-dibromoethane is broken down into other substances which leave the body quickly in the urine and to a lesser extent in stool. Some of the 1,2-dibromoethane that is inhaled exits the body during exhalation [31].

### Adverse Health Effects

Clinical signs (such as depression) in people exposed to high levels of 1,2-dibromoethane are indicative of neurological effects. Except for adverse reproductive effects in men after occupational exposure, chronic non-cancer effects have not been documented in humans. In animals, the liver, kidney, and testis can be affected by 1,2-dibromoethane regardless of the route of exposure [31].

### Carcinogenicity

The human evidence supporting 1,2-dibromoethane as a human carcinogen is considered inadequate; however, the evidence of its ability to cause cancer in animals is considered sufficient enough for it to be considered a probable human carcinogen. In male rats, an increased incidence in forestomach squamous cell carcinoma, hemangiosarcoma, and thyroid follicular cell adenoma was observed. At high doses, female rats also exhibited treatment-related hepatocellular and adrenocortical carcinoma. In male and female mice, forestomach squamous cell carcinoma was the most common treatment-related cancer. Mice of both sexes also exhibited lung adenomas that were considered treatment related [32].

### Health Assessment Comparison Values

The following HAC values have been established for inhalation exposures to 1,2-dibromoethane:

- CREG 0.000217 ppb (0.00167  $\mu\text{g}/\text{m}^3$ )
- Chronic Inhalation RfC 1.21 ppb (9.30  $\mu\text{g}/\text{m}^3$ )
- EPA's Inhalation Unit Risk  $6.0 \times 10^{-4}$  ( $\mu\text{g}/\text{m}^3$ )<sup>-1</sup>

### Results

1,2-Dibromoethane was detected at quantifiable levels in 3 (0.45%) of the 663 ambient air samples. 1,2-Dibromoethane concentrations ranged from an assigned, non-detection value of 0.0005 ppb (one-half the detection limit) to 0.12 ppb, with an average concentration of 0.000840. Through Monte Carlo analysis, the 95% UCL of the arithmetic mean for 1,2-dibromoethane was estimated to be 0.00138 ppb (0.0106  $\mu\text{g}/\text{m}^3$ ). The combined average background level of 1,2-

dibromoethane found in 67 Texas sites in 2005 and 14 New York State sites in 2003 was calculated to be 0.0193 ppb. The resulting BQ of 0.0435 for Midlothian indicates that the average concentration found in Midlothian was 23.0 times lower than average background.

### Public Health Implications

*Non-Carcinogenic Health Effects Evaluation:* The chronic inhalation RfC is based on BMD modeling of a chronic inhalation study in mice in which nasal inflammation was the critical effect. BMD modeling yielded a human equivalent BMCL of 364 ppb. The RfC of 1.21 ppb was derived by dividing the BMCL (adjusted for frequency and duration) by an overall uncertainty factor of 300 (3 for interspecies variability, 10 for intraspecies variability in sensitivity, and 10 for database uncertainty) [32].

None of the 1,2-dibromoethane measurements exceeded the chronic inhalation RfC. The highest observed air level in Midlothian (0.12 ppb) is approximately 10 times lower than the RfC. The 95% UCL of the arithmetic mean for 1,2-dibromoethane (0.00138 ppb), which would be the most representative concentration for chronic exposure, is over 870 times lower than the RfC. Based on available toxicological information we would not expect to see adverse non-cancer health effects from either short-term or long-term exposure to 1,2-dibromethane at the concentrations found in Midlothian.

*Carcinogenic Health Effects Evaluation:* The CREG for 1,2-dibromoethane (0.000217 ppb) is below the detection limit for this compound; consequently, we were not able to determine the number of samples that may have exceeded this value. We multiplied the IUR by the 95% UCL of the arithmetic mean (converted to  $\mu\text{g}/\text{m}^3$ ), the most representative concentration for chronic lifetime exposure, to obtain an estimate of the cancer risk from lifetime exposure at the concentrations found in Midlothian. Based on these conservative assumptions, we calculated an increased lifetime risk of  $6.36 \times 10^{-6}$ . This means that if 157,237 people were exposed to the levels of 1,2-dibromoethane found in Midlothian every day for 70-years, theoretically, we would predict that one additional person might get cancer as a result of that exposure. Qualitatively, we would describe a risk of this magnitude as posing no apparent increased lifetime risk for cancer.

### *1,2-Dichloroethane*

1,2-Dichloroethane is a clear, manufactured liquid that has a pleasant smell and a sweet taste. The most common use of 1,2-dichloroethane is to make vinyl chloride for the production of plastic and vinyl products. It also is used as a solvent and to remove lead from leaded gasoline. Historically, it was found in industrial degreasing products, some household cleaning solutions, pesticides, varnish, finish removers, and adhesives [33].

### Absorption, Distribution, & Elimination

When animals inhale 1,2-dichloroethane it goes to many organs of the body, but usually leaves in the breath within 1 or 2 days. The breakdown products of 1,2-dichloroethane leave the body quickly in the urine [33].

### Adverse Health Effects

People exposed to large amounts of 1,2-dichloroethane in the air often developed nervous system disorders as well as liver and kidney disease. Lung effects also occur after inhaling a large amount of 1,2-dichloroethane; some people exposed to high levels died from heart failure. We

do not know the levels of 1,2-dichloroethane that cause these effects, but they are probably high. Studies in laboratory animals also found that breathing or swallowing large amounts of 1,2-dichloroethane produced nervous system disorders, kidney disease, lung effects, and a reduced ability to fight infection. Long-term exposure to low doses may cause kidney disease in animals [33].

### Carcinogenicity

Exposure to 1,2-dichloroethane has not been associated with cancer in humans. Cancer was found in laboratory animals who were fed large doses of 1,2-dichloroethane. When 1,2-dichloroethane was put on the skin of laboratory animals, they developed lung tumors. We are not sure whether breathing 1,2-dichloroethane causes cancer in animals. Because of the cancer findings in animals we cannot rule out the possibility that it can cause cancer in humans. Thus, EPA has determined that 1,2-dichloroethane is a probable human carcinogen [34].

### Health Assessment Comparison Values

The following HAC values have been established for inhalation exposures to 1,2-dichloroethane:

- CREG 0.00950 ppb (0.0385  $\mu\text{g}/\text{m}^3$ )
- Chronic Inhalation MRL 600 ppb (2,428  $\mu\text{g}/\text{m}^3$ )
- EPA's Inhalation Unit Risk  $2.6 \times 10^{-5}$  ( $\mu\text{g}/\text{m}^3$ )<sup>-1</sup>

### Results

1,2-Dichloroethane was detected at quantifiable levels in 87 (9.14%) of the 952 ambient air samples. 1,2-Dichloroethane concentrations ranged from an assigned, non-detection value of 0.0005 ppb (one-half the detection limit) to 0.46 ppb, with an average concentration of 0.00631 ppb. Through Monte Carlo analysis, the 95% UCL of the arithmetic mean for 1,2-dichloroethane was estimated to be 0.00805 ppb (0.0326  $\mu\text{g}/\text{m}^3$ ). The combined average background level of 1,2-dichloroethane found in 67 Texas sites in 2005 and 14 New York State sites in 2003 was calculated to be 0.0223 ppb. The resulting BQ of 0.284 indicates that the average concentration found in Midlothian was 3.53 times lower than average background.

### Public Health Implications

*Non-Carcinogenic Health Effects Evaluation:* The chronic inhalation MRL for 1,2-dichloroethane was derived by dividing the NOAEL for liver histopathology in rats (50,000 ppb) by an uncertainty factor of 90 (3 for interspecies adjustment after dosimetric adjustment, 10 for human variability, and 3 for database deficiencies) [33].

In Midlothian, none of the 1,2-dichloroethane measurements exceeded the chronic inhalation MRL. The maximum measured value of 0.46 ppb is 1,300 times lower than the chronic inhalation MRL. Based on available toxicological information we would not expect to see adverse non-cancer health effects from either short-term or long-term exposure to 1,2-dichloroethane at the concentrations found in Midlothian.

*Carcinogenic Health Effects Evaluation:* In Midlothian, 87 of the 952 ambient air samples (9.14%) exceeded the CREG of 0.00950 ppb, the most conservative HAC value for this contaminant. Using the 95% UCL as a projected lifetime average exposure level and multiplying by the inhalation unit risk factor, the cancer risk from lifetime exposure to 1,2-

dichloroethane at the concentrations found in Midlothian was estimated to be  $8.47 \times 10^{-7}$ . This means that if 1,180,057 people were exposed to the levels of 1,2-dichloroethane found in Midlothian every day for 70-years, theoretically, we would predict that one additional person might get cancer as a result of that exposure. Qualitatively we would describe a risk of this magnitude as posing no increased lifetime risk for cancer.

### ***Methylene Chloride***

Methylene chloride, also known as dichloromethane, is a colorless liquid with a slightly sweet smell that is made from methane gas or wood alcohol. It is commonly used as a paint stripper or chemical solvent and as a component in some aerosol products and pesticides. Most of the methylene chloride released to the environment results from its use as an end product by various industries and through its use in aerosol products and paint removers in the home. There is no evidence to suggest it occurs naturally [35].

#### Absorption, Distribution, & Elimination

Since methylene chloride quickly evaporates into the air, exposure by breathing is the most likely way that it can get into the body. Most of the methylene chloride that enters the lungs (over 70%) enters the bloodstream and is quickly transported throughout the body. Most of the methylene chloride in the blood goes to the liver, kidney, brain, lungs, and fatty tissue. Some of the methylene chloride in the body is broken down to other chemicals such as carbon monoxide and some accumulates in body fat. About half of the methylene chloride in the blood leaves the body within 40 minutes mainly in exhaled air; a small amount can leave in the urine within 48 hours after exposure [35].

#### Adverse Health Effects

Breathing large concentrations of methylene chloride (800,000 ppb) can reduce reaction time, cause unsteadiness, and affect the ability to perform tasks that require precise hand movement. Breathing it for a long enough period of time may cause dizziness, nausea, tingling in the fingers and toes, and drunkenness. Smaller concentrations of methylene chloride may impair hand eye coordination and decrease attentiveness. In most instances these types of affects disappear shortly after stopping the exposure. Animals studies suggest that exposure to higher concentrations (8,000,000 to 20,000,000 ppb) can lead to unconsciousness and death and that exposure to concentrations as low as 490,000 ppb has resulted in eye irritation and cornea affects. Animals exposed to methylene chloride also have exhibited changes in the liver and kidney; however, these effects have not been observed in humans [35].

No studies have been performed to determine whether methylene chloride affects children differently than adults. There is no information regarding the effects of methylene chloride on reproductive health or whether it is likely to cause birth defect in humans. Some birth defects have been seen in animals inhaling very high levels of methylene chloride [35].

#### Carcinogenicity

Information on whether methylene chloride causes cancer in humans is considered to be inadequate. However, there is sufficient evidence that it can cause cancer in animals. Mice exposed to methylene chloride by inhalation (dose groups ranging from 0 to 4,000,000 ppb) exhibited increased incidences of hepatocellular and alveolar-bronchiolar adenomas and carcinomas. Based on the evidence in animals the World Health Organization (WHO) has

determined that methylene chloride may cause cancer in humans. The Department of Health and Human Services (DHHS) has determined that methylene chloride can be reasonably anticipated to be a cancer-causing chemical. The EPA has determined that methylene chloride is a probable cancer-causing agent in humans [36].

#### Health Assessment Comparison Values

The following HAC values have been established for inhalation exposures to methylene chloride:

- CREG 0.613 ppb (2.13  $\mu\text{g}/\text{m}^3$ )
- Chronic Inhalation MRL 300 ppb (1,042  $\mu\text{g}/\text{m}^3$ )
- Acute Inhalation MRL 600 ppb (2,084  $\mu\text{g}/\text{m}^3$ )
- EPA's Inhalation Unit Risk  $4.7 \times 10^{-7} (\mu\text{g}/\text{m}^3)^{-1}$

#### Results

Methylene chloride was detected at quantifiable levels in 262 (27.5%) of the 952 ambient air samples. Methylene chloride concentrations ranged from an assigned, non-detection value of 0.0005 ppb (one-half the detection limit) to 1.58 ppb, with an average concentration of 0.0304 ppb. Through Monte Carlo analysis, the 95% UCL of the arithmetic mean for methylene chloride was estimated to be 0.0351 ppb (0.122  $\mu\text{g}/\text{m}^3$ ). The combined average background level of methylene chloride found in 67 Texas sites in 2005, 14 New York State sites in 2003, and 4 New Jersey cities was calculated to be 0.153 ppb. The resulting BQ of 0.199 indicates that the average concentration found in Midlothian was 5.0 times lower than average background.

#### Public Health Implications

*Non-Carcinogenic Health Effects Evaluation:* The chronic inhalation MRL of 300 ppb is based on an animal study in which rats, exposed to methylene chloride 6 hours per day/ 5 days per week for two years, exhibited a slight increase in the incidence of vacuoles (holes) in the liver. The human equivalent adjusted lowest observed adverse effect level for this study was determined to be 36,000 ppb. The human equivalent adjusted no observed adverse effect level (NOAEL) was 8,920 ppb. The chronic inhalation MRL was derived by dividing the identified NOAEL of 8,920 ppb by an uncertainty factor of 30 (3 for extrapolation from animals to humans and 10 for human variability) [35].

In Midlothian, none of the measured samples exceeded either the acute or the chronic inhalation MRL, the most conservative non-cancer HAC value for this contaminant. The highest reported concentration (1.58 ppb) was over 300 times lower than the acute inhalation MRL. Likewise, it was 190 times lower than the chronic inhalation MRL, 5,600 times lower than the human equivalent NOAEL, and 22,000 times lower than the human equivalent LOAEL. The 95% UCL of the arithmetic mean, the value most representative of long-term exposure was 8,500 times lower than the chronic inhalation MRL, 250,000 times lower than the human equivalent NOAEL, and over 1,000,000 times lower than the human equivalent LOAEL. Based on available toxicological information we would not expect to see adverse non-cancer health effects from either short-term or long-term exposure to methylene chloride at the concentrations found in Midlothian.

*Carcinogenic Health Effects Evaluation:* In Midlothian, 3 out of 952 ambient air samples (0.315%) exceeded the CREG, the most conservative HAC value for this contaminant. Using the 95% UCL as a projected lifetime average exposure level and multiplying by the inhalation unit risk factor, the cancer risk from lifetime exposure to methylene chloride at the concentrations found in Midlothian was estimated to be  $5.73 \times 10^{-8}$ . This means that if 17,438,716 people were exposed to the levels of methylene chloride found in Midlothian every day for 70-years, theoretically, we would predict that one additional person might get cancer as a result of that exposure. Qualitatively we would describe a risk of this magnitude as posing no increased lifetime risk for cancer.

### ***1,1,2,2-Tetrachloroethane***

1,1,2,2-Tetrachloroethane is a synthetic, colorless, dense liquid with a penetrating, sweet odor similar to chloroform. Historically, 1,1,2,2-tetrachloroethane was used as an industrial solvent and to produce other chemicals; however, large-scale commercial production has stopped because other chemicals now are available to perform the same functions [37].

#### Absorption, Distribution, & Elimination

1,1,2,2-Tetrachloroethane can enter the body when a person breathes air containing the chemical. Studies on people have shown that approximately 97% contained in a single breath can be absorbed into the blood stream. Based on animal studies, when 1,1,2,2-tetrachloroethane is metabolized it is converted to more harmful metabolites. Once in the body most of the chemical and its metabolites leave the body within a few days through the breath or through the urine [37].

#### Adverse Health Effects

Inhalation of 1,1,2,2-tetrachloroethane has been shown to cause death both in animals and humans. Although the concentrations in air that have caused death in humans are not known, concentrations ranging from 1,000,000 ppb to 6,000,000 ppb have been shown to cause death in animals. Exposure to lower concentrations of 1,1,2,2-tetrachloroethane has caused mucosal irritation (13,000 ppb) and gastrointestinal problems (1,000 to 248,000 ppb) in humans. Perhaps the most significant effect seen in humans is on the liver, the major target organ both in humans and animals. People exposed in the workplace have developed jaundice and an enlarged liver; though specific exposure levels were not determined, the concentrations in the workplace ranged from 1,500 to 248,000 ppb. Mice exposed to 600,000 to 800,000 ppb for 3 hours showed fatty changes in the liver. Rats exposed to 130,000 ppb for 5 hours per day, 5 days per week for 15 weeks had increased liver weights, signs of hyperplasia, granulation, and vacuolization of the liver. Rabbits exposed to 15,000 ppb for 7 to 11 months showed early signs of liver degeneration. 1,1,2,2-Tetrachloroethane also may have effects on the eye; people exposed to 130,000 ppb for 10 minutes experienced irritation of the ocular mucosa. The ocular effects are likely due to direct contact rather than true systemic effects [37].

People who inhaled 116,000 ppb of 1,1,2,2-tetrachloroethane for 10 to 30 minutes reported getting dizzy. These effects did not occur at a concentration of 13,000 ppb. Exposure to concentrations ranging from 9,000 to 98,000 ppb for 18 months or less reported symptoms such as headache, tremors, dizziness, numbness, and drowsiness. Recovery from many of the effects of 1,1,2,2-tetrachloroethane cease once the exposure stops. The health effects on people from long-term exposure to small amounts of 1,1,2,2-tetrachloroethane are not known [37].

### Carcinogenicity

No data are available evaluating whether 1,1,2,2-tetrachloroethane causes cancer in people. In a long-term animal study, 1,1,2,2-tetrachloroethane caused an increase in liver carcinomas in mice, but not in rats. The International Agency for Research on Cancer (IARC) has determined that 1,1,2,2-tetrachloroethane cannot be classified as to its ability to cause cancer in humans, while the Environmental Protection Agency (EPA) has determined that the chemical is a possible human carcinogen [38].

### Health Assessment Comparison Values

The following HAC values have been established (or calculated) for inhalation exposures to 1,1,2,2-tetrachloroethane:

- |                                      |                       |  |
|--------------------------------------|-----------------------|--|
| • CREG                               | 0.00251 ppb           | (0.0172 $\mu\text{g}/\text{m}^3$ )         |
| • Provisional Chronic Inhalation MRL | 18.6 ppb (calculated) | (128 $\mu\text{g}/\text{m}^3$ )            |
| • Intermediate Inhalation MRL        | 400 ppb               | (2,746 $\mu\text{g}/\text{m}^3$ )          |
| • EPA's Inhalation Unit Risk         | $5.8 \times 10^{-5}$  | ( $\mu\text{g}/\text{m}^3$ ) <sup>-1</sup> |

### Results

1,1,2,2-Tetrachlorethane was detected at quantifiable levels in 3 of the 407 non-zero ambient air samples. 1,1,2,2-Tetrachloroethane concentrations ranged from an assigned, non-detection value of 0.0005 ppb (one-half the detection limit) to 0.150 ppb, with an average concentration of 0.000914 ppb. Through Monte Carlo analysis, the 95% UCL of the arithmetic mean for 1,1,2,2-tetrachloroethane was estimated to be 0.00158 ppb (0.0109  $\mu\text{g}/\text{m}^3$ ). The combined average background level of 1,1,2,2-tetrachloroethane found in 67 Texas sites in 2005 and 14 New York State sites in 2003 was calculated to be 0.0195 ppb. The resulting BQ of 0.0469 for Midlothian indicates that the average concentration of 1,1,2,2-tetrachlorethane found in Midlothian air was 21.3 times lower than average background.

### Public Health Implications

*Non-Carcinogenic Health Effects Evaluation:* The intermediate inhalation MRL of 400 ppb is based on an animal study in which rats, exposed to 1,1,2,2-tetrachloroethane 5 hours per day/ 5 days per week for 15 weeks, exhibited increased liver weights as well as granulation and vacuolization in liver cells. The lowest observed adverse effect level for this study was determined to be 130,000 ppb. The intermediate inhalation MRL was derived by dividing the identified LOAEL of 130,000 by an uncertainty factor of 300 (3 for use of a LOAEL, 10 for extrapolation of animals to humans, and 10 for human variability) [37].

Neither a chronic RfC nor a chronic inhalation MRL was available for this compound, so we derived a chronic inhalation MRL using the same LOAEL as in the study above. We adjusted for a chronic duration exposure by adjusting the observed LOAEL for 24 hour per day exposures, 7 days per week, 52 weeks per year ( $130,000 \times 5/7 \times 5/24 \times 15/52 = 5,580$  ppb) and dividing the resulting concentration by an uncertainty of 300 ( $5,580/300 = 18.6$  ppb) [37].

In Midlothian, none of the measured concentrations exceeded the chronic inhalation MRL of 18.6 ppb, the most conservative non-cancer HAC value for this contaminant. The highest measured level of 0.15 ppb is 125 times lower than the calculated chronic inhalation MRL. The

95% UCL of the arithmetic mean (0.00158 ppb) is over 11,000 times lower than the calculated chronic inhalation MRL. Additionally, 3 out of 407 samples (0.74%) were detected at quantifiable levels. Based on available toxicological information we would not expect to see adverse non-cancer health effects from either short-term or long-term exposure to 1,1,2,2-tetrachloroethane at the concentrations found in Midlothian.

*Carcinogenic Health Effects Evaluation:* In Midlothian, 3 out of 407 ambient air samples (0.74%) exceeded the CREG for 1,1,2,2-tetrachloroethane of 0.0197 ppb, the most conservative HAC value for this contaminant. Using the 95% UCL as a projected lifetime average exposure level and multiplying by the inhalation unit risk factor, the cancer risk from lifetime exposure to 1,1,2,2-tetrachloroethane at the concentrations found in Midlothian was estimated to be  $6.31 \times 10^{-7}$ . This means that if 1,585,457 people were exposed to the levels of 1,1,2,2-tetrachloroethane found in Midlothian every day for 70-years, theoretically, we would predict that one additional person might get cancer as a result of that exposure. Qualitatively we would describe a risk of this magnitude as posing no increased lifetime risk for cancer.

### ***1,1,2-Trichloroethane***

1,1,2-Trichloroethane is a colorless, sweet-smelling volatile liquid that boils at a higher temperature than water. There is limited information available regarding how much is made and how it is used; although it is known to be used as a solvent. 1,1,2-Trichloroethane is known to be formed in landfills when 1,1,2,2-tetrachloroethane is broken down. Most of the 1,1,2-trichloroethane released into the environment ends up in the air. The half life of 1,1,2-trichloroethane in air is 49 days, meaning that half of the released contaminant will break down in 49 days so it is likely to spread far from where it is released before breaking down [39].

#### Absorption, Distribution, & Elimination

About 90% of the 1,1,2-trichloroethane that enters the lungs is absorbed into the blood and carried throughout the body to the liver, kidney, brain, heart, spleen, and fatty tissue. Most of the 1,1,2-trichloroethane that gets into the body leaves the body unchanged in the breath or as breakdown products in the urine in about one day; very little stays in the body for more than two days [39].

#### Adverse Health Effects

Direct skin contact with 1,1,2-trichloroethane can cause a temporary stinging or burning sensation at the site of contact. There is no information on the health effects caused by inhalation of 1,1,2-trichloroethane in humans. Although the information on other health effects in humans is limited, animals exposed to various concentrations of this compound in the air have exhibited a variety of effects, mostly involving the nervous system (excitation followed by sleepiness) and the liver (increases in certain liver enzymes) [39].

#### Carcinogenicity

Information on whether 1,1,2-trichloroethane causes cancer in humans is not available; however, mice fed 1,1,2-trichloroethane by gavage 5 times per week for 78 weeks exhibited an increased incidence of hepatocellular carcinomas. A dose related increase in pheochromocytomas (tumors of the adrenal gland) was also observed in female mice. Rats fed lower doses of the substances did not exhibit any statistically significant increase in tumor incidence as a function of treatment; however, adrenal cortex carcinomas, carcinomas of the kidney, and hemangiosarcomas of the

spleen, pancreas, and abdomen were found in treated animals but not in controls. Based on the animal data EPA has classified this compound as a Class C, possible human carcinogen [40].

#### Health Assessment Comparison Values

The following HAC values have been established (or calculated) for inhalation exposures to 1,1,2-trichloroethane:

- CREG 0.0115 ppb (0.0627  $\mu\text{g}/\text{m}^3$ )
- Provisional Chronic Inhalation RfD 2.57 ppb (calculated) (14.0  $\mu\text{g}/\text{m}^3$ )
- EPA's Inhalation Unit Risk  $1.6 \times 10^{-5}$  ( $\mu\text{g}/\text{m}^3$ )<sup>-1</sup>

#### Results

1,1,2-Trichloroethane was detected at quantifiable levels in 1 (0.13%) of the 831 ambient air samples. 1,1,2-Trichloroethane concentrations ranged from an assigned, non-detection value of 0.0005 ppb (one-half the detection limit) to 0.150 ppb, with an average concentration of 0.000681 ppb. Through Monte Carlo analysis, the 95% UCL of the arithmetic mean for 1,1,2-trichloroethane was estimated to be 0.00101 ppb (0.00551  $\mu\text{g}/\text{m}^3$ ). The combined average background level of 1,1,2-trichloroethane found in 67 Texas sites in 2005 and 14 New York State sites in 2003 was calculated to be 0.0213 ppb. The resulting BQ of 0.0320 for Midlothian indicates that the average concentration of 1,1,2-trichloroethane found in Midlothian air was 31.3 times lower than average background.

#### Public Health Implications

*Non-Carcinogenic Health Effects Evaluation:* No acute, intermediate, or chronic inhalation MRLs were available for 1,1,2-trichloroethane. Consequently, we derived a chronic inhalation RfD by calculating the air concentration necessary to produce an inhalation dose equivalent to the RfD for 1,1,2-trichloroethane ( $4.0 \times 10^{-3}$  mg/kg/day), assuming a 70 kg body weight, and a respiratory daily volume of 20 m<sup>3</sup> per day [40].

In Midlothian, none of the measured 1,1,2-trichloroethane levels exceeded the calculated chronic inhalation RfD. The 95% UCL of the arithmetic mean, the value considered to be most representative of chronic exposure, is over 2,500 times lower than the chronic inhalation RfD. Based on available toxicological information we would not expect to see adverse non-cancer health effects from either short-term or long-term exposure to 1,1,2-trichloroethane at the concentrations found in Midlothian.

*Carcinogenic Health Effects Evaluation:* In Midlothian, 1 out of 831 ambient air samples (0.12%) exceeded the CREG, the most conservative HAC value for this contaminant. Using the 95% UCL as the projected lifetime average exposure level and multiplying by the inhalation unit risk factor, the cancer risk for lifetime exposure to 1,1,2-trichloroethane at the concentrations found in Midlothian was estimated to be  $8.81 \times 10^{-8}$ . This means that if 11,345,339 people were exposed to the levels of 1,1,2-trichloroethane found in Midlothian every day for 70-years, theoretically, we would predict that one additional person might get cancer as a result of that exposure. Qualitatively we would describe a risk of this magnitude as posing no increased lifetime risk for cancer.

### ***Trimethylbenzenes***

The trimethylbenzenes (1,2,4-trimethylbenzene a.k.a. pseudocumene and 1,3,5-trimethylbenzene a.k.a. mesitylene) are colorless flammable liquids in pure form. They are found in paint thinners and paints, and are naturally occurring in petroleum products, including coal, crude oil, and gasoline. They are used as a solvent in coatings, cleaners, pesticides, and printing inks [41].

#### Absorption, Distribution, & Elimination

Trimethylbenzenes are absorbed into the blood stream through the lungs. Approximately, 85% of the chemical in the blood is bound to red blood cells. Trimethylbenzene is mainly eliminated from the body in exhaled air (parent compound) and in the urine (metabolites). The half-life for the elimination of metabolites in urine ranges from 9.5 to 37.6 hours, depending on the metabolite [41].

#### Adverse Health Effects

Trimethylbenzenes are classified as CNS depressants and skin, eye, and respiratory irritants. Workers breathing high concentrations of trimethylbenzene in the air experienced headache, fatigue, drowsiness, and bronchitis. Long-term exposure to 1,2,4-trimethylbenzene and/or 1,3,5-trimethylbenzene may cause nervousness, tension, and bronchitis. Painters who worked for several years with solvents containing various trimethylbenzenes showed nervousness, tension, anxiety, asthmatic bronchitis, anemia, and alterations in the ability of the blood to clot. Hydrocarbon vapor concentrations ranged from 10,000 to 60,000 ppb [42,43].

#### Carcinogenicity

We were not able to find any information as to the ability of trimethylbenzene to cause cancer in animals or humans [41].

#### Health Assessment Comparison Values

The following HAC values have been established for inhalation exposures to trimethylbenzene:

- Provisional Chronic Inhalation RfC            1.221 ppb [44]            (6.00  $\mu\text{g}/\text{m}^3$ )

#### Results – 1,2,4-Trimethylbenzene

1,2,4-Trimethylbenzene was detected at quantifiable levels in 287 (33.4%) out of 858 ambient air samples. 1,2,4-Trimethylbenzene concentrations ranged from an assigned, non-detection value of 0.0005 ppb (one-half the detection limit) to 7.33 ppb, with an average concentration of 0.0498 ppb. Through Monte Carlo analysis, the 95% UCL of the arithmetic mean for 1,2,4-trimethylbenzene was estimated to be 0.0709 ppb (0.349  $\mu\text{g}/\text{m}^3$ ). The combined average background level of 1,2,4-trimethylbenzene found in 77 Texas sites in 2005, 14 New York sites in 2003, and 4 New Jersey cities was calculated to be 0.125 ppb. The resulting BQ of 0.399 for Midlothian indicates that the average concentration found in Midlothian was 2.5 times lower than average background.

### Public Health Implications – 1,2,4-Trimethylbenzene

*Non-Carcinogenic Health Effects Evaluation:* Levels of 1,2,4-trimethylbenzene exceeded the provisional chronic inhalation RfC of 1.221 ppb, the most conservative HAC value for this contaminant, in 5 out of 855 non-zero ambient air samples (0.58%).

The 95% UCL of the arithmetic mean for 1,2,4-trimethylbenzene, the concentrations most representative of chronic exposure, was over 17 times lower than the provisional chronic inhalation RfC. Based on available toxicological information we would not expect to see adverse non-cancer health effects from either short-term or long-term exposure to 1,2,4-trimethylbenzene at the concentrations found in Midlothian.

### Results – 1,3,5-Trimethylbenzene

1,3,5-Trimethylbenzene was detected at quantifiable levels in 160 (18.6%) out of 858 ambient air samples. 1,3,5-Trimethylbenzene concentrations ranged from an assigned, non-detection value of 0.0005 ppb (one-half the detection limit) to 2.030 ppb, with an average concentration of 0.0154 ppb. Through Monte Carlo analysis, the 95% UCL of the arithmetic mean for 1,3,5-trimethylbenzene was estimated to be 0.0215 ppb (0.106  $\mu\text{g}/\text{m}^3$ ). The combined average background level of 1,3,5-trimethylbenzene found in 77 Texas sites in 2005, 14 New York sites in 2003, and 4 New Jersey cities was calculated to be 0.0401 ppb. The resulting BQ of 0.384 for Midlothian indicates that the average concentration found in Midlothian was 2.6 times lower than average background.

### Public Health Implications – 1,3,5-Trimethylbenzene

*Non-Carcinogenic Health Effects Evaluation:* Levels of 1,3,5-trimethylbenzene exceeded the provisional chronic inhalation RfC of 1.221 ppb, the most conservative HAC value for this contaminant, in 2 out of 858 non-zero ambient air samples (0.23%).

The 95% UCL of the arithmetic mean for 1,3,5-trimethylbenzene, the concentrations most representative of chronic exposure, was over 56 times lower than the provisional chronic RfC. Based on available toxicological information we would not expect to see adverse non-cancer health effects from either short-term or long-term exposure to 1,3,5-trimethylbenzene at the concentrations found in Midlothian.

### ***Vinyl Chloride***

Vinyl chloride, also called chloroethene, chloroethylene, ethylene monochloride, or monochloroethylene is a colorless gas that is unstable at high temperatures. It is a manufactured substance that does not occur naturally; but it can be formed in the environment when other substances such as trichloroethylene break down. In the United States, most vinyl chloride is used to make polyvinyl chloride (PVC), a component of several plastic products including PVC pipe, packaging materials, automobile upholstery, house wares, and wall coverings. Historically, vinyl chloride was used as a coolant, as a propellant in spray cans, and in cosmetics; although these uses were discontinued in the mid-1970s [45].

### Absorption, Distribution, & Elimination

At low concentrations in air (<20,000 ppb) most of the vinyl chloride that is inhaled quickly enters the bloodstream where it is transported throughout the body. Vinyl chloride that enters the body can be excreted through the urine; however, some is changed to other substances by the

liver. Some of the substances produced by the liver are more harmful than the vinyl chloride and can cause damage. Eventually, even these new compounds leave the body. If you take in more vinyl chloride than the liver can handle, the vinyl chloride is released from the body in expired air [45].

### Adverse Health Effects

Vinyl chloride at high concentrations (10,000,000 ppb) will result in dizziness or sleepiness within five minutes, and higher concentrations (25,000,000 ppb vinyl chloride) may cause unconsciousness. These effects will stop when exposure to the vinyl chloride stops. There have been contradictory reports on the effects of vinyl chloride on the lungs; however, adverse respiratory effects have been reported in several epidemiologic studies. The reports include: increased incidence of emphysema, decreased respiratory volume, decreased vital capacity, decreased oxygen and carbon dioxide diffusion, pulmonary fibrosis, abnormal chest x-rays, and dyspnea. Animal studies have supported the ability of vinyl chloride to adversely affect the lungs. Vinyl chloride also may affect the blood. Female workers exposed to vinyl chloride at concentrations ranging from 200 to 130,700 ppb had a significantly lower number of platelets than non-exposed workers during the early part of their pregnancies [45].

Workers who breathed vinyl chloride for several years exhibited changes in the structure of their livers. These liver changes are more likely to occur in people who breathe high levels of vinyl chloride. Vinyl chloride-induced liver damage includes: hypertrophy and hyperplasia of liver cells, fibrosis of the portal tracts and other areas, and focal areas of liver degeneration. Working with vinyl chloride also has been associated with nerve damage, immune reactions, scleroderma like skin changes, decreased blood flow to the hands, and resorption of the bones in the tips of the fingers. Studies in animals generally support the adverse health effects observed in people [45].

No information is available to determine if vinyl chloride affects children differently than adults. While effects reported in exposed workers could occur in children, the levels used in these studies were much higher than those found in ambient air. Some studies suggest a possible association between birth defects and vinyl chloride exposure of the parents of affected children. Animal studies also suggest that infants and young children might be more susceptible to vinyl chloride-induced cancers than adults [45].

### Carcinogenicity

Based on sufficient evidence from human epidemiology studies vinyl chloride is considered to be a known human carcinogen by the inhalation route of exposure. This classification is supported by a consistent causal association between occupational exposure to vinyl chloride and the development of angiosarcoma, an extremely rare tumor; consistent evidence of carcinogenicity in rats, mice, and hamsters both by the oral and inhalation routes of exposure; and mutagenicity and deoxyribonucleic acid (DNA) adduct formation by vinyl chloride and its metabolites in numerous in vivo and in vitro test systems [46]. The International Agency for Research on Cancer (IARC) has concluded that vinyl chloride is carcinogenic to humans [47].

### Health Assessment Comparison Values

The following HAC values have been established for inhalation exposures to vinyl chloride:

- CREG 0.0445 ppb (0.114  $\mu\text{g}/\text{m}^3$ )

- Chronic Inhalation RfC 39.1 ppb (100  $\mu\text{g}/\text{m}^3$ )
- Acute Inhalation MRL 500 ppb (1,278  $\mu\text{g}/\text{m}^3$ )
- EPA's Inhalation Unit Risk  $8.8 \times 10^{-6}$  ( $\mu\text{g}/\text{m}^3$ )<sup>-1</sup>

### Results

Vinyl chloride was detected at quantifiable levels in 14 (1.47%) of the 952 ambient air samples. Vinyl chloride concentrations ranged from an assigned, non-detection value of 0.0005 ppb (one-half the detection limit) to 0.120 ppb, with an average concentration of 0.00126 ppb. Through Monte Carlo analysis, the 95% UCL of the arithmetic mean for vinyl chloride was estimated to be 0.00171 ppb (0.00438  $\mu\text{g}/\text{m}^3$ ). The combined average background level of vinyl chloride found in 67 Texas sites in 2005 and 14 New York State sites in 2003 was calculated to be 0.0141 ppb. The resulting BQ of 0.0898 for Midlothian indicates that the average concentration found in Midlothian was 11.1 times lower than average background.

### Public Health Implications

*Non-Carcinogenic Health Effects Evaluation:* The acute inhalation MRL of 500 ppb was derived from a NOAEL of 50,000 ppb for developmental effects in mice exposed 7 hours/day. The NOAEL was then multiplied by 7/24 in order to convert from intermittent to continuous exposure to give a duration-adjusted NOAEL of 15,000 ppb. Since the blood-gas partition coefficient is greater in animals than in humans, no additional factors were needed to derive the human equivalent concentration (NOAEL<sub>HEC</sub>), of 15,000 ppb. A total uncertainty factor of 30 (3 for extrapolation from animals to humans and 10 for human variability) was applied to the NOAEL<sub>HEC</sub> to arrive at the acute inhalation MRL of 500 ppb [45].

In Midlothian, none of the measured vinyl chloride levels exceeded the acute inhalation MRL (500 ppb). The highest level measured (0.12 ppb) was 4,167 times lower than the acute inhalation MRL and 125,000 times lower than the NOAEL<sub>HEC</sub>. Based on these data, we would not expect to see signs or symptoms of acute exposure to vinyl chloride in Midlothian.

The chronic inhalation RfC of 39.1 ppb is based on a chronic dietary study in rats reporting a NOAEL for liver cell polymorphism of 0.13 mg/kg/day [46]. The rationale for basing an inhalation RfC on an oral study is based on evidence for a mode of action common to exposures from either route (liver toxicity) and availability of PBPK models to perform route-to-route extrapolations. Conversion of the study NOAEL was then accomplished by dividing the animal dose metric for this concentration (3.00) by the conversion factor (1.18) to arrive at a NOAEL<sub>HEC</sub> of 2,500  $\mu\text{g}/\text{m}^3$ . The chronic inhalation RfC of 39.1 ppb (100  $\mu\text{g}/\text{m}^3$ ) was derived by dividing the NOAEL<sub>HEC</sub> by a total uncertainty factor of 30 (3 for extrapolation from animals to humans and 10 for human variability) [46].

None of the measured vinyl chloride levels exceeded the chronic inhalation RfC (39.1 ppb), the most conservative non-cancer HAC value for this contaminant. The maximum detected value of 0.120 ppb was over 4,100 times lower than the acute inhalation MRL. The 95% UCL of the arithmetic mean, the value considered to be most representative of chronic exposure, is over 22,800 times lower than the chronic inhalation RfC. Based on available toxicological information we would not expect to see adverse non-cancer health effects from either short-term or long-term exposure to vinyl chloride at the concentrations found in Midlothian.

*Carcinogenic Health Effects Evaluation:* In Midlothian, 7 out of 952 ambient air samples (0.74%) exceeded the CREG, the most conservative HAC value for this contaminant. Using the 95% UCL as the projected lifetime average exposure level and multiplying by the inhalation unit risk factor, the cancer risk from lifetime exposure to vinyl chloride at the concentrations found in Midlothian was estimated to be  $3.86 \times 10^{-8}$ . This means that if 25,933,909 people were exposed to the levels of vinyl chloride found in Midlothian every day for 70-years, theoretically, we would predict that one additional person might get cancer as a result of that exposure. Qualitatively we would describe a risk of this magnitude as posing no increased lifetime risk for cancer. Using the recommended additional age-dependent adjustment factors of 10 for exposures occurring between birth and 2.0 years, and 3 for exposures occurring between the ages of 2.0 and 6.0 years, we would anticipate a cancer risk of  $5.06 \times 10^{-8}$ . This means that if 19,751,644 people were exposed to the levels of vinyl chloride found in Midlothian every day for 70-years, theoretically, we would predict that one additional person might get cancer as a result of that exposure. Qualitatively we would describe a risk of this magnitude as posing no increased lifetime risk for cancer.

### *Xylenes*

M-, o-, and p-xylene are the three isomers or forms of xylene. They differ in the positioning of methyl groups on the benzene ring. The term “total xylenes” refers to all three isomers of xylene (m-, o-, and p-xylene). Chemical industries produce xylene from petroleum. Xylene also occurs naturally in petroleum and coal tar and is formed during forest fires, to a small extent. It is a colorless, flammable liquid with a sweet odor, and it is used as a solvent, cleaning agent, and paint thinner. Xylene also is used to manufacture plastics and coating for fabrics and paper [48].

#### Absorption, Distribution, & Elimination

When xylene enters the lungs it is rapidly absorbed into the blood. In both people and animals most of the xylene that is inhaled is broken down by the liver into other more water soluble chemicals which may be quickly excreted in the urine. Some of the xylene that is inhaled leaves in expired air. Some of the breakdown products have appeared in the urine as soon as 2 hours after inhalation. One of the breakdown products of xylene, methylbenzaldehyde, is harmful to the lungs of some animals; however, this chemical has not been found in humans. Most xylene that is taken in leaves the body within 18 hours after exposure ends; however, storage of xylene in fat or muscle may prolong the time needed for xylene to leave the body [48].

#### Adverse Health Effects

People exposed to xylene concentrations ranging from 50,000 ppb to 200,000 ppb for short periods of time (3 to 5 minutes to 2 hours) have reported nose and throat irritation. Exposure to high concentrations (10,000,000 ppb) for several hours has resulted in intra-alveolar hemorrhage and pulmonary edema. Chronic (long-term) exposure to a geometric mean xylene concentration of 14,000 ppb also has resulted in respiratory (nose and throat irritation) and neurological (reduced muscle power) effects. Workers exposed to unspecified concentrations of xylene in the air exhibited symptoms of nausea, vomiting, and gastric discomfort. Workers exposed to 700,000 ppb xylene exhibited transient elevations in certain liver enzymes and others exposed to lower levels (14,000 ppb) for an average of 7 years did not show any changes in liver enzymes that would be indicative of altered liver function [48].

There are no studies to determine if xylene exposure affects children differently than it affects adults. Exposure of pregnant women to high levels of xylene may cause harmful effects to the fetus. Studies of unborn animals indicate that high concentrations of xylene may cause increased numbers of deaths, decreased weight, skeletal changes, and delayed skeletal development. In many instances, these same high concentrations also cause damage to the mothers [48].

### Carcinogenicity

Information from either human or animal studies is not adequate to determine whether or not xylene causes cancer in humans. Both the International Agency for Research on Cancer (IARC) and EPA have found that there is insufficient information to determine whether or not xylene is carcinogenic and consider xylene not classifiable as to its human carcinogenicity [49].

### Health Assessment Comparison Values

The following HAC values have been established for inhalation exposures to total xylene:

- Chronic Inhalation RfC      23.0 ppb      (100  $\mu\text{g}/\text{m}^3$ )
- Chronic Inhalation MRL      50.0 ppb      (217  $\mu\text{g}/\text{m}^3$ )
- Acute Inhalation MRL      2,000 ppb      (8,684  $\mu\text{g}/\text{m}^3$ )

### Results

M- and p-xylene concentrations were combined in the analytical data provided. Because the concentrations were combined, m- and p-xylene values were compared to the HAC values for total xylene. Xylene was detected at quantifiable levels in 561 (61.5%) of the 912 ambient air samples. M- and p-xylene concentrations ranged from an assigned, non-detection value of 0.0005 ppb (one-half the detection limit) to 32.05 ppb, with an average concentration of 0.178 ppb. Through Monte Carlo analysis, the 95% UCL of the arithmetic mean for m- and p-xylene was estimated to be 0.263 ppb (1.14  $\mu\text{g}/\text{m}^3$ ). The combined average background level of m- and p-xylene found in 76 Texas sites in 2005, 14 New York State sites in 2003, and 4 New Jersey cities was calculated to be 0.371 ppb. The resulting BQ of 0.480 for Midlothian indicates that the average concentration found in Midlothian was 2.08 times lower than average background.

### Public Health Implications

*Non-Carcinogenic Health Effects Evaluation:* The acute inhalation MRL for xylene is based on a human study of 56 healthy volunteers exposed to 50,000 ppb or 150,000 ppb of mixed xylenes for 2 hours at a time, every 2 weeks, for a total of 3 treatments. The minimal LOAEL for reduced lung function, respiratory symptoms, and CNS effects was found to be 50,000 ppb. This value was divided by a total uncertainty factor of 30 (3 for use of a minimal LOAEL and 10 for human variability) to arrive at the acute inhalation MRL of 2,000 ppb [48].

None of the measured levels in Midlothian exceeded the acute inhalation MRL for xylene. The highest recorded level (32.05 ppb) was over 62 times lower than the acute inhalation MRL and over 1,560 times lower than the LOAEL upon which the acute MRL is based. Consequently, we would not expect to see signs or symptoms of acute exposure to xylene at the levels seen in Midlothian.

The chronic inhalation RfC was based on a subchronic inhalation study in male rats that reported a NOAEL of 50,000 ppb (217,117  $\mu\text{g}/\text{m}^3$ ) for impaired motor coordination (decreased rotarod

performance) in animals exposed for 6 hours/day, 5 days/week. After adjusting for exposure duration ( $217,117 \times 6/24 \times 5/7 = 38,771 \approx 39,000$ ) and blood/gas partition coefficients (factor = 1), the  $\text{NOAEL}_{\text{HEC}}$  was determined to be  $39,000 \mu\text{g}/\text{m}^3$ . This value was divided by a total uncertainty factor of 300 (3 for extrapolation from animals to humans, 10 for human variability, 3 for extrapolation from subchronic to chronic, and 3 for uncertainties in the database) to arrive at a chronic inhalation RfC of  $100 \mu\text{g}/\text{m}^3$  (23.0 ppb) [49].

Out of 912 ambient air samples, 1 measurement (0.11%) exceeded the chronic inhalation RfC for xylene, the most conservative HAC value for this contaminant. The 95% UCL of the sample average, the value most representative of chronic exposure, was over 87 times lower than the chronic inhalation RfC and over 34,100 times lower than the human equivalent NOAEL from which the RfC was derived. Based on the toxicological information used to derive the RfC we would not expect to see adverse non-cancer health effects from either short-term or long-term exposure to m- and p-xylene at the concentrations found in Midlothian.

## Metals and Other Inorganic Contaminants

The following metals & other inorganic compounds were identified as having one or more measured concentration at or above the most stringent health-based screening level for that contaminant: arsenic, beryllium, cadmium, chlorine, chromium, lead, and manganese (Table 4b). Figures 16 through 29 show individual maximum concentrations for each contaminant obtained in any 24-hour period plotted by sample date, the 95% UCL on the sample average, and the most conservative comparison value for each contaminant.

Below we discuss the various physical and chemical properties of each compound along with an evaluation that integrates the relevant toxicological information with plausible exposures. In this section, all references to TSP metal concentrations refer to TSP STP, all references to  $\text{PM}_{2.5}$  metal concentrations refer to  $\text{PM}_{2.5}$  LC (local conditions), and references to  $\text{PM}_{10}$  metal concentrations refer to  $\text{PM}_{10}$  STP. Wherever a plausible exposure exceeded the most stringent health-based comparison value we compared the estimated exposures to known toxicological endpoints to determine whether adverse health effects are possible or probable.

### *Arsenic ( $\text{PM}_{10}$ , $\text{PM}_{2.5}$ , & TSP)*

Arsenic is an element that is widely distributed in the Earth's crust. It is usually found in the environment combined with other elements such as oxygen, chlorine, and sulfur. When combined with these elements it is called inorganic arsenic. When combined with carbon and hydrogen it is referred to as organic arsenic. Most arsenic compounds are white or colorless powders. They have no smell, and most have no special taste. Inorganic arsenic occurs naturally in soil and in many kinds of rock. Arsenic is no longer produced in the United States [50].

Arsenic has been used as a wood preservative (copper chromated arsenic or CCA). Wood treated with CCA is referred to as "pressure-treated." In 2003, U.S. manufacturers of wood preservatives containing arsenic began a voluntary transition from CCA to other wood preservatives for use in residential structures. Historically, inorganic arsenic compounds were used as pesticides, but now only organic arsenic compounds can be used in pesticide formulations. Small quantities of arsenic metal are added to other metals to form alloys. The greatest use of arsenic alloys is in lead-acid batteries for automobiles. Another important use of arsenic compounds is in semiconductors and light-emitting diodes [50].

Arsenic is naturally occurring, and may be present in soils, dust, and minerals; small amounts of arsenic also may be released from coal-fired power plants and incinerators. Arsenic cannot be destroyed, but it can change form in the environment. It also may become attached to or separated from soil and dust particles [50].

#### Absorption, Distribution, & Elimination

When dust containing arsenic is inhaled many of the dust particles settle onto the lining of the lungs and most of the arsenic on these particles is taken up into the body. After exposure, the liver changes some of the arsenic to a less harmful organic form and both inorganic and organic forms leave your body in your urine. Most arsenic is excreted within days following exposure; however, some will remain in the body for several months or longer [50].

#### Adverse Health Effects

Inorganic arsenic has been recognized as a human poison since ancient times, and large oral doses (above 60,000 ppb in food or water) can produce death. If you swallow lower levels of inorganic arsenic (ranging from about 300 to 30,000 ppb in food or water), you may experience irritation of your stomach and intestines, with symptoms such as stomach ache, nausea, vomiting, and diarrhea. Other effects you might experience from swallowing inorganic arsenic include decreased production of red and white blood cells which may cause fatigue, abnormal heart rhythm, blood-vessel damage resulting in bruising, and impaired nerve function causing a "pins and needles" sensation in your hands and feet. Inhalation of high concentrations of inorganic arsenic (approximately greater than  $100 \mu\text{g}/\text{m}^3$ ) for a short time period may result in a sore throat and irritated lungs. Perhaps the single most characteristic effect of long-term oral exposure to inorganic arsenic is a pattern of skin changes. These include a darkening of the skin and the appearance of small "corns" or "warts" on the palms, soles, and torso. Exposure to lower concentrations for a longer time period can also result in circulatory and peripheral nervous disorders. Some data suggests that inhalation of inorganic arsenic may cause problems in the developing fetus. Inhalation of inorganic arsenic is associated with an increased risk of lung cancer, particularly in people who work at smelters, mines, and chemical factories [50].

#### Carcinogenicity

Individuals with skin changes from long-term exposure to arsenic may ultimately develop skin cancer. Long-term ingestion of arsenic has also been reported to increase the risk of cancer in the liver, bladder, kidneys, prostate, and lungs. The Department of Health and Human Services (DHHS) has determined that inorganic arsenic is a known carcinogen. The International Agency for Research on Cancer (IARC) has determined that inorganic arsenic is carcinogenic to humans. Both the EPA and the National Toxicology Program (NTP) have classified inorganic arsenic as a known human carcinogen [51].

#### Health Assessment Comparison Values

The following HAC values have been established (or calculated) for inhalation exposures to arsenic:

- CREG  $0.000233 \mu\text{g}/\text{m}^3$
- Provisional Chronic Inhalation RfD  $1.05 \mu\text{g}/\text{m}^3$  (calculated value)
- EPA's Inhalation Unit Risk  $4.3 \times 10^{-3} (\mu\text{g}/\text{m}^3)^{-1}$

### Results – Arsenic (PM<sub>10</sub>)

Arsenic (PM<sub>10</sub>) was detected at quantifiable levels in all 181 ambient air samples. Arsenic (PM<sub>10</sub>) concentrations ranged from 0.011 µg/m<sup>3</sup> to 0.012 µg/m<sup>3</sup>, with an average concentration of 0.0116 µg/m<sup>3</sup>. Through Monte Carlo analysis, the 95% UCL of the arithmetic mean for arsenic (PM<sub>10</sub>) was estimated to be 0.0116 µg/m<sup>3</sup>. The combined average background level of arsenic (PM<sub>10</sub>) found at 19 other Texas sites from 2001 through 2005 was calculated to be 0.00602 µg/m<sup>3</sup>. The resulting BQ of 1.92 for Midlothian indicates that the average concentration found in Midlothian was almost two times higher than average background.

### Public Health Implications – Arsenic (PM<sub>10</sub>)

*Non-Carcinogenic Health Effects Evaluation:* No acute, intermediate, or chronic inhalation MRLs or RfCs were available for arsenic [50, 51]. Consequently, we derived a chronic inhalation RfD by calculating the air concentration necessary to produce an inhalation dose equivalent to the RfD for arsenic (3.0×10<sup>-4</sup> mg/kg/day), assuming a 70 kg body weight, and a respiratory daily volume of 20 m<sup>3</sup> per day (3.0×10<sup>-4</sup> × 1000 × 70/20 = 1.05 µg/m<sup>3</sup>).

In Midlothian, none of the 181 non-zero ambient air samples exceed the chronic inhalation RfD for arsenic (1.05 µg/m<sup>3</sup>), the most conservative non-cancer HAC value for this substance. The highest level measured (0.012 µg/m<sup>3</sup>) was over 87 times lower than the chronic inhalation RfD. Based on available toxicological information we would not expect to see adverse non-cancer health effects from either short-term or long-term exposure to arsenic (PM<sub>10</sub>) at the concentrations found in Midlothian.

*Carcinogenic Health Effects Evaluation:* All of the 181 non-zero ambient air samples exceed the CREG for arsenic (0.000233 µg/m<sup>3</sup>), the most conservative HAC value for this contaminant. Using the 95% UCL as a projected lifetime average exposure level and multiplying by the inhalation unit risk factor, the cancer risk from lifetime exposure to arsenic (PM<sub>10</sub>) at the concentrations found in Midlothian was estimated to be 4.99×10<sup>-5</sup>. This means that if 20,025 people were exposed to the levels of arsenic (PM<sub>10</sub>) found in Midlothian every day for 70-years, theoretically, we would predict that one additional person might get cancer as a result of that exposure. Qualitatively we would describe a risk of this magnitude as posing a low increased lifetime risk for cancer.

### Results – Arsenic (PM<sub>2.5</sub>)

Arsenic (PM<sub>2.5</sub>) was detected at quantifiable levels in 50 of the 162 ambient air samples. Arsenic (PM<sub>2.5</sub>) concentrations ranged from an assigned, non-detection value of 0.0005 µg/m<sup>3</sup> (one-half the detection limit) to 0.00982 µg/m<sup>3</sup>, with an average concentration of 0.000972 µg/m<sup>3</sup>. The most conservative HAC value for this contaminant (CREG of 0.000233 µg/m<sup>3</sup>) is below the detection limit for arsenic (PM<sub>2.5</sub>), thus it is not possible to determine how many samples exceed the HAC value. Through Monte Carlo analysis, the 95% UCL of the arithmetic mean for arsenic (PM<sub>2.5</sub>) was estimated to be 0.00111 µg/m<sup>3</sup>. The combined average background level of arsenic (PM<sub>2.5</sub>) found at 45 Texas sites in 2005 and 9 New York State sites in 2003 was calculated to be 0.00100 µg/m<sup>3</sup>. The resulting BQ of 0.968 for Midlothian indicates that the average concentration found in Midlothian was slightly lower than average background.

### Public Health Implications – Arsenic (PM<sub>2.5</sub>)

*Non-Carcinogenic Health Effects Evaluation:* None of the 162 ambient air samples exceeded the chronic inhalation RfD for arsenic, the most conservative non-cancer HAC value for this substance. The highest level measured (0.00982  $\mu\text{g}/\text{m}^3$ ) was over 106 times lower than the chronic inhalation RfD. Based on available toxicological information we would not expect to see adverse non-cancer health effects from either short-term or long-term exposure to arsenic (PM<sub>2.5</sub>) at the concentrations found in Midlothian.

*Carcinogenic Health Effects Evaluation:* All of the 157 non-zero ambient air samples exceeded the CREG for arsenic (0.000233  $\mu\text{g}/\text{m}^3$ ), the most conservative HAC value for this substance. Using the 95% UCL as a projected lifetime average exposure level and multiplying by the inhalation unit risk factor, the cancer risk from lifetime exposure to arsenic (PM<sub>2.5</sub>) at the concentrations found in Midlothian was estimated to be  $4.77 \times 10^{-6}$ . This means that if 209,520 people were exposed to the levels of arsenic (PM<sub>2.5</sub>) found in Midlothian every day for 70-years, theoretically, we would predict that one additional person might get cancer as a result of that exposure. Qualitatively we would describe a risk of this magnitude as posing no apparent increased lifetime risk for cancer.

### Results – Arsenic (TSP)

Arsenic (TSP) was detected at quantifiable levels in 33 of the 40 ambient air samples. Arsenic (TSP) concentrations ranged from an assigned, non-detection value of 0.0005  $\mu\text{g}/\text{m}^3$  (one-half the detection limit) to 0.0580  $\mu\text{g}/\text{m}^3$ , with an average concentration of 0.0181  $\mu\text{g}/\text{m}^3$ . The most conservative HAC value for this contaminant (CREG of 0.000233  $\mu\text{g}/\text{m}^3$ ) is below the detection limit for arsenic (TSP), thus it is not possible to determine how many samples exceed the HAC value. Through Monte Carlo analysis, the 95% UCL of the arithmetic mean for arsenic (TSP) was estimated to be 0.0216  $\mu\text{g}/\text{m}^3$ . The combined average background level of arsenic (TSP) found at 114 other Texas monitoring sites in 1981, was calculated to be 0.0531  $\mu\text{g}/\text{m}^3$ . The resulting BQ of 0.341 for Midlothian indicates that the average concentration found in Midlothian was 2.93 times lower than average background.

### Public Health Implications – Arsenic (TSP)

*Non-Carcinogenic Health Effects Evaluation:* In Midlothian, none of the 40 ambient air samples exceed the chronic inhalation RfD for arsenic, the most conservative non-cancer HAC value for this substance. The highest level measured (0.058  $\mu\text{g}/\text{m}^3$ ) was over 18 times lower than the chronic inhalation RfD. Based on available toxicological information we would not expect to see adverse non-cancer health effects from either short-term or long-term exposure to arsenic (TSP) at the concentrations found in Midlothian.

*Carcinogenic Health Effects Evaluation:* In Midlothian, all 40 of the ambient air samples exceed the CREG for arsenic (0.000233  $\mu\text{g}/\text{m}^3$ ), the most conservative HAC value for this contaminant. Using the 95% UCL as a projected lifetime average exposure level and multiplying by the inhalation unit risk factor, the cancer risk from lifetime exposure to arsenic (TSP) at the concentrations found in Midlothian was estimated to be  $9.30 \times 10^{-5}$ . This means that if 10,748 people were exposed to the levels of arsenic (TSP) found in Midlothian every day for 70-years, theoretically, we would predict that one additional person might get cancer as a result of that exposure. All 40 of the ambient air samples for arsenic (TSP) were collected during calendar

year 1981, consequently, we would categorize past exposures (prior to 1982) as a posing a low increased lifetime risk for cancer.

### ***Beryllium (PM<sub>10</sub>)***

Beryllium is an extremely lightweight, naturally occurring metal that is present in a variety of rocks, coal and oil, soil, and volcanic dust. Mineral rocks, bertrandite and beryl, which contain beryllium are commercially mined. Very pure gem-quality beryl (a silicate of beryllium and aluminum) is sold as emerald, aquamarine, Morganite, or heliodor gemstones [52,53].

Most of the beryllium ore that is mined is mixed with other metals to form alloys, which are then used to make electrical or electronic parts or molds for plastics, among other uses. Other uses include computers, automobiles, golf clubs, bicycle frames, and dental bridges. Pure beryllium metal is used for very specific technical purposes, including parts in nuclear weapons and reactors, aircraft and space vehicle structures, instruments, x-ray machines, and mirrors. Beryllium oxide is used to make specialty ceramics for electrical and high-technology applications. As with the other metals, beryllium may change form in the environment, but it cannot be destroyed.

#### Absorption, Distribution, & Elimination

Beryllium and its compounds are poorly absorbed from the gastrointestinal tract, but can be absorbed through the lungs. Due to an accidental leakage of beryllium dust in a laboratory, 25 people were exposed to an undetermined concentration for 10 to 20 hours [54]. The day after exposure, serum beryllium levels were  $3.5 \pm 0.47$  ppb beryllium, compared to 1.0 ppb in unexposed controls. Six days later, the serum level decreased to  $2.4 \pm 0.3$  ppb beryllium, and 2 to 8 weeks after exposure the serum levels returned to normal. The biological half-time of beryllium was calculated to be 2 to 8 weeks. Beryllium is widely distributed to the organs of animals after pulmonary absorption, particularly the lungs (60%), skeleton (13.5%), muscle (9.5%), blood (5%), kidney (1.5%), brain (1.4%), liver (0.9%), heart (0.4%), and spleen (0.1%). Beryllium is eliminated primarily in the feces, but small amounts are excreted in the urine [52].

#### Adverse Health Effects

Exposure to beryllium can result in two types of non-neoplastic respiratory disease, acute beryllium disease (ABD) and chronic beryllium disease (CBD); both forms can be fatal. Inhalation of beryllium (greater than  $1000 \mu\text{g}/\text{m}^3$ ) can result in an acute condition that resembles pneumonia and is referred to as ABD. Death may occur in some individuals with fulminating disease as a result of massive pulmonary edema. Some individuals may become sensitized to beryllium and develop an inflammatory respiratory reaction, referred to as CBD or berylliosis. This may occur many years after exposure to higher than normal levels of beryllium (greater than  $0.5 \mu\text{g}/\text{m}^3$ ). CBD is an inflammatory lung disease characterized by the formation of granulomas with varying degrees of interstitial fibrosis. Signs and symptoms of CBD include fatigue, difficult breathing, anorexia, weight loss, and possibly heart disease in advanced cases. The general population is unlikely to develop ABD or CBD because ambient air levels of beryllium are normally very low ( $0.00003$  to  $0.0002 \mu\text{g}/\text{m}^3$ ) [52].

No studies are available to determine if beryllium affects children differently, but health effects seen in children exposed to beryllium will likely be similar to the effects seen in adults. No information is available to determine if exposure to beryllium will result in birth defects or other

developmental effects in humans. Animal studies to determine developmental effects are not conclusive [52].

### Carcinogenicity

Long term exposure to beryllium can increase the risk of developing lung cancer in people. The Department of Health and Human Services (DHHS) and the International Agency for Research on Cancer (IARC) have determined that beryllium is a human carcinogen. The EPA has determined that beryllium is a probable human carcinogen and has estimated that lifetime exposure to  $0.04 \mu\text{g}/\text{m}^3$  beryllium can result in a one in ten thousand chance of developing cancer [55].

### Health Assessment Comparison Values

The following HAC values have been established for inhalation exposures to beryllium:

- CREG  $0.000417 \mu\text{g}/\text{m}^3$
- Chronic Inhalation RfC  $0.02 \mu\text{g}/\text{m}^3$
- EPA's Inhalation Unit Risk  $2.4 \times 10^{-3} (\mu\text{g}/\text{m}^3)^{-1}$

### Results

Beryllium ( $\text{PM}_{10}$ ) was not detected at quantifiable levels in any of the 181 ambient air samples, and all measurements were given assigned values of  $0.0005 \mu\text{g}/\text{m}^3$  (one-half the detection limit). However, the most conservative HAC value for this contaminant (CREG of  $0.000417 \mu\text{g}/\text{m}^3$ ) is below the detection limit for beryllium ( $\text{PM}_{10}$ ), thus it is not possible to determine how many samples may have exceeded the HAC value. Through Monte Carlo analysis, the 95% UCL of the arithmetic mean for beryllium ( $\text{PM}_{10}$ ) was estimated to be  $0.0005 \mu\text{g}/\text{m}^3$ . The combined average background level of beryllium ( $\text{PM}_{10}$ ) found at 14 other Texas sites, from 2001 through 2005, was calculated to be  $0.0003 \mu\text{g}/\text{m}^3$ . The resulting BQ of 1.67 for Midlothian indicates that the estimated average concentration found in Midlothian was higher than average background.

### Public Health Implications

*Non-Carcinogenic Health Effects Evaluation:* The chronic inhalation RfC is based on beryllium sensitization and progression to CBD identified among workers at a beryllium plant. The study identified a LOAEL for beryllium sensitization in workers exposed to  $0.55 \mu\text{g}/\text{m}^3$ . After adjusting for respiratory daily volume and 7-days-per-week exposure, the  $\text{LOAEL}_{\text{HEC}}$  was calculated as  $0.20 \mu\text{g}/\text{m}^3$  ( $0.55 \times 10/20 \times 5/7 = 0.196$ ). The chronic inhalation RfC of  $0.02 \mu\text{g}/\text{m}^3$  was derived by dividing the LOAEL by an overall uncertainty factor of 10 (3 to account for the sensitive nature of the subclinical endpoint (beryllium sensitization) and a database uncertainty factor of 3 was used to account for the poor quality of exposure monitoring in supporting studies) [55].

The 95% UCL of the sample average, the value most representative of chronic exposure, was over 40 times lower than the chronic inhalation RfC and over 400 times lower than the human equivalent LOAEL from which the RfC was derived. Based on the toxicological information used to derive the RfC we would not expect to see adverse non-cancer health effects from either short-term or long-term exposure to beryllium ( $\text{PM}_{10}$ ) at the concentrations found in Midlothian.

*Carcinogenic Health Effects Evaluation:* All 181 beryllium (PM<sub>10</sub>) measurements were below the detection limit of 0.001 µg/m<sup>3</sup> and hence, were assigned a value of one-half the detection limit (i.e., 0.0005 µg/m<sup>3</sup>). Using the 95% UCL as a projected lifetime average exposure level and multiplying by the inhalation unit risk factor, the cancer risk from lifetime exposure to beryllium (PM<sub>10</sub>) at the concentrations found in Midlothian was estimated to be 1.20×10<sup>-6</sup>. This means that if 833,333 people were exposed to the levels of beryllium (PM<sub>10</sub>) found in Midlothian every day for 70-years, theoretically, over the course of a 70 year period one person might get cancer as a result of that exposure. Qualitatively we would describe a risk of this magnitude as posing no apparent increased lifetime risk for cancer.

### ***Cadmium (PM<sub>10</sub>, PM<sub>2.5</sub>, & TSP)***

Cadmium is a metallic element that occurs naturally in the earth's crust. Pure cadmium is a soft, silver-white metal. Cadmium is not usually present in the environment as a pure metal, but as a mineral combined with other elements [56].

Most cadmium used in the United States is extracted as a by-product during the production of other metals such as zinc, lead, or copper. Cadmium has many uses in industry and consumer products, mainly in batteries, pigments, metal coatings, plastics, and some metal alloys [56].

Approximately half of the cadmium released each year is from the weathering of rocks. Forest fires and volcanoes also release some cadmium to the air. Human activities, including mining and burning of fossil fuels and household garbage also contribute to cadmium in air. Cadmium cannot be destroyed in the environment, but it can change forms [56].

#### Absorption, Distribution, & Elimination

Cadmium metal and cadmium salts have low volatility and exist in air primarily as fine suspended particulate matter. When inhaled, some fraction of this particulate matter is deposited in the airways or the lungs, and the rest is exhaled. Large particles (greater than about 10 µm in diameter) tend to be deposited in the upper airway, while small particles (approximately 0.1 µm) tend to penetrate into the alveoli. Mucociliary clearance removes cadmium particles from the upper tract. Some soluble cadmium compounds (cadmium chloride and cadmium sulfate) may undergo limited absorption from particles deposited in the respiratory tree, but the major site of absorption is the alveoli. About one-quarter of the total inhaled cadmium is absorbed. Cadmium absorption from cigarettes appears to be higher than absorption from cadmium aerosols, probably due to the very small size of particles in cigarette smoke. Cadmium can be stored in the liver and kidneys for many years, and the body can change cadmium to harmless forms if the system is not overloaded. If air containing cadmium is breathed over a long time period, it may cause fragile bones, and it may build up in the lungs and the kidneys, causing damage and/or disease [56].

#### Adverse Health Effects

Numerous studies have shown that acute inhalation exposure to cadmium can cause death in humans and animals. In humans, several fatal inhalation exposures have occurred in occupational accidents. During the acute exposure, the general symptoms are relatively mild but, within a few days following exposure, severe pulmonary edema and chemical pneumonitis develop, leading to death due to respiratory failure. For example exposure to cadmium oxide (CdO) in air at a level of 8,630 µg/m<sup>3</sup> for 5 hours led to the deaths of 5 workers. Longer-term

occupational exposures to levels of cadmium, below those that cause lung inflammation, however, have been reported to cause emphysema and dyspnea in humans [56].

The toxic effects of chronic cadmium exposure occur primarily in the lungs and in the kidneys. Pulmonary effects are associated solely with inhalation exposures, while the kidney effects may occur after either oral or inhalation exposures [56,57]. It has been hypothesized that there is a critical concentration of cadmium in the kidney, above which, cadmium-induced nephropathy will occur. Most cadmium-induced renal toxicity is probably associated with cadmium not bound to metallothionein. However, brush border membranes of the renal tubule may be damaged by metallothionein-bound cadmium. Damage is thought to occur when the renal cortical cadmium concentration exceeds the "critical" level of 200  $\mu\text{g/g}$  wet weight [56,57]. At these levels, the amount of cadmium not bound to metallothionein becomes high enough to begin causing tubular damage. However, other researchers have proposed that, for the general population, the amount of cadmium accumulated in the renal cortex should not exceed 50  $\mu\text{g/g}$  – a level corresponding to a urinary excretion of 2  $\mu\text{g}$  of cadmium per 24-hours [58].

Long-term exposure to excessive cadmium can affect the kidneys, causing proximal tubular necrosis, lesions in the renal cortex, and kidney dysfunction. Common laboratory findings include the presence of protein, amino acids, and glucose in the urine. Average kidney cadmium levels in non-occupationally exposed 50 year-olds are approximately 15 to 30  $\mu\text{g Cd/g-wet-weight}$  [59]. Cigarette smoking can double renal cortical cadmium concentrations. Because of the high amount of cadmium ingested through diet, the margin of safety for exposure to cadmium from other sources may be relatively small, particularly for smokers.

There is no conclusive information to determine if breathing cadmium can cause reproductive or developmental effects, but animal studies have shown that rats and mice had fewer litters and higher rates of defects in offspring when dams were exposed to high concentrations of cadmium. The effect of cadmium inhalation exposure on human birth defect rates is undetermined. Animal studies show that high levels of cadmium can affect the nervous system, resulting in learning and behavioral effects. High exposure levels in animals have also resulted in decreased body weight and skeleton formation effects in the young. These effects have not been observed in humans, possibly because the levels of cadmium used in the animal studies are considerably higher than any known human exposure. Animal studies have also indicated that liver damage and immune system effects resulted when rats and mice breathe cadmium-containing air. There is no reliable information to indicate that breathing cadmium harms the liver, heart, nervous system, or immune system in humans [56].

#### Carcinogenicity

Animal studies in which mice and hamsters breathed cadmium did not result in cancer; however, similar studies in rats did result in lung cancer. As a conservative approach, and based on the limited human data and the studies in rats, the United States Department of Health and Human Services (DHHS) has determined that cadmium and cadmium compounds may reasonably be anticipated to be carcinogens. The International Agency for Research on Cancer (IARC) has determined that cadmium is carcinogenic to humans. The EPA has determined that cadmium is a probable human carcinogen by inhalation [60].

### Health Assessment Comparison Values

The following HAC values have been established (or calculated) for inhalation exposures to cadmium:

- CREG 0.000556  $\mu\text{g}/\text{m}^3$
- Chronic Inhalation RfD 1.75  $\mu\text{g}/\text{m}^3$  (calculated value)
- EPA's Inhalation Unit Risk  $1.8 \times 10^{-3} (\mu\text{g}/\text{m}^3)^{-1}$

### Results – Cadmium (PM<sub>10</sub>)

Cadmium (PM<sub>10</sub>) was detected at quantifiable levels in 2 (1.1%) of the 181 ambient air samples. Cadmium (PM<sub>10</sub>) concentrations ranged from an assigned, non-detection value of 0.001  $\mu\text{g}/\text{m}^3$  (one-half the detection limit) to 0.004  $\mu\text{g}/\text{m}^3$ , with an average concentration of 0.00103  $\mu\text{g}/\text{m}^3$ . The most conservative HAC value for this contaminant (CREG of 0.000556  $\mu\text{g}/\text{m}^3$ ) is below the detection limit for cadmium (PM<sub>10</sub>), thus it is not possible to determine how many samples exceed the HAC value. Through Monte Carlo analysis, the 95% UCL of the arithmetic mean for cadmium (PM<sub>10</sub>) was estimated to be 0.00106  $\mu\text{g}/\text{m}^3$ . The combined average background level of cadmium (PM<sub>10</sub>) found at 17 other Texas sites from 2001 through 2005 was calculated to be 0.00082  $\mu\text{g}/\text{m}^3$ . The resulting BQ of 1.25 for Midlothian indicates that the average concentration found in Midlothian was higher than average background.

### Public Health Implications – Cadmium (PM<sub>10</sub>)

*Non-Carcinogenic Health Effects Evaluation:* No acute, intermediate, or chronic inhalation MRLs or RfCs were available for cadmium [0,0]. Consequently, we derived a chronic inhalation RfD (1.75  $\mu\text{g}/\text{m}^3$ ) from EPA's oral RfD; this value is defined as the concentration of cadmium in air which would deliver a dose equivalent to the oral RfD – assuming 100% absorption from the lung, a 70 kg body weight, and a respiratory daily volume of 20  $\text{m}^3/\text{day}$ .

None of the measured levels of cadmium (PM<sub>10</sub>) exceeded the chronic inhalation RfD, the most conservative non-cancer HAC value for this substance. The highest level measured in Midlothian (0.004  $\mu\text{g}/\text{m}^3$ ) was over 437 times lower than the chronic inhalation RfD. Based on available toxicological information we would not expect to see adverse non-cancer health effects from either short-term or long-term exposure to cadmium (PM<sub>10</sub>) at the concentrations found in Midlothian.

*Carcinogenic Health Effects Evaluation:* Using the 95% UCL as a projected lifetime average exposure level and multiplying by the inhalation unit risk factor, the cancer risk from lifetime exposure to cadmium (PM<sub>10</sub>) at the concentrations found in Midlothian was estimated to be  $1.91 \times 10^{-6}$ . This means that if 524,559 people were exposed to the levels of cadmium (PM<sub>10</sub>) found in Midlothian every day for 70-years, theoretically, we would predict that one additional person might get cancer as a result of that exposure. Qualitatively we would describe a risk of this magnitude as posing no apparent increased lifetime risk for cancer.

### Results – Cadmium (PM<sub>2.5</sub>)

Cadmium (PM<sub>2.5</sub>) was detected at quantifiable levels in 57 (35.2%) of the 162 ambient air samples. Cadmium (PM<sub>2.5</sub>) concentrations ranged from an assigned, non-detection value of 0.0005  $\mu\text{g}/\text{m}^3$  (one-half the detection limit) to 0.0092  $\mu\text{g}/\text{m}^3$ , with an average concentration of 0.00147  $\mu\text{g}/\text{m}^3$ , and 57 samples (36.31%) exceeded the CREG for cadmium (0.000556  $\mu\text{g}/\text{m}^3$ ),

the most conservative HAC value for this contaminant. Through Monte Carlo analysis, the 95% UCL of the arithmetic mean for cadmium (PM<sub>2.5</sub>) was estimated to be 0.00166 µg/m<sup>3</sup>. The combined average background level of cadmium (PM<sub>2.5</sub>) found at 45 Texas sites in 2005 and 9 New York State sites in 2003 was calculated to be 0.00325 µg/m<sup>3</sup>. The resulting BQ of 0.451 for Midlothian indicates that the average concentration found in Midlothian was 2.22 times lower than average background.

#### Public Health Implications – Cadmium (PM<sub>2.5</sub>)

*Non-Carcinogenic Health Effects Evaluation:* None of the 162 ambient air samples for cadmium (PM<sub>2.5</sub>) exceeded the minimum non-cancer HAC value; the highest level measured (0.0092 µg/m<sup>3</sup>) was over 190 times lower than the chronic inhalation RfD. Based on available toxicological information we would not expect to see adverse non-cancer health effects from either short-term or long-term exposure to cadmium (PM<sub>2.5</sub>) at the concentrations found in Midlothian.

*Carcinogenic Health Effects Evaluation:* Using the 95% UCL as a projected lifetime average exposure level and multiplying by the inhalation unit risk factor, the cancer risk from lifetime exposure to cadmium (PM<sub>2.5</sub>) at the concentrations found in Midlothian was estimated to be 2.99×10<sup>-6</sup>. This means that if 334,114 people were exposed to the levels of cadmium (PM<sub>2.5</sub>) found in Midlothian every day for 70-years, theoretically, we would predict that one additional person might get cancer as a result of that exposure. Qualitatively we would describe a risk of this magnitude as posing no apparent increased lifetime risk for cancer.

#### Results – Cadmium (TSP)

Cadmium (TSP) was detected at quantifiable levels in 27 of the 40 ambient air samples. Cadmium (TSP) concentrations ranged from an assigned, non-detection value of 0.0005 µg/m<sup>3</sup> (one-half the detection limit) to 0.129 µg/m<sup>3</sup>. The average concentration was 0.0298 µg/m<sup>3</sup>, and 27 samples (67.5%) exceed the CREG for cadmium (0.000556 µg/m<sup>3</sup>), the most conservative HAC value for this contaminant. Through Monte Carlo analysis, the 95% UCL of the arithmetic mean for cadmium (TSP) was estimated to be 0.0299 µg/m<sup>3</sup>. The combined average background level of cadmium (TSP) found at 114 other Texas sites in 1981 was calculated to be 0.0452 µg/m<sup>3</sup>. The resulting BQ of 0.660 for Midlothian indicates that the average concentration found in Midlothian was 1.51 times lower than average background.

#### Public Health Implications – Cadmium (TSP)

*Non-Carcinogenic Health Effects Evaluation:* None of the 40 ambient air samples for cadmium (TSP) exceeded the minimum non-cancer HAC value; the highest level measured (0.129 µg/m<sup>3</sup>) was over 13 times lower than the chronic inhalation RfD. Based on available toxicological information we would not expect to see adverse non-cancer health effects from either short-term or long-term exposure to cadmium (TSP) at the concentrations found in Midlothian.

*Carcinogenic Health Effects Evaluation:* Using the 95% UCL as a projected lifetime average exposure level and multiplying by the inhalation unit risk factor, the cancer risk from lifetime exposure to cadmium (TSP) at the concentrations found in Midlothian was estimated to be 5.38×10<sup>-5</sup>. This means that if 18,597 people were exposed to the levels of cadmium (TSP) found in Midlothian every day for 70-years, theoretically, we would predict that one additional person might get cancer as a result of that exposure. All 40 of the non-zero ambient air samples for

cadmium (TSP) were collected during calendar year 1981, consequently, we would categorize past exposures (prior to 1982) as a posing a low increased lifetime risk for cancer.

### *Chlorine (PM<sub>2.5</sub>)*

At standard temperature and pressure, chlorine is a yellow-green gas with an irritating odor. Chlorine is used widely as a bleaching agent for cloth and paper products, as a disinfectant, and in the manufacture of many chemicals, plastics, and resins. When it enters the environment, it reacts with water to form hypochloric and hydrochloric acids, which break down rapidly. Many large water-treatment facilities (serving more than 100,000 persons) in the United States use chlorine to treat drinking water. Aqueous chlorine is effective in reducing surface spoilage bacterial counts on carcasses of slaughter animals and is presently being used by some packing plants for washing beef, pork, lamb, and poultry [61].

#### Absorption, Distribution, & Elimination

Chlorine gas in inhaled air is very rapidly hydrolyzed to hydrochloric acid (HCl) in the upper airways, and very little reaches the respiratory air spaces [62]. Some is absorbed through the mucus membranes into the blood stream, and the remainder, through muco-ciliary action, is coughed up and swallowed where it joins the HCl already in the stomach. Excess chloride is eliminated primarily in the urine.

#### Adverse Health Effects

Breathing air containing low concentrations of chlorine gas (1 to 10 ppm) is irritating and corrosive to the respiratory tract, eyes, and skin and may result in coughing. Exposure to higher levels could cause burning of the eyes and skin, rapid breathing, narrowing of the bronchi, wheezing, blue coloring of the skin, accumulation of fluid in the lungs, and pain in the lung region. Exposure to even higher levels can produce severe eye and skin burns, lung collapse, and death. An inflammatory reaction called reactive airways dysfunction syndrome (RADS) is a type of asthma caused by some irritating and corrosive substances that some people may develop. No information is available to determine if exposure to chlorine can result in reproductive effects [61].

No information is available to determine if children are more susceptible to the health effects of chlorine than adults. Children may be more vulnerable to corrosive agents, such as chlorine, because of the smaller diameter of their airways. No studies are available to determine if exposure to chlorine gas can result in birth defects or other developmental effects [61].

#### Carcinogenicity

The Department of Health and Human Services (DHHS), the International Agency for Research on Cancer (IARC), and the EPA have not classified chlorine as to its carcinogenicity. There is no additional information to determine whether exposure to chlorine might cause cancer [63].

#### Health Assessment Comparison Values

The following HAC value has been established for inhalation exposures to chlorine:

- Chronic Inhalation RfC      0.232  $\mu\text{g}/\text{m}^3$  (provisional) [64]

## Results

Chlorine (PM<sub>2.5</sub>) was detected at quantifiable levels in 55 (34%) of the 162 ambient air samples. Chlorine (PM<sub>2.5</sub>) concentrations ranged from an assigned, non-detection value of 0.0005 µg/m<sup>3</sup> (one-half the detection limit) to 0.407 µg/m<sup>3</sup>, with an average concentration of 0.011 µg/m<sup>3</sup>. Through Monte Carlo analysis, the 95% UCL of the arithmetic mean for chlorine (PM<sub>2.5</sub>) was estimated to be 0.0113 µg/m<sup>3</sup>. The combined average background level of chlorine (PM<sub>2.5</sub>) found at 45 Texas sites in 2005 and 9 New York State sites in 2003 was calculated to be 0.0565 µg/m<sup>3</sup>. The resulting BQ of 0.195 for Midlothian indicates that the average concentration found in Midlothian was 5.13 times lower than average background.

## Public Health Implications

*Non-Carcinogenic Health Effects Evaluation:* The chronic inhalation RfC of 0.232 µg/m<sup>3</sup> was derived from an animal study in which rats exposed to chlorine gas intermittently (6 hours per day, 3 days per week) for 2 years exhibited damage (hyperplasia) to the upper respiratory tissues. The lowest observed adverse effects level (LOAEL) in the study was 0.4 ppm (1160 µg/m<sup>3</sup>) [64]. The benchmark concentration associated with a 5% increase in the incidence of lesions in the upper respiratory tissues in the study animals (the BMC<sub>05</sub>) was calculated to be 0.14 ppm (406 µg/m<sup>3</sup>). Adjusting for 24-hour exposure 7 days per week, the BMC<sub>05</sub> was multiplied by 6/24 and by 3/7 to arrive at the LOAEL<sub>HEC</sub> of 0.0024 ppm (6.96 µg/m<sup>3</sup>). The chronic inhalation RfC of 0.232 µg/m<sup>3</sup> (0.08 ppb) was derived by dividing the LOAEL<sub>HEC</sub> by an overall uncertainty factor of 30 (3 for extrapolation from animals to humans and 10 for human variability).

In Midlothian, 2 ambient air samples (1.23%) exceeded the provisional chronic inhalation RfC for chlorine (0.232 µg/m<sup>3</sup>), the most conservative HAC value for this contaminant. However, the 95% UCL of the sample average, the value most representative of chronic exposure, was over 17 times lower than the chronic inhalation RfC and over 500 times lower than the human equivalent LOAEL from which the RfC was derived. Based on available toxicological information we would not expect to see adverse non-cancer health effects from either short-term or long-term exposure to chlorine (PM<sub>2.5</sub>) at the concentrations found in Midlothian.

## *Chromium (PM<sub>10</sub>, PM<sub>2.5</sub>, & TSP)*

Chromium is found in rocks, animals, plants, soil, and in volcanic dust and gases in several different forms. The most common forms are chromium(0) (metallic), chromium(III) (trivalent), and chromium(VI) (hexavalent). Trivalent chromium is naturally occurring and is a nutrient required by the human body, whereas metallic chromium and hexavalent chromium are produced by industrial process. Additionally, trivalent chromium can be converted to hexavalent chromium through natural processes and human activities [65].

Chromium compounds are used for chrome plating, the manufacture of dyes and pigments, leather tanning, and wood preserving, as well as other purposes [65].

In air, chromium compounds are present mostly as fine dust particles, and the chromium will remain in the air for less than 10 days [65].

Chromium(III) is an essential nutrient that helps the body use sugar, protein, and fat. An intake of 50 to 200 µg of chromium(III) per day is recommended for adults. On the average, adults in the United States take in an estimated 60 to 80 µg of chromium per day in food. Therefore, many people's diets may not provide enough chromium(III). Without chromium(III) in the diet,

the body loses its ability to use sugars, proteins, and fat properly, which may result in weight loss or decreased growth, improper function of the nervous system, and a diabetic-like condition. Therefore, chromium(III) compounds have been used as dietary supplements and are beneficial if taken in recommended dosages [65].

#### Absorption, Distribution, & Elimination

In general, chromium(VI) is absorbed by the body more easily than chromium(III), but once inside the body, chromium(VI) is changed to chromium(III). When air containing chromium is inhaled, some of the chromium from air particles is absorbed to the bloodstream, where it is distributed throughout the body and leaves through the kidneys in the urine stream within a few days after exposure [65].

#### Adverse Health Effects

Breathing in small amounts of chromium(VI) for short or long periods does not cause a problem in most people. However, high levels of chromium in the workplace have caused asthma attacks in people who are allergic to chromium [65].

Breathing in chromium trioxide (CrO<sub>3</sub>), chromic acid (H<sub>2</sub>CrO<sub>4</sub>), or other chromium(VI) compounds at levels greater than 2 µg/m<sup>3</sup> can irritate the nose and cause runny nose, sneezing, itching, nosebleeds, ulcers, and holes in the nasal septum. These effects are generally seen only in factory workers who have been exposed to chromium(VI) compounds for several months to many years [65].

Breathing in chromium(III) compounds generally does not cause irritation to the nose or mouth in most people. Reliable information is not available to determine if any form of chromium has harmful effects on reproduction or causes birth defects in humans, though it does not seem likely that the amount of chromium that most people are exposed to will result in reproductive or developmental effects [65].

Little information is available to determine how chromium can affect the health of children. Children need small amounts of chromium(III) for normal growth and development. It is likely that the health effects seen in children exposed to high amounts of chromium will be similar to the effects seen in adults, but it is unknown if children will be more or less susceptible to health effects of chromium [65].

One animal study showed that more chromium(III) will enter the body of a newborn than an adult, but similar information is not available for chromium(VI). There is no information to determine if a child's body will store chromium differently than an adult, or how quickly chromium will leave a child's body. Animal studies indicate that chromium is transferred from a mother to her developing fetus [65].

In animals that breathed high levels of chromium, harmful effects on the respiratory system and a lower ability to fight disease were noted. However, information is not available to determine if chromium can lower a person's ability to fight disease [65].

#### Carcinogenicity

Long-term exposure to chromium has been associated with lung cancer in workers exposed to levels in air that were 100 to 1,000 times higher than those found in the natural environment. Lung cancer may occur long after exposure to chromium has ended. Some chromium(VI)

compounds produced lung cancer in animals that breathed in the particles or had the particles placed directly in their lungs [66].

Because some chromium(VI) compounds have been associated with lung cancer in workers and caused cancer in animals, the Department of Health and Human Services has determined that certain chromium(VI) compounds (calcium chromate, chromium trioxide, lead chromate, strontium chromate, and zinc chromate) are known human carcinogens. The International Agency for Research on Cancer (IARC) has determined that chromium(VI) is carcinogenic to humans, based on sufficient evidence in humans for the carcinogenicity of chromium(VI) compounds as found in chromate production, chromate pigment production, and chromium plating industries. IARC's determination is also based on sufficient evidence in experimental animals for the carcinogenicity of calcium chromate, zinc chromate, strontium chromate, and lead chromate; and limited evidence in experimental animals for the carcinogenicity of chromium trioxide (chromic acid) and sodium dichromate. IARC has also determined that chromium(0) and chromium(III) compounds are not classifiable as to their carcinogenicity to humans. The EPA has determined that chromium(VI) in air is a human carcinogen. The EPA has also determined that there is insufficient information to determine whether chromium(VI) in water or food and chromium(III) are human carcinogens [66].

#### Health Assessment Comparison Values

The following HAC values have been established for inhalation exposures to particulate chromium(VI):

- CREG 0.0000833  $\mu\text{g}/\text{m}^3$
- Chronic Inhalation RfC 0.10  $\mu\text{g}/\text{m}^3$
- Intermediate Inhalation MRL 1.00  $\mu\text{g}/\text{m}^3$
- EPA's Inhalation Unit Risk 0.012  $(\mu\text{g}/\text{m}^3)^{-1}$

#### Method

Chromium samples were collected from (PM<sub>10</sub>), from (TSP), and from (PM<sub>2.5</sub>). The data from these samples were provided separately and evaluated independent of each other for this consultation. The data provided from the air samples collected in Midlothian represents total chromium, which includes chromium(0), chromium(III), and chromium(VI). For the risk estimates and HQs outlined below, we have made the conservative assumption that all the chromium was chromium VI, the most toxic form.

#### Results – Chromium (PM<sub>10</sub>)

Chromium (PM<sub>10</sub>) was detected at quantifiable levels in 148 (81.8%) of the 181 ambient air samples. Chromium (PM<sub>10</sub>) concentrations ranged from an assigned, non-detection value of 0.001  $\mu\text{g}/\text{m}^3$  (one-half the detection limit) to 0.025  $\mu\text{g}/\text{m}^3$ , with an average concentration of 0.00520  $\mu\text{g}/\text{m}^3$ . Through Monte Carlo analysis, the 95% UCL of the arithmetic mean for chromium (PM<sub>10</sub>) was estimated to be 0.00566  $\mu\text{g}/\text{m}^3$ . The combined average background level of chromium (PM<sub>10</sub>) found at 16 other Texas sites from 2001 through 2005 was calculated to be 0.00418  $\mu\text{g}/\text{m}^3$ . The resulting BQ of 1.24 for Midlothian indicates that the average concentration found in Midlothian was higher than average background.

### Public Health Implications – Chromium (PM<sub>10</sub>)

*Non-Carcinogenic Health Effects Evaluation:* None of the 181 ambient air samples for chromium (PM<sub>10</sub>) exceeded the chronic inhalation RfC of 0.10 µg/m<sup>3</sup> (the minimum non-cancer HAC value for this substance). The highest level measured (0.025 µg/m<sup>3</sup>) was 4 times lower than the chronic inhalation RfC and 40 times lower than the intermediate inhalation MRL.

Based on available toxicological information, we would not expect to see adverse non-cancer health effects from either short-term or long-term exposure to chromium (PM<sub>10</sub>) at the concentrations found in Midlothian.

*Carcinogenic Health Effects Evaluation:* The CREG (0.0000833 µg/m<sup>3</sup>), the most conservative HAC value for this contaminant, is below the detection limit for chromium (PM<sub>10</sub>), thus it is not possible to determine how many samples exceeded the HAC value. Using the 95% UCL as a projected lifetime average exposure level and multiplying by the inhalation unit risk factor, the cancer risk resulting from lifetime exposure to chromium (PM<sub>10</sub>) at the concentrations found in Midlothian was estimated to be 6.8×10<sup>-5</sup>. This means that if 14,714 people were exposed to the levels of chromium (PM<sub>10</sub>) found in Midlothian every day for 70-years, theoretically, we would predict that one additional person might get cancer as a result of that exposure. This risk estimate is based on the assumption that all chromium present in PM<sub>10</sub> particulates is in the form of chromium(VI) and, consequently, this figure very likely over-estimates the true lifetime risk. Speciated chromium levels from other monitoring sites in the state (from Deer Park near Houston and from Karnack near Marshall) have shown that the fraction of chromium present as chromium(VI) is approximately 1.4 to 4.5% of the total chromium present. If chromium (PM<sub>10</sub>) turns out to be largely chromium(III) instead of chromium(VI), then the risk estimate would essentially approach zero. Qualitatively we would describe the risks associated with the worst case scenario (100% chromium (VI)) as posing a low increased lifetime risk for cancer.

### Results – Chromium (PM<sub>2.5</sub>)

Chromium (PM<sub>2.5</sub>) was detected at quantifiable levels in 46 (28.4%) out of 162 ambient air samples (28.4%). Chromium (PM<sub>2.5</sub>) concentrations ranged from an assigned, non-detection value of 0.0005 µg/m<sup>3</sup> to 0.0287 µg/m<sup>3</sup>, with an average concentration of 0.0011 µg/m<sup>3</sup>. The most conservative HAC value for this contaminant (CREG of 0.0000833 µg/m<sup>3</sup>) is below the detection limit for chromium (PM<sub>2.5</sub>), thus it is not possible to determine how many samples exceeded the HAC value. Through Monte Carlo analysis, the 95% UCL of the arithmetic mean for chromium (PM<sub>2.5</sub>) was estimated to be 0.0014 µg/m<sup>3</sup>. The combined average background level of chromium (PM<sub>2.5</sub>) found at 45 Texas sites in 2005 and 9 New York State sites in 2003 was calculated to be 0.00114 µg/m<sup>3</sup>. The resulting BQ of 0.961 for Midlothian indicates that the average concentration found in Midlothian was slightly lower than average background.

### Public Health Implications – Chromium (PM<sub>2.5</sub>)

*Non-Carcinogenic Health Effects Evaluation:* None of the 162 ambient air samples for chromium (PM<sub>2.5</sub>) exceeded the chronic inhalation RfC of 0.10 µg/m<sup>3</sup> (the minimum non-cancer HAC value for this substance). The highest level measured (0.0287 µg/m<sup>3</sup>) was over 3 times lower than the chronic inhalation RfC and over 34 times lower than the intermediate inhalation MRL. Based on available toxicological information we would not expect to see adverse non-cancer health effects from either short-term or long-term exposure to chromium (PM<sub>2.5</sub>) at the concentrations found in Midlothian.

*Carcinogenic Health Effects Evaluation:* The CREG ( $0.0000833 \mu\text{g}/\text{m}^3$ ), the most conservative HAC value for this contaminant, is below the detection limit for chromium ( $\text{PM}_{2.5}$ ), thus it is not possible to determine how many samples exceeded the HAC value. Using the 95% UCL as a projected lifetime average exposure level and multiplying by the inhalation unit risk factor, the cancer risk from lifetime exposure to chromium ( $\text{PM}_{2.5}$ ) at the concentrations found in Midlothian was estimated to be  $1.68 \times 10^{-5}$ . This means that if 59,689 people were exposed to the levels of chromium ( $\text{PM}_{2.5}$ ) found in Midlothian every day for 70-years, theoretically, we would predict that one additional person might get cancer as a result of that exposure. This risk estimate is based on the assumption that all chromium present in  $\text{PM}_{2.5}$  particulates is in the form of chromium(VI) and, consequently, this figure very likely over-estimates the true lifetime risk. If chromium ( $\text{PM}_{2.5}$ ) turns out to be largely chromium(III) instead of chromium(VI), then the risk estimate would essentially approach zero. Qualitatively we would describe the magnitude of the risk associated with the worst case scenario (100% chromium (VI)) as posing a low increased lifetime risk for cancer.

#### Results – Chromium (TSP)

Chromium (TSP) was detected at quantifiable levels in 14 (35%) of the 40 ambient air samples. Chromium (TSP) concentrations ranged from an assigned, non-detection value of  $0.0005 \mu\text{g}/\text{m}^3$  to  $0.0270 \mu\text{g}/\text{m}^3$ , with an average concentration of  $0.00423 \mu\text{g}/\text{m}^3$ . The most conservative HAC value for this contaminant (CREG of  $0.00008 \mu\text{g}/\text{m}^3$ ) is below the detection limit for chromium (TSP), thus it is not possible to determine how many samples exceeded the minimum HAC value. Through Monte Carlo analysis, the 95% UCL of the arithmetic mean for chromium (TSP) was estimated to be  $0.00577 \mu\text{g}/\text{m}^3$ . The combined average background level of chromium (TSP) found at 100 other Texas sites in 1981 was calculated to be  $0.0273 \mu\text{g}/\text{m}^3$ . The resulting BQ of 0.155 for Midlothian indicates that the average concentration found in Midlothian was 6.47 times lower than average background.

#### Public Health Implications – Chromium (TSP)

*Non-Carcinogenic Health Effects Evaluation:* None of the 40 ambient air samples for chromium (TSP) exceeded the chronic inhalation RfC of  $0.10 \mu\text{g}/\text{m}^3$  (the minimum non-cancer HAC value for this substance). The highest level measured ( $0.027 \mu\text{g}/\text{m}^3$ ) was over 3 times lower than the chronic inhalation RfC and over 37 times lower than the intermediate inhalation MRL. Based on the toxicological information used to derive the RfC we would not expect to see adverse non-cancer health effects from either short-term or long-term exposure to chromium (TSP) at the concentrations found in Midlothian.

*Carcinogenic Health Effects Evaluation:* The CREG ( $0.0000833 \mu\text{g}/\text{m}^3$ ), the most conservative HAC value for this contaminant, is below the detection limit for chromium (TSP), thus it is not possible to determine how many samples exceeded the HAC value. Using the 95% UCL as a projected lifetime average exposure level and multiplying by the inhalation unit risk factor, the cancer risk from lifetime exposure to chromium (TSP) at the concentrations found in Midlothian was estimated to be  $6.93 \times 10^{-5}$ . This means that if 14,436 people were exposed to the levels of chromium (TSP) found in Midlothian every day for 70-years, theoretically, we would predict that one additional person might get cancer as a result of that exposure. This risk estimate is based on the assumption that all chromium present in TSP is in the form of chromium(VI) and, consequently, this figure very likely over-estimates the true lifetime risk. All 40 of the non-zero ambient air samples for chromium (TSP) were collected during calendar year 1981,

consequently, assuming the worst case scenario we would categorize past exposures (prior to 1982) as a posing a low increased lifetime risk for cancer.

### ***Lead (TSP)***

Lead is a naturally occurring bluish-gray metal found in small amounts in the earth's crust. It has no characteristic taste or smell. Metallic lead does not dissolve in water and does not burn. Lead can combine with other chemicals to form what are usually known as lead compounds or lead salts. Some lead salts dissolve in water better than others. Some natural and manufactured substances contain lead but do not look like lead in its metallic form. Some of these substances can burn—for example, organic lead compounds in some types of gasoline. Lead has many different uses including automobile and truck batteries, ammunition, some brass and bronze products, and in solder for making electrical connections in scientific and electronic equipment and computers. Lead compounds have been used as major constituents of paint and as fuel additives to increase octane ratings of gasoline [67].

#### Absorption, Distribution, & Elimination

Shortly after lead gets into your body, it travels in the blood to the "soft tissues" (such as the liver, kidneys, lungs, brain, spleen, muscles, and heart). After several weeks, most of the lead moves into your bones and teeth. In adults, about 94% of the total amount of lead in the body is contained in the bones and teeth. About 73% of the lead in children's bodies is stored in their bones. Some of the lead can stay in your bones for decades; however, some lead can leave your bones and reenter your blood and organs under certain circumstances, for example, during pregnancy and periods of breast feeding, after a bone is broken, and during advancing age [67].

#### Adverse Health Effects

The effects of lead are the same whether it enters the body through breathing or swallowing. The main target for lead toxicity is the nervous system, both in adults and in children. Long-term exposure of adults to lead at work has resulted in decreased performance in some tests that measure functions of the nervous system. Lead exposure may also cause weakness in fingers, wrists, or ankles. Some studies in humans have suggested that lead exposure may increase blood pressure, but the evidence is inconclusive. Lead exposure may also cause anemia, a low number of blood cells. The connection between the occurrence of some of these effects (e.g., increased blood pressure, altered function of the nervous system) and low levels of exposure to lead is not certain. At high levels of exposure, lead can severely damage the brain and kidneys in adults or children. In pregnant women, high levels of exposure to lead may cause miscarriage. High-level exposure in men can damage the organs responsible for sperm production [67].

#### Carcinogenicity

We have no proof that lead causes cancer in humans. Kidney tumors have developed in rats and mice given large doses of lead. The animal studies have been criticized because of the very high doses used, among other things. The results of high-dose studies should not be used to predict whether lead may cause cancer in humans. The Department of Health and Human Services (DHHS) has determined that lead acetate and lead phosphate may reasonably be expected to be capable of causing cancer, based on sufficient evidence from animal studies, but there is inadequate evidence from human studies [68].

### Child Health Considerations

Children are more vulnerable to lead poisoning than adults. They can be exposed to lead in the womb if their mothers have lead in their bodies. Babies can swallow lead when they breast feed, or eat other foods and drink water that contains lead. Babies and children can swallow and breathe lead in dirt, dust, or sand while they play on the floor or ground. These activities make it easier for children to be exposed to lead than adults. The dirt or dust on their hands, toys, and other items may have lead particles in it. In some cases children swallow nonfood items such as paint chips; these may contain very large amounts of lead, particularly in and around older houses that were painted with lead-based paint. The paint in these houses often chips off and mixes with dust and dirt. Some old paint is 5 to 40% lead. Also, compared to adults, a bigger proportion of the amount of lead swallowed will enter the blood in children [67].

### Health Assessment Comparison Values

The ATSDR has not established an inhalation MRL for lead, and the EPA has not set an RfD, an RfC, an OSF, or an IUR for lead. However, the EPA has set a NAAQS primary standard of  $1.5 \mu\text{g}/\text{m}^3$  that stipulates that the quarterly average level of airborne lead shall be less than  $1.5 \mu\text{g}/\text{m}^3$ . Thus, this value was used as a starting point for evaluating inhalation exposures to lead:

- NAAQS primary standard for lead (quarterly average)  $1.5 \mu\text{g}/\text{m}^3$
- Chronic Inhalation RfC (provisional)  $0.375 \mu\text{g}/\text{m}^3$  (calculated)

### Results

Lead (TSP) was detected at quantifiable levels in 410 (99.3%) of the 413 ambient air samples. Lead (TSP) concentrations ranged from an assigned, non-detection value of  $0.0005 \mu\text{g}/\text{m}^3$  to  $1.51 \mu\text{g}/\text{m}^3$ , with an average concentration of  $0.199 \mu\text{g}/\text{m}^3$ . Two of the measured values were greater than or equal to the NAAQS primary standard of  $1.5 \mu\text{g}/\text{m}^3$ . Through Monte Carlo analysis, the 95% UCL of the arithmetic mean for lead (TSP) was estimated to be  $0.217 \mu\text{g}/\text{m}^3$ . The combined average background level of lead (TSP) found at 34 other Texas sites in 1996 was calculated to be  $0.0673 \mu\text{g}/\text{m}^3$ . The resulting BQ of 2.96 for Midlothian indicates that the average concentration found in Midlothian was almost 3 times higher than average background.

### Public Health Implications

*Non-Carcinogenic Health Effects Evaluation:* Because the EPA's NAAQS primary standard for lead (which stipulates that the quarterly average air lead level shall not exceed  $1.5 \mu\text{g}/\text{m}^3$ ) is a regulatory standard and not a health-based standard, we evaluated the significance of average air lead levels of  $1.5 \mu\text{g}/\text{m}^3$ . Using the EPA's Integrated Exposure Uptake Biokinetic (IEUBK) model for multiple-pathway lead exposures and assuming all other exposures remain the same (at program defaults), a long-term average air lead level of  $1.5 \mu\text{g}/\text{m}^3$  appears to increase the geometric mean blood lead level in children, ages 0 to 84 months, by approximately  $1.075 \mu\text{g}/\text{dl}$  ( $4.484 \mu\text{g}/\text{dl}$  vs.  $3.409 \mu\text{g}/\text{dl}$ ). At average air levels equal to the NAAQS primary standard, air would be contributing 24.0% of the total lead exposure for the child. The maximum quarterly average air lead level observed in Midlothian was  $0.443 \mu\text{g}/\text{m}^3$ , recorded in the 2<sup>nd</sup> quarter of 1995.

We calculated a provisional chronic inhalation RfC of  $0.375 \mu\text{g}/\text{m}^3$  by adjusting the exposure duration from quarterly (3 months) to chronic (12 months) ( $1.5 \times 3 \div 12 = 0.375 \mu\text{g}/\text{m}^3$ ). At the

provisional chronic inhalation RfC, the IEUBK model predicts a geometric mean blood lead level increase of 0.214 µg/dl (3.623 µg/dl vs. 3.409 µg/dl). For long-term exposures to lead (TSP) at the provisional chronic inhalation RfC, air would be contributing 5.9% of the total lead exposure for the child, and less than 1.54% of children ages 0 to 84 months would be expected to exceed a blood lead level of 10 µg/dl.

A total of 65 out of 413 ambient air samples (15.7%) were greater than or equal to the provisional chronic inhalation RfC of 0.375 µg/m<sup>3</sup>. Using the 95% UCL of the arithmetic mean concentration of lead (TSP) (0.217 µg/m<sup>3</sup>) as a projected long-term average exposure level, the IEUBK model predicts a geometric mean blood lead level increase of 0.091 µg/dl (3.500 µg/dl vs. 3.409 µg/dl). For long-term exposures at the 95% UCL level for lead (TSP), air would be contributing 2.6% of the total lead exposure for the child. Based on available toxicological information we would not expect to see adverse non-cancer health effects from either short-term or long-term exposure to lead (TSP) at the concentrations found in Midlothian.

### ***Manganese (PM<sub>10</sub> & TSP)***

Manganese is a naturally occurring metallic substance found in many types of rock, but it does not occur naturally as a pure metal. Manganese is found combined with other elements such as oxygen, sulfur, and chlorine to form compounds. Manganese can change from one compound to another and be carried on dust particles. Although it can change form, it does not break down in the environment [69].

Rocks with high levels of manganese compounds are mined, and manganese metal is produced, mixed with iron, and made into steel. Some manganese compounds are found in batteries, dietary supplements, some ceramics, pesticides, and fertilizers. Manganese can be released into the air by industries that burn fossil fuels. Additional sources can include iron- and steel-producing plants, power plants, coke ovens, and dust from mining operations [69].

It is an essential nutrient for humans and animals and plays a role in bone mineralization, protein and energy metabolism, metabolic regulation, cellular protection from damaging free radical species, and the formation of glycosaminoglycans. The Estimated Safe and Adequate Daily Dietary Intake (ESADDI) for adults for manganese is 2 to 5 mg/day [70].

#### Absorption, Distribution, & Elimination

No studies were located regarding the absolute amount of manganese that is absorbed by humans or animals after inhalation exposure to manganese dusts. In general, the extent of inhalation absorption is a function of particle size (because size determines the extent and location of particle deposition in the respiratory tract) and solubility of the specific manganese compounds. Particles that are deposited in the lower airway are probably mainly absorbed, while particles deposited in the upper airways may be moved by mucociliary transport to the throat, where they are swallowed and enter the stomach. Thus manganese may be absorbed both from the lungs and in the gastrointestinal tract following inhalation of manganese dust. However, the relative amounts absorbed from each site are not accurately known [69].

Manganese is a normal component of human and animal tissues and fluids. In humans, most tissue concentrations range between 0.1 and 1 µg manganese/g wet weight, with the highest levels in the liver, pancreas, and kidney and the lowest levels in bone and fat [69].

In humans, absorbed manganese is removed from the blood by the liver where it conjugates with bile and is excreted into the intestine and eliminated in the feces. Small amounts of manganese can also be found in urine, sweat, and breast milk. In humans who inhaled manganese dichloride ( $MnCl_2$ ) or manganese trioxide ( $Mn_2O_3$ ), about 60% of the material originally deposited in the lung was excreted in the feces within 4 days [69].

### Adverse Health Effects

Although it is an essential element, too much manganese in the body may cause serious illness. A combination of symptoms, including mental and emotional disturbances accompanied by slow, clumsy, body movement, has been observed in manganese miners or steel workers exposed to high levels of manganese dust in air. This combination of symptoms is known as ‘manganism,’ but workers do not usually develop these symptoms unless they have been exposed to high levels of manganese dust for many months or years [69,71].

Breathing too much manganese dust over a short or long time can cause irritation of the lungs. Sometimes this makes breathing difficult, and it can also increase the chances of getting a lung infection, such as pneumonia. However, this can happen from breathing in many kinds of dust particles and not just those that contain manganese [69].

Results from occupational studies show that men exposed to manganese in air over a long time period may develop impotence. Animal studies further indicate that inhalation of manganese may affect the testes. No information is available to determine if female reproductive health is affected [69].

The negative adverse effects of exposure to excess levels of manganese have been observed in all ages. Several studies in humans and animals indicate that the elderly may be a potentially susceptible population to the adverse effects of manganese exposure. Further, studies show that the young may also be a susceptible population [69].

Studies conducted to determine if exposure to high levels of manganese could cause birth defects in humans were inconclusive. One animal study indicated that exposure of pregnant females to manganese resulted in decreased birth weight [69].

### Carcinogenicity

No studies have been done to determine whether breathing manganese dust causes cancer in humans. The EPA lists manganese as class D (not classifiable as to human carcinogenicity) and DHHS/NTP lists it as class 3 (not classified) [72].

### Health Assessment Comparison Values

The following HAC values have been established (or calculated) for inhalation exposures to manganese:

- Chronic Inhalation RfC       $0.05 \mu\text{g}/\text{m}^3$
- Chronic Inhalation MRL       $0.04 \mu\text{g}/\text{m}^3$
- Chronic Inhalation RDA       $1.00 \mu\text{g}/\text{m}^3$  (calculated value)

### Results – Manganese (PM<sub>10</sub>)

Manganese (PM<sub>10</sub>) was detected at quantifiable levels in all 181 ambient air samples. Manganese (PM<sub>10</sub>) concentrations ranged from 0.004 µg/m<sup>3</sup> to 0.171 µg/m<sup>3</sup>, with an average concentration of 0.0413 µg/m<sup>3</sup>. Through Monte Carlo analysis, the 95% UCL of the arithmetic mean for manganese (PM<sub>10</sub>) was estimated to be 0.0454 µg/m<sup>3</sup>. The combined average background level of manganese (PM<sub>10</sub>) found at 14 other Texas monitoring sites from 2001 through 2005 was calculated to be 0.0273 µg/m<sup>3</sup>. The resulting BQ of 1.52 for Midlothian indicates that the average concentration found in Midlothian was approximately 52% higher than average background.

### Public Health Implications – Manganese (PM<sub>10</sub>)

*Non-Carcinogenic Health Effects Evaluation:* The chronic inhalation MRL (the most conservative HAC value for this contaminant) is based on BMD modeling of neurological deficits from an occupational study at a battery plant in which workers were exposed to MnO<sub>2</sub> dust by inhalation [71]. The BMDL<sub>10</sub> (interpreted as being equivalent to the NOAEL) was calculated to be 74 µg/m<sup>3</sup>. The chronic inhalation MRL was derived by dividing the NOAEL by a total uncertainty factor of 2,100 (10 for human variability, 7/5 and 24/8 to convert from intermittent to chronic exposures, 10 to account for limitations in the inhalation database, and 5 for the potential for increased susceptibility in children) [69].

A total of 71 out of 181 ambient air samples (39.23%) exceeded the chronic inhalation MRL for manganese (0.04 µg/m<sup>3</sup>), the most conservative HAC value for this contaminant. The 95% UCL on the average exposure level is 1.13 times higher than the chronic inhalation MRL but is 1,630 times lower than the study NOAEL upon which the chronic inhalation MRL is based. Considering these results and the uncertainty factors used in deriving the chronic inhalation MRL and a review of available toxicological data, we would not expect to see signs or symptoms of non-cancer health effects from acute or chronic exposure to manganese (PM<sub>10</sub>) at the levels seen in Midlothian.

### Results – Manganese (TSP)

Manganese (TSP) was detected at quantifiable levels in all 40 ambient air samples. Manganese (TSP) concentrations ranged from 0.009 µg/m<sup>3</sup> to 0.076 µg/m<sup>3</sup>, with an average concentration of 0.0407 µg/m<sup>3</sup>. Through Monte Carlo analysis, the 95% UCL of the arithmetic mean for manganese (TSP) was estimated to be 0.0446 µg/m<sup>3</sup>. The combined average background level of manganese (TSP) found at 102 other Texas sites in 1981 was calculated to be 0.0262 µg/m<sup>3</sup>. The resulting BQ of 1.56 for Midlothian indicates that the average concentration found in Midlothian was approximately 56% higher than average background.

### Public Health Implications – Manganese (TSP)

*Non-Carcinogenic Health Effects Evaluation:* A total of 20 out of 40 ambient air samples (50%) exceeded the chronic inhalation MRL for manganese (0.04 µg/m<sup>3</sup>), the most conservative HAC value for this contaminant. The 95% UCL on the average exposure level is 1.12 times higher than the chronic inhalation MRL (HQ = 1.12, margin of safety = 0.897) but is 1,659 times lower than the study NOAEL upon which the chronic inhalation MRL is based. All 40 of the ambient air samples for manganese (TSP) were collected during calendar year 1981. Considering these results and the uncertainty factors used in deriving the chronic inhalation MRL and a review of

available toxicological data, we would not expect to see signs or symptoms of non-cancer health effects from acute or chronic exposure to manganese (TSP) at the levels seen in Midlothian.

## **Aggregate Exposures to Chemical Mixtures – Non-Cancer Effects**

All 99 substances for which HAC values have been established by the ATSDR or the EPA (or for which provisional inhalation HAC values could be calculated from an RfD or RDA) were included in this analysis regardless of whether or not they had any measurements exceeding their respective HAC values. Measurements of metals and other inorganics on total suspended particulates (i.e., TSP measurements) were only obtained during calendar year 1981, whereas measurements of metals and other inorganics on PM<sub>10</sub> and PM<sub>2.5</sub> were obtained from 2001 through 2004. Also, since PM<sub>10</sub> particulates by definition include particles from 0 to 10 µm in size, they also include PM<sub>2.5</sub> particulates as a subset (i.e., particles from 0 to 2.5 µm in size). Consequently, only the hazard quotients for measurements of metals or other inorganics on PM<sub>10</sub> were used to calculate the overall hazard indices for the non-cancer critical effects. The 1993 through 2005 data set was used for this analysis because it included data points for inorganic compounds and VOCs, and is therefore most representative of aggregate exposures.

### Results

Only one critical non-cancer effect had a HI greater than or equal to 1.0 (see Appendix E, Table 7a). The HI for CNS/neurological effects was 1.18, and compounds that contributed to this HI included: bromomethane, chloromethane, n-hexane, styrene, tetrachloroethylene, toluene, trichloroethylene, xylene, lead (PM<sub>10</sub>), manganese (PM<sub>10</sub>), and mercury (PM<sub>2.5</sub>).

### Public Health Implications

The cumulative HI for CNS/neurological effects exceeded 1 by a small margin, primarily due to the contribution from manganese, which contributed 96% of the combined HI. As we have seen in the section above, the chronic inhalation MRL for manganese is 2,100 times lower than the study NOAEL upon which it is based. Considering these results and the large uncertainty factor built into the most conservative HAC value for manganese, we would not expect to see adverse CNS/neurological effects from either short-term or long-term exposure to the chemical mixtures found in Midlothian.

For all other non-cancer critical effects, the HI was less than 1.0; therefore, we would not expect to see any of the other non-cancer critical effects as a result of either short-term or long-term exposure to chemical mixtures at the concentrations found in Midlothian.

## **Aggregate Exposures to Chemical Mixtures – Cancer Effects**

Risk estimates for all 22 substances considered to be carcinogenic were included in this analysis, regardless of whether or not they had any measurements greater than or equal to the CREG for that substance. Measurements of metals and other inorganics on total suspended particulates (i.e., TSP measurements) were only obtained during calendar year 1981, whereas measurements of metals and other inorganics on PM<sub>10</sub> and PM<sub>2.5</sub> were obtained from 2001 through 2004. Also, since PM<sub>10</sub> particulates by definition include particles from 0 to 10 µm in size, they also include PM<sub>2.5</sub> particulates as a subset (i.e., particles from 0 to 2.5 µm in size). Consequently, only the cancer risk estimates derived from measurements of metals or other inorganics on PM<sub>10</sub> were

used to calculate the overall cancer risk for each cancer site and for total cancer (all cancer sites combined). The 1993 through 2005 data set was used for this analysis because it included data points for inorganic compounds and VOCs, and is therefore most representative of aggregate exposures.

### Results

Only total cancers (all cancer sites combined) had a cumulative risk for aggregate exposures that exceeded  $1 \times 10^{-4}$  (see Appendix E, Table 7b). The cumulative risk for total cancer was  $1.49 \times 10^{-4}$  (1 theoretical excess cancer in 6,712 persons exposed for a 70-year lifetime). Compounds that contributed to this risk included: benzene, 1,3-butadiene, carbon tetrachloride, chloroform, 1,2-dibromoethane, 1,2-dichloroethane, cis-1,3-dichloropropylene, trans-1,3-dichloropropylene, methylene chloride, 1,1,2,2-tetrachloroethane, 1,1,2-trichloroethane, vinyl chloride, arsenic ( $PM_{10}$ ), beryllium ( $PM_{10}$ ), cadmium ( $PM_{10}$ ), and chromium ( $PM_{10}$ ).

### Public Health Implications

The total cancer risk estimate can be interpreted as implying that if we followed the reported cancer cases occurring in current residents of Midlothian (a city of approximately 12,000) for 70 years, theoretically, over a period of 70 years, two people might get cancer as a result of their exposure to these contaminants. Qualitatively, we would interpret a risk of this magnitude as a posing a moderately increased lifetime risk for cancer. This interpretation is based on the assumption that all the chromium ( $PM_{10}$ ) present in the air is chromium(VI), an assumption that is unlikely to be true based upon our knowledge of the relative proportions of the two common oxidation states of chromium in air. Speciated chromium levels from other monitoring sites in the state (i.e., from Deer Park near Houston and from Karnack near Marshall) have shown that the fraction of chromium present as chromium(VI) is approximately 1.4 to 4.5% of the total chromium present. If the chromium ( $PM_{10}$ ) turns out to be largely chromium(III) instead of chromium(VI), then the aggregate-exposure risk estimate for total cancers (all cancer sites combined) would decrease to  $8.10 \times 10^{-5}$  (1 in 12,342). Qualitatively we would interpret a cancer risk of this magnitude as posing no apparent public health hazard as a result of long-term aggregate exposure to multiple contaminants. Additional sampling is needed to determine the specific proportions of the chromium species present in  $PM_{10}$  in Midlothian air and, thereby, to further refine the overall cancer risk estimate.

All other specific cancer sites had cumulative risks estimates less than  $1 \times 10^{-4}$ ; therefore, we would not expect to see any discernable effect on cancer incidence or mortality rates as a result of aggregate exposures to the particular mixture of carcinogenic contaminants at the concentrations found in Midlothian.

## **Response to Petitioner and Community Concerns**

The 4 different lists (A, B, C, & D) of petitioner and community concerns are given in Appendix B. Each list contains from 4 to 8 individual (numbered) concerns. Responses to one or more of these concerns are addressed in the paragraphs below (identified by the list letter and concern number, e.g. C.3. refers to list C, concern number 3).

A.1. While it is true that “all the chemicals being released from cement kilns and steel mills have not been fully identified,” this health consultation has evaluated 237 individual contaminants including 119 VOCs and 108 metals and other inorganic substances.

A.2. It is also true that “All the chemicals currently being incinerated and released have not been tested for carcinogenicity and endocrine disrupting potential.” However, based on historical reviews of cancer incidence and/or mortality rates in Midlothian and Ellis County, no individual or aggregate cancer rates were significantly elevated with respect to the rest of the state.

A.4., C.3., & D.3. The community was concerned about the health effects of dioxins, metals, and mixtures of compounds. Air data for dioxins are not routinely collected in Texas; therefore it is not possible to evaluate the potential adverse health effects associated with these compounds. We evaluated available VOCs and metals air contaminant data with respect to its potential for causing adverse health effects in humans due to acute, intermediate, and/or chronic exposures. Only manganese exceeded its health based screening value for chronic inhalation exposures. However, based upon a review of the toxicological data, we would not expect to see adverse health effects due to either long-term or short-term exposure to manganese. Mixtures of compounds also were evaluated in this consultation. Long-term aggregate exposures to air contaminants in Midlothian are not expected to result in adverse non-cancer or cancer health effects.

A.5., A.7., & C.1. In this health consultation, DSHS has analyzed each and every individual air sampling result collected from all TCEQ sampling locations in the Midlothian area and has not relied on any TCEQ-summarized data. Also, DSHS has not relied on any of the TCEQ’s effects screening levels (ESLs) for determining potential health risks associated with exposures to airborne contaminants in Midlothian.

A.6. & D.4. The community was concerned that the potential for adverse health effects may be underestimated due to averaging of contaminant data over time. The initial screening of the air data involved comparing the maximum concentration for each contaminant to its most conservative health-based screening value. Contaminants whose maximum concentrations exceeded the most conservative health-based screening value were evaluated for acute, intermediate, and long-term exposures. None of the compounds examined (with the exception of benzene) had a single 24-hour measurement that exceeded its acute exposure guideline. The acute inhalation MRL for benzene was exceeded 3 isolated times in 13 years. Consequently, after reviewing all of the available data (which includes 94,932 individual 24-hour measurements), we find no evidence to suggest that adverse health effects would be anticipated as a result of any of the short-term or peak exposures to VOCs or Metals. The potential for adverse health effects due to exposure to EPA’s NAAQS compounds will be evaluated in a future health consultation.

A.8., B.4., C.4., & D.1. The community was concerned about asthma, allergies, immune system deficiencies, and other health problems in adults as well as children. Data for these health problems are not routinely collected in Texas. Therefore, we were not able to systematically assess whether the levels of these conditions in Midlothian are different than in other areas of the state.

B.1., B.2., & D.2. Over the years, the Texas Cancer Registry and Texas Birth Defects Registry have conducted incidence, mortality, and prevalence investigations to determine if cancer and birth defect rates were higher or lower in the Midlothian area compared to the rest of the state (Appendix D). No statistically significant elevations of specific or total cancers were found. The prevalences for a few birth defects were higher than expected and for a few other birth defects were lower than expected based on state rates. These higher prevalence rates were not unique to Midlothian/Ellis County but were also observed throughout Health Service Region 3

(which includes 18 other counties primarily north and west of Ellis County). Because of the numerous factors involved, it is not possible to determine if these increases are due to environmental exposures or differences in reporting practices in this region compared with the rest of the state. Furthermore, it should be noted that only 3 of the 99 compounds with health based comparison values (i.e., ethylbenzene, 2-butanone, and methyl isobutyl ketone) listed “developmental effects” as the critical effect (i.e., the first observable physiological or adverse health effect occurring at the lowest exposure dose known to produce any effect at all). Hazard quotients for those 3 compounds were 0.000352, 0.0000653, and 0.00000793 respectively, levels that are far below levels that might be expected to result in an increased risk for birth defects.

B.3. It has been suggested that the Down syndrome cluster reported in Ellis, Hood, and Somervell Counties in 1991-1994 may have been related to a cesium-137 source melt that occurred at Chaparral Steel on September 16, 1993. This might seem plausible in that one of the risk factors for Down syndrome is exposure of the mother or the father to excessive radiation prior to conception of the child. However, the time line is not right for this to have been a possibility, because the non-disjunction of chromosome 21 that results in the manifestations of Down syndrome would have had to have occurred **prior** to the date of the cesium-137 source melt for 15 out of 18 of the reported Down syndrome cases (based on the estimated date of conception for each of the children with Down syndrome). Also, analysis of the wind rose patterns for Midlothian during a similar time period to the cluster (i.e., 1992-94), revealed that the wind would have been blowing in the direction of one of the Down syndrome cases for less than 2% of the time during the 3-year period. Although the precise wind direction on the exact day of the source melt is not known, the predominant winds are out of the SSE during September, which would have been blowing toward none of the three Down syndrome cases whose estimated date of conception was after the cesium-137 source melt (two of these cases were from Granbury, which is approximately 44 miles west of Midlothian, and the other was from Palmer which is 21 miles ESE of Midlothian). And finally, although the exact quantity of radiation released is unknown, modeling of this release as though the entire source (approximately 89 millicuries of cesium-137) was vaporized and released into the air (and not caught in baghouse dust as most of it was), indicates that the additional radiation would not have been detectable above background radiation levels.

C.2. This concern turned out to be unfounded, in that all three CAMS monitoring locations have collected air sampling data on 97-99 of the 119 different VOCs, amounting to 60,396 individual contaminant measurements. The CAMS-94 location collected air sampling data on 52 metals or other inorganics present in PM<sub>2.5</sub> particulate matter amounting to 8,164 individual contaminant measurements, and the CAMS-302 location collected air sampling data on 24 metals or other inorganics present in PM<sub>10</sub> particulate matter, amounting to 4,344 individual contaminant measurements. Only the CAMS-52 location collected no air samples for metals or other inorganics present in particulate matter. The confusion may have arisen because the CAM sites only collect data for the NAAQS compounds on a continuous basis (i.e., 24 one-hour-average levels per day). The other contaminants (VOCs and metals) are collected noncontinuously as one 24-hour-average level collected once every 6 days.

C.4. & D.5. Health problems reported in domesticated animals and livestock were shared with veterinarians at Texas A&M University. While DSHS does not have animal-species-specific health-based comparison values to evaluate the risks for health effects in animals, many of the health-based comparison values used in our evaluation of human exposures are derived from

animal studies and consequently, we would expect these human HAC values to be equally conservative in protecting animal health for most common domestic and farm animals.

## Conclusions

### *General Findings*

1. One hundred thirteen contaminants (47 VOCs and 66 metals or other inorganic compounds) had no levels exceeding the most conservative HAC value (or had no reported levels above the detection limit). No known health effects are associated with exposure to these contaminants at the concentrations measured in Midlothian; therefore, exposure to these contaminants would not be expected to result in adverse health effects.
2. Health based screening values were not available for 87 contaminants (59 VOCs and 28 metals or other inorganic compounds). Additional information is needed to determine the public health significance of these contaminants.
3. Thirteen VOCs had one or more measured level above the most protective health-based screening value. Three of the VOCs (1,1,2-trimethylbenzene; 1,3,5-trimethylbenzene; and m- and p- xylene) had one or more level above the most conservative contaminant-specific non-cancer screening value. Ten of the VOCs (benzene; 1,3-butadiene; carbon tetrachloride; chloroform; 1,2,-dibromoethane; 1,2-dichloroethane; methylene chloride; 1,1,2,2-tetrachloroethane; 1,1,2-trichloroethane; and vinyl chloride) had one or more level above the most conservative contaminant-specific cancer screening value.
4. Fourteen metals or other inorganic compounds had one or more measured level above the most protective health-based screening value. Four of the metals or other inorganic compounds [chlorine (PM<sub>2.5</sub>), lead (TSP), manganese (TSP), and manganese (PM<sub>10</sub>)] had one or more level above the most conservative contaminant-specific non-cancer screening value. Ten metals [arsenic (PM<sub>10</sub>), arsenic (PM<sub>2.5</sub>), arsenic (TSP), beryllium (PM<sub>10</sub>), cadmium (PM<sub>10</sub>), cadmium (PM<sub>2.5</sub>), cadmium (TSP), chromium (PM<sub>10</sub>), chromium (PM<sub>2.5</sub>), and chromium (TSP)] had one or more level above the most conservative contaminant-specific cancer screening value.

### *Background Comparisons*

1. Five out of 47 VOCs and 11 out of 66 metals or other inorganics that were below health-based screening levels nevertheless slightly exceeded average background levels (levels obtained from other areas in Texas and/or the US).
2. Sixteen out of 59 VOCs and 2 out of 28 metals or other inorganic compounds for which HAC values were not available had average levels slightly above average background.
3. All 13 VOCs having one or more level exceeding its minimum HAC value nevertheless had average levels that were below average background.
4. Seven out of 14 metals having one or more level exceeding its minimum HAC value [arsenic (PM<sub>10</sub>), beryllium (PM<sub>10</sub>), cadmium (PM<sub>10</sub>), chromium (PM<sub>10</sub>), lead (TSP), manganese (TSP), and manganese (PM<sub>10</sub>)] had average levels that also were above average background.

### *Individual Contaminants – Non-Cancer Health Effects Evaluation*

Using reasonable maximum exposure scenarios, only manganese (both as PM<sub>10</sub> and as TSP) exceeded ATSDR's chronic inhalation MRL by a small margin. After an in-depth review of the toxicological information and the uncertainty factors used in deriving the chronic inhalation MRL, we concluded that it is highly unlikely that the manganese levels seen in Midlothian would result in any observable adverse health effects, even after long-term exposure.

### ***Individual Contaminants – Cancer Health Effects Evaluation***

#### **Exposures Prior to 1982:**

Based on ambient air samples collected prior to calendar year 1982, the estimated excess lifetime cancer risks associated with reasonable maximal exposure to arsenic (PM<sub>10</sub>), cadmium (PM<sub>10</sub>), and chromium (PM<sub>10</sub>) ranged from  $9.30 \times 10^{-5}$  (a total of 1 excess cancer in 10,748 people exposed for 70 years) to  $5.38 \times 10^{-5}$  (a total of 1 excess cancer in 18,597 people exposed for 70 years). If these exposures were to continue for 70 years, they would pose a low increased lifetime risk for cancer and would not be expected to result in measurable harmful health effects. Past exposures to these compounds (prior to 1982) therefore posed “no apparent public health hazard.”

#### **Exposures through 2005:**

1. The estimated lifetime cancer risks associated with reasonable maximal exposure to arsenic (PM<sub>10</sub>), chromium (PM<sub>10</sub>), and chromium (PM<sub>2.5</sub>) ranged from  $1.68 \times 10^{-5}$  (a total of 1 excess cancer in 59,689 people exposed for 70 years) to  $6.8 \times 10^{-5}$  (a total of 1 excess cancer in 14,714 people exposed for 70 years). Based on available information, we have concluded that exposures to these contaminants pose a low increased lifetime risk for cancer and would not be expected to result in measurable harmful health effects.
2. The estimated lifetime cancer risks associated with reasonable maximal exposure to benzene, carbon tetrachloride, 1,2-dibromoethane, arsenic (PM<sub>2.5</sub>), beryllium (PM<sub>10</sub>), cadmium (PM<sub>10</sub>), and cadmium (PM<sub>2.5</sub>) ranged from  $1.2 \times 10^{-6}$  (a total of 1 excess cancer in 833,333 people exposed for 70 years) to  $9.66 \times 10^{-6}$  (a total of 1 excess cancer in 103,548 people exposed for 70 years). Based on available information we have concluded that exposures to these contaminants pose no apparent increased lifetime risk for cancer and would not be expected to result in measurable harmful health effects.
3. The estimated lifetime cancer risks associated with reasonable maximal exposure to 1,3-butadiene, chloroform, 1,2-dichloroethane, methylene chloride, 1,1,2,2-tetrachloroethane, 1,1,2-trichloroethane, and vinyl chloride ranged from  $5.06 \times 10^{-8}$  (a total of 1 excess cancer in 19,751,644 people exposed for 70 years) to  $8.47 \times 10^{-7}$  (a total of 1 excess cancer in 1,180,057 people exposed for 70 years). Based on available information we have concluded that exposures to these contaminants pose no increased lifetime risk for cancer and would not be expected to result in measurable harmful health effects.

### ***Aggregate Exposures – Non-Cancer Health Effects***

Only one critical non-cancer effect had a HI greater than or equal to 1.0 – the HI for CNS/neurological effects. Although several compounds contributed to CNS/neurological effects, manganese (PM<sub>10</sub>) contributed 96% of this result. The toxicological basis for the health-based criteria for manganese is based on a no-effects level that is over 2,100 times lower than the estimated reasonable maximal exposure estimates used in this analysis. Based on available

information, long-term aggregate exposures to air contaminants in Midlothian would not be likely to result in CNS/neurological effects, either under current or anticipated future conditions.

### ***Aggregate Exposures – Cancer Health Effects***

Total cancers had a cumulative risk for aggregate exposures that exceeded  $1 \times 10^{-4}$  (i.e., exceeded a total of 1 excess cancer in 10,000 people exposed for 70 years). However, this cancer risk estimate is based on the assumption that all chromium (PM<sub>10</sub>) present in the air is chromium(VI), an assumption that is inconsistent with information obtained from other areas of the state. Additional sampling is needed to determine the specific proportions of the major chromium oxidation states and to further refine the total cancer risk estimate.

### ***Overall Conclusions***

We found that the majority of the risks associated with exposure to the chemicals analyzed in this health consultation were low. However, we are classifying this site as an Indeterminate Public Health Hazard because further information is needed to fully characterize the extent of the public health hazard posed by air contaminants in Midlothian. This classification is based on the following facts:

1. Sixteen out of 59 VOCs and 2 out of 28 metals or other inorganic compounds for which health-based screening values were not available had average levels above average background (levels obtained from other areas in Texas and/or the US). Additional information is needed to determine the public health significance of these contaminants.
2. While individual contaminants produced, at most, a low increased lifetime risk for cancer and no apparent public health hazard, under the aggregate exposure scenario, total excess lifetime cancer risk for all cancers combined could be interpreted as posing a public health hazard. However, this conclusion is based on the assumption that all the chromium detected in the air is of the most toxic form [i.e., chromium(VI)], an assumption that is inconsistent with information obtained from other areas of the state. The relative proportions of chromium(III) and chromium(VI) will need to be determined in order to accurately define the risk estimate for total cancer (all sites combined).
3. While this health consultation reviewed the majority of the contaminants measured in Midlothian air (119 VOCs and 108 metals and other inorganics), EPA's NAAQS compounds still need to be evaluated in a future consultation.
4. There are data gaps both in sampling locations and parameters of interest. No air data for the analysis of VOCs were collected prior to 1993. Air data for the analysis of metals and other inorganic compounds were collected from only one location from 1981 through 1984, no air data for these contaminants were collected prior to 1981, and none were collected between 1985 and 1992. For the time periods that air data does exist, data were collected from a limited number of monitoring stations and may not reflect conditions throughout the community. However, since the major monitoring locations were relatively close to one or more of the primary emission sources, we do not anticipate that air pollutant levels for much of the city would be too much higher than those observed.

## Recommendations

We have made the following recommendations in response to these findings:

1. As resources allow, research the toxicology literature for contaminants measured in Midlothian air for which health-based screening values were not available, and determine the potential public health impact of exposures to these substances.
2. Collect additional ambient air samples from previously sampled locations to determine the specific distribution of chromium species and to refine the risk estimates for this contaminant.
3. Evaluate the levels of EPA's NAAQS compounds in the continuous air monitoring data.
4. Where possible identify and fill data gaps with additional data from TCEQ to identify any additional air contaminants that might need evaluation and/or sampling.

## Public Health Action Plan

### Actions Completed

1. Historically, the TCEQ has collected a vast amount of environmental data in Midlothian, Texas, including air monitoring samples, soil samples, vegetation samples, and others dating back to the early 1980's.
2. Earlier data were analyzed by the TCEQ using EPA methodology and TCEQ's screening levels [4, 10].
3. DSHS staff reviewed summarized monitoring data (1993 through 1995), attended numerous meetings with TCEQ staff and area residents, and distributed questionnaires to see if there were consistent reports of odors, or signs or symptoms of illnesses that might be related to environmental pollution.
4. The Texas Cancer Registry analyzed cancer morbidity and mortality data for Midlothian and Ellis County, looking for any significant increases in cancer rates in this area over the period 1993 through 2002.
5. The Texas Birth Defects Registry analyzed birth defect data for Midlothian, Ellis County, and Health Service Region 3, looking for any significant birth defect elevations during the period 1999 through 2003.
6. DSHS staff conducted site visits in 2005 to determine community concerns, as well as to gather information about the major industries in town. Data from the door-to-door survey (conducted in December 2005) and from mailers which were distributed to ascertain public health concerns were compiled and evaluated to determine additional community health concerns. These concerns were addressed in the "Response to Community Health Concerns" section of this document.
7. DSHS staff obtained detailed (not summarized) TCEQ air monitoring data from 1981 through 1984 and from January 1993 through March 2005 in an electronic format and created a database of monitoring results. With the completion of this health consultation, DSHS has analyzed this data for VOCs and metals or other inorganic compounds and compared these data to health-based screening levels published by ATSDR and EPA. A

conservative exposure scenario was generated, and carcinogenic and non-carcinogenic risk estimates were calculated, assuming 70-year lifetime and/or chronic exposures at the reasonable maximal exposure levels seen in the Midlothian area.

### **Actions Under Way**

Currently, DSHS staff are analyzing the hourly NAAQS data (sulfur dioxide, hydrogen sulfide, nitric oxide, nitrogen dioxide, nitrogen oxides, ozone, and particulates) and preparing a health consultation to address these compounds.

### **Actions Planned**

1. DSHS and ATSDR will make this health consultation available to the public, local industries, the local government, and state and federal health/environmental agencies.
2. DSHS and ATSDR will continue to address the community's health concerns relating to air quality.
3. DSHS will discuss with ATSDR the possibility of researching the toxicology literature for contaminants measured in Midlothian air that were at levels above background and for which health-based screening values were not available.
4. DSHS will discuss with TCEQ the potential for determining the specific distribution of chromium species in Midlothian air.
5. DSHS will discuss with TCEQ the potential for identifying and filling data gaps and identifying any additional air contaminants that might need evaluation and/or sampling
6. DSHS will complete the analysis of the hourly NAAQS data.

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## **Appendices**

Appendix A: Acronyms and Abbreviations

Appendix B: List of Petitioner and Community Concerns

Appendix C: Figures

Appendix D: Birth Defects and Cancer Registries Data Reviews

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Appendix F: Monte Carlo Methodology

## **Appendix A – Acronyms and Abbreviations**

## Acronyms and Abbreviations

ABD	Acute Beryllium Disease
ALC	Absolute Lymphocyte Count
AML	Acute Myeloid Leukemia
ATSDR	Agency for Toxic Substances and Disease Registry
BMC	Benchmark Concentration
BMC <sub>05</sub>	Benchmark Concentration for a 5% response rate
BMCL	95% Lower Confidence Limit on the Benchmark Concentration
BMD	Benchmark Dose
BMDL	95% Lower Confidence Limit on the Benchmark Dose
BMDL <sub>10</sub>	95% Lower Confidence Limit on the exposure expected to result in a 10% response rate
CAMS	Continuous Air Monitoring Station
CBD	Chronic Beryllium Disease
CCA	Copper Chromated Arsenic
CNS	Central Nervous System
CrO <sub>3</sub>	Chromium Trioxide
CREG	Carcinogenic Risk Evaluation Guide
CSF	Cancer Slope Factor
°F	Degrees Fahrenheit
DHHS	US Department of Health and Human Services
DNA	Deoxyribonucleic Acid
DSHS	Texas Department of State Health Services
EPA	US Environmental Protection Agency
ESL	Effects Screening Level
ESADDI	Estimated Safe and Adequate Daily Dietary Intake
ETJ	Extra-Territorial Jurisdiction
f/k/a	Formerly Known As
H <sub>2</sub> CrO <sub>4</sub>	Chromic Acid
H <sub>2</sub> S	Hydrogen sulfide
HAC Value	Health Assessment Comparison Value
HBSL	Health Based Screening Levels
HEC	Human Equivalent Concentration
HI	Hazard Index
HQ	Hazard Quotient
HWDF	Hazardous Waste-Derived Fuel
IARC	International Agency for Research on Cancer
IEUBK	Integrated Exposure Uptake Biokinetic Model
IRIS	US Environmental Protection Agency's Integrated Risk Information System
IUR	Inhalation Unit Risk
LC	Local Conditions
LOAEL	Lowest Observed Adverse Effects Level
LOAEL <sub>Adj</sub>	Adjusted Lowest Observed Adverse Effects Level
LOAEL <sub>HEC</sub>	Lowest Observed Adverse Effects Level - Human Equivalent Concentration

m <sup>3</sup> /day	Cubic meters per day
mg/kg	Milligrams per kilogram
mg/L	Milligrams per liter
MnCl <sub>2</sub>	Manganese Chloride
MnO <sub>2</sub>	Manganese Dioxide
Mn <sub>2</sub> O <sub>3</sub>	Manganese Trioxide
MRL	Minimal Risk Level
MTL	Maximum Tolerable Levels
NAAQS	National Ambient Air Quality Standards
NTP	National Toxicology Program
NOAEL	No Observed Adverse Effects Level
NOAEL <sub>Adj</sub>	Adjusted No Observed Adverse Effects Level
NOAEL <sub>HEC</sub>	No Observed Adverse Effects Level - Human Equivalent Concentration
NO <sub>x</sub>	Nitrogen oxides
O <sub>3</sub>	Ozone
OSF	Oral Slope Factor
PCDDs	Polychlorinated dibenzodioxins
PCDFs	Polychlorinated dibenzofurans
PM <sub>10</sub>	Particulate matter up to 10 microns in size
PM <sub>2.5</sub>	Particulate matter up to 2.5 microns in size
ppb	Parts per billion
ppm	Parts per million
ppt	Parts per trillion
PVC	Polyvinyl Chloride
QA/QC	Quality Assurance/Quality Control
RADS	Reactive Airways Dysfunction Syndrome
RfC	Reference Concentration
RfD	Reference Dose
SIR	Standardized Incidence Ratio
SMR	Standardized Mortality Ratio
SNCR	Selective Non-Catalytic Reduction
SO <sub>2</sub>	Sulfur dioxide
STP	Standard Temperature and Pressure
TCDD	Tetrachlorodibenzodioxin
TCDF	Tetrachlorodibenzofuran
TCEQ	Texas Commission on Environmental Quality
TDF	Tire-Derived Fuel
TEF	Toxicity Equivalency Factor
TEQ	Toxicity Equivalency Quotient
TNRCC	Texas Natural Resource Conservation Commission
TSP	Total Suspended Particulates
TXI	Texas Industries, Inc.
95% UCL	95% Upper Confidence Limit
µg/m <sup>3</sup>	Micrograms per cubic meter
µm	Micrometer
VOCs	Volatile Organic Compounds
WHO	World Health Organization

## **Appendix B – List of Petitioner and Community Concerns**

## List of Petitioner and Community Concerns

A. The specific concerns expressed by the petitioners include the following:

1. All the chemicals being released from cement kilns and steel mills have not been fully identified.
2. All the chemicals currently being incinerated and released have not been tested for carcinogenicity and endocrine disrupting potential.
3. Some of the airborne emissions may be persistent in the environment leading to long-term exposure potential.
4. The impact of aggregate low-level exposures on pregnant women, infants, children, the elderly, and the immunosuppressed has not been evaluated.
5. The TCEQ's risk assessment methodology, because of its dependence on effects screening levels (ESLs), may not be sufficiently protective of public health.
6. Results from TCEQ's air monitoring stations may not be representative of actual exposures, in part, because samples are collected only once every 6 days, but also because the collection sites may not be optimally positioned to accurately characterize air emissions in Midlothian.
7. In the past, DSHS has relied on summarized data from the TCEQ who, in turn, has used screening levels of questionable validity to determine whether or not contaminant levels were in a safe range.
8. While DSHS has, to some extent, evaluated cancers and birth defects, they have failed to evaluate health issues such as breathing problems, immune deficiencies, and other conditions, which are not categorized as cancers or birth defects.

B. The petition included the following statements regarding the health of Midlothian and other Ellis County residents:

1. The rates for cancers, including leukemia, CNS/brain cancer, and childhood total cancer, are higher in Ellis County when compared to state-wide rates.
2. The rates for birth defects, including hypospadias/epispadias and others not specified in the petition are higher in Midlothian when compared to state-wide rates.
3. A study by DSHS of a Down syndrome cluster in Ellis County was conducted but not designed to consider environmental factors.
4. A higher incidence of respiratory problems has been identified in Midlothian residents (when compared to Waxahachie residents), as reported in a symptom survey conducted by Legator, et al. (1998) [73].

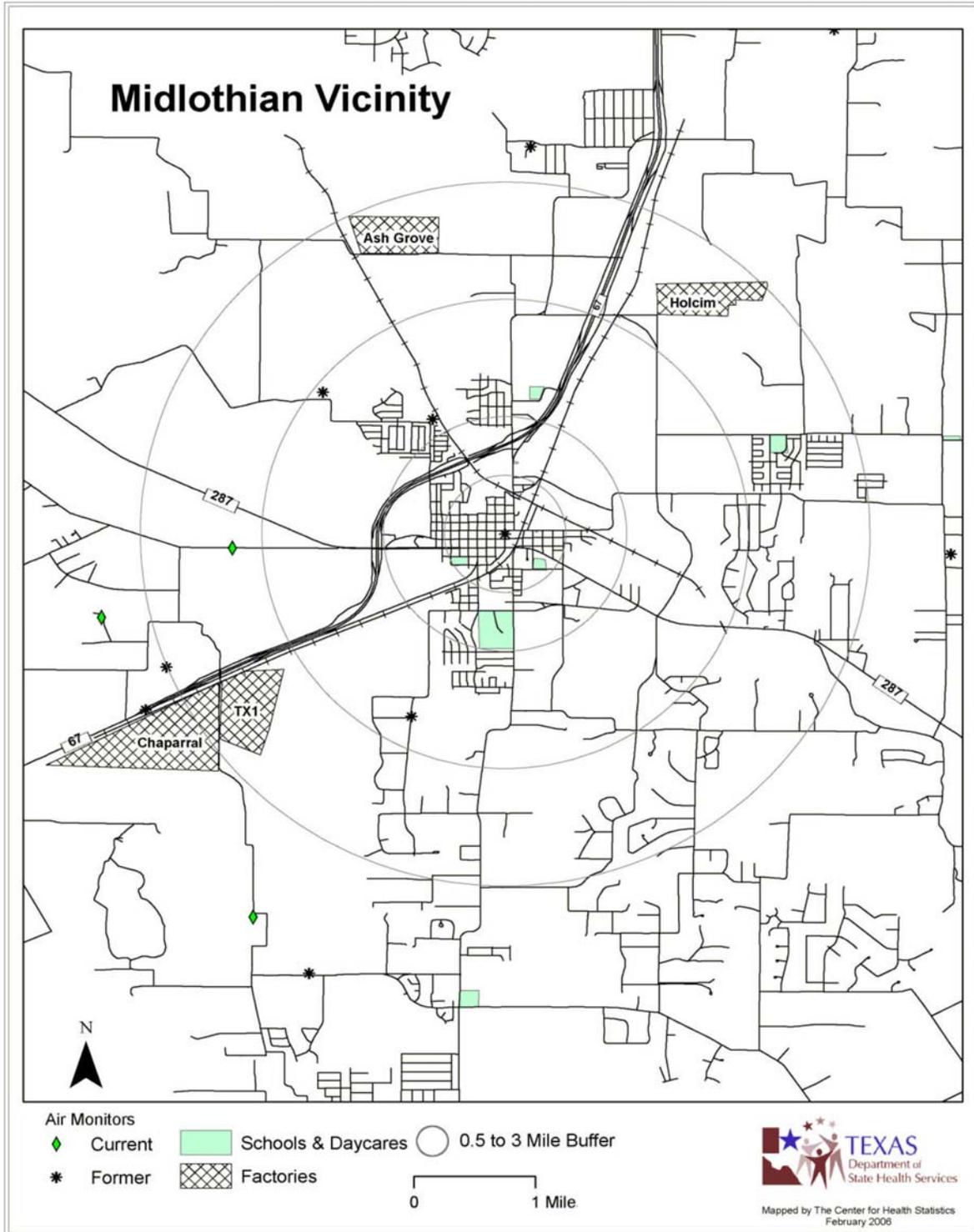
C. An August 12<sup>th</sup> addendum to the initial petition document requested that ATSDR and DSHS consider the following additional items of concern:

1. An independent peer review group found major problems with TCEQ's ESLs, claiming they are almost 300 times as high as those of New Jersey and Vermont and 12 times as high as Louisiana.
2. The 3 CAM sites in Midlothian (CAMS 302, CAMS 52, and CAMS 94) do not monitor for the almost 100 VOCs and heavy metals as the petitioners had been previously informed.

3. The potential impact of “aggregate” exposures to the emissions from the four major local industries and “aggregate” health conditions among Midlothian area residents should be evaluated.
  4. There is a concern that there is a high incidence of immune system deficiencies manifesting not only in animals but in the human population as well.
- D. DSHS gathered information about the community and their concerns from previous reports prepared by the TNRCC, EPA, and DSHS, the petition to ATSDR, and through meetings, conversations, e-mailed comments, and door-to-door surveys conducted in the community during the preparation of this document. Based on the information gathered, DSHS determined that the community is concerned about the potential health impacts of emissions from the major industries in Midlothian. Community concerns include the following:
1. Asthma, allergies, and other health problems in adults as well as children
  2. Cancer and birth defects
  3. Health effects of dioxins, metals, and mixtures of compounds
  4. The potential for adverse health effects to be underestimated by averaging of contaminant levels over time, which may tend to mask the peak exposures
  5. Health problems with their domesticated animals and livestock such as, but not limited to, spontaneous abortions or stillbirths, difficulties with reproduction or breeding, birth defects, and immune system disorders. Residents have attributed these problems to environmental pollution.

## **Appendix C – Figures**

**Figure 1. Map of Midlothian and Vicinity with Schools, Day Care Centers, and Major Industries**



**Figure 2. Aerial Photo of Midlothian and Surrounding Area with Historical Air Monitoring Site Locations**

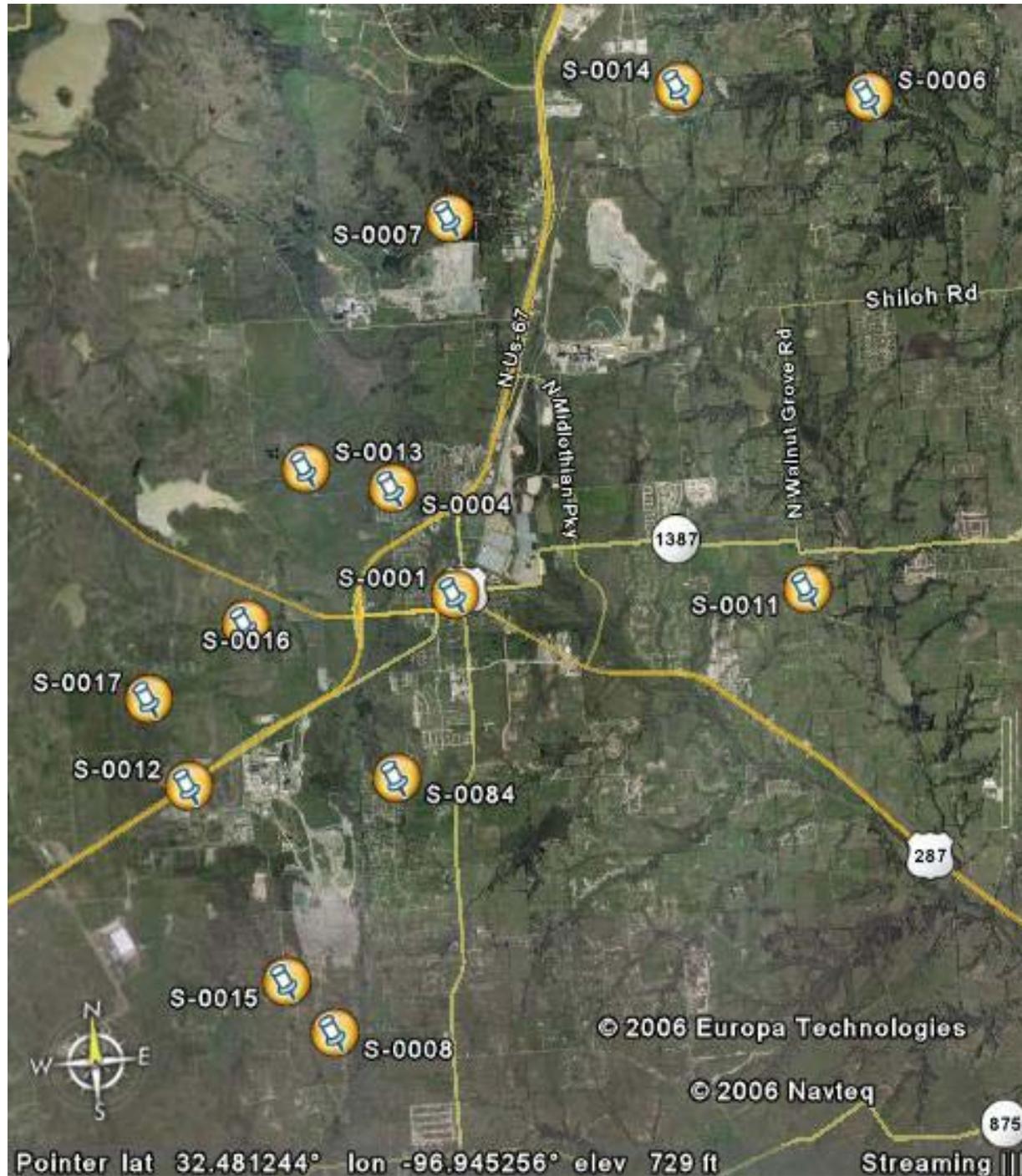


Figure 3. Benzene

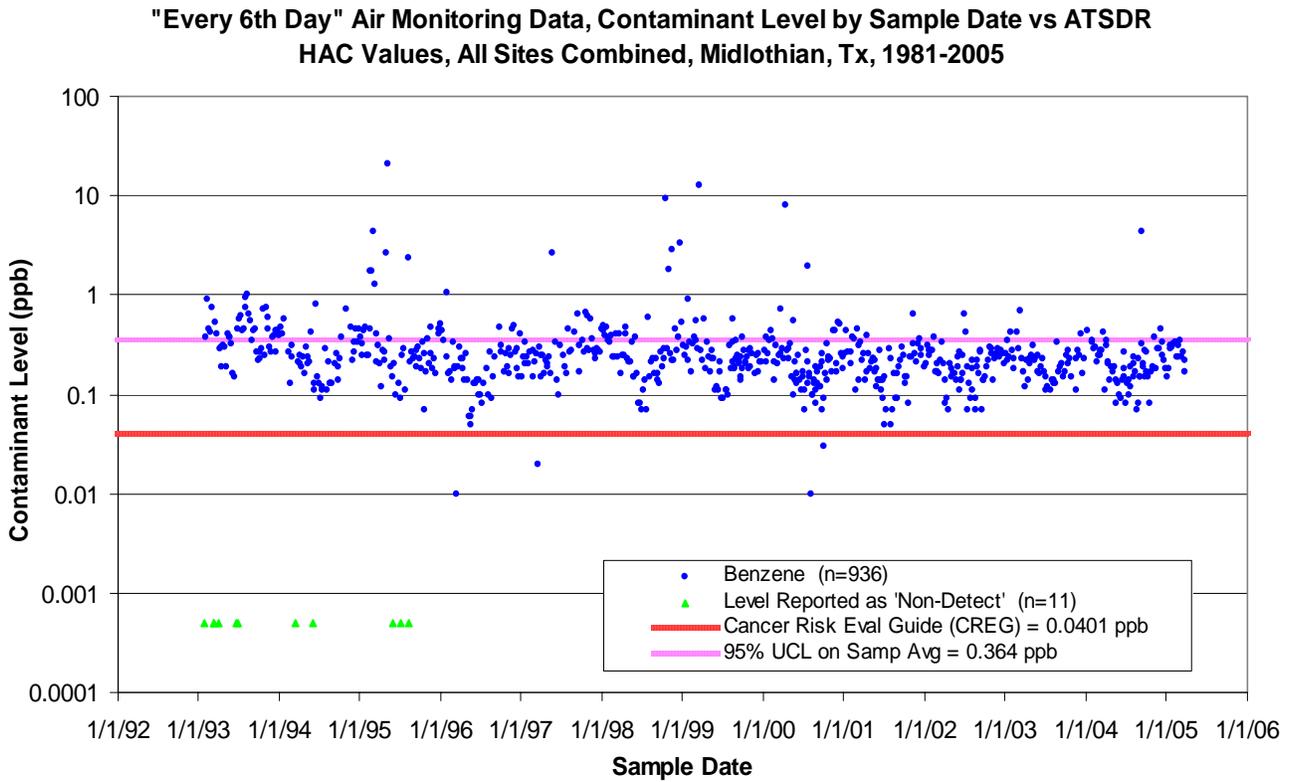


Figure 4. 1,3-Butadiene

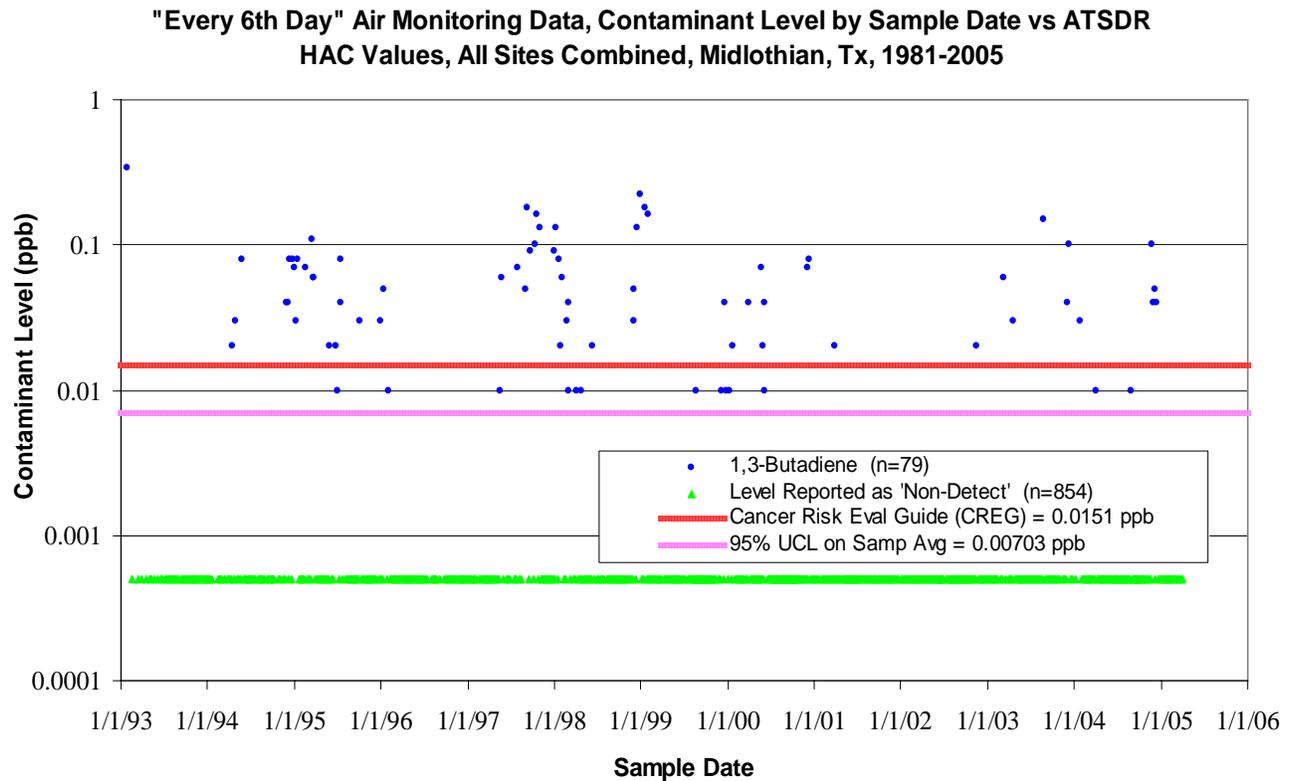


Figure 5. Carbon Tetrachloride

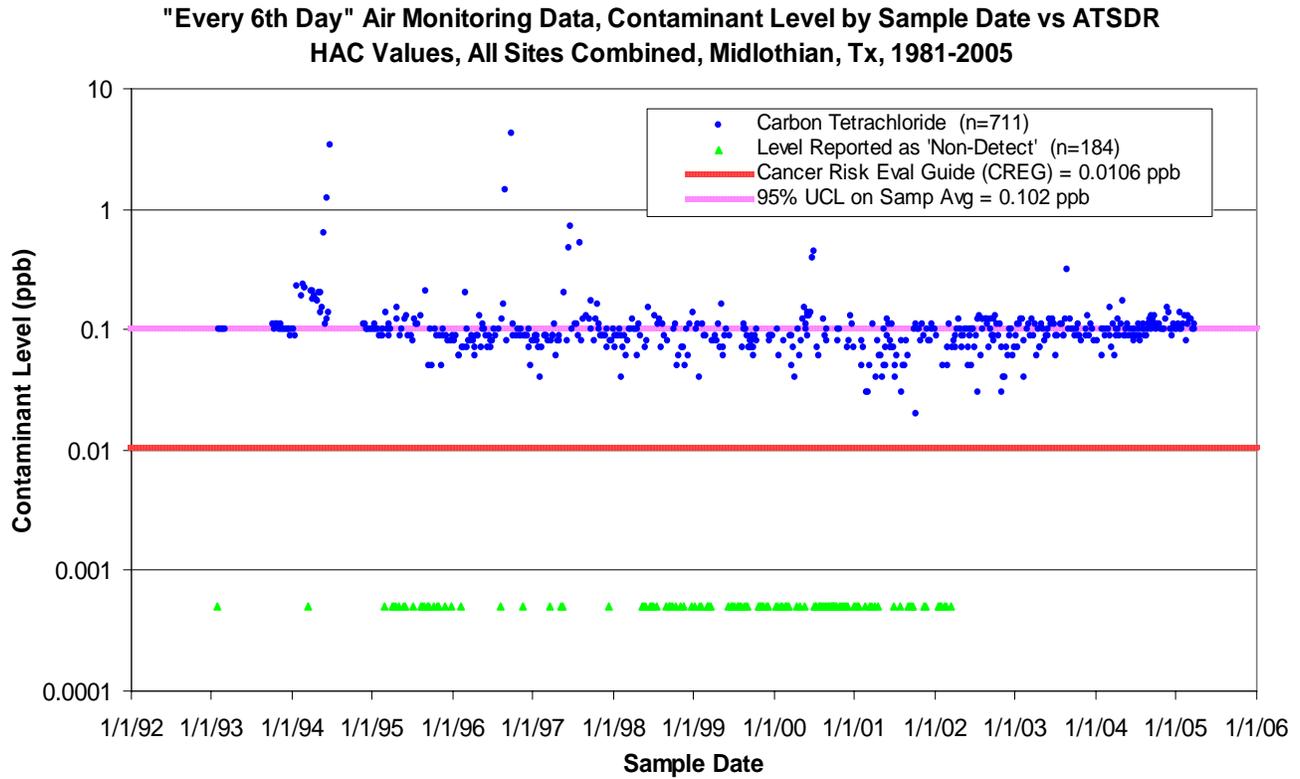


Figure 6. Chloroform

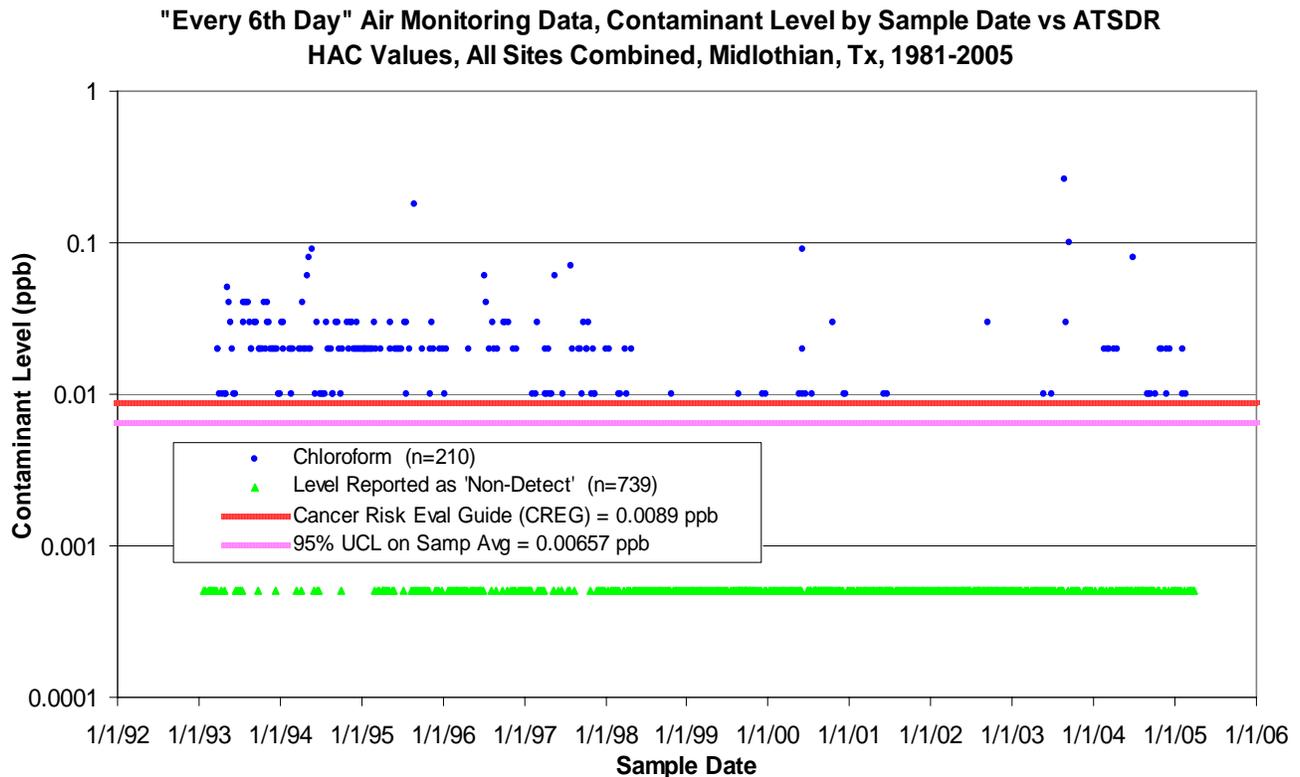


Figure 7. 1,2-Dibromoethane

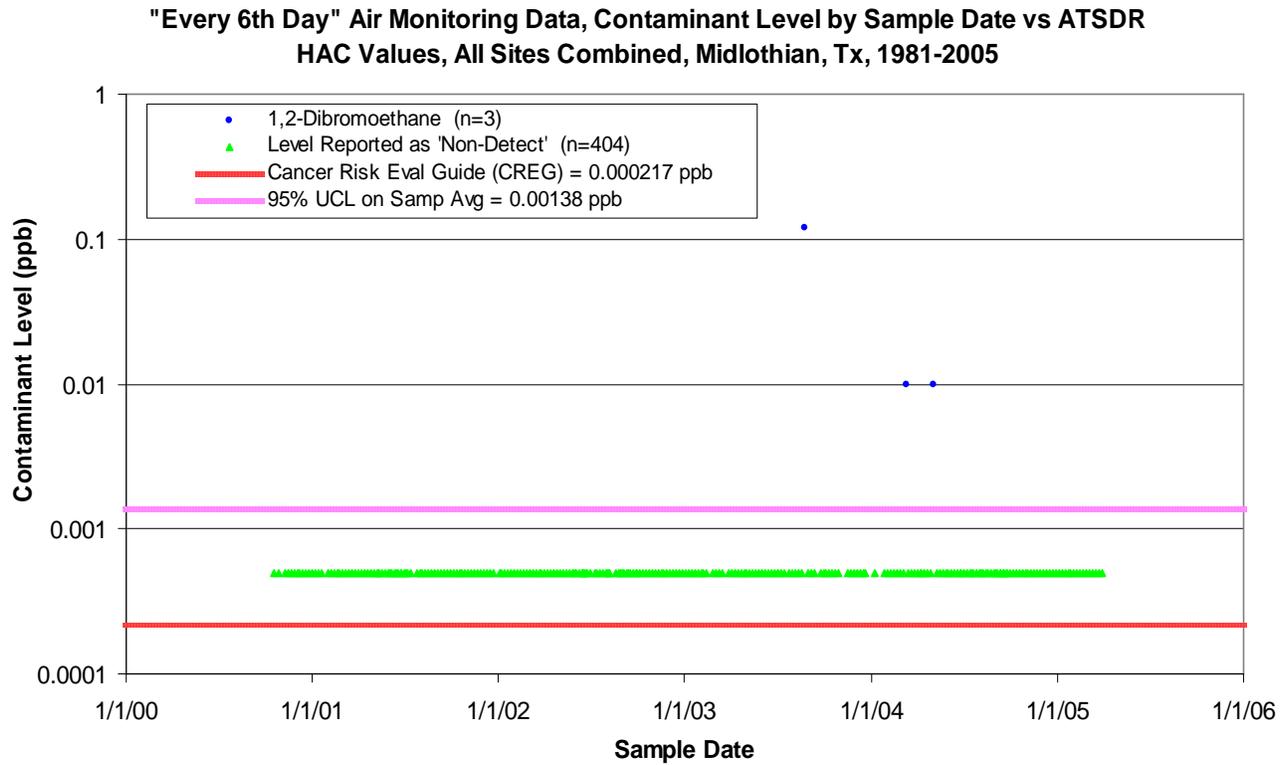


Figure 8. 1,2-Dichloroethane

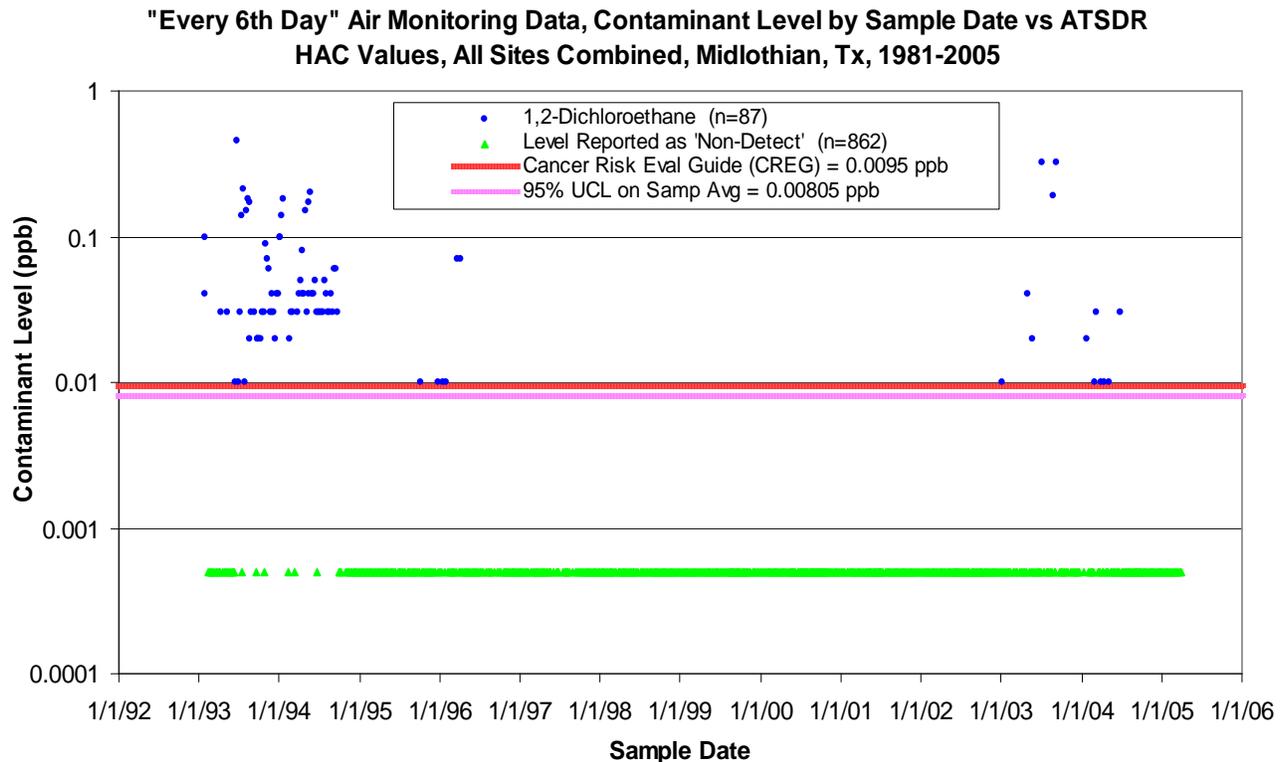


Figure 9. Methylene Chloride

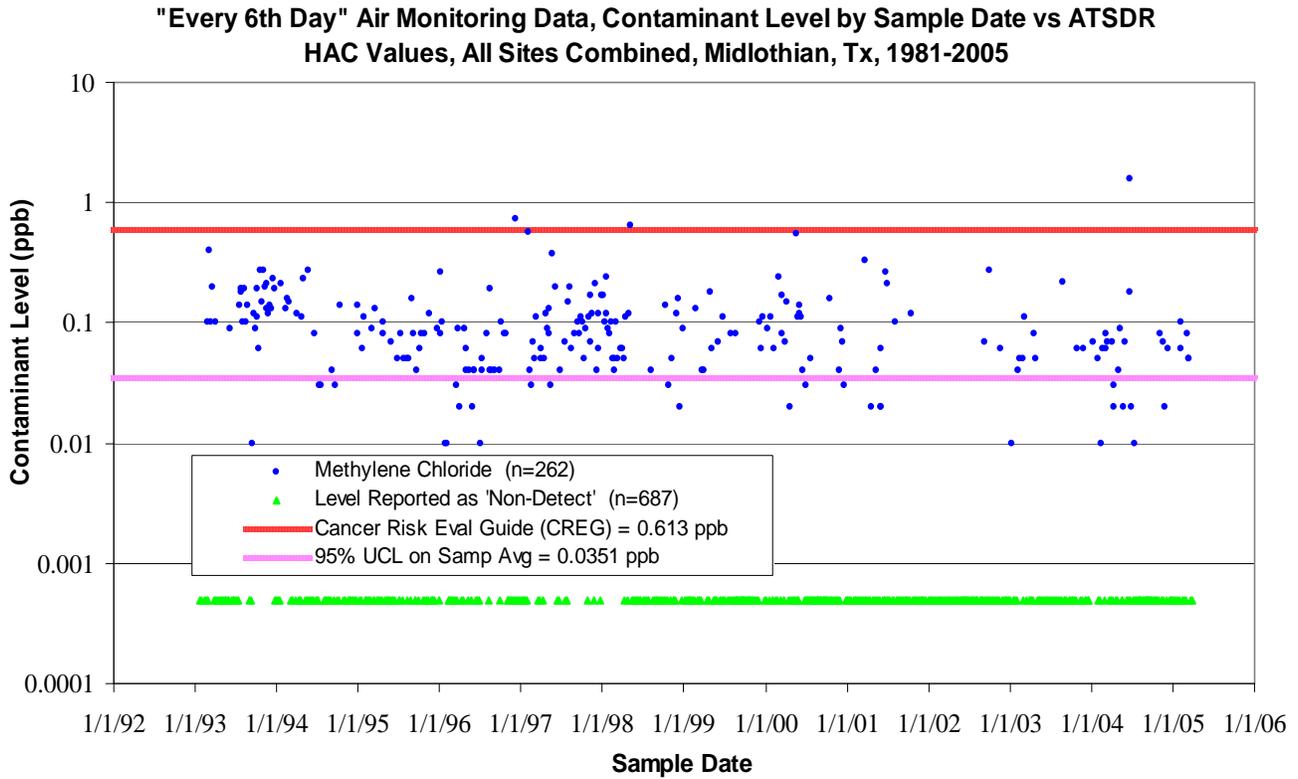


Figure 10. 1,1,2,2-Tetrachloroethane

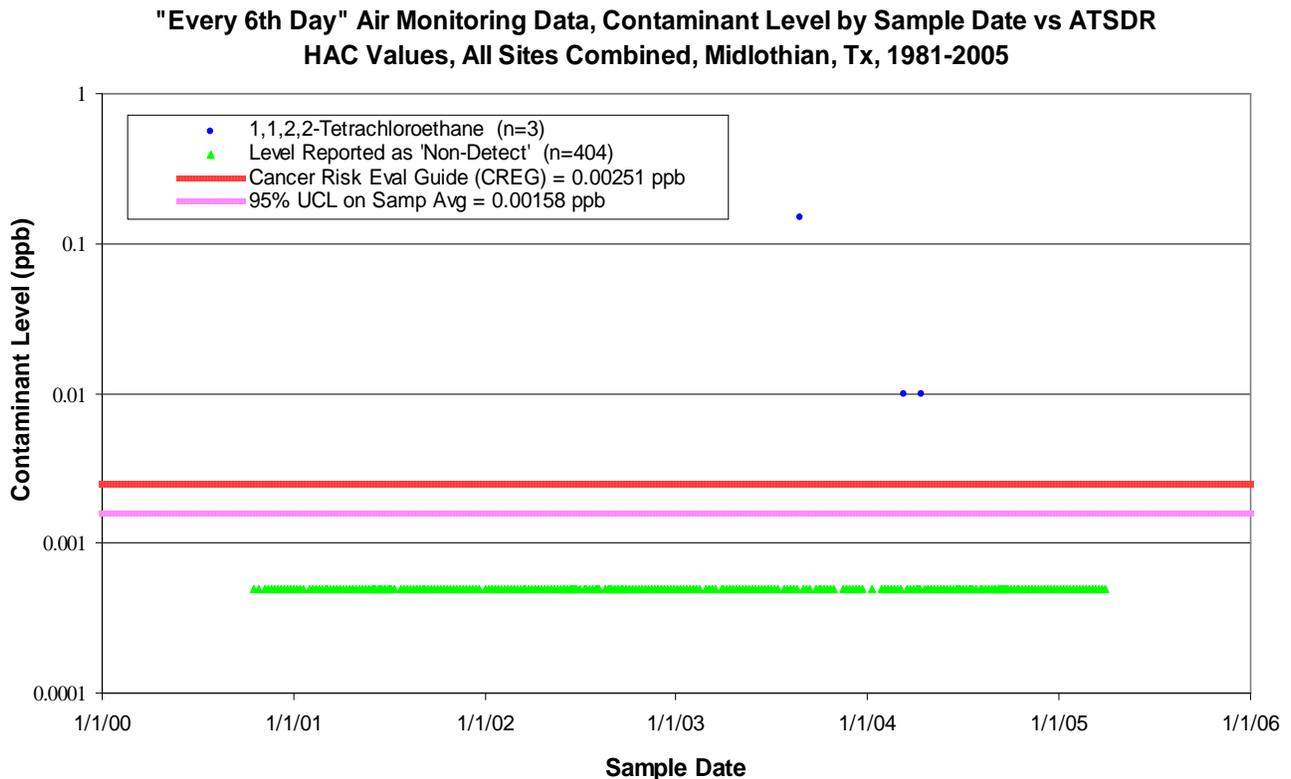


Figure 11. 1,1,2-Trichloroethane

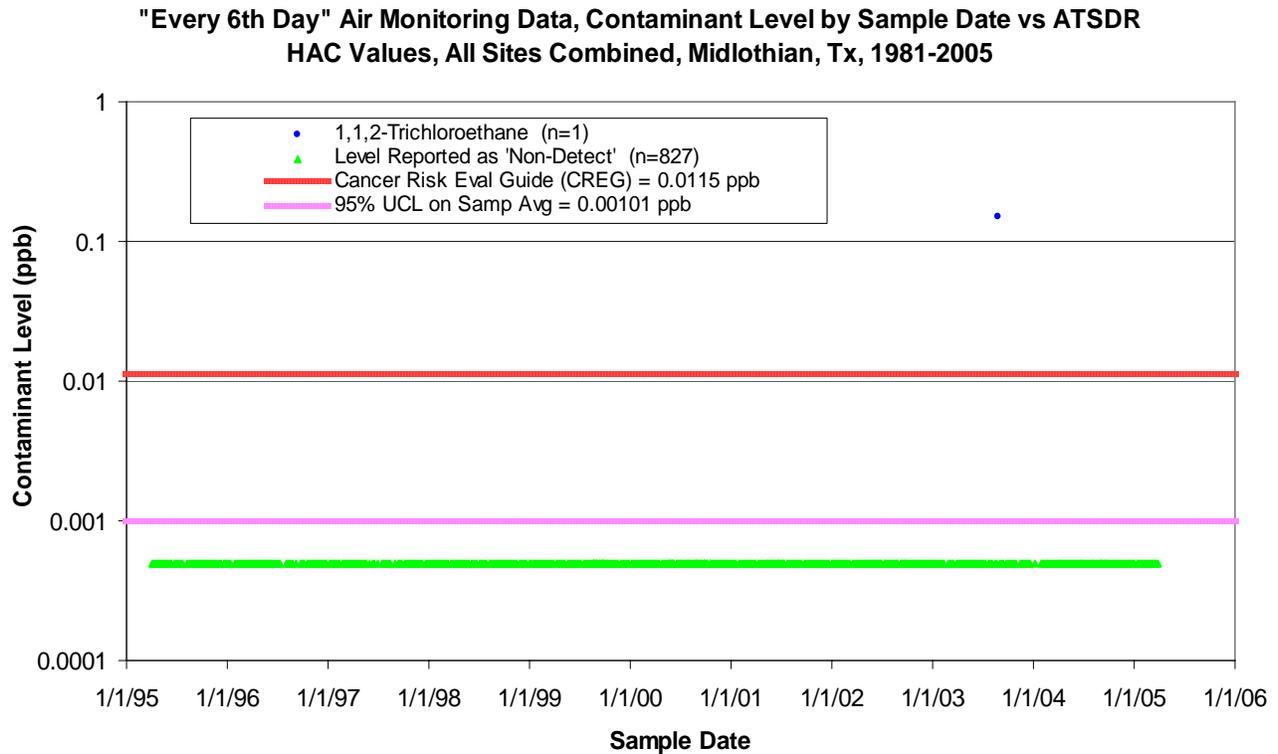


Figure 12. 1,2,4-Trimethylbenzene

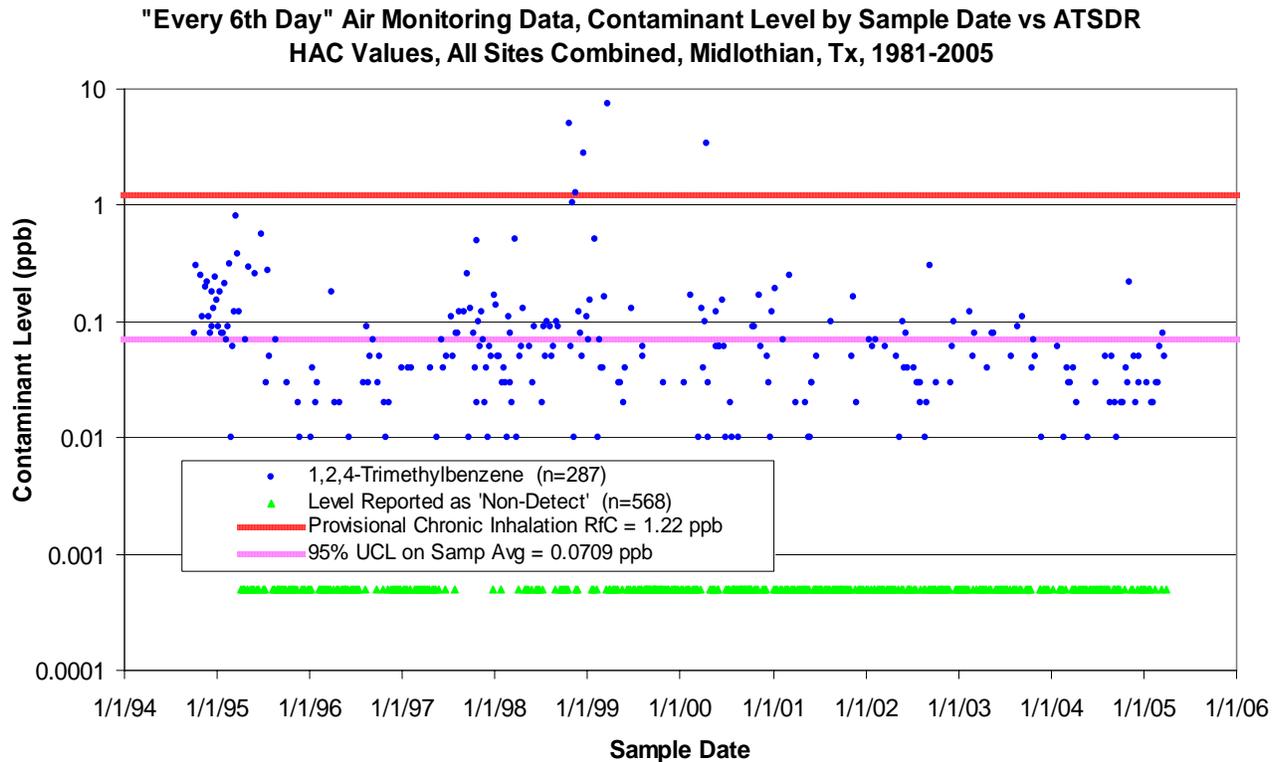


Figure 13. 1,3,5-Trimethylbenzene

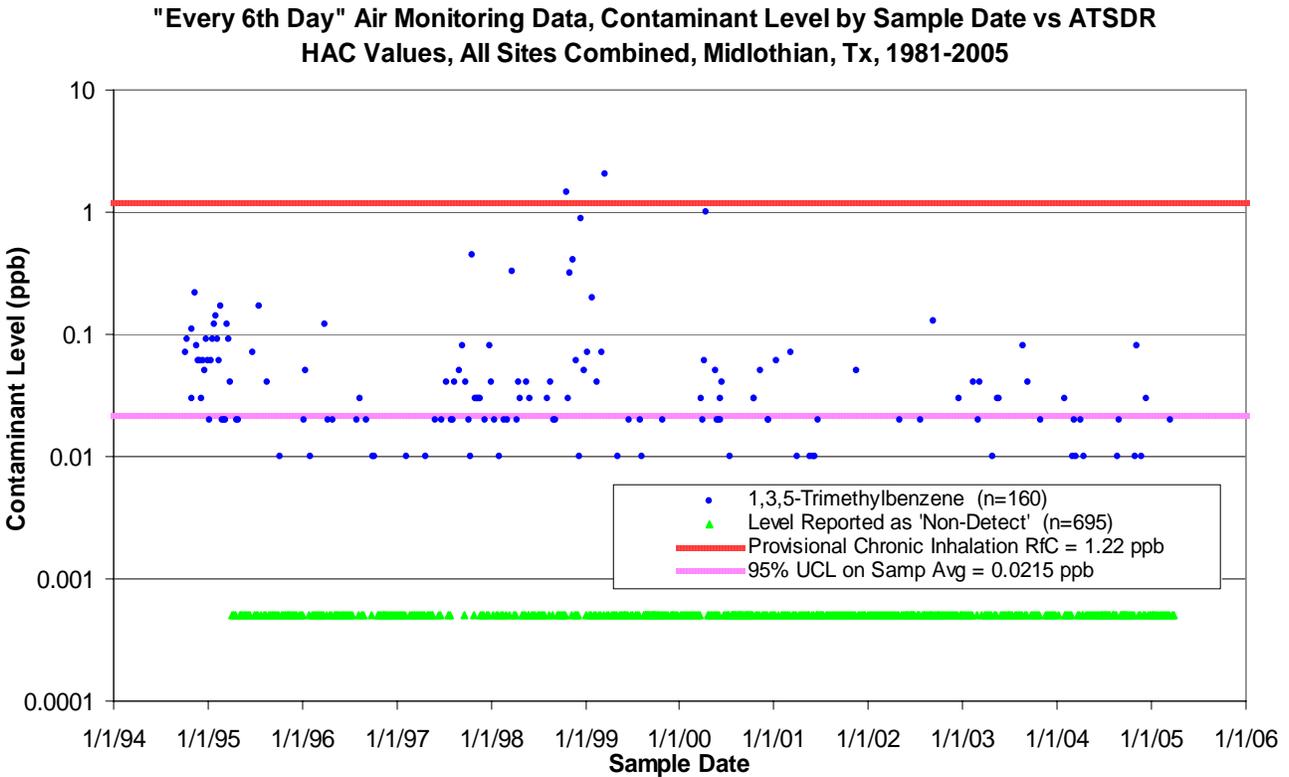


Figure 14. Vinyl Chloride

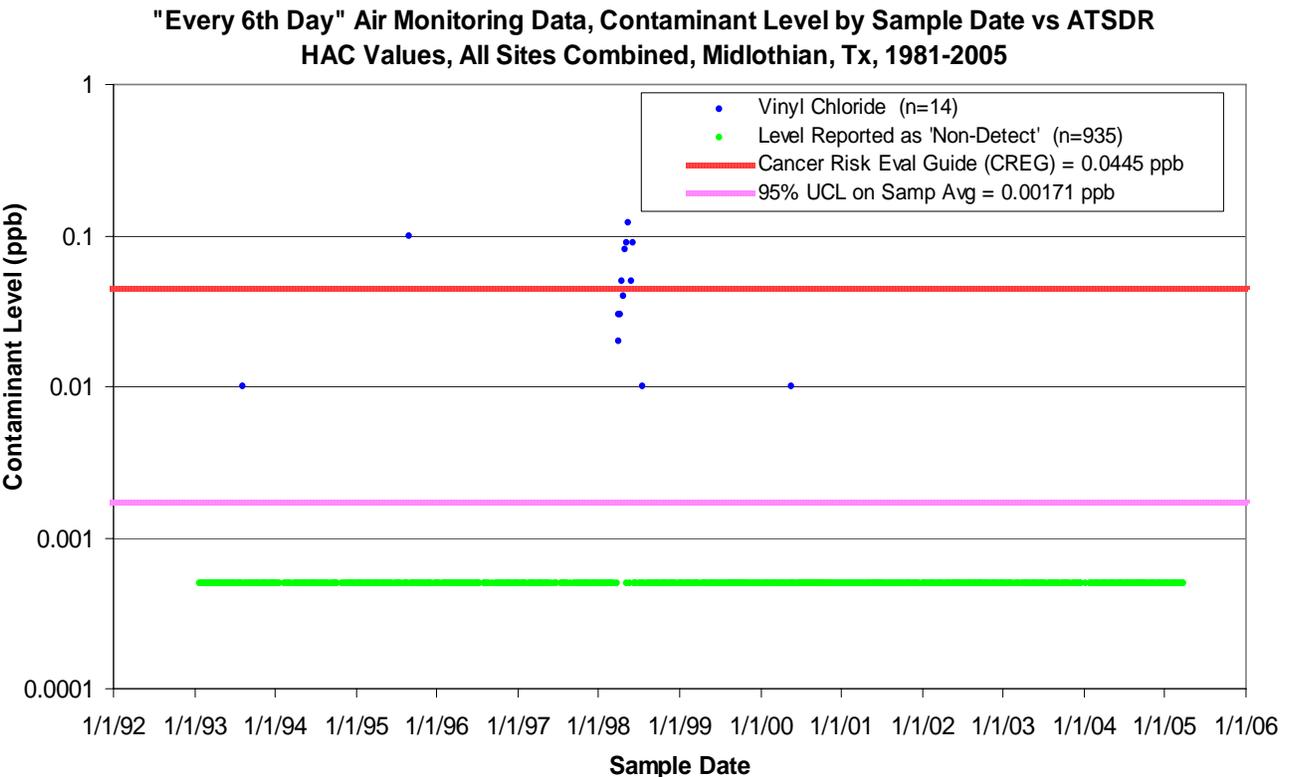


Figure 15. M+P-Xylene

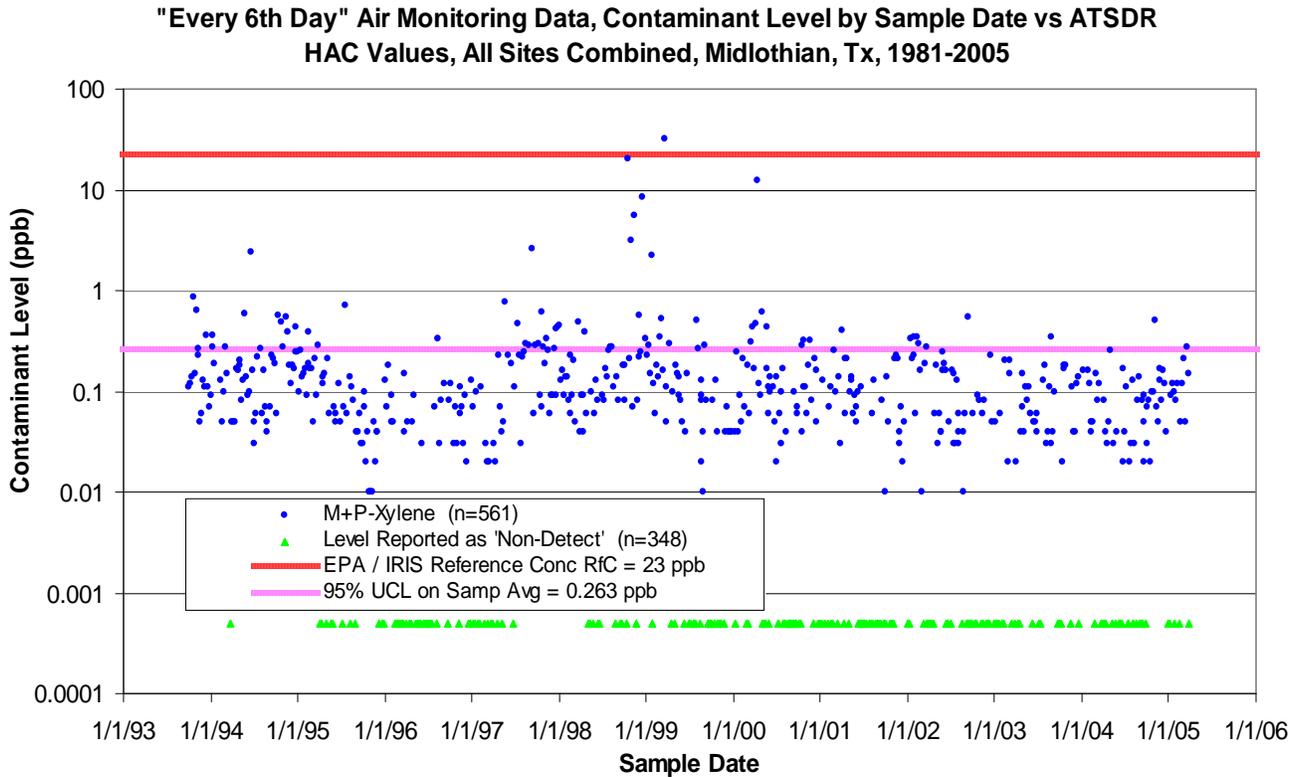


Figure 16. Arsenic (PM<sub>10</sub>)

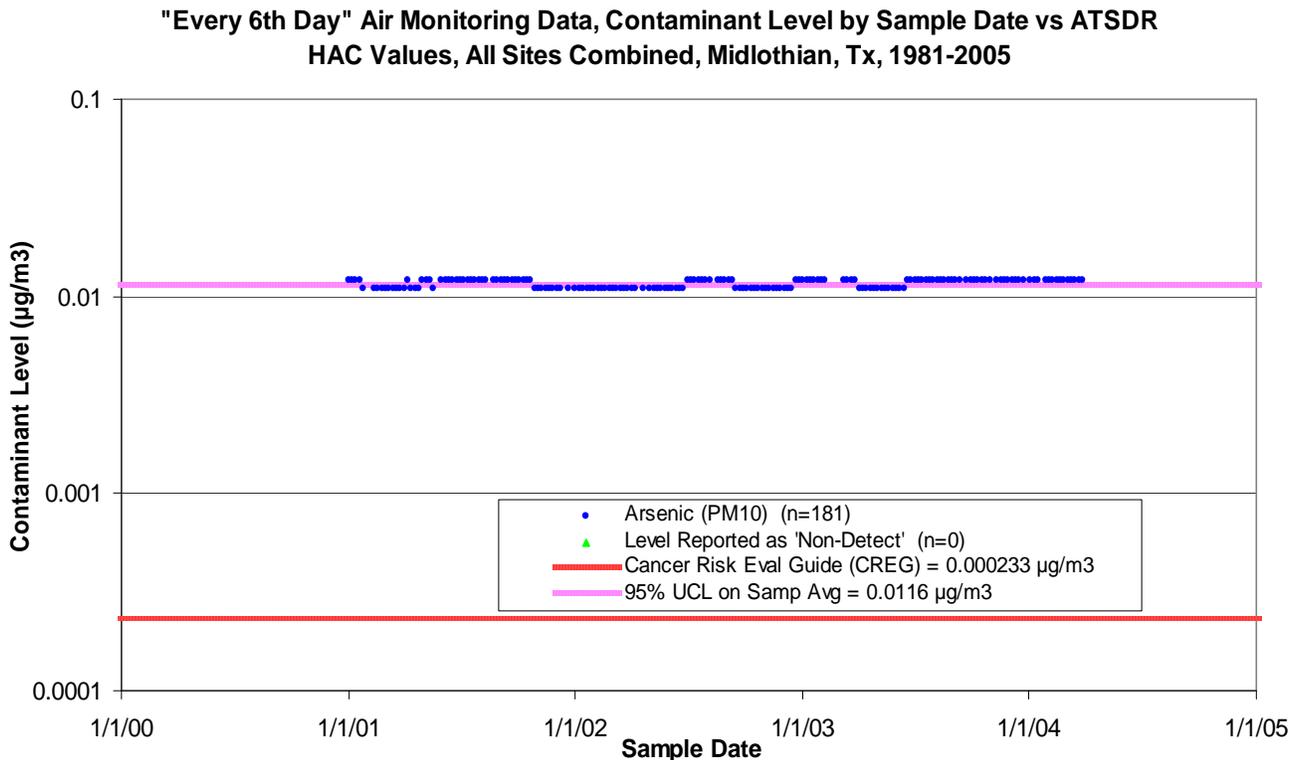


Figure 17. Arsenic (TSP)

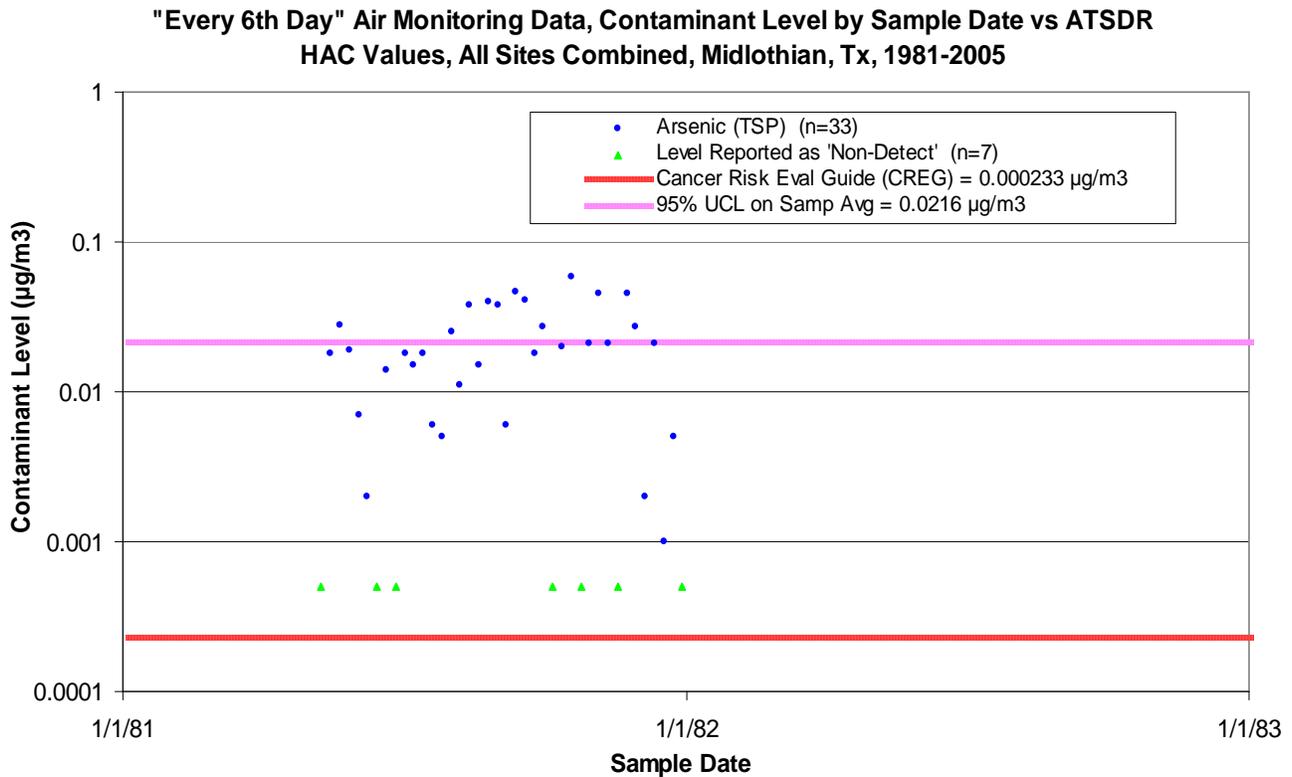


Figure 18. Arsenic (PM<sub>2.5</sub>)

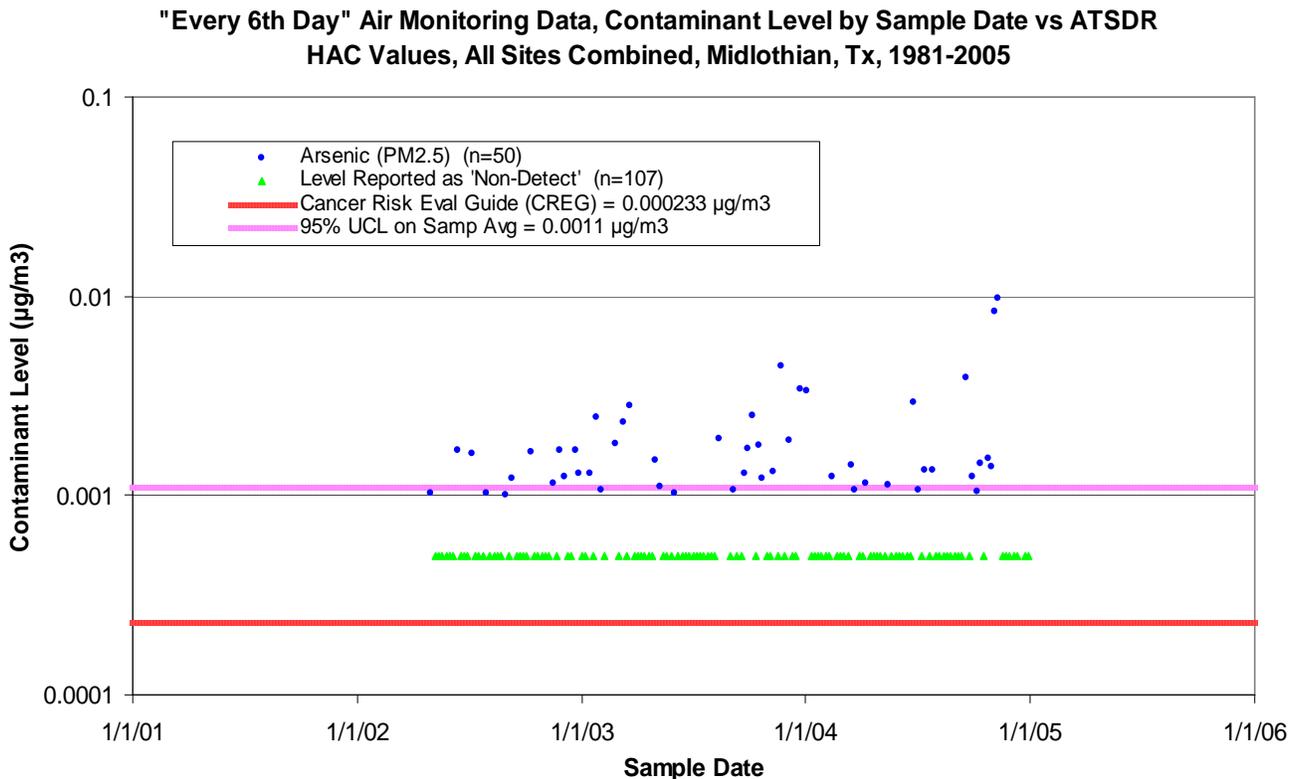


Figure 19. Beryllium (PM<sub>10</sub>)

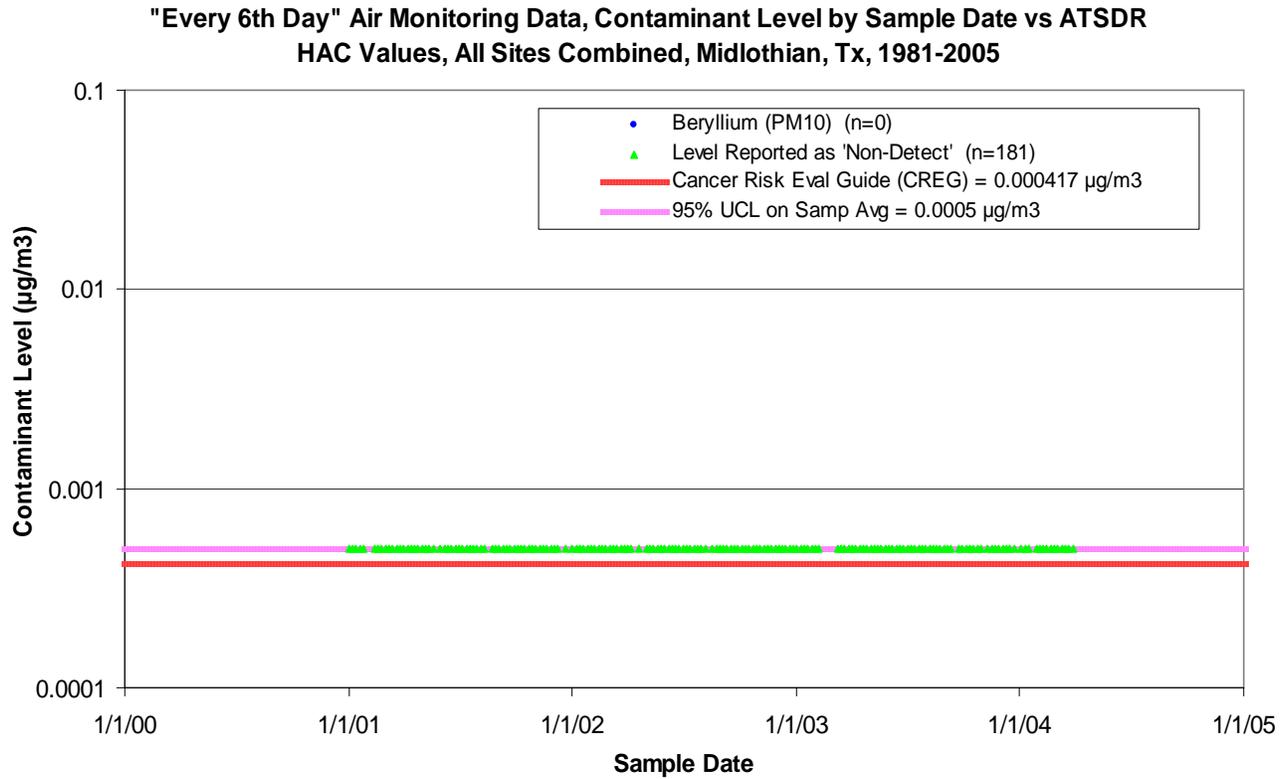


Figure 20. Cadmium (PM<sub>10</sub>)

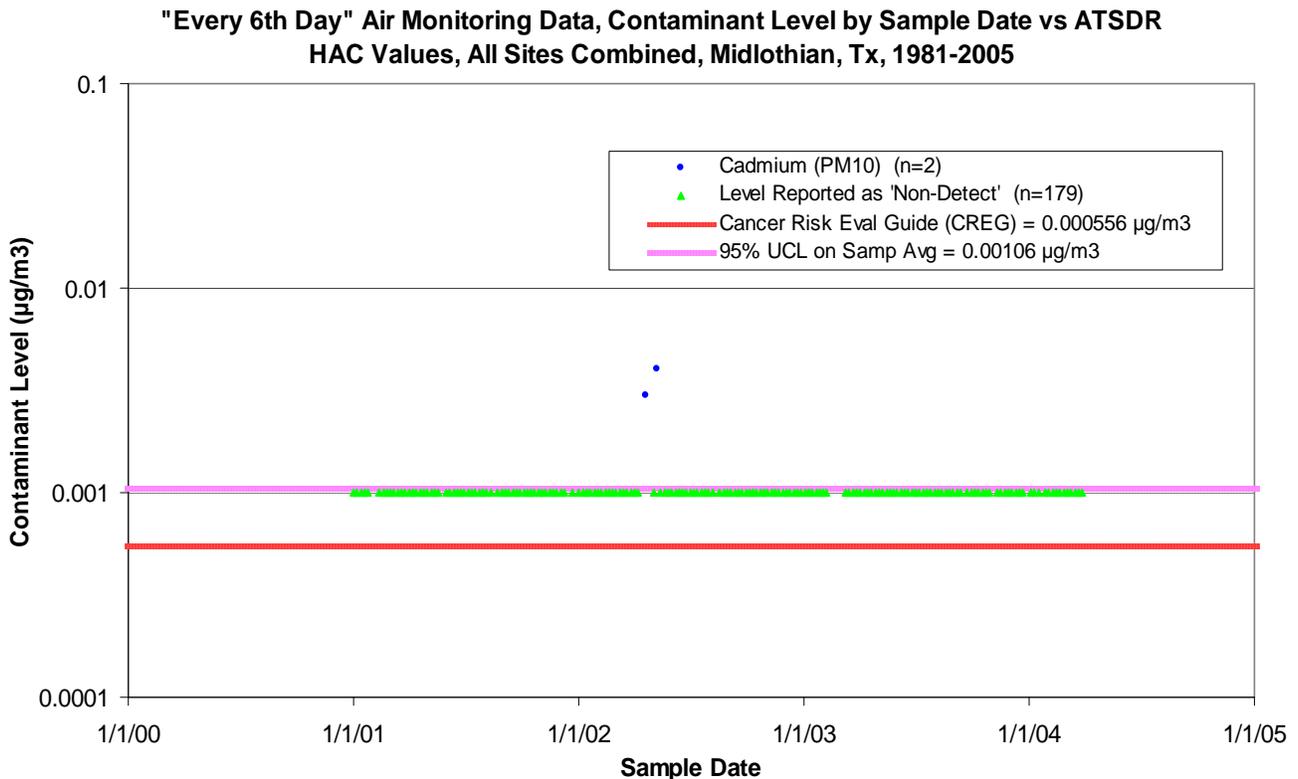


Figure 21. Cadmium (TSP)

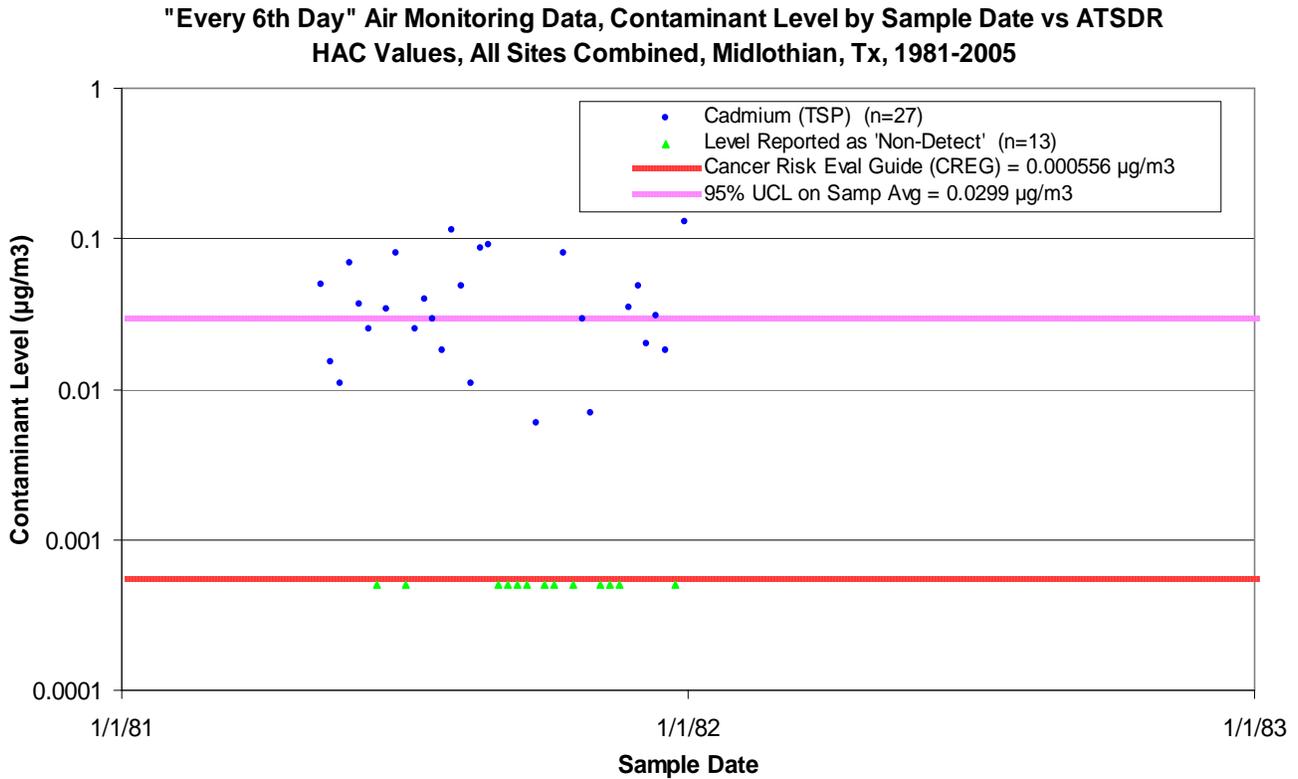


Figure 22. Cadmium (PM<sub>2.5</sub>)

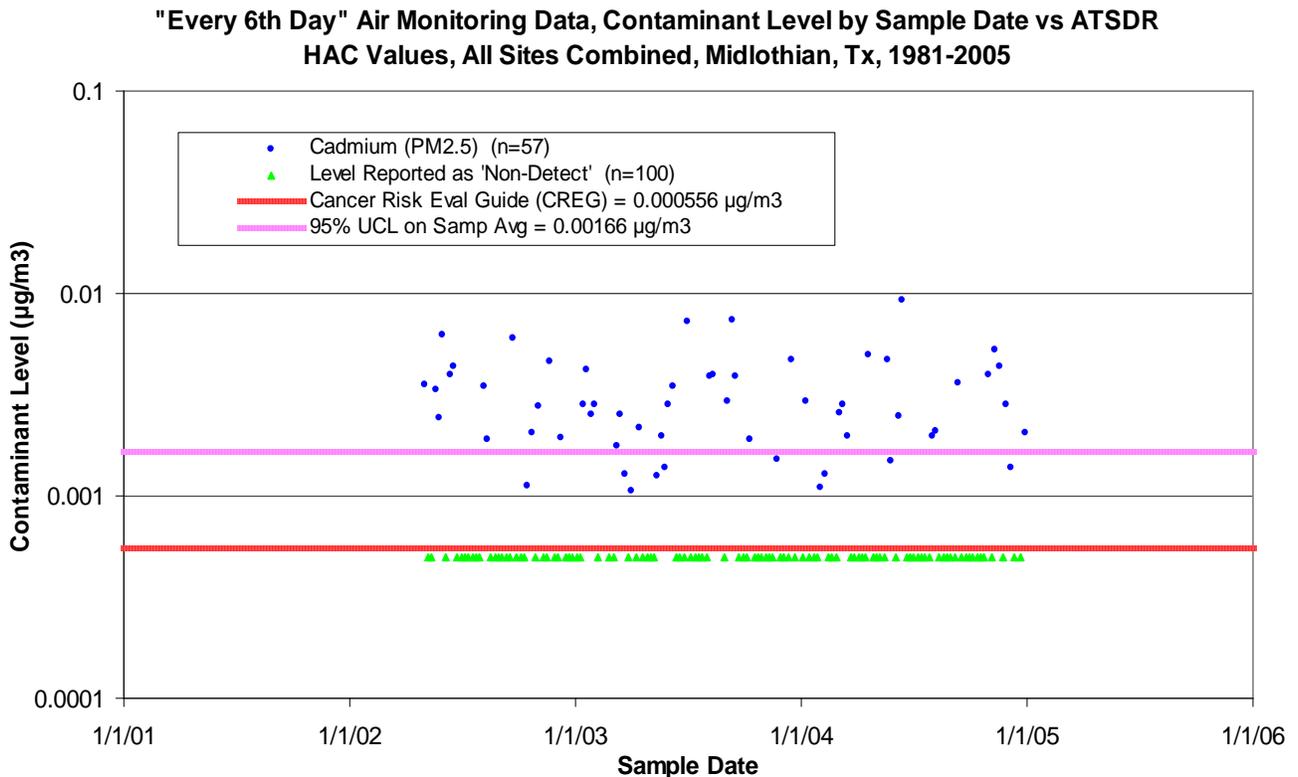


Figure 23. Chlorine (PM<sub>2.5</sub>)

**"Every 6th Day" Air Monitoring Data, Contaminant Level by Sample Date vs ATSDR HAC Values, All Sites Combined, Midlothian, Tx, 1981-2005**

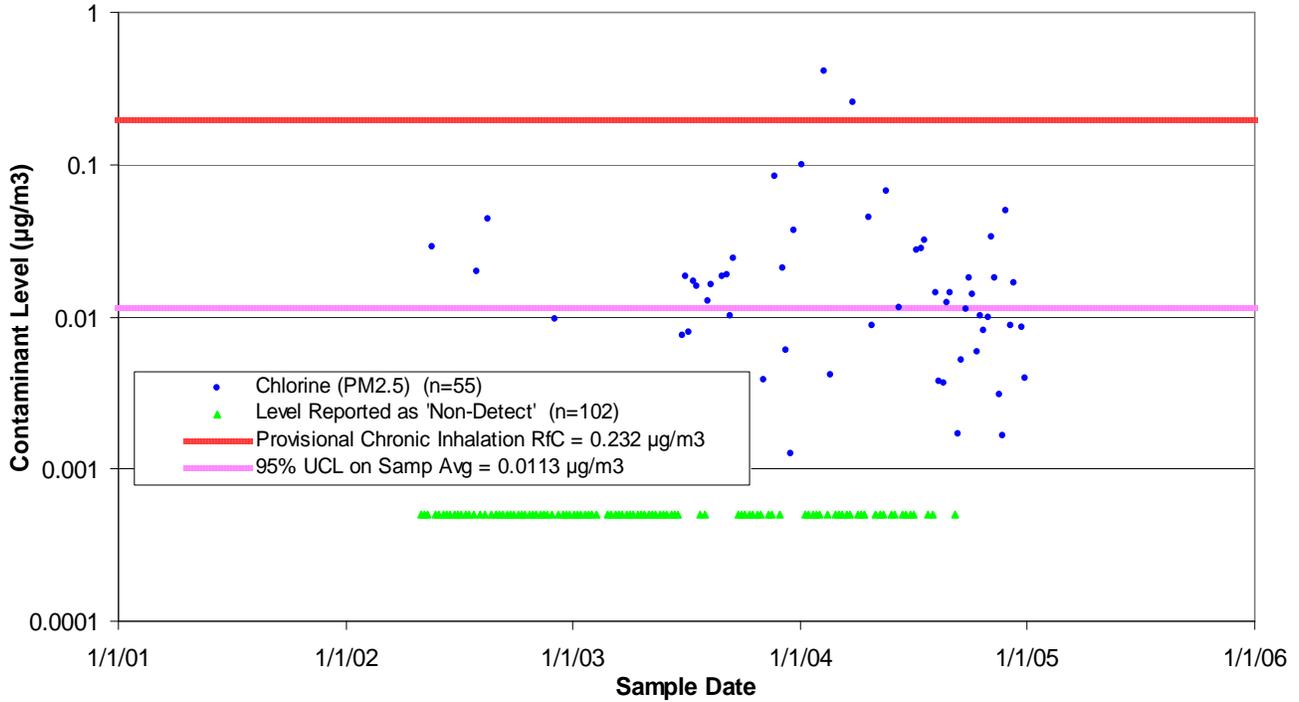


Figure 24. Chromium (PM<sub>10</sub>)

**"Every 6th Day" Air Monitoring Data, Contaminant Level by Sample Date vs ATSDR HAC Values, All Sites Combined, Midlothian, Tx, 1981-2005**

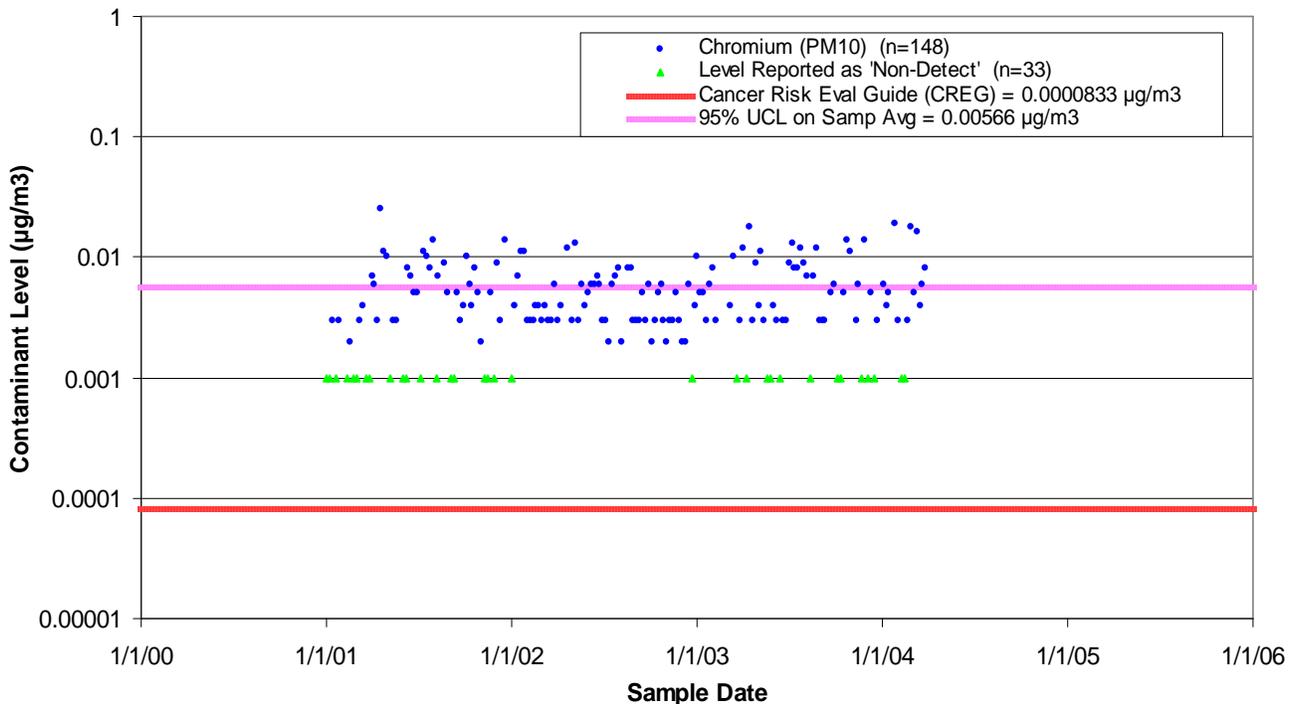


Figure 25. Chromium (TSP)

**"Every 6th Day" Air Monitoring Data, Contaminant Level by Sample Date vs ATSDR HAC Values, All Sites Combined, Midlothian, Tx, 1981-2005**

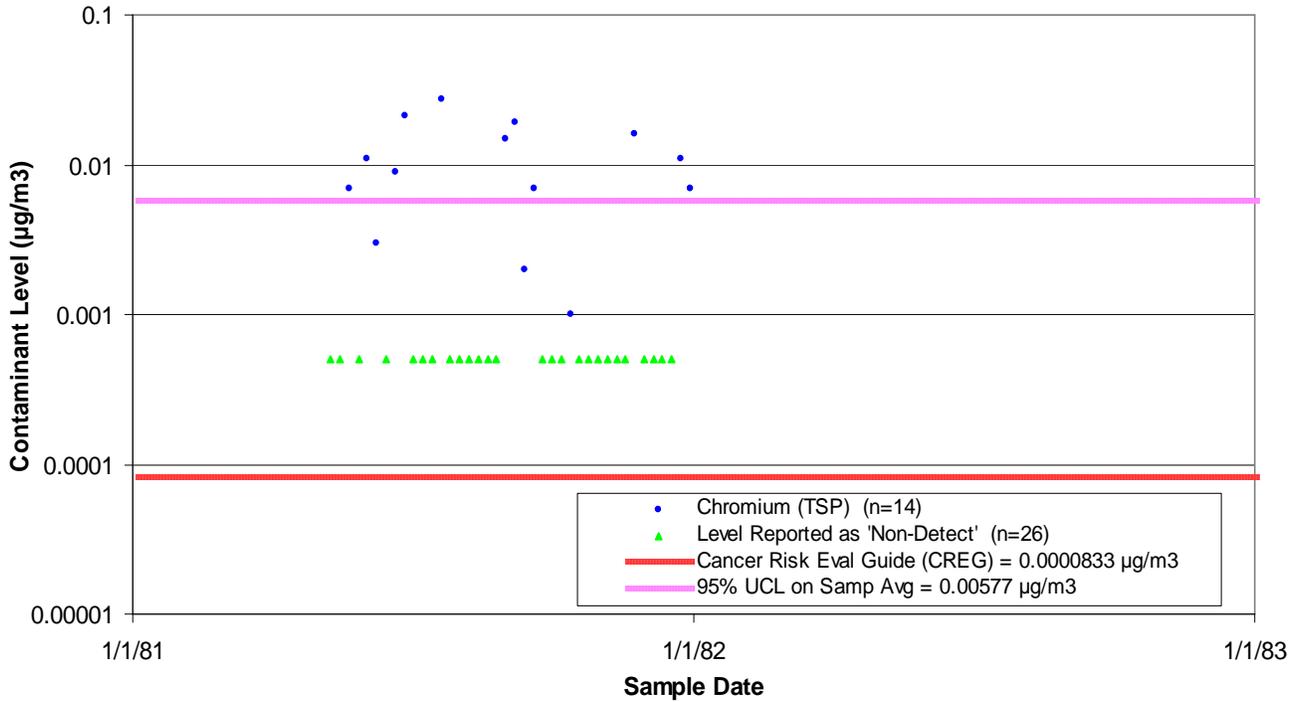


Figure 26. Chromium (PM<sub>2.5</sub>)

**"Every 6th Day" Air Monitoring Data, Contaminant Level by Sample Date vs ATSDR HAC Values, All Sites Combined, Midlothian, Tx, 1981-2005**

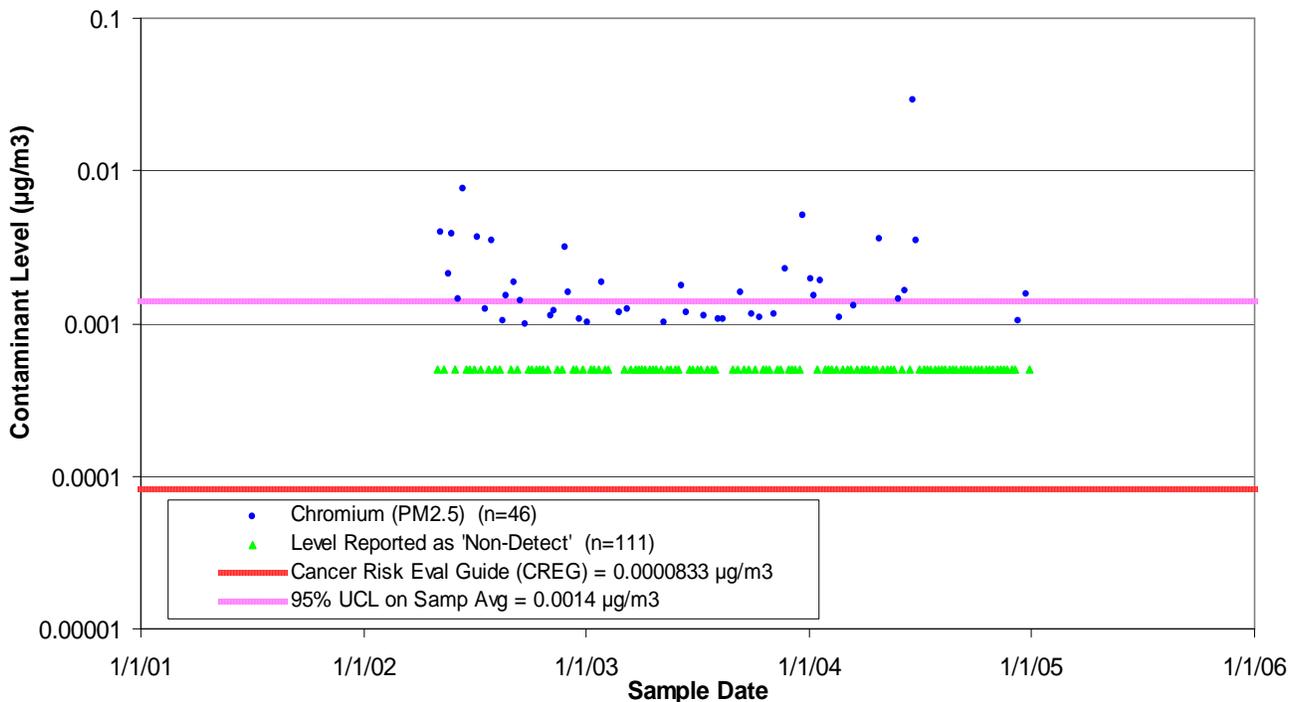


Figure 27. Lead (TSP)

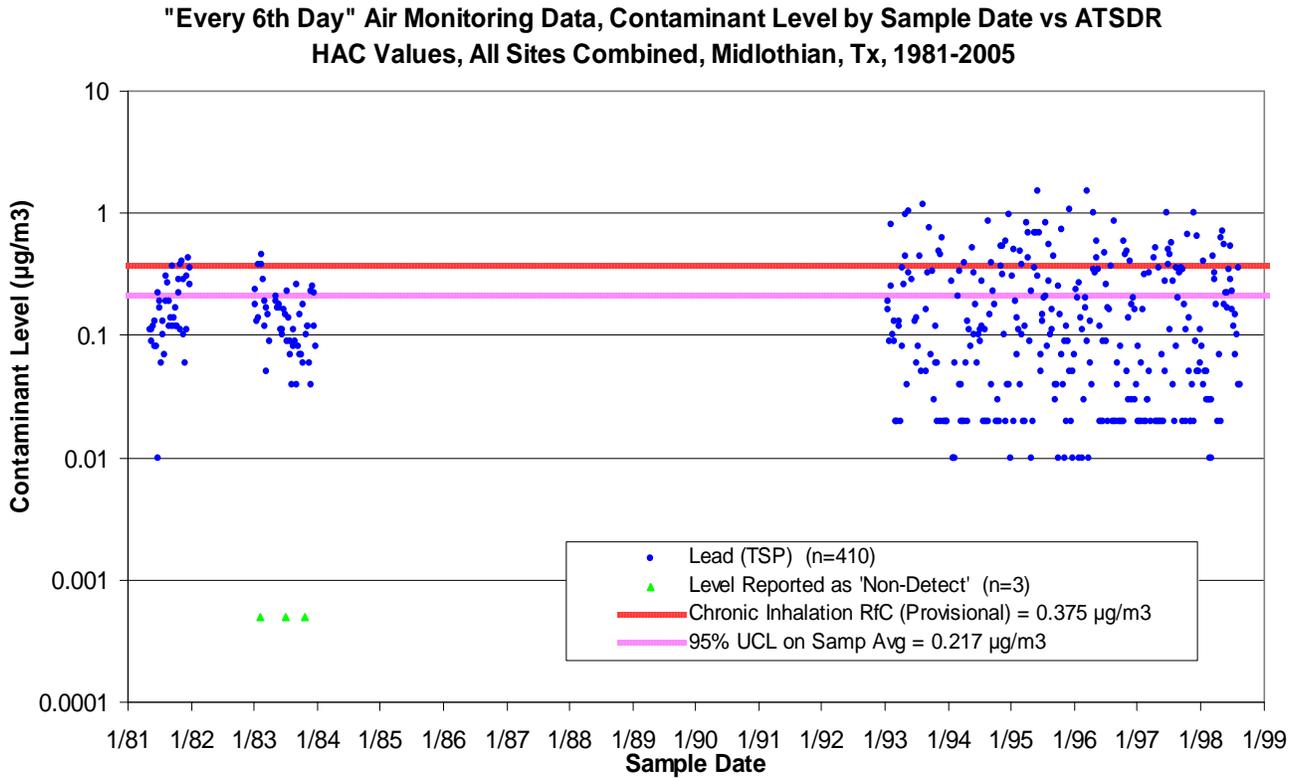
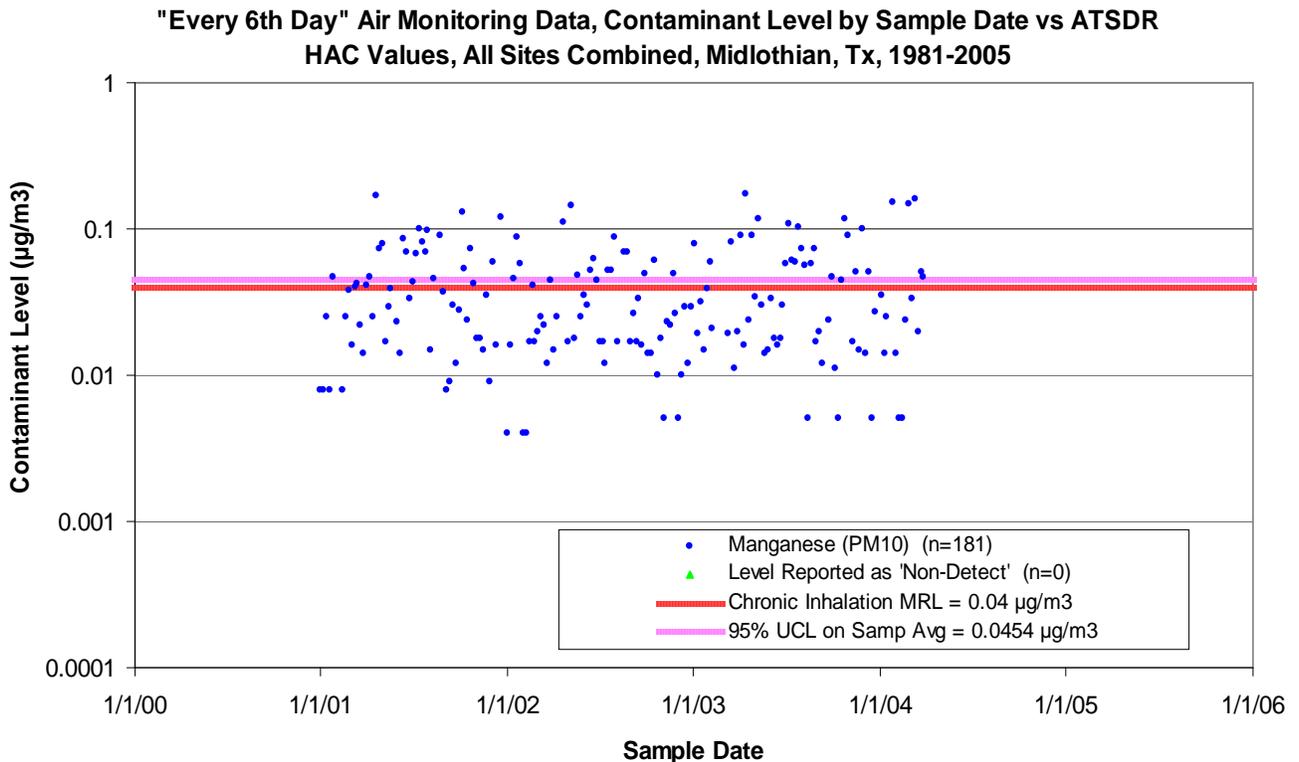


Figure 28. Manganese ( $\text{PM}_{10}$ )





## **Appendix D – Birth Defects and Cancer Registries Report Summaries**

### ***Birth Defects Registry Report Summaries***

A Down syndrome cluster investigation released in 1996 reported that the number of Down syndrome cases in Ellis, Hood, and Somervell Counties among deliveries in 1992 through 1994 was 3.4 times higher than expected based on statewide rates [74]. Those results, which included adjustment for maternal age, were statistically significant at the 95% level. While that study did not provide evidence that environmental factors were associated with the excess occurrence of Down syndrome cases, its ability to do so was limited.

In response to a citizen request, the DSHS Texas Birth Defects registry completed an additional review of birth defects registry data in June 2005 [75]. They examined the occurrence of 48 specific types of birth defects as well as “any monitored birth defect” among deliveries to residents of Midlothian, Venus, and Cedar Hill over the period from 1997 through 2001 and compared those rates to the state as a whole (1999 through 2001). Adjusting for maternal age, the prevalence rate for the occurrence of one type of birth defect related to urinary tract development (hypospadias or epispadias) was approximately 3.7 times higher than the prevalence rate observed for Texas (1999 through 2001). Adjusting for maternal race/ethnicity, the prevalence rate for hypospadias or epispadias was approximately 4.2 times higher than the prevalence rate observed for Texas (1999 through 2001). These results were statistically significant at the 95% level. Similarly, the prevalence of any monitored birth defect among Midlothian residents (1997 through 2001), adjusted for maternal age, was 1.5 times the prevalence rate for Texas (1999 through 2001), and the result was statistically significant at the 95% level. However, adjusting for maternal race/ethnicity, caused the prevalence ratio to drop to 1.2, and the result was no longer statistically significant. It is not clear what effect if any the different time periods for data inclusion in Midlothian vs. Texas may have had on the birth defect prevalence rates.

In response to additional inquiries in August and October 2006, DSHS Texas Birth Defects registry completed an additional review of birth defects registry data in November 2006. They examined the prevalence of total birth defects as well as 48 specific types of birth defects in the 11 Health Service Regions of Texas over the period from 1999 through 2003. The standardized prevalence ratio (SPR) for any monitored birth defect, adjusted for maternal age and race/ethnicity, in Health Service Region 3 (which includes Ellis County and 18 other counties in the Dallas-Fort Worth area) was found to be 18% higher than the state as a whole, and those results were statistically significant at the 95% level. Specific defects found to be significantly elevated at the 95% level included hypospadias/epispadias (SPR=1.14), obstructive genitourinary defects (SPR=1.11), microcephaly (SPR=1.31), and craniosynostosis (SPR=1.33). Pyloric stenosis was significantly lower in Health Service Region 3 than Texas as a whole (SPR=0.84). The maternal age and race/ethnicity adjusted prevalence rate (per 10,000 live births) for total birth defects in Ellis County was 483.66 compared with 360.70 in Texas as a whole (SPR=1.34); these results also were statistically significant at the 95% level. Out of 48 specific birth defects (after adjustment for maternal age and race/ethnicity), only craniosynostosis (SPR=3.61) was significantly elevated in Ellis County with respect to Texas as a whole.

### ***Cancer Registry Report Summaries***

The Texas Department of State Health Services completed cancer incidence and/or mortality investigations in November 1995 [76], February 1998 [77], June 1998 [78], and May 2005 [79] in response to citizen's concerns regarding cancer statistics in Midlothian and Ellis County.

In November 1995, the Texas Cancer Registry (TCR) evaluated cancer mortality data for leukemia and all cancer sites combined in Midlothian, Texas, during the period 1984 through 1993 [8]. The Standardized Mortality Ratios (SMRs) for leukemia were: males, SMR=0.59 (95% CI, 0.02 to 3.32); females, SMR=2.77 (95% CI, 0.76 to 7.11). The SMRs for all sites combined were: males, SMR=1.27 (95% CI, 0.95 to 1.67); females, SMR=1.17 (95% CI, 0.84 to 1.59). These minor deviations from the rates for the State of Texas were appropriately described as "not statistically significant."

In February 1998, the TCR evaluated mortality data for cancers of the colon, pancreas, lung, trachea, prostate, breast, brain, and leukemia in Midlothian, Texas, zip code 76065, during the period 1990 through 1996 [9]. Lung and brain cancers in males were slightly higher than state rates while colon, pancreas, trachea, prostate, and leukemia were slightly lower than state rates. Similarly, colon, breast, brain, and leukemia in females were slightly higher than state rates, while pancreas, lung, and trachea were slightly lower than state rates. None of the deviations from State of Texas rates were statistically significant at the 95% level.

In June 1998, the TCR evaluated the mortality data for cancers of the liver and breast in Midlothian, Texas, zip code 76065 during the period 1990 through 1996 [11]. Liver and breast cancers were slightly lower than expected in males and slightly higher than expected in females. None of the deviations from State of Texas rates were statistically significant at the 95% level.

DSHS completed another cancer morbidity/mortality investigation in May 2005 [79]. Local residents were concerned that cement kiln dust, benzene, 1,3-butadiene, radiation, or other emissions from the nearby cement plants or other industry may be causing increased cancer rates in their community. A literature review revealed that increased risk for laryngeal cancer has been observed in workers exposed to cement kiln dust [80,81]. Likewise, an increased risk for acute myeloid leukemia and non-Hodgkin's lymphoma has been associated with exposure to benzene [17,18,19,20]. Similarly, an increased risk for leukemia has been associated with exposure to 1,3-butadiene [82]. Finally, increased risks for several leukemia subtypes, non-Hodgkin's lymphoma, and brain cancer have been weakly linked with radiation exposure [82]. To address these concerns, the TCR evaluated incidence data (1995 through 2002) and mortality data (1993 through 2002) for cancers of the female breast, prostate, lung and bronchus, colon and rectum, male bladder, corpus and uterus, non-Hodgkin's lymphoma, brain/central nervous system (brain/CNS), larynx, selected leukemia subtypes, and total childhood cancers. The investigation looked at the individual cancer incidence and mortality data for zip codes 76065, 75104, and 76084 (Midlothian, Cedar Hill, and Venus, TX, respectively) and concluded (using 99% confidence intervals) that prostate cancer mortality was statistically lower than expected in zip code 76065 and prostate cancer incidence was statistically lower than expected in zip code 76084 [79]. The incidence and mortality of the other cancer types were not significantly different than what would be expected when compared to the rest of the state.

## **Appendix E – Tables**



**TABLE 1a**

**Number of Measurements Made for Volatile Organic Compound Parameters by Monitoring Site and Year of Sample Collection, Midlothian, Texas, 1981 through 2005**

Year\Site	235 No 8th St City Hall/Fs roof	801 Auger Rd Midlothian	5050 Gorman Rd	440 Tayman Dr Water Treatment Plant	462 Waterworks Rd Mt Creek H2O Supp	491 Hidden Valley Trail	2060 South Hwy 67	Auger Rd Water Treatment Plant	2 Tar Road Box 485 Midlothian	4252 Waterworks Rd -- CAMS-94	2725 Old Fort Worth Rd -- CAMS-52	1241 East Wyatt Rd -- CAMS-302	1120A Cedar Dr	All Sites Combined Total
1981														-
1982														-
1983														-
1984														-
1985														-
1986														-
1987														-
1988														-
1989														-
1990														-
1991														-
1992														-
1993				1,193										1,193
1994				2,090										2,090
1995				5,197										5,197
1996				5,076										5,076
1997				1,274							3,978			5,252
1998											5,182			5,182
1999										2,853	4,510			7,363
2000										6,416	4,520			10,936
2001										1,680	4,872			6,552
2002										4,032	4,946			8,978
2003											4,993			4,993
2004										6,528	4,224	1,152		11,904
2005										1,447		1,447		2,894
Total All Years	-	-	-	14,830	-	-	-	-	-	22,956	37,225	2,599	-	77,610



**TABLE 1b**

**Number of Measurements Made for Metals and Other Inorganic Parameters by Monitoring Site and Year of Sample Collection, Midlothian, Texas, 1981 through 2005**

Metals and Other Inorganics	235 No 8th St City Hall/Fs roof	801 Auger Rd Midlothian	5050 Gorman Rd	440 Tayman Dr Water Treatment Plant	462 Waterworks Rd Mt Creek H2O Supp	491 Hidden Valley Trail	2060 South Hwy 67	Auger Rd Water Treatment Plant	2 Tar Road Box 485 Midlothian	4252 Waterworks Rd -- CAMS-94	2725 Old Fort Worth Rd -- CAMS-52	1241 East Wyatt Rd -- CAMS-302	1120A Cedar Dr	All Sites Combined
Year\Site	0001	0004	0006	0007	0008	0011	0012	0013	0014	0015	0016	0017	0084	Total
1981	1,320													1,320
1982	61													61
1983	113													113
1984	59													59
1985														-
1986														-
1987														-
1988														-
1989														-
1990														-
1991														-
1992														-
1993		5	17	56	17	47	49	49	8				61	309
1994				57			53	46	55	9	9		46	275
1995				59			61		3	61	61			245
1996				59			118			61	61			299
1997							120			61	59			240
1998							160			122	120			402
1999										120	120	7		247
2000										61	61	61		183
2001										61	61	1,381		1,503
2002										2,234	61	1,453		3,748
2003										3,241	61	1,357		4,659
2004										3,263	30	366		3,659
2005														-
Total All Years	1,553	5	17	231	17	47	561	95	66	9,294	704	4,625	107	17,322

**TABLE 2a**

**Volatile Organic Compounds Identified at Concentrations That are Either Below the Detection Limit or Below the Minimum Health-Based Screening Level, Midlothian, Texas, 1981 through 2005<sup>4</sup>**

Item No.	Contaminant Name	Number Samples	Midlothian Avg (ppb)	Midlothian Max (ppb)	Background Quotient	Min HAC (ppb)	Type of HAC
1	Acetaldehyde	179	-	-	N/A - NonDet	0.252	CREG
2	Acetone	260	-	-	N/A - NonDet	13,000	Chr I MRL
3	Acrolein	179	-	-	N/A - NonDet	0.00872	Chr I RfC
4	N-Amyl Alcohol	179	-	-	N/A - NonDet	-	-
5	Benzaldehyde	179	-	-	N/A - NonDet	80.6	Chr I RfD
6	Bromomethane	890	0.00150	0.190	0.0099	1.29	Chr I RfC
7	N-Butyl Alcohol	260	-	-	N/A - NonDet	115	Chr I RfD
<b>8</b>	<b>Chlorobenzene</b>	<b>952</b>	<b>0.0114</b>	<b>0.770</b>	<b>1.2081</b>	<b>4.34</b>	<b>p-Chr I RfC</b>
9	Chloromethane	189	0.583	1.25	0.9441	43.6	Chr I RfC
10	Chloroprene	553	0.0005	0.0005	0.0184	19.3	Chr I RfD
11	trans-Crotonaldehyde	179	-	-	N/A - NonDet	-	-
<b>12</b>	<b>Cyclohexane</b>	<b>858</b>	<b>0.0466</b>	<b>5.72</b>	<b>3.6266</b>	<b>1,743</b>	<b>Chr I RfC</b>
13	Dichlorodifluoromethane	189	0.494	0.990	0.8501	40.4	p-Chr I RfC
14	1,1-Dichloroethane	705	0.000778	0.0500	0.0661	124	p-Chr I RfC
15	1,1-Dichloroethene	893	0.00118	0.440	0.0622	20.0	Int I MRL
16	1,2-Dichloropropane	952	0.00134	0.440	0.0989	0.866	Chr I RfC
17	cis-1,3-Dichloropropene	407	0.000523	0.0100	0.0326	0.0551	CREG
18	trans-1,3-Dichloropropene	407	0.0005	0.0005	0.0331	0.0551	CREG
19	Ethyl Acetate	449	0.0005	0.0005	0.1000	874	Chr I RfD
20	Ethyl Alcohol	179	-	-	N/A - NonDet	-	-
21	Ethylbenzene	952	0.0601	8.18	0.4773	230	Chr I RfC
22	Heptanal	179	-	-	N/A - NonDet	-	-
23	3-Heptanone	44	0.0005	0.0005	N/A - NonDet	-	-
24	Heptene	27	0.0005	0.0005	N/A - NonDet	-	-
25	Hexaldehyde	179	-	-	N/A - NonDet	-	-
<b>26</b>	<b>N-Hexane</b>	<b>858</b>	<b>0.397</b>	<b>79.9</b>	<b>1.0690</b>	<b>199</b>	<b>Chr I RfC</b>
27	1-Hexanol	179	-	-	N/A - NonDet	-	-
28	Isopropylbenzene	858	0.00466	0.500	0.6842	81.4	Chr I RfC
29	Isovaleraldehyde	179	-	-	N/A - NonDet	-	-
30	Methyl Ethyl Ketone	449	0.0860	1.86	0.1215	1,695	Chr I RfC
31	Methyl Isoamyl Ketone	28	-	-	N/A - NonDet	-	-
32	Methyl Isobutyl Ketone	449	0.00283	0.270	0.2831	732	Chr I RfC
33	Methyl t-Butyl Ether	912	0.178	31.6	0.3225	700	Chr I MRL
<b>34</b>	<b>Methylcyclohexane</b>	<b>858</b>	<b>0.0704</b>	<b>11.7</b>	<b>1.3243</b>	<b>750</b>	<b>Chr I RfD</b>
35	3-Pentanone	173	0.0005	0.0005	0.1000	-	-

<sup>4</sup> Contaminants in bold indicate that average levels in Midlothian exceeded average background levels in Texas or the US.

**TABLE 2a (Cont)**

**Volatile Organic Compounds Identified at Concentrations That are Either Below the Detection Limit or Below the Minimum Health-Based Screening Level, Midlothian, Texas, 1981 through 2005<sup>5</sup>**

Item No.	Contaminant Name	Number Samples	Midlothian Avg (ppb)	Midlothian Max (ppb)	Background Quotient	Min HAC (ppb)	Type of HAC
36	2-Propanol	260	-	-	N/A - NonDet	-	-
37	Propionaldehyde	179	-	-	N/A - NonDet	-	-
38	N-Propyl Acetate	368	0.0005	0.0005	0.1000	-	-
39	N-Propyl Alcohol	179	-	-	N/A - NonDet	-	-
40	Styrene	950	0.0112	0.840	0.4241	60.0	Chr I MRL
41	Tetrachloroethylene	952	0.0129	0.440	0.1344	40.0	Chr I MRL
42	Toluene	952	0.417	40.3	0.5456	80.0	Chr I MRL
<b>43</b>	<b>1,1,1-Trichloroethane</b>	<b>952</b>	<b>0.0539</b>	<b>2.43</b>	<b>1.9971</b>	<b>700</b>	<b>Int I MRL</b>
44	Trichloroethylene	952	0.0108	1.12	0.5325	100	Int I MRL
45	Trichlorofluoromethane	952	0.285	5.85	0.9125	125	p-Chr I RfC
46	Valeraldehyde	179	-	-	N/A - NonDet	-	-
47	O-Xylene	952	0.0671	10.3	0.6394	23.0	Chr I RfC

<sup>5</sup> Contaminants in bold indicate that average levels in Midlothian exceeded average background levels in Texas or the US.

**TABLE 2b**

**Metals and Other Inorganic Compounds Identified at Concentrations That are Either Below the Detection Limit or Below the Minimum Health-Based Screening Level, Midlothian, Texas, 1981 - 2005<sup>6</sup>**

Item No.	Contaminant Name	Number Samples	Midlothian Avg (µg/m <sup>3</sup> )	Midlothian Max (µg/m <sup>3</sup> )	Background Quotient	Min HAC (µg/m <sup>3</sup> )	Type of HAC
1	Aluminum (PM <sub>10</sub> )	181	0.188	1.74	0.9545	5.00	p-Chr I RfC
2	Aluminum (PM <sub>2.5</sub> )	162	0.0411	1.24	0.0977	5.00	p-Chr I RfC
3	Aluminum (TSP)	40	0.649	2.19	0.7017	5.00	p-Chr I RfC
4	Ammonium Ion (PM <sub>2.5</sub> )	162	1.22	5.40	0.7689	73.8	Chr I MRL
5	Antimony (PM <sub>10</sub> )	181	0.00939	0.0210	0.7946	0.400	Int I RfC
6	Antimony (PM <sub>2.5</sub> )	162	0.00379	0.0254	0.8003	0.400	Int I RfC
7	Antimony (TSP)	40	0.0147	0.118	0.4555	0.400	Int I RfC
8	Barium (PM <sub>10</sub> )	181	0.0119	0.190	0.5418	0.500	p-Chr I RfC
9	Barium (PM <sub>2.5</sub> )	162	0.00437	0.0228	0.3727	0.500	p-Chr I RfC
10	Barium (TSP)	40	0.0153	0.164	0.2076	0.500	p-Chr I RfC
11	Calcium (PM <sub>10</sub> )	181	1.89	6.13	0.6132	500	p-Chr I RDA
12	Calcium (PM <sub>2.5</sub> )	162	0.124	0.567	0.0484	500	p-Chr I RDA
<b>13</b>	<b>Calcium (TSP)</b>	<b>40</b>	<b>11.9</b>	<b>24.03</b>	<b>1.9771</b>	<b>500</b>	<b>p-Chr I RDA</b>
14	Cerium (PM <sub>2.5</sub> )	162	0.00148	0.0305	0.1437	0.200	Chr I RfC
15	Chloride (TSP)	40	0.379	3.98	0.3758	1,700	p-Chr I RDA
16	Cobalt (PM <sub>10</sub> )	181	0.00104	0.00900	0.5261	0.0200	Chr I RfC
17	Cobalt (PM <sub>2.5</sub> )	162	0.000527	0.00475	0.5253	0.0200	Chr I RfC
18	Cobalt (TSP)	40	0.00491	0.0190	0.3224	0.0200	Chr I RfC
19	Copper (PM <sub>10</sub> )	181	0.0167	0.0890	0.4413	1.00	p-Chr I RDA
20	Copper (PM <sub>2.5</sub> )	162	0.00373	0.0273	0.2642	1.00	p-Chr I RDA
21	Copper (TSP)	40	0.0151	0.0380	0.4003	1.00	p-Chr I RDA
22	Iodide (TSP)	40	0.0117	0.0720	0.3081	0.0750	p-Chr I RDA
<b>23</b>	<b>Iron (PM<sub>10</sub>)</b>	<b>181</b>	<b>1.10</b>	<b>3.58</b>	<b>1.1183</b>	<b>9.00</b>	<b>p-Chr I RDA</b>
24	Iron (PM <sub>2.5</sub> )	162	0.0857	0.925	0.2621	9.00	p-Chr I RDA
25	Iron (TSP)	40	0.866	1.93	0.9756	9.00	p-Chr I RDA
26	Lead (PM <sub>10</sub> )	181	0.0155	0.0800	0.0940	0.375	p-Chr I RfC
27	Lead (PM <sub>2.5</sub> )	162	0.00328	0.0294	0.0574	0.375	p-Chr I RfC
28	Magnesium (PM <sub>10</sub> )	181	0.172	0.756	0.7035	200	p-Chr I RDA
29	Magnesium (PM <sub>2.5</sub> )	162	0.0139	0.208	0.5373	200	p-Chr I RDA
30	Manganese (PM <sub>2.5</sub> )	162	0.00157	0.0128	0.2396	0.0400	Chr I MRL
31	Mercury (PM <sub>2.5</sub> )	162	0.000797	0.00452	0.9010	0.200	Chr I MRL
32	Molybdenum (PM <sub>10</sub> )	181	0.00401	0.00900	0.8735	17.5	p-Chr I RfD
<b>33</b>	<b>Molybdenum (PM<sub>2.5</sub>)</b>	<b>162</b>	<b>0.00119</b>	<b>0.00681</b>	<b>1.0381</b>	<b>17.5</b>	<b>p-Chr I RfD</b>
34	Molybdenum (TSP)	40	0.000825	0.00900	0.1628	17.5	p-Chr I RfD
35	Nickel (PM <sub>10</sub> )	181	0.00404	0.0100	0.9018	0.0900	Chr I MRL
36	Nickel (PM <sub>2.5</sub> )	162	0.000624	0.00394	0.1469	0.0900	Chr I MRL
37	Nickel (TSP)	40	0.00253	0.0160	0.2362	0.0900	Chr I MRL

<sup>6</sup> Contaminants in bold indicate that average levels in Midlothian exceeded average background levels in Texas or the US.

**TABLE 2b (Cont)**

**Metals and Other Inorganic Compounds Identified at Concentrations That are Either Below the Detection Limit or Below the Minimum Health-Based Screening Level, Midlothian, Texas, 1981 - 2005<sup>7</sup>**

Item No.	Contaminant Name	Number Samples	Midlothian Avg (µg/m <sup>3</sup> )	Midlothian Max (µg/m <sup>3</sup> )	Background Quotient	Min HAC (µg/m <sup>3</sup> )	Type of HAC
38	Phosphorus (PM <sub>2.5</sub> )	162	0.00347	0.114	0.1097	0.0700	p-Chr I RfD
<b>39</b>	<b>Phosphorus (TSP)</b>	<b>40</b>	<b>0.104</b>	<b>0.266</b>	<b>1.0140</b>	<b>0.0700</b>	<b>p-Chr I RfD</b>
40	Potassium (PM <sub>10</sub> )	181	0.166	0.604	0.4053	1,750	p-Chr I RDA
41	Potassium (PM <sub>2.5</sub> )	162	0.0717	0.525	0.3891	1,750	p-Chr I RDA
42	Potassium (TSP)	40	0.420	0.940	0.9136	1,750	p-Chr I RDA
43	Potassium Ion (PM <sub>2.5</sub> )	162	0.0366	0.285	0.2131	1,750	p-Chr I RDA
<b>44</b>	<b>Selenium (PM<sub>10</sub>)</b>	<b>181</b>	<b>0.0130</b>	<b>0.0150</b>	<b>1.4387</b>	<b>17.5</b>	<b>p-Chr I RfD</b>
45	Selenium (PM <sub>2.5</sub> )	162	0.000906	0.00320	0.7780	17.5	p-Chr I RfD
46	Selenium (TSP)	40	0.00241	0.0170	0.1595	17.5	p-Chr I RfD
47	Silver (PM <sub>10</sub> )	181	0.00313	0.00800	0.8654	17.5	p-Chr I RfD
48	Silver (PM <sub>2.5</sub> )	162	0.00170	0.0113	0.7195	17.5	p-Chr I RfD
<b>49</b>	<b>Sodium (PM<sub>10</sub>)</b>	<b>181</b>	<b>1.01</b>	<b>3.71</b>	<b>1.0056</b>	<b>35.0</b>	<b>p-Chr I RDA</b>
50	Sodium (PM <sub>2.5</sub> )	162	0.0672	0.718	0.3985	35.0	p-Chr I RDA
<b>51</b>	<b>Sodium Ion (PM<sub>2.5</sub>)</b>	<b>162</b>	<b>0.114</b>	<b>0.857</b>	<b>1.0045</b>	<b>17.5</b>	<b>p-Chr I RfD</b>
52	Strontium (PM <sub>2.5</sub> )	162	0.00127	0.00709	0.3697	2100	p-Chr I RfD
<b>53</b>	<b>Strontium (TSP)</b>	<b>40</b>	<b>0.0156</b>	<b>0.0400</b>	<b>1.1912</b>	<b>2100</b>	<b>p-Chr I RfD</b>
<b>54</b>	<b>Thallium (PM<sub>10</sub>)</b>	<b>181</b>	<b>0.0186</b>	<b>0.185</b>	<b>1.3272</b>	<b>0.280</b>	<b>p-Chr I RfD</b>
55	Thallium (TSP)	40	0.00464	0.0370	0.1545	0.280	p-Chr I RfD
<b>56</b>	<b>Tin (PM<sub>10</sub>)</b>	<b>181</b>	<b>0.00802</b>	<b>0.00900</b>	<b>1.2292</b>	<b>2,190</b>	<b>Chr I RBC</b>
57	Tin (PM <sub>2.5</sub> )	162	0.00396	0.0193	0.7821	2,190	Chr I RBC
58	Tin (TSP)	40	0.0209	0.124	0.6205	2,190	Chr I RBC
59	Titanium (PM <sub>2.5</sub> )	162	0.00655	0.106	0.2167	30.1	p-Chr I RfD
60	Titanium (TSP)	40	0.0562	0.186	0.8287	30.1	p-Chr I RfD
61	Vanadium (PM <sub>10</sub> )	181	0.00171	0.00600	0.4032	0.200	Acu I MRL
62	Vanadium (PM <sub>2.5</sub> )	162	0.00119	0.00644	0.5501	0.200	Acu I MRL
63	Vanadium (TSP)	40	0.00223	0.00700	0.6235	0.200	Acu I MRL
<b>64</b>	<b>Zinc (PM<sub>10</sub>)</b>	<b>181</b>	<b>0.101</b>	<b>0.870</b>	<b>2.0128</b>	<b>1,050</b>	<b>p-Chr I RfD</b>
65	Zinc (PM <sub>2.5</sub> )	162	0.0164	0.193	0.7912	1,050	p-Chr I RfD
66	Zinc (TSP)	40	0.0566	0.281	0.2433	1,050	p-Chr I RfD

<sup>7</sup> Contaminants in bold indicate that average levels in Midlothian exceeded average background levels in Texas or the US.

**TABLE 3a**

**Volatile Organic Compounds Identified for which There are No Published Health-Based Screening Levels, Midlothian, Texas, 1981 through 2005<sup>8</sup>**

Item No.	Contaminant Name	CAS No	Number Samples	Midlothian Avg (ppb)	Midlothian Max (ppb)	Background Quotient
1	Acetylene	74-86-2	720	0.593	6.63	0.5917
2	N-Butane	106-97-8	858	1.35	44.5	0.2611
3	1-Butene	106-98-9	858	0.206	2.01	0.1338
4	cis-2-Butene	590-18-1	858	0.0140	2.60	0.0419
5	trans-2-Butene	624-64-6	858	0.0165	3.09	0.0404
6	Butyl Acetate	123-86-4	449	0.000883	0.0400	0.1766
7	Butyraldehyde	123-72-8	352	0.0554	2.21	0.4260
<b>8</b>	<b>2-Chloropentane</b>	<b>625-29-6</b>	<b>885</b>	<b>0.00618</b>	<b>0.890</b>	<b>1.2363</b>
9	Cyclopentane	287-92-3	858	0.0312	3.48	0.1736
<b>10</b>	<b>Cyclopentene</b>	<b>142-29-0</b>	<b>642</b>	<b>0.00599</b>	<b>1.65</b>	<b>1.1389</b>
11	N-Decane	124-18-5	858	0.0141	2.88	0.0779
12	M-Diethylbenzene	141-93-5	734	0.00420	0.720	0.1442
13	P-Diethylbenzene	105-05-5	734	0.00769	1.25	0.0601
14	2,2-Dimethylbutane	75-83-2	858	0.0152	1.43	0.3006
15	2,3-Dimethylbutane	79-29-8	858	0.0410	3.09	0.3810
<b>16</b>	<b>2,3-Dimethylpentane</b>	<b>565-59-3</b>	<b>858</b>	<b>0.0329</b>	<b>3.18</b>	<b>4.5898</b>
<b>17</b>	<b>2,4-Dimethylpentane</b>	<b>108-08-7</b>	<b>858</b>	<b>0.0170</b>	<b>1.31</b>	<b>3.2302</b>
18	Ethane	74-84-0	720	4.94	21.6	0.6622
<b>19</b>	<b>Ethylene</b>	<b>74-85-1</b>	<b>720</b>	<b>0.758</b>	<b>3.93</b>	<b>1.0684</b>
<b>20</b>	<b>M-Ethyltoluene</b>	<b>620-14-4</b>	<b>734</b>	<b>0.0282</b>	<b>4.63</b>	<b>4.6074</b>
<b>21</b>	<b>O-Ethyltoluene</b>	<b>611-14-3</b>	<b>734</b>	<b>0.0109</b>	<b>1.57</b>	<b>1.7869</b>
<b>22</b>	<b>P-Ethyltoluene</b>	<b>622-96-8</b>	<b>734</b>	<b>0.0184</b>	<b>2.75</b>	<b>2.7657</b>
<b>23</b>	<b>N-Heptane</b>	<b>142-82-5</b>	<b>858</b>	<b>0.0558</b>	<b>7.15</b>	<b>1.9829</b>
24	3-Hexanone	589-38-8	189	0.000562	0.0100	0.1123
25	1-Hexene + 2-Methyl-1-Pentene	592-41-6 + 763-29-1	407	0.000572	0.0300	0.0044
26	cis-2-Hexene	7688-21-3	807	0.00346	0.750	0.3671
27	trans-2-Hexene	4050-45-7	858	0.00695	1.66	0.4646
28	Isobutane	75-28-5	858	0.736	9.05	0.2301
29	Isobutyraldehyde	78-84-2	189	0.0480	3.67	0.8334
30	Isopentane	78-78-4	858	0.836	45.7	0.1737
31	Isoprene	78-79-5	858	0.0203	1.51	0.1928
32	3-Methyl-1-Butene	563-45-1	807	0.00328	0.560	0.6557
33	2-Methyl-1-Pentene	763-29-1	450	0.0166	1.35	0.3405
34	4-Methyl-1-Pentene	691-37-2	858	0.00262	0.650	0.5235
35	2-Methyl-2-Butene	513-35-9	858	0.0413	8.53	0.2024

<sup>8</sup> Contaminants in bold indicate that average levels in Midlothian exceeded average background levels in Texas or the US.

**TABLE 3a (Cont)**

**Volatile Organic Compounds Identified for which There are No Published Health-Based Screening Levels, Midlothian, Texas, 1981 through 2005<sup>9</sup>**

Item No.	Contaminant Name	CAS No	Number Samples	Midlothian Avg (ppb)	Midlothian Max (ppb)	Background Quotient
36	2-Methyl-3-Hexanone	7379-12-6	189	0.00139	0.0500	0.2779
37	Methyl Butyl Ketone	591-78-6	189	0.00139	0.0600	0.1393
38	Methylcyclopentane	96-37-7	858	0.142	29.4	0.2682
<b>39</b>	<b>2-Methylheptane</b>	<b>592-27-8</b>	<b>858</b>	<b>0.00972</b>	<b>1.17</b>	<b>1.9441</b>
<b>40</b>	<b>3-Methylheptane</b>	<b>589-81-1</b>	<b>858</b>	<b>0.00686</b>	<b>1.18</b>	<b>1.3719</b>
41	2-Methylhexane	591-76-4	858	0.0776	8.73	0.7197
<b>42</b>	<b>3-Methylhexane</b>	<b>589-34-4</b>	<b>858</b>	<b>0.0974</b>	<b>10.5</b>	<b>1.0268</b>
43	2-Methylpentane	107-83-5	858	0.245	14.7	0.0584
44	3-Methylpentane	96-14-0	858	0.191	26.2	0.2457
45	N-Nonane	111-84-2	858	0.00541	0.760	0.0351
46	N-Octane	111-65-9	858	0.0107	1.81	0.0894
47	N-Pentane	109-66-0	858	0.520	31.2	0.3693
48	1-Pentene	109-67-1	858	0.0275	3.21	0.1577
49	cis-2-Pentene	627-20-3	858	0.0192	3.64	0.0342
50	trans-2-Pentene	646-04-8	858	0.0364	6.95	0.2959
51	Alpha-Pinene	80-56-8	124	0.0106	1.02	0.0722
52	Beta-Pinene	127-91-3	124	0.00396	0.0700	0.5546
53	Propane	74-98-6	858	3.36	54.3	0.7551
<b>54</b>	<b>N-Propylbenzene</b>	<b>103-65-1</b>	<b>858</b>	<b>0.0116</b>	<b>1.36</b>	<b>1.6582</b>
55	Propylene	115-07-1	858	0.485	7.30	0.3879
<b>56</b>	<b>1,2,3-Trimethylbenzene</b>	<b>526-73-8</b>	<b>734</b>	<b>0.00980</b>	<b>1.21</b>	<b>1.7494</b>
57	2,2,4-Trimethylpentane	540-84-1	831	0.0421	4.13	0.0979
<b>58</b>	<b>2,3,4-Trimethylpentane</b>	<b>565-75-3</b>	<b>858</b>	<b>0.0123</b>	<b>1.43</b>	<b>2.4174</b>
<b>59</b>	<b>N-Undecane</b>	<b>1120-21-4</b>	<b>858</b>	<b>0.0164</b>	<b>4.72</b>	<b>2.1606</b>

<sup>9</sup> Contaminants in bold indicate that average levels in Midlothian exceeded average background levels in Texas or the US.

**TABLE 3b**

**Metals and Other Inorganic Compounds Identified for which There are No Published Health-Based Screening Levels, Midlothian, Texas, 1981 through 2005<sup>10</sup>**

Item No.	Contaminant Name	CAS No	Number Samples	Midlothian Avg (µg/m <sup>3</sup> )	Midlothian Max (µg/m <sup>3</sup> )	Background Quotient
1	Bromide (TSP)	24959-67-9	40	0.0460	0.118	0.6735
2	Bromine (PM <sub>2.5</sub> )	7726-95-6	162	0.00462	0.0481	0.2115
3	Cesium (PM <sub>2.5</sub> )	7440-46-2	162	0.00242	0.0183	0.4669
4	Europium (PM <sub>2.5</sub> )	7440-53-1	162	0.00502	0.0719	0.6146
5	Gallium (PM <sub>2.5</sub> )	7440-55-3	162	0.000523	0.00335	0.4911
6	Germanium (TSP)	7440-56-4	40	0.00375	0.0190	0.1500
7	Gold (PM <sub>2.5</sub> )	7440-57-5	162	0.000954	0.00386	0.6830
8	Hafnium (PM <sub>2.5</sub> )	7440-58-6	162	0.00266	0.0277	0.7642
9	Indium (PM <sub>2.5</sub> )	7440-74-6	162	0.00223	0.0130	0.8994
10	Iridium (PM <sub>2.5</sub> )	7439-88-5	162	0.000691	0.00411	0.4622
11	Lanthanum (PM <sub>2.5</sub> )	7439-91-0	162	0.00147	0.0109	0.1273
12	Lanthanum (TSP)	7439-91-0	40	0.0314	0.209	0.1689
13	Niobium (PM <sub>2.5</sub> )	7440-03-1	162	0.000623	0.00599	0.6486
14	Rubidium (PM <sub>2.5</sub> )	7440-17-7	162	0.000509	0.00132	0.5909
15	Rubidium (TSP)	7440-17-7	40	0.00260	0.0140	0.1728
16	Samarium (PM <sub>2.5</sub> )	7440-19-9	162	0.00185	0.107	0.3659
<b>17</b>	<b>Scandium (PM<sub>2.5</sub>)</b>	<b>7440-20-2</b>	<b>162</b>	<b>0.000623</b>	<b>0.00650</b>	<b>1.5072</b>
18	Silicon (PM <sub>2.5</sub> )	7440-21-3	162	0.205	3.13	0.1477
19	Silicon (TSP)	7440-21-3	40	3.47	7.80	0.8551
20	Sulfate (PM <sub>2.5</sub> )	14808-79-8	162	3.44	18.3	0.8249
21	Sulfur (PM <sub>2.5</sub> )	7704-34-9	162	1.17	6.60	0.7012
<b>22</b>	<b>Sulfur (TSP)</b>	<b>7704-34-9</b>	<b>40</b>	<b>3.38</b>	<b>10.4</b>	<b>1.1496</b>
23	Tantalum (PM <sub>2.5</sub> )	7440-25-7	162	0.00152	0.0107	0.3219
24	Terbium (PM <sub>2.5</sub> )	7440-27-9	162	0.00232	0.0795	0.2336
25	Tungsten (PM <sub>2.5</sub> )	7440-33-7	162	0.00141	0.00622	0.5298
26	Yttrium (PM <sub>2.5</sub> )	7440-65-5	162	0.000527	0.00214	0.8469
27	Zirconium (PM <sub>2.5</sub> )	7440-67-7	162	0.000711	0.00491	0.4765
28	Zirconium (TSP)	7440-67-7	40	0.00200	0.0160	0.2987

<sup>10</sup> Contaminants in bold indicate that average levels in Midlothian exceeded average background levels in Texas or the US.

**TABLE 4a**

**Volatile Organic Compounds Identified as Having One or More Measurements at or Above the Minimum Health-Based Screening Level, Midlothian, Texas, 1981 through 2005**

Item No.	Contaminant Name	CAS No	Number Samples	Midlothian Avg (ppb)	Midlothian Max (ppb)	Background Quotient	Min HAC (ppb)	Type of HAC	#>=Min HAC
1	Benzene	71-43-2	952	0.308	20.6	0.7097	0.0401	CREG	926
2	1,3-Butadiene	106-99-0	952	0.00561	0.340	0.0359	0.0151	CREG	66
3	Carbon Tetrachloride	56-23-5	952	0.0907	4.27	0.9522	0.0106	CREG	711
4	Chloroform	67-66-3	952	0.00567	0.260	0.1667	0.00890	CREG	210
5	1,2-Dibromoethane	106-93-4	663	0.000840	0.120	0.0435	0.000217	CREG	407
6	1,2-Dichloroethane	107-06-2	952	0.00631	0.460	0.2835	0.00950	CREG	87
7	Methylene Chloride	75-09-2	952	0.0304	1.58	0.1987	0.613	CREG	3
8	1,1,2,2-Tetrachloroethane	79-34-5	407	0.000914	0.150	0.0469	0.00251	CREG	3
9	1,1,2-Trichloroethane	79-00-5	831	0.000681	0.150	0.0320	0.0115	CREG	1
10	1,2,4-Trimethylbenzene	95-63-6	858	0.0498	7.33	0.3994	1.22	p-Chr I RfC	5
11	1,3,5-Trimethylbenzene	108-67-8	858	0.0154	2.03	0.3843	1.22	p-Chr I RfC	2
12	Vinyl Chloride	75-01-4	952	0.00126	0.120	0.0898	0.0445	CREG	7
13	M+P-Xylene	108-38-3 + 106-42-3	912	0.178	32.1	0.4797	23.0	Chr I RfC	1

**TABLE 4b**

**Metals and Other Inorganic Compounds Identified as Having One or More Measurements at or Above the Minimum Health-Based Screening Level, Midlothian, Texas, 1981 through 2005<sup>11</sup>**

Item No.	Contaminant Name	CAS No	Number Samples	Midlothian Avg (µg/m <sup>3</sup> )	Midlothian Max (µg/m <sup>3</sup> )	Background Quotient	Min HAC (µg/m <sup>3</sup> )	Type of HAC	#>=Min HAC
<b>1</b>	<b>Arsenic (PM<sub>10</sub>)</b>	<b>7440-38-2</b>	<b>181</b>	<b>0.0116</b>	<b>0.0120</b>	<b>1.9369</b>	<b>0.000233</b>	<b>CREG</b>	<b>181</b>
2	Arsenic (PM <sub>2.5</sub> )	7440-38-2	162	0.000972	0.00982	0.9680	0.000233	CREG	157
3	Arsenic (TSP)	7440-38-2	40	0.0181	0.0580	0.3413	0.000233	CREG	40
<b>4</b>	<b>Beryllium (PM<sub>10</sub>)</b>	<b>7440-41-7</b>	<b>181</b>	<b>0.0005</b>	<b>0.0005</b>	<b>1.6667</b>	<b>0.000417</b>	<b>CREG</b>	<b>181</b>
<b>5</b>	<b>Cadmium (PM<sub>10</sub>)</b>	<b>7440-43-9</b>	<b>181</b>	<b>0.00103</b>	<b>0.00400</b>	<b>1.2482</b>	<b>0.000556</b>	<b>CREG</b>	<b>181</b>
6	Cadmium (PM <sub>2.5</sub> )	7440-43-9	162	0.00147	0.00920	0.4512	0.000556	CREG	57
7	Cadmium (TSP)	7440-43-9	40	0.0298	0.129	0.6602	0.000556	CREG	27
8	Chlorine (PM <sub>2.5</sub> )	7782-50-5	162	0.0110	0.407	0.1950	0.232	p-Chr I RfC	2
<b>9</b>	<b>Chromium (PM<sub>10</sub>)</b>	<b>7440-47-3</b>	<b>181</b>	<b>0.00520</b>	<b>0.0250</b>	<b>1.2440</b>	<b>0.0000833</b>	<b>CREG</b>	<b>181</b>
10	Chromium (PM <sub>2.5</sub> )	7440-47-3	162	0.00110	0.0287	0.9609	0.0000833	CREG	157
11	Chromium (TSP)	7440-47-3	40	0.00423	0.0270	0.1545	0.0000833	CREG	40
<b>12</b>	<b>Lead (TSP)</b>	<b>7439-92-1</b>	<b>413</b>	<b>0.199</b>	<b>1.51</b>	<b>2.9589</b>	<b>0.375</b>	<b>p-Chr I RfC</b>	<b>65</b>
<b>13</b>	<b>Manganese (PM<sub>10</sub>)</b>	<b>7439-96-5</b>	<b>181</b>	<b>0.0413</b>	<b>0.171</b>	<b>1.5164</b>	<b>0.0400</b>	<b>Chr I MRL</b>	<b>71</b>
<b>14</b>	<b>Manganese (TSP)</b>	<b>7439-96-5</b>	<b>40</b>	<b>0.0407</b>	<b>0.0760</b>	<b>1.5566</b>	<b>0.0400</b>	<b>Chr I MRL</b>	<b>20</b>

<sup>11</sup> Contaminants in bold indicate that average levels in Midlothian exceeded average background levels in Texas or the US.

**TABLE 5a**

**Summary of Hazard Quotients for Chronic or Sub-Chronic Exposures to each of the VOCs that Had One or More Measurement That Exceeded the Initial Screening HAC Value, Midlothian, Texas, 1981 through 2005**

Item No.	Contaminant Name	MC 95% UCL on Avg (ppb)	Minimum Non-Ca HAC (ppb)	Type HAC (Non-Ca)	Hazard Quotient for 95% UCL	Margin of Safety
1	Benzene	0.364	3.00	Chr I MRL	1.21E-01	8.24
2	1,3-Butadiene	0.00703	0.904	Chr I RfC	7.77E-03	129
3	Carbon Tetrachloride	0.102	30.0	Chr I MRL	3.41E-03	293
4	Chloroform	0.00657	20.0	Chr I MRL	3.29E-04	3,043
5	1,2-Dibromoethane	0.00138	1.21	Chr I RfC	1.14E-03	880
6	1,2-Dichloroethane	0.00805	600	Chr I MRL	1.34E-05	74,508
7	Methylene Chloride	0.0351	300	Chr I MRL	1.17E-04	8,541
8	1,1,2,2-Tetrachloroethane	0.00158	18.6	Chr I MRL	8.52E-05	11,742
9	1,1,2-Trichloroethane	0.00101	2.57	p-Chr I RfD	3.93E-04	2,541
10	1,2,4-Trimethylbenzene	0.0709	1.22	p-Chr I RfC	5.81E-02	17.2
11	1,3,5-Trimethylbenzene	0.0215	1.22	p-Chr I RfC	1.76E-02	56.7
12	Vinyl Chloride	0.00171	30.0	Int I MRL	5.71E-05	17,501
13	M+P-Xylene	0.263	23.0	Chr I RfC	1.14E-02	87.6

**TABLE 5b**

**Summary of Hazard Quotients for Chronic Exposures to each of the Metals and Other Inorganic Compounds that Had One or More Measurement That Exceeded the Initial Screening HAC Value, Midlothian, Texas, 1981 through 2005<sup>12</sup>**

Item No.	Contaminant Name	MC 95% UCL on Avg ( $\mu\text{g}/\text{m}^3$ )	Minimum Non-Ca HAC ( $\mu\text{g}/\text{m}^3$ )	Type HAC (Non-Ca)	Hazard Quotient for 95% UCL	Margin of Safety
1	Arsenic (PM <sub>10</sub> )	0.0116	1.05	p-Chr I RfD	1.11E-02	90.4
2	Arsenic (TSP)	0.0216	1.05	p-Chr I RfD	2.06E-02	48.5
3	Arsenic (PM <sub>2.5</sub> )	0.00111	1.05	p-Chr I RfD	1.06E-03	946
4	Beryllium (PM <sub>10</sub> )	0.0005	0.0200	Chr I RfC	2.50E-02	40.0
5	Cadmium (PM <sub>10</sub> )	0.00106	1.75	p-Chr I RfD	6.05E-04	1,652
6	Cadmium (TSP)	0.0299	1.75	p-Chr I RfD	1.71E-02	58.6
7	Cadmium (PM <sub>2.5</sub> )	0.00166	1.75	p-Chr I RfD	9.50E-04	1,052
8	Chlorine (PM <sub>2.5</sub> )	0.0113	0.200	p-Chr I RfC	4.89E-02	20.5
9	Chromium (PM <sub>10</sub> )	0.00566	0.100	Chr I RfC	5.66E-02	17.7
10	Chromium (TSP)	0.00577	0.100	Chr I RfC	5.77E-02	17.3
11	Chromium (PM <sub>2.5</sub> )	0.00140	0.100	Chr I RfC	1.40E-02	71.6
12	Lead (TSP)	0.217	0.375	p-Chr I RfC	5.78E-01	1.73
<b>13</b>	<b>Manganese (PM<sub>10</sub>)</b>	<b>0.0454</b>	<b>0.0400</b>	<b>Chr I MRL</b>	<b>1.13E+00</b>	<b>0.882</b>
<b>14</b>	<b>Manganese (TSP)</b>	<b>0.0446</b>	<b>0.0400</b>	<b>Chr I MRL</b>	<b>1.12E+00</b>	<b>0.897</b>

<sup>12</sup> Items in bold indicate contaminants for which the hazard quotient for long-term exposure exceeds 1.0 (i.e., contaminants for which the 95% UCL of the arithmetic mean exposure level exceeds the minimum long-term health-based comparison value).

**TABLE 6a**

**Summary of Cancer Risk Estimates and Qualitative Cancer Risk Statements for Lifetime Exposures to each of the VOCs that Had One or More Measurement That Exceeded the Initial Screening HAC Value, Midlothian, Texas, 1981 through 2005**

Item No.	Contaminant Name	MC 95% UCL on Avg ( $\mu\text{g}/\text{m}^3$ )	Inhalation Unit Risk ( $\mu\text{g}/\text{m}^3$ ) <sup>-1</sup>	Type HAC (Cancer)	Ca Risk Estimates for 95% UCL	Ca Risk Expressed as 1 in:	Qualitative Cancer Risk Statement
1	Benzene	1.16	7.80E-06	CREG	9.08E-06	110,170	No Apparent Increased Lifetime Risk
2	1,3-Butadiene	0.0155	3.00E-05	CREG	4.66E-07	2,144,299	No Increased Lifetime Risk
3	Carbon Tetrachloride	0.644	1.50E-05	CREG	9.66E-06	103,548	No Apparent Increased Lifetime Risk
4	Chloroform	0.0321	2.30E-05	CREG	7.38E-07	1,354,716	No Increased Lifetime Risk
5	1,2-Dibromoethane	0.0106	6.00E-04	CREG	6.36E-06	157,237	No Apparent Increased Lifetime Risk
6	1,2-Dichloroethane	0.0326	2.60E-05	CREG	8.47E-07	1,180,057	No Increased Lifetime Risk
7	Methylene Chloride	0.122	4.70E-07	CREG	5.73E-08	17,438,716	No Increased Lifetime Risk
8	1,1,2,2-Tetrachloroethane	0.0109	5.80E-05	CREG	6.31E-07	1,585,457	No Increased Lifetime Risk
9	1,1,2-Trichloroethane	0.00551	1.60E-05	CREG	8.81E-08	11,345,339	No Increased Lifetime Risk
10	1,2,4-Trimethylbenzene	0.349	N/A	N/A	N/A	N/A	N/A - Non-Carcinogenic
11	1,3,5-Trimethylbenzene	0.106	N/A	N/A	N/A	N/A	N/A - Non-Carcinogenic
12	Vinyl Chloride	0.00438	8.80E-06	CREG	5.06E-08	19,751,644	No Increased Lifetime Risk
13	M+P-Xylene	1.14	N/A	N/A	N/A	N/A	N/A - Non-Carcinogenic

**TABLE 6b**

**Cancer Risk Estimates and Qualitative Cancer Risk Statements for Lifetime Exposures to each of the Metals and Other Inorganic Compounds that Had One or More Measurement That Exceeded the Initial Screening HAC Value, Midlothian, Texas, 1981 through 2005**

Item No.	Contaminant Name	MC 95% UCL on Avg (µg/m3)	Inhalation Unit Risk (µg/m3)-1	Type HAC (Cancer)	Ca Risk Estimates for 95% UCL	Ca Risk Expressed as 1 in:	Qualitative Cancer Risk Statement
1	Arsenic (PM <sub>10</sub> )	0.0116	4.30E-03	CREG	4.99E-05	20,025	Low Increased Lifetime Risk
2	Arsenic (TSP)	0.0216	4.30E-03	CREG	9.30E-05	10,748	Low Increased Lifetime Risk
3	Arsenic (PM <sub>2.5</sub> )	0.00111	4.30E-03	CREG	4.77E-06	209,520	No Apparent Increased Lifetime Risk
4	Beryllium (PM <sub>10</sub> )	0.0005	2.40E-03	CREG	1.20E-06	833,333	No Apparent Increased Lifetime Risk
5	Cadmium (PM <sub>10</sub> )	0.00106	1.80E-03	CREG	1.91E-06	524,559	No Apparent Increased Lifetime Risk
6	Cadmium (TSP)	0.0299	1.80E-03	CREG	5.38E-05	18,597	Low Increased Lifetime Risk
7	Cadmium (PM <sub>2.5</sub> )	0.00166	1.80E-03	CREG	2.99E-06	334,114	No Apparent Increased Lifetime Risk
8	Chlorine (PM <sub>2.5</sub> )	0.0113	N/A	N/A	N/A	N/A	N/A - Non-Carcinogenic
9	Chromium (PM <sub>10</sub> )	0.00566	1.20E-02	CREG	6.80E-05	14,714	Low Increased Lifetime Risk
10	Chromium (TSP)	0.00577	1.20E-02	CREG	6.93E-05	14,436	Low Increased Lifetime Risk
11	Chromium (PM <sub>2.5</sub> )	0.00140	1.20E-02	CREG	1.68E-05	59,689	Low Increased Lifetime Risk
12	Lead (TSP)	0.217	N/A	N/A	N/A	N/A	N/A - Non-Carcinogenic
13	Manganese (PM <sub>10</sub> )	0.0454	N/A	N/A	N/A	N/A	N/A - Non-Carcinogenic
14	Manganese (TSP)	0.0446	N/A	N/A	N/A	N/A	N/A - Non-Carcinogenic

**TABLE 7a**

**Cumulative Non-Cancer Hazard Index from Aggregate Exposures to All 99 Substances Identified for which HAC Values Have Been Established by the ATSDR or the EPA, Midlothian, Texas, 1981 through 2005<sup>13</sup>**

<b>Item No.</b>	<b>Non-Cancer Critical Effect</b>	<b>Cumulative Hazard Index from Aggregate Exposures</b>	<b>Margin of Safety over the Most Conservative Reported HAC Value</b>
<b>1</b>	<b>CNS/Neurologic</b>	<b>1.18</b>	<b>0.848</b>
2	Blood	0.699	1.43
3	Neurobehavioral	0.578	1.73
4	Lung	0.273	3.66
5	Immune System	0.146	6.83
6	Liver	0.0887	11.3
7	Kidney	0.0285	35.0
8	Nasal	0.0230	43.5
9	Hair	0.0160	62.4
10	Weight Loss	0.0126	79.5
11	Skin	0.0120	83.3
12	Vascular	0.0111	90.4
13	Ovarian	0.00777	129
14	Parturition	0.00599	167
15	Survival	0.00239	419
16	Selenosis	0.000747	1,339
17	Developmental	0.000425	2,352
18	Serum Chemistries	0.000393	2,541
19	Increased Uric Acid	0.000232	4,302
20	Eye	0.000199	5,026
21	Offspring BW	0.0000368	27,195
22	Bone	0.00000867	115,385

<sup>13</sup> Items in bold indicate critical effects for which the cumulative hazard index for aggregate exposures exceeds 1.0.

**TABLE 7b**

**Cumulative Cancer Risk from Aggregate Exposures to All 22  
 Substances Identified that are Classified as Known or Probable  
 Human Carcinogens, Midlothian, Texas, 1981 through 2005<sup>14</sup>**

<b>Item No.</b>	<b>Cancer Critical Effect</b>	<b>Cumulative Cancer Risk from Aggregate Exposures</b>	<b>Cumulative Cancer Risk Expressed as 1 Excess Ca in n,nnn Population Exposed</b>	<b>Qualitative Cancer Risk Statement</b>
<b>1</b>	<b>Total Cancer</b>	<b>1.49E-04</b>	<b>6,712</b>	<b>Moderate Increased Lifetime Risk</b>
2	Lung Cancer	9.60E-05	10,412	Low Increased Lifetime Risk
3	Skin Cancer	2.50E-05	40,049	Low Increased Lifetime Risk
4	Liver Cancer	1.12E-05	89,153	Low Increased Lifetime Risk
5	Leukemia	9.54E-06	104,787	No Apparent Increased Lifetime Risk
6	Vascular Cancer	2.12E-06	471,836	No Apparent Increased Lifetime Risk
7	Mesothelioma	1.27E-06	786,185	No Apparent Increased Lifetime Risk
8	Nasal Cancer	1.27E-06	786,185	No Apparent Increased Lifetime Risk
9	Stomach Cancer	1.27E-06	786,185	No Apparent Increased Lifetime Risk
10	Thyroid Cancer	1.27E-06	786,185	No Apparent Increased Lifetime Risk
11	Bladder Cancer	6.44E-09	155,195,998	No Increased Lifetime Risk

<sup>14</sup> Items in bold indicate cancer sites for which the cumulative cancer risk estimate for aggregate exposures exceeds 1.00E-04 (i.e., a total of 1 excess cancer case per 10,000 population exposed for 70 years).

## **Appendix F – Monte Carlo Methodology**

### ***Monte Carlo Methodology***

The term *Monte Carlo simulation* refers to a process in which a series of random numbers is drawn from a random number table (or generated by computer) and used to select values from a probability density function. The technique often is used to simulate natural processes that would be impractical if not impossible to observe and for which exact solutions are mathematically difficult. Although the simulation results only approximate the underlying values and the numbers vary slightly from run to run, accuracy may be improved by conducting a large enough number of trials.

In this application, historically measured contaminant levels were used to define the shape of the probability density functions to be used in the simulations. Frequency distributions of measured values were obtained for each of the contaminants with one or more measured values that exceeded the most conservative HAC value. Each frequency distribution was used to generate a contaminant-specific cumulative probability distribution, taking on values between 0 and 1 and having anywhere from 1 to 144 different elements depending on the number of different values observed in the original data set. The cumulative distributions, paired with the corresponding frequency distribution values, formed a “lookup” table which was then sampled using the built-in RAND function of Microsoft® Excel 2003. Samples drawn from the lookup table were then averaged together to arrive at that specific run average. The number of samples drawn from the lookup table varied from 55 to 880 (in multiples of 55) depending on approximately how many samples for that specific contaminant were present in the original data set. The number of runs for each contaminant varied from 1,200 to 19,200, depending on how many values were drawn at one time from the lookup table so that the product of the number of samples per run times the number of runs amounted to 1,056,000 for each contaminant (i.e.,  $55 \times 19,200 = 110 \times 9,600 = \dots = 880 \times 1,200 = 1,056,000$ ). The arrays of sample averages were queried using the built-in SMALL function of Microsoft® Excel 2003 to arrive at the 95<sup>th</sup> percentile value in each contaminant array. These values correspond to the 95% UCLs on the arrays of run averages, and represent the 95% UCLs for the average daily exposure levels for the various contaminants.