

HAZARDOUS AIR POLLUTANTS SPECIAL REPORT

Special report/study: An epidemiologic investigation of the relationship between the presence of ambient hazardous air pollutants (HAPs) and cancer incidence in Harris County.

[About the study](#)

[Executive summary](#)

[The study \(PDF\)](#)

[Butadiene Trend Analyses Houston East End](#)

Epidemiologic Investigation

Preliminary epidemiologic investigation of the relationship between the presence of ambient hazardous air pollutants (HAPs) and cancer incidence in Harris County.

About the study...

This study was initiated by the City of Houston Health Department and conducted by the University of Texas School of Public Health.

This study is a preliminary epidemiologic investigation of the relationship between the presence of ambient hazardous air pollutants (HAPs) and cancer incidence in Harris County. The cancer investigated is lymphohematopoietic.

The investigation consists of two analyses:

In the first analysis, home address in proximity to the Houston Ship Channel (HSC) is used as a proxy for exposure to HAPs. The rationale for this proxy is the large presence of major industrial HAP emission sources along the ship channel. The difference between cancer incidence rates in people living near the ship channel is compared with those living farther away from the ship channel.

In the second analysis, the average ambient air concentration of the individual hazardous air pollutants benzene and 1,3 butadiene are used as a proxy for exposure to the HAPs family. The rationale for this proxy is that these specific HAPs are two of the top seven air toxics identified, in a probabilistic human health risk screening study released by the Mayor's Task Force in the spring of 2006, as posing a definite risk to human health in Houston. These chemicals are found in high concentration in the HSC area.

In general, the main objective of an epidemiological study is to find out if, within a group of people, or cohort, there is evidence of a cause and effect relationship between the incidence of disease and exposure to a chemical or physical agent. In the study the health or disease patterns in a population group exposed to the chemical or agent, are compared with those of an unexposed or lesser exposed "control" group. The differences in the numbers of the sick vs. healthy people between the groups are statistically analyzed to determine if the differences between the number of sick people within the groups are those that would likely occur by chance alone or if in fact, the disease pattern differences occur outside the realm of those expected from chance and are caused by exposure to the chemical.

What this study does do?

1. The strongest and most important findings of this study are the trend of increased incidence of:

a particular cancer (acute lymphocytic leukemia) in children living in Harris County in close proximity to the Houston Ship Channel compared to those living farther away and any type of leukemia in children living in Harris County in regions with higher average ambient air 1,3 butadiene concentrations compared to those with lower average ambient air 1,3 butadiene concentrations.

2. The link between leukemia and 1,3 butadiene exposure association found in this study is consistent with and supports the findings of many previous studies in other geographic regions. It is the first study of this type to relate incidence of cancer and these HAPs in Harris County.
3. This study underscores and supports the need for control of HAPs in ambient air.
4. This study emphasizes the need for a more in depth epidemiological study which better handles impacts of meteorology, refined source location, ambient concentration statistics and identified confounding factors.

What this study does not do?

1. This study does not provide the first or new insight into the carcinogenicity of these two HAPs. EPA has previously characterized benzene and 1, 3 butadiene as human carcinogens based on the weight of evidence from many sources.
2. Because of the complexity of this type of study and the many levels of inherent uncertainty, it does not and cannot identify a bright line of exact concentrations or geographic boundaries associated with increased cancer incidence.
3. Although the study did not find consistent statistically significant overall trends in increased cancer incidence associated with adults:

living in close proximity to the ship channel compared with those living farther from the ship channel and

living in Harris County in regions with higher average ambient 1,3 butadiene and benzene concentrations compared to those with lower average ambient air 1,3 butadiene and benzene concentrations,

the study results cannot be extrapolated to indicate that there is no such relationship. A more detailed assessment of the statistical power of the analysis would need to be conducted to ascertain if there was enough data to reject the hypothesis of increased cancer with increased exposure to HAPs. In addition, it is possible that the relationship does exist but cannot be seen with this preliminary assessment. There are many confounding factors which may not be completely controlled with this assessment. A confounding factor is a particular circumstance that could influence the outcome of the study. The statistics must include control or correction for the impact of confounding factors so that there is a clear path between the chemical and response.

Association in adults is more complicated because of additional confounding factors more easily accounted for in children. For example, mobility or changing residences throughout life (the study assumes that the address when cancer was reported is the lifetime address although there is a long latency period of cancer), occupational exposure (comparing male and female rates may tease out impacts from occupational exposure however this requires more observations to attain statistical power), lifestyle choices (e.g., smoking) may not be accurately corrected, the ambient concentrations used in this study may not be representative of the actual exposure concentration/duration causing the cancer because of long and varying latency periods.

4. Although the study did not find consistent statistically significant overall trends in increased cancer incidence associated with children living in Harris County in regions with higher average ambient benzene concentrations compared to those with lower average ambient air benzene concentrations, the study results cannot be extrapolated to indicate that there is no such relationship. In addition to statistical power issues and adequate control of confounding factors as discussed above, the differences in results found for 1,3 butadiene compared to benzene may stem from other causes such as the calculation of the exposure statistic and the nature of the chemical (e.g., 1,3 butadiene is a highly reactive chemical that is localized around its area of production in the HSC region while benzene is a ubiquitous pollutant in Harris County and a more refined concentration estimation procedure incorporating variability and confidence is recommended).

What is the City of Houston currently doing about this problem?

In addition to the routine oversight and enforcement of air pollution sources conducted by the City's Bureau of Air Quality Control, over the past 12 to 18 months, the City of Houston has initiated specific actions to address 1,3-butadiene and benzene air pollution problems through many avenues including:

Negotiations of emission reductions with specific facilities emitting 1,3-butadiene: The City entered into an emission reduction agreement with the facility that emits the highest quantity of 1,3-butadiene emissions in the City so that reductions of emissions are quantified and verified; .

Air monitoring and data surveillance of these pollutants has been increased: The Milby Park canister monitor was upgraded to an automatic gas chromatograph so that near real time hourly data are available. The data are routinely reviewed for spikes and trends. A mobile air pollution monitor and laboratory is slated to be online in May to quantify impacts from specific 1,3-butadiene and benzene sources.

The City has advocated for legislative and regulatory improvements to include: state ambient air pollutant standards for these and other air toxics; more stringent source emission controls from EPA.

The City is implementing a major source voluntary emissions reduction plan for benzene.

Epidemiologic Investigation - Executive Summary

Preliminary epidemiologic investigation of the relationship between the presence of ambient hazardous air pollutants (HAPs) and cancer incidence in Harris County.

Executive Summary

Title: A preliminary investigation of the association between hazardous air pollutants and lymphohematopoietic cancer risk among residents of Harris County Texas

Authors: Kristina M. Walker, Ann L. Coker, Elaine Symanski, Philip J. Lupo

Affiliation: University of Texas Health Science at Houston, School of Public Health

In the recent months, there has been a great deal of interest surrounding Houston's air quality and the potential for high levels of pollutants in the ambient environment to adversely affect citizens' health. Historically, attention has focused on criteria pollutants such as ozone. However, there is increasing concern regarding another group of pollutants, known as hazardous air pollutants (HAPs). HAPs are a class of 189 chemicals which are known or suspected to have adverse effects on health.¹ Unlike criteria pollutants, there are no national standards regulating acceptable levels of these compounds in the ambient environment. Two HAPs, benzene and 1,3-butadiene, may be of particular concern in Houston due to the large volume of emissions as well as their potential to cause cancer in humans.

While recent reports have commented on the elevated ambient levels of hazardous air pollutants (HAPs) in certain areas of Houston, relative to other cities, few studies have assessed the health effects of HAPs for Houstonians and none have evaluated the association between ambient levels of these pollutants and lymphohematopoietic cancer risk in this population. Accordingly, we conducted a population based analysis of ambient environmental levels of HAPs and leukemia and lymphoma incidence in Harris County. We identified all cancer cases, including adult and childhood cases, diagnosed and reported to the Texas Cancer Registry from 1995-2003. We then used existing air monitoring data collected from 1992-2003 by the Texas Commission on Environmental Quality (TCEQ) to estimate ambient census tract levels to benzene and 1,3-butadiene. Additionally, we estimated risk of developing leukemia and lymphoma associated with residential proximity to the Houston Ship Channel (HSC) as another surrogate of HAP exposure. We assigned cancer cases to a particular census tract based on their residence at diagnosis as reported to the TCR. We then calculated cancer rates separately for adult and childhood cancers for each census tract. We further accounted for gender, age, socio-economic status, and ethnicity.

We found a 56% increased risk of acute lymphocytic leukemia among children living within two miles of the HSC (p-value=0.01) compared with children living more than 10 miles from the HSC. We found no increased risks of developing any other childhood leukemia or lymphoma associated with living within two miles of the HSC. We did, however, find that compared with children living in areas with the lowest estimated 1,3-butadiene levels estimated from monitoring data collected by the Texas Commission on Environmental Quality (TCEQ), children living in areas with the highest levels had a 40% (p-value=0.02), 38% (p-value=0.05) and 153% (p-value=0.03) increased risk of developing any type of leukemia, acute lymphocytic leukemia and acute myeloid leukemia, respectively. Higher 1,3-butadiene levels were not associated with childhood lymphoma or with adult leukemia or lymphoma. Higher estimated ambient benzene levels based on TCEQ monitoring data were not associated with childhood leukemia or lymphoma rates.

At the suggestion of several environmental scientists, we repeated our analyses for childhood leukemia using the United States Environmental Protective Agency's 1999 National Air Toxics Assessment (NATA) modeled ambient 1,3-butadiene and benzene levels. In general, we saw a similar pattern to that observed using the TCEQ monitoring data. When comparing childhood leukemia rates in those census tracts with the highest 1,3-butadiene levels with those in the lowest, a 32% increase in cancer risk was noted (p=0.09). This compares with a 40% increase (p=0.02) in risk for all types of leukemia in children based on the TCEQ monitoring data estimates for 1,3 butadiene.

Among adults, neither proximity to the Houston Ship Channel, nor levels of benzene or 1,3-butadiene was consistently associated with leukemia or lymphoma risk. Additional analytic studies with more refined exposure assessment methods are planned. However, observing a specific health effect of HAPs in light of recently documented elevated levels of two known carcinogens, benzene and 1,3 butadiene, in Houston,² strongly suggests a need to explore this issue further and possibly take action to limit potential exposure to HAPs in Houston.

1. About Air Toxics, Health and Ecological Effects. 2006. Retrieved On: 4-2-06. <http://www.epa.gov/air/toxicair/newtoxics.html>
2. A closer look at air pollution in Houston. Identifying priority health risks: Report of the Mayor's task force on the health effects of air pollution. Houston, Texas: University of Texas, Health Science Center. Institute for Health Policy.

Title: An investigation of the association between hazardous air pollutants and lymphohematopoietic cancer risk among residents of Harris County, Texas

Authors: Kristina M. Walker, Ann L. Coker, Elaine Symanski, Philip J. Lupo

Affiliation: University of Texas Health Science at Houston, School of Public Health

Abstract

While recent reports have commented on the elevated ambient levels of hazardous air pollutants (HAPs) in certain areas of Houston, relative to other cities, few studies have assessed the health effects of HAPs for Houstonians and none have evaluated the association between ambient levels of these pollutants and lymphohematopoietic cancer risk in this population. To begin to address this deficit in the literature, we conducted a population based analysis of ambient levels of HAPs and lymphohematopoietic cancer incidence in Harris County. Cancer cases were obtained from the Texas Cancer Registry (TCR), 1995-2003, and included adult and childhood cancers. We used existing monitoring data collected from 1992-2003 by the Texas Commission on Environmental Quality (TCEQ) to estimate ambient benzene and 1,3 butadiene levels for each census tract, which were then collapsed into 3 or 4 categories, respectively, in the analyses that were carried out. We also estimated risk of lymphohematopoietic cancer associated with residential proximity to the Houston Ship Channel (HSC) as another proxy (albeit a crude measure) of HAP exposure. Poisson regression was used to estimate rate ratios for increasing HAP levels and cancer rates, adjusting for gender, age, and a composite index for socioeconomic status and race / ethnicity. We observed an association among children living within two miles of the HSC and higher rates of acute lymphocytic leukemia (adjusted rate ratio [aRR] = 1.56; p=0.01) compared with those living greater than 10 miles from the HSC. Further, higher 1,3-butadiene levels (≥ 1.15 parts per billion [ppbV] relative to <0.266 ppbV) were associated with acute lymphocytic leukemia, acute myeloid leukemia and all leukemias in children. While we did not observe statistically significant associations for the other two categories (i.e., 0.266 - 0.381 ppbV or 0.382 - 1.15 ppbV) as compared to the referent group of <0.226 ppbV, a trend of increasing 1,3-butadiene levels and increasing leukemia rates was noted for these cancers. Higher benzene levels were not associated with lymphohematopoietic cancer by types in children. Among adults, neither proximity to the HSC, nor ambient levels of benzene or 1,3-butadiene levels were associated with lymphohematopoietic cancers in a dose dependent manner for either males or females. Additional analyses using more sophisticated methods to assess exposure are planned to confirm our findings. Additionally, future analyses will address many of the limitations of the current analysis. However, observing a specific health effect of HAPs in light of recently documented elevated levels of two known carcinogens, benzene and 1,3 butadiene, in Houston,(1) suggests a need to explore this issue further and to potentially take action to limit potential exposure to HAPs.

Introduction

In 1997, Texas ranked among the top three states in the number of chemical and allied products manufacturing facilities and in the number of petroleum and coal products manufacturing facilities, with more facilities located in Harris County (Houston) than in any other Texas county. (2) In a recent report on the effects of air pollution, the Mayor's Task Force determined that ambient levels of both benzene and 1,3-butadiene pose a 'definite' cancer risk to Houstonians. (1) According to the International Agency for Research on Cancer (IARC), benzene is classified as a known human carcinogen and 1,3-butadiene is classified as a probable human carcinogen. (3) Few studies have assessed the health effects of hazardous air pollutants (HAP) for Houstonians and none have addressed ambient levels of these pollutants and risk of lymphohematopoietic cancer, which includes leukemia, lymphoma and myeloma.

The majority of studies that have examined the potential health effects of HAPs have been conducted in occupational workers. However, most have not directly measured individual exposures to pollutants. The HAPs of primary concern in Houston, benzene and 1,3-butadiene, are mainly produced by petrochemical and synthetic rubber and plastics manufacturers. Occupational studies conducted in these settings typically compare worker populations with the population at large and report lower cancer mortality and incidence rates, for all cancers combined. (4-23)

Four studies reported more deaths from lymphohematopoietic cancers than expected among refinery/petrochemical plant employees (10, 13) or those exposed to 1,3-butadiene (7) or benzene (24) while findings from eight studies do not support this association. (6, 16, 17, 19, 21, 25-27) When looking at risk of developing cancer, four worker based cohort studies found higher risk of leukemia associated with exposure to benzene, (28, 29) or 1,3-butadiene (5, 30) and ten studies did not find an association. (6, 8-10, 12, 13, 16, 19-21) A nested case-control study (31) reported a significant increased risk of leukemia associated with an average exposure to 1,3-butadiene at levels of 1 ppm and another study reported that workers exposed to benzene at levels below 1 ppm had significantly decreased levels of white blood cells compared with unexposed workers. (32)

Few studies have looked at HAPs and risk of cancer in community populations. In these studies, exposure to HAPs is usually measured with a proxy measure such as residential distance from a plant or spill. One study compared actual and expected number of cancers in an area affected by a gasoline spill as a measure of high exposure; residents' leukemia rates were 4.40 (95% CI: 1.09-10.24) higher than expected by chance. (33) Another study found no effect of proximity (three kilometer area radius) to a petrochemical plant on the risk for leukemia (standardized incidence ratio [SIR]=0.99, 95% CI: 0.66-1.51) yet they did note a significant increased risk of multiple myeloma (SIR=2.15, 95% CI: 1.25-3.67). (34) Three other studies found no increased risk of leukemia in areas thought to be affected by industrial pollution. (35-37) Five have found a significant association with residence near industrial facilities and specific lymphohematopoietic cancers (38-42) while three others have found no association (34, 42, 43)

Only a small number of studies have explored the potential association between HAPs and childhood cancers. In a large California based study including 7,143 childhood cancer cases, Reynolds et al. (15) found a slight but non significant increase in leukemias (aRR=1.15; 95% CI 0.97-1.37) and proximity to road ways; there was no evidence that cancer rates increase with increasing vehicle or road density. In a follow-up for this study, Reynolds et al. (14) used data from the same 7,143 cases but used a dispersion model developed by the EPA to assign census tract level exposure scores to 25 potentially carcinogenic HAPs. Exposure scores for individual HAPs were summed to create a combined HAP

exposure score. Reynolds et al. found a significant ($p < 0.05$) trend of increasing childhood leukemia rates with increasing HAP exposure levels with a 21% (aRR = 1.21; 95% CI: 1.03-1.42) increased risk of childhood leukemia in census tracts with the highest combined exposure score. They further report a 32% increase in leukemia risk (aRR = 1.32; 95% CI: 1.11-1.57) associated with the highest levels of HAP exposure from point sources, compared with non-significant leukemia risks associated with the highest levels of HAP exposures from mobile (aRR=1.18) and area (aRR=1.16) sources. In the United Kingdom, Knox found correlations between residence at birth near geographically defined “hotspots” for several criteria pollutants as well as volatile organic compounds (VOCs), benzene, dioxins, 1,3-butadiene, and benz(a)pyrene and childhood leukemias. (11) Additional studies are needed to confirm the findings from these studies.

Because there have been few community level studies of HAP and cancer risk and because most of these studies have not utilized data on ambient levels of specific air pollutants, we have conducted a population based analysis of the association between HAPs and lymphohematopoietic cancer incidence in Harris County. We used existing monitoring data collected from 1992-2003 by the Texas Commission on Environmental Quality (TCEQ) to estimate census tract ambient levels of benzene and 1,3-butadiene. Cancer cases were obtained from the Texas Cancer Registry (TCR), 1995-2003, and included cases in adults and children.

Methods:

Overview and Study Population:

Two ecologic analyses utilizing existing data sources were performed to begin to address the question of whether air pollution is associated with increased rates of lymphohematopoietic cancer in Harris County, Texas. Our focus was on two specific hazardous air pollutants, benzene and 1,3-butadiene, identified as posing a ‘definite’ cancer risk in a recent report to the Houston Mayor’s Office. (1) The two analyses presented in this report serve as preliminary results for a future planned study incorporating more complex methodology. In the first analysis, we assessed the association between distance from the Houston Ship Channel (HSC) and cancer rates. We then evaluated the association between cancer rates and ambient levels of benzene and 1,3-butadiene. Both analyses were performed at the census tract level.

Our study population included all residents of Harris County, Texas between 1995 and 2003. We identified cases of lymphohematopoietic cancer diagnosed and reported to the Texas Cancer Registry (TCR) during the same time period. All incident cases of lymphohematopoietic cancer, independent of age or stage of disease were eligible. The specific lymphohematopoietic cancers we examined include leukemia (ICD-10 codes C91-C95), myeloma (ICD10 codes C88, C90, C96), non-Hodgkin’s lymphoma (ICD-10 codes C82-C85) and Hodgkin’s disease (7.8% of total) (ICD-10 code C81). We excluded those cancer cases with a prior cancer diagnosis. No primary data were collected for this study and no patients, physicians or hospitals were contacted. Since only cancer registry data was used, no identifying information other than age, race/ethnicity and address at the time of diagnosis was available. Addresses were used solely for the purposes of assignment of cases to census tracts. At the census tract level, rates of cancer were calculated using the Census 2000 estimates for the population. All rate ratios were adjusted for census tract level age, gender, and socioeconomic status and race / ethnicity indicators.

Exposure Assessment:

Distance from Houston Ship Channel:

We evaluated distance of census tracts from the HSC using geographic information system (GIS) methodology. Using ArcGIS(44) we constructed a set of five mutually exclusive buffers two miles apart around the HSC (see figure 1). We then assigned census tracts to a particular buffer based on the buffer in which the centroid of the census tract fell. Consequently, there were five exposure groups corresponding to the five buffers that we will refer to as the following: : 0-2 miles, 2-4 miles, 4-6 miles, 6-8 miles, 8-10 miles. Census tracts with centroids that fell outside all five buffers (i.e., > 10 miles) served as the referent group. We hypothesized that if distance from the Houston Ship Channel were correlated with and therefore a proxy for industrial air pollution levels and this proxy was related to lymphohematopoietic cancer risk, rates of cancer would be higher in those buffers closest to the HSC. We do recognize that distance from the Houston Ship Channel is only one proxy measure of industrial air pollution and that there are other industrial pollutants outside the HSC.

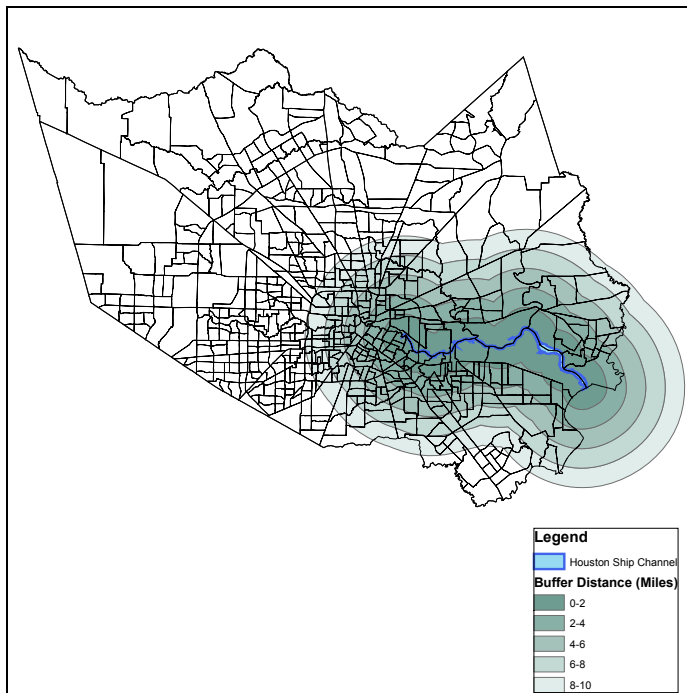


Figure 1. Map of Harris County, Texas depicting five buffer zones around the Houston Ship Channel

Compound-Specific Exposure:

Notwithstanding the limitations of using ambient air levels as a surrogate of personal exposure (45-47), we utilized data from the Texas Commission on Environmental Quality (TCEQ) to construct an assessment based on the potential for exposure to specific compounds. The TCEQ operates an extensive air monitoring network in the state of Texas, with Houston being one of the most monitored cities in the nation.(48) Although the TCEQ began monitoring air quality in the early 1970's, they did

not begin monitoring for HAPs until 1992. Between the years 1992 and 2005, 15 TCEQ monitoring sites were recording data on HAPs in Harris County. However, not all 15 sites were operable for the entire period. Because the cancer data obtained from the TCR extends only to 2003, we limited our use of the TCEQ data through this year as well. One monitoring site was not operable before 2005; therefore, the data used for this assessment comes from a total of 14 monitoring sites noted as red dots in Figure 2. As shown in the figure, these monitors are not geographically distributed throughout the Houston area as many are located within or near the Ship Channel, which poses difficulty in any assessment that attempts to utilize such data to estimate ambient levels at distances remote from the monitoring stations.

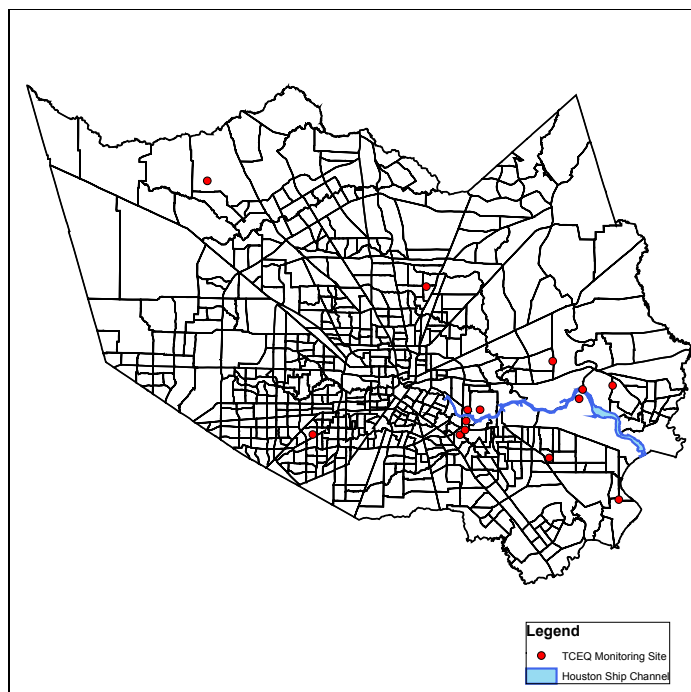


Figure 2. Map of Harris County, Texas with TCEQ monitoring site locations

In order to assess potential exposure to benzene and 1,3-butadiene, we developed a method to estimate ambient levels of these compounds for each census tract, based on the data obtained from the 14 monitoring sites without any adjustment for meteorological conditions (a limitation that we note later in the report). We first calculated the annual arithmetic mean concentrations of benzene and 1,3-butadiene for each monitoring location. We refer to this value as the site-specific annual mean. We then calculated the arithmetic mean concentrations for each of these two compounds over the entire period, 1992-2003, for each site. We refer to this value as the site-specific period mean. Because not all sites were active for all 12 years, we calculated the site-specific period mean as a weighted average of the annual means. Next, we determined, for each census tract, the distance of the centroid to each of the 14 monitoring sites, using ArcGIS(44) software. We were then able to assign each census tract an ambient value for benzene and for 1,3-butadiene based on the average of the site specific period means from the three closest monitoring sites. This value is the tract-specific mean.

The tract-specific means for benzene and 1,3-butadiene were categorized based on risk concentrations calculated using the California Office of Environmental Health Hazard Assessment (OEHHA) unit risk estimates (URE) as compiled by the Houston Mayor's Task Force on the Health Effects of Air Pollution. (1) These UREs reflect the excess cancer risk expected to occur as a result from lifetime exposure to 1 $\mu\text{g}/\text{m}^3$ of the chemical. (1) In order to calculate a risk concentration for a given lifetime cancer risk (1 in a million, for example), the lifetime cancer risk is divided by the URE. (1) Because the UREs are reported by the Task Force in units of $(\mu\text{g}/\text{m}^3)^{-1}$ and the TCEQ data is reported in units of ppbV, we first calculated the risk concentrations in units of $\mu\text{g}/\text{m}^3$ and then transformed them based on the following equation:

$$\mu\text{g} / \text{m}^3 = \frac{\text{ppbV} * \text{MolecularWeight} * 273\text{K}}{22.4 * 298\text{K}}$$

For example, given a unit risk estimate for benzene of 0.000029, the risk concentration based on 1 in a million risk is 0.034 $\mu\text{g}/\text{m}^3$ or 0.0107 ppbV. For 1,3-butadiene, the risk concentration based on 1 in a million risk is 0.00266 ppbV. Because the minimum site-specific period means for each benzene and 1,3-butadiene were greater than these concentrations, we chose to base our categorization of the tract-specific means on a lifetime risk of 1 in 10,000 rather than 1 in 1,000,000. The values corresponding to a 1 in 10,000 lifetime cancer risk for benzene and 1,3-butadiene were: 1.07 ppbV and 0.266 ppbV, respectively. For benzene, the tract-specific means were categorized into three groups, <1.07 ppbV, 1.07 - 1.19 ppbV and ≥ 1.20 ppbV with the referent group being those values below 1.07 ppbV. For 1,3-butadiene, the tract-specific means were categorized into four groups, with the referent group being those values below 0.266 ppbV. The remaining three categories were: 0.266 - 0.381, 0.382 - 1.14 and ≥ 1.15 . The benzene categorization resulted in one less category than that for 1,3-butadiene because only 10% of census tracts had tract-specific means greater than 1.07 ppbV; therefore, more than three exposure categories would have resulted in very sparse numbers. We chose to categorize tract-specific means for benzene and 1,3-butadiene in this manner rather than by percentiles because we felt using the UREs to guide the categorization would result in more biologically meaningful categories. The use of UREs were chosen over reference exposure levels (REL) because, unlike the derivation of RELs, the approach to deriving UREs does not assume a threshold level.(49) Therefore, the URE is more appropriate when considering cancer risk.

Statistical Analyses:

Confounding Factors:

For all analyses, we considered age at diagnosis, race / ethnicity and socioeconomic status as potential confounders. Further, because the etiology of childhood cancers (age at diagnosis <20 years) is likely different than adult cancers (age at diagnosis ≥ 20 years), we analyzed these two groups separately. To further account for the potential influence of age at diagnosis on cancer incidence within these groups, we adjusted for age in 5 year intervals within the children and adult groups. Additionally, we stratified analyses of adult cancers by gender. For analyses of childhood cancers, we adjusted for gender. Because socioeconomic status is a complex term and involves such attributes as income, education level and household characteristics,(50) and because it is difficult to obtain individual measures of these attributes, we constructed a composite index of socioeconomic status based on census tract level data from the 2000 Census, using principal component analysis to identify the most relevant factors. This variable was then categorized into quartiles based on its distribution in the state of Texas. While we did not include race as an independent variable in the statistical models, we did include a factor representing the percent of the census tract population that is Hispanic and the percent

of the census tract population that is African-American in the composite socioeconomic status variable. Including race as an additional independent variable would have produced too fine a stratification for constructing census tract level case counts and population totals, resulting in a loss of power and potentially affecting the ability of the models to converge properly.

Analysis #1 – Distance from the Houston Ship Channel:

To test the association between distance from the HSC and lymphohematopoietic cancer incidence, we used poisson regression. The dependent variable in this regression was case count, indexed by census tract, gender and age group. The buffer zones indicating distance from the HSC was modeled as an ordinal variable with 6 levels, as previously discussed. Other independent variables included in the model were gender (childhood cancers only), age group, and race/ ethnicity and socioeconomic status. An offset equal to the log of the population total for each census tract, gender and age group combination was used to account for the different population sizes in each of these groups. Additionally, generalized estimating equations were used to account for clustering of incidence rates within census tracts.

Because the etiology of lymphohematopoietic cancer differs by cancer subtypes, analyses were conducted separately for adult and childhood cancers as well as by the following cancer types: Hodgkin's lymphoma, non-Hodgkin's lymphoma, Myeloma, all leukemias, acute lymphocytic leukemia, acute myeloid leukemia, chronic lymphocytic leukemia, and chronic myeloid leukemia.

Analysis #2 – Compound-Specific Exposure:

We conducted a second poisson regression analysis to test the association between lymphohematopoietic cancer incidence and ambient levels of benzene and 1,3-butadiene, separately. As in the previous regression, the dependent variable for this second analysis was case count, indexed by census tract, gender and age group. The independent variables were gender, age group, socioeconomic status and the tract-specific mean for either benzene or 1,3-butadiene, categorized as already discussed. An offset equal to the log of the population total for each census tract, gender and age group combination was used to account for the different population sizes in each of these groups. Generalized estimating equations were also used to account for clustering of incidence rates within census tracts.

This analysis was conducted separately for adult and childhood cancers as well as by the following cancer types: Hodgkin's lymphoma, non-Hodgkin's lymphoma, myeloma, all leukemias, acute lymphocytic leukemia, acute myeloid leukemia, chronic lymphocytic leukemia, and chronic myeloid leukemia

Results

Analysis among children, adjusting for gender, age, and socio-economic status:

Based on the buffer analysis (table 1), no trend toward increasing risk of any lymphohematopoietic cancer by distance from the HSC in 2 mile intervals was noted. However, rates of acute lymphocytic leukemia were significantly higher for those living 0-2 miles from the HSC (aRR = 1.56; p=0.0105) compared with those living greater than 10 miles from the HSC. Additionally, higher 1,3-butadiene levels (≥ 1.15 ppbV relative to < 0.266 ppbV) were associated with acute lymphocytic leukemia (p=0.05), acute myeloid leukemia (p 0.03) and all leukemias (p = 0.02) and a trend of increasing 1,3-butadiene levels and increasing leukemia rates was noted for these cancers (table 2). Higher benzene

levels (1.07-1.19 ppbV or ≥ 1.20 ppbV relative to <1.07 ppbV) were not associated with any lymphohematopoietic cancer in children (table 3).

Analysis among adults, stratifying by gender and adjusting for age and socio-economic status:

No significant trends of increasing lymphohematopoietic cancer risk with increasing proximity to the HSC was observed for men or women for any specific cancer type (table 4). Although we did observe several significant associations between Hodgkin's disease, non-Hodgkin's Lymphoma and myeloma and specific distances from the HSC, no consistent pattern of risk emerged for either gender (table 4). Finding an association for both genders would provide greater support for an environmental versus an occupational route of exposure. Among male adults, higher 1,3-butadiene levels were associated with non-Hodgkin's lymphoma and a significant trend was also noted ($p=0.010$) (table 5). A similar pattern was not observed for females. Although we also found a 57% increased risk of Hodgkin's disease among females associated with 1,3-butadiene levels of 0.382 - 1.14 ppbV relative to the lowest level of <0.226 ppbV, there was not a significant increasing trend (table 5). We did also observe an increased risk of chronic myeloid leukemia in men associated with benzene levels between 1.07 -1.19 ppbV (table 6). However, higher benzene levels were not associated with a significant trend toward increasing lymphohematopoietic cancer rates for men or women for any specific cancer type (table 6).

Discussion

Our findings and methods are similar to those reported by Reynolds et al. (14); they report a 21% increase in childhood leukemia associated with those census tracts with higher modeled ambient levels of HAPs. On the basis of our assessment using residential proximity to the HSC, those living within 2 miles had a 56% higher childhood lymphocytic leukemia rate than did those living greater than 10 miles from the HSC ($p=0.01$). When we evaluated risks using monitored ambient levels of specific HAPs, those living in areas with the highest estimated 1,3-butadiene levels had significantly higher childhood acute lymphocytic leukemia, acute myeloid leukemia and all leukemias. Neither proximity to the HSC nor levels of benzene or 1,3-butadiene were associated with adult lymphohematopoietic cancer. Our finding that higher 1,3-butadiene levels were associated with higher non-Hodgkin's lymphoma rates (among adult males only) may be due to an occupational exposure confounding the association. Had we found that non-Hodgkin's lymphoma rates were associated with higher 1,3-butadiene levels in both males and females, we would have better evidence of an unconfounded association.

Our team has taken the following efforts to limit bias and improve study validity. We limited selection bias by using population based TCR data. To the extent that TCR is complete and accurate for lymphohematopoietic cancer from years 1995-2003, we have included all cases in the population. We used Census 2000 as the data source for the denominator at the census tract level which is the smallest unit of analysis possible for rare cancers while maintaining sufficient study power. One limitation of ecologic studies is that we do not have individual data on exposure, disease, and confounding factors. Because we used the smallest geographic unit possible, census tracts, we got closer to having individual level data and therefore have reduced the impact of ecological misclassification bias on study findings. Our use of census data to additionally control for important confounders including age, gender and socioeconomic status also limits confounding bias common in ecological studies. Because cancer rates vary by age and socioeconomic status and because there is considerable variation in the distributions of these factors within census tracts across Houston, we adjusted our analyses for both factors. Smoking may also confound the putative association between exposure to ambient benzene and 1,3-butadiene since smoking may provide another source of exposure to these chemicals. It has

been shown in numerous studies that aggregate measures of socioeconomic status may be viewed as a proxy for individual level smoking status. (51-54) By adjusting rate ratios for socioeconomic status we are also attempting to control for confounding due to occupational exposures, although residual confounding is likely still present. We attempted to address this residual confounding in adults through stratification by gender, as males are more likely to be affected by this type of exposure.

At the suggestion of several environmental scientists, we repeated our analyses for childhood leukemias using the USEPA's 1999 National Air Toxics Assessment (NATA) modeled ambient 1,3-butadiene and benzene levels at the census tract level. In general we saw a similar pattern to that reported in table 2 based on our TCEQ estimates of ambient levels. From table 2, for all leukemias in children, the rate ratio for comparing the highest grouped ambient level to the lowest was 1.40 and the p-value was 0.02. Using the NATA data for childhood leukemias, again comparing the highest with the lowest 1,3-butadiene levels, this rate ratio was 1.32 ($p=0.09$). The latter was of borderline statistical significance. Note however that the actual levels of 1,3-butadiene differ for the two analyses yet the relative grouping based on quartiles are similar. Further and more sophisticated analyses are planned to compare the NATA modeled estimates with the actual TCEQ monitoring data to determine reliability of our categorizations and the extent of exposure misclassification. Additionally, techniques known as kriging will be used to better estimate ambient benzene and 1,3-butadiene levels by census tracts based on TCEQ monitoring data.

This is the one of the first health effects studies to use monitoring data as a measure of ambient air levels of HAPs in Houston. However, there are challenges to using these data. The strategy that we used to estimate ambient levels for each census tract was limited by several factors. First, the TCEQ monitoring sites are not distributed randomly across Houston and not all monitors have been active over the entire period of interest (1995-2003). This presented a particular challenge for those census tracts in the presumed low exposed area where few monitors exist (see Figure 2). The distribution of monitors raises a question related to the appropriateness of using monitoring data collected for one purpose (i.e., assessing compliance) for another (i.e., assessing an exposure-response relationship). Note however that these data are the only existing source of potential ambient HAP exposures collected over time. Secondly, the methods used in this analysis to estimate ambient levels of pollutants for each census tract did not account for meteorological conditions, which affect the transport and fate of air pollutants. Analyses are planned that will improve upon the methods used to estimate ambient levels by census tract and thereby limit the biases that may have been introduced in the current investigation. Finally, we opted to estimate ambient levels for each census tract in this investigation by averaging data collected over the entire period (along with cancer incidence). While long-term HAP exposures are more likely to be etiologically related to cancer development, this approach raises questions regarding temporality of the potential exposure-disease relationship. We did exclude those cancers that occurred prior to the recorded years of HAP monitoring data reported by TCEQ as a means to address this issue of temporal sequence. In subsequent analyses we will attempt a more comprehensive assessment of lagged exposures relative to cancer incidence. However, these analyses will be limited by study power as the annual number of cancer cases are relatively small. We opted to explore HAP ambient levels with these specific cancers because of the relatively short latency period from exposure to cancer development. Because the TCEQ monitoring data and the TCR cancer data have only been collecting data over the past decade, this preliminary report represents the first opportunity to correlate ambient HAP levels with those cancers for which we might expect to see an association given the shorter latency periods. Additionally, because the California OEHHA unit risk estimates (URE) are not the same as URE obtained from other sources, such as the United States Environment Protection Agency, had we used another source, our resulting classification would have been different which could then have affected our final results.

We used distance of census tracts from the HSC as a crude surrogate for proximity to higher ambient levels of air pollutants given the concentration of petroleum and chemical manufacturing facilities in the HSC. However, we recognize that there is variability in ambient air levels within the HSC, that there may be industrial facilities that emit benzene and 1,3-butadiene located outside the HSC, and that mobile sources contribute to air pollution as well. Nonetheless, our preliminary results of an association between childhood leukemia rates and both proximity to industrial sources (defined as distance from the HSC) and ambient levels of HAPs, derived from TCEQ monitoring data, suggest that a true association may exist. Given the increased sensitivity of children to environmental toxicants including carcinogens(55) and that children metabolize toxins differently than adults with behaviors which present a unique opportunity for exposure(56), it is plausible that we might observe an association between HAPs and lymphohematopoietic cancer in children but not adults. Other than the increased sensitivity of children, studying childhood cancer development presents advantages over that of adults since there are few known risk factors for childhood cancer and because the latency of childhood cancers is much shorter. Additional studies in child and adult populations will be necessary to allow for more definitive statements to be made regarding whether increased cancer risk is associated with ambient air pollution.

In conclusion, the primary limitation of this study lies in the 'exposure assessment' and the potential for biases that may have affected study validity. In fact, we do not have individual exposure data nor were we able to assess the validity of using monitoring data as a surrogate measure for personal exposure. Thus, the results presented here should be viewed as preliminary. Nonetheless, additional analytic studies with more refined methodology are warranted to further evaluate the association between increased lymphohematopoietic cancer risk and ambient levels of hazardous air pollutants, the results of which might inform policies intended to mitigate health risks of Houstonians in the future.

Table 1. Crude annual incidence rates and adjusted rate ratios for association between distance from the Houston Ship Channel (HSC) and lymphohematopoietic cancer incidence among children (aged <20)

Cancer Type	Distance from HSC (Miles)	# Census Tracts	# Cases	Crude Annual Incidence Rate (per 100,000)	Rate Ratio*	p-value	p-value for trend
Hodgkin's Disease	>10	344	64	1.168	1.00		0.1522
	8-10	60	10	1.444	1.17	0.6169	
	6-8	62	9	1.452	1.15	0.6917	
	4-6	71	11	1.348	1.06	0.8794	
	2-4	77	7	0.609	0.47	0.0995	
	0-2	35	4	0.867	0.61	0.3708	
non-Hodgkin's Lymphoma	>10	344	46	0.839	1.00		0.7031
	8-10	60	7	1.011	1.28	0.5825	
	6-8	62	5	0.807	1.05	0.9076	
	4-6	71	8	0.981	1.30	0.4597	
	2-4	77	10	0.870	1.14	0.7044	
	0-2	35	3	0.650	0.91	0.8947	
All Leukemia	>10	344	274	5.000	1.00		0.6189
	8-10	60	38	5.487	1.09	0.6412	
	6-8	62	36	5.809	1.17	0.4335	
	4-6	71	44	5.393	1.11	0.5282	
	2-4	77	55	4.783	0.96	0.8322	
	0-2	35	29	6.285	1.22	0.2688	
Acute Lymphocytic Leukemia	>10	344	207	3.777	1.00		0.3274
	8-10	60	32	4.620	1.21	0.2974	
	6-8	62	23	3.712	0.98	0.9917	
	4-6	71	34	4.167	1.16	0.4405	
	2-4	77	40	3.479	0.96	0.8280	
	0-2	35	27	5.852	1.56	0.0105	
Acute Myeloid Leukemia	>10	344	40	0.730	1.00		0.3470
	8-10	60	4	0.578	0.71	0.5215	
	6-8	62	8	1.291	1.58	0.3301	
	4-6	71	5	0.613	0.74	0.5577	
	2-4	77	9	0.783	0.87	0.7434	
	0-2	35	1	0.217	0.20	0.1290	

*Rate Ratios Adjusted for Age, Gender, Ethnicity, and Socioeconomic Status

Note: There were no childhood Myeloma cases; Results are not shown for chronic lymphocytic leukemia or chronic myeloid leukemia due to small numbers (4 and 16 total cases, respectively)

Table 2. Crude annual incidence rates and adjusted rate ratios for association between ambient 1,3-Butadiene levels and cancer incidence among children aged 0-19

Cancer Type	1,3-Butadiene Level*	# Census Tracts	# Cases	Crude Annual Incidence Rate (per 100,000)	Rate Ratio**	p-value	p-value for trend
Hodgkin's Disease	1	148	22	0.86	1.00		
	2	158	25	1.21	1.53	0.171	
	3	137	22	1.23	1.67	0.152	
	4	206	36	1.29	1.80	0.071	0.099
non-Hodgkin's Lymphoma	1	148	20	0.78	1.00		
	2	158	20	0.96	1.44	0.264	
	3	137	18	1.01	1.73	0.128	
	4	206	21	0.75	1.25	0.510	0.559
All Leukemia	1	148	116	4.51	1.00		
	2	158	99	4.77	1.10	0.530	
	3	137	93	5.21	1.19	0.292	
	4	206	168	6.02	1.40	0.024	0.017
Acute Lymphocytic Leukemia	1	148	89	3.46	1.00		
	2	158	76	3.67	1.09	0.629	
	3	137	68	3.81	1.11	0.528	
	4	206	130	4.66	1.38	0.051	0.041
Acute Myeloid Leukemia	1	148	12	0.47	1.00		
	2	158	13	0.63	1.50	0.359	
	3	137	14	0.78	1.79	0.217	
	4	206	28	1.00	2.53	0.033	0.026

*1,3-Butadiene levels correspond to: level 1 =<0.266 ppbV; level 2 = 0.266 - 0.381 ppbV; level 3 = 0.382-1.14 ppbV; and level 4 = ≥1.15 ppbV

**Rate Ratios Adjusted for Age, Gender, Ethnicity and Socioeconomic Status

Note: There were no childhood Myeloma cases; Results are not shown for chronic lymphocytic leukemia or chronic myeloid leukemia due to small numbers (4 and 16 total cases, respectively)

Table 3. Crude annual incidence rates and adjusted rate ratios for association between ambient Benzene levels and cancer Incidence among children aged 0-19

Cancer Type	Benzene Level*	# Census Tracts	# Cases	Crude Annual Incidence Rate (per 100,000)	Rate Ratio**	p-value	p-value for trend
Hodgkin's Disease	1	543	85	1.11	1.00		
	2	79	14	1.27	1.14	0.721	
	3	27	6	1.27	1.16	0.745	0.673
non-Hodgkin's Lymphoma	1	543	66	0.86	1.00		
	2	79	8	0.73	0.98	0.965	
	3	27	5	1.06	1.36	0.487	0.587
All Leukemia	1	543	399	5.22	1.00		
	2	79	62	5.63	1.01	0.943	
	3	27	15	3.17	0.60	0.078	0.164
Acute Lymphocytic Leukemia	1	543	308	4.03	1.00		
	2	79	42	3.81	0.86	0.450	
	3	27	13	2.75	0.66	0.183	0.147
Acute Myeloid Leukemia	1	543	55	0.72	1.00		
	2	79	11	1.00	1.17	0.733	
	3	27	1	0.21	0.27	0.186	0.372

*Benzene Levels correspond to: level 1 = <1.07 ppbV; level 2 = 1.07 - 1.19 ppbV; and level 3= ≥1.20 ppbV

**Rate Ratios Adjusted for Age, Gender, Ethnicity, and Socioeconomic Status

Note: There were no childhood Myeloma cases; Results are not shown for chronic lymphocytic leukemia or chronic myeloid leukemia due to small numbers (4 and 16 total cases, respectively)

Table 4. Crude annual incidence rates and adjusted rate ratios for association between distance from the Houston Ship Channel (HSC) and lymphohematopoietic cancer incidence among adults (aged ≥20)

Cancer Type	Gender	Distance from HSC (Miles)	# Census Tracts	# Cases	Crude Annual		p-value	p-value for trend
					Incidence (per 100,000)	Rate Ratio*		
Acute Myeloid Leukemia	Male	>10	344	169	2.79	1.00	0.052	0.431
		8-10	60	37	4.38	1.37		
		6-8	62	277	33.97	1.14		
		4-6	71	37	3.84	1.34		
		2-4	77	37	3.26	1.25		
	0-2	35	16	3.78	1.49			
	Female	>10	344	188	2.94	1.00		
		8-10	60	27	3.02	0.89		
		6-8	62	31	3.66	1.19		
		4-6	71	37	3.77	1.22		
2-4		77	34	3.00	1.07			
0-2	35	11	2.71	1.00				
Chronic Lymphocytic Leukemia	Male	>10	344	233	3.85	1.00	0.327	0.303
		8-10	60	37	4.38	0.92		
		6-8	62	41	5.03	1.14		
		4-6	71	45	4.67	1.04		
		2-4	77	35	3.08	0.76		
	0-2	35	15	3.54	0.82			
	Female	>10	344	162	2.53	1.00		
		8-10	60	31	3.47	1.07		
		6-8	62	22	2.60	0.95		
		4-6	71	36	3.67	1.29		
2-4		77	39	3.44	1.37			
0-2	35	7	1.72	0.69				
Chronic Myeloid Leukemia	Male	>10	344	106	1.75	1.00	0.613	0.608
		8-10	60	18	2.13	1.04		
		6-8	62	24	2.94	1.50		
		4-6	71	20	2.08	1.04		
		2-4	77	16	1.41	0.73		
	0-2	35	8	1.89	0.88			
	Female	>10	344	87	1.36	1.00		
		8-10	60	9	1.01	0.65		
		6-8	62	8	0.94	0.65		
		4-6	71	19	1.94	1.25		
2-4		77	11	0.97	0.67			
0-2	35	6	1.48	1.03				
Myeloma	Male	>10	344	304	5.03	1.00	0.111	0.483
		8-10	60	53	6.28	1.04		
		6-8	62	62	7.60	1.29		
		4-6	71	84	8.72	1.40		
		2-4	77	69	6.08	1.07		
	0-2	35	29	6.85	1.16			
	Female	>10	344	310	4.84	1.00		
		8-10	60	43	4.81	0.77		
		6-8	62	58	6.85	1.09		
		4-6	71	83	8.46	1.19		
2-4		77	66	5.83	0.89			
0-2	35	18	4.43	0.62				

Table 4. (cont.) Crude annual incidence rates and adjusted rate ratios for association between distance from the Houston Ship Channel (HSC) and lymphohematopoietic cancer incidence among adults (aged ≥20)

Cancer Type	Gender	Distance from HSC (Miles)	# Census Tracts	# Cases	Crude Annual Incidence (per 100,000)	Rate Ratio*	p-value	p-value for trend			
Acute Myeloid Leukemia	Male	>10	344	169	2.79	1.00		0.052			
		8-10	60	37	4.38	1.37	0.1272				
		6-8	62	277	33.97	1.14	0.4942				
		4-6	71	37	3.84	1.34	0.0956				
		2-4	77	37	3.26	1.25	0.2300				
		0-2	35	16	3.78	1.49	0.1376				
	Female	>10	344	188	2.94	1.00					
		8-10	60	27	3.02	0.89	0.5832				
		6-8	62	31	3.66	1.19	0.3712				
		4-6	71	37	3.77	1.22	0.2758				
		2-4	77	34	3.00	1.07	0.7170				
		0-2	35	11	2.71	1.00	0.9966				
		Chronic Lymphocytic Leukemia	Male	>10	344	233	3.85		1.00		0.327
				8-10	60	37	4.38		0.92	0.6582	
6-8	62			41	5.03	1.14	0.4452				
4-6	71			45	4.67	1.04	0.8375				
2-4	77			35	3.08	0.76	0.1467				
0-2	35			15	3.54	0.82	0.4916				
Female	>10		344	162	2.53	1.00					
	8-10		60	31	3.47	1.07	0.7148				
	6-8		62	22	2.60	0.95	0.8166				
	4-6		71	36	3.67	1.29	0.1922				
	2-4		77	39	3.44	1.37	0.1091				
	0-2		35	7	1.72	0.69	0.3529				
	Chronic Myeloid Leukemia		Male	>10	344	106	1.75	1.00		0.613	
				8-10	60	18	2.13	1.04	0.8925		
6-8		62		24	2.94	1.50	0.1996				
4-6		71		20	2.08	1.04	0.8713				
2-4		77		16	1.41	0.73	0.2530				
0-2		35		8	1.89	0.88	0.7650				
Female		>10	344	87	1.36	1.00					
		8-10	60	9	1.01	0.65	0.1769				
		6-8	62	8	0.94	0.65	0.2090				
		4-6	71	19	1.94	1.25	0.3999				
		2-4	77	11	0.97	0.67	0.2202				
		0-2	35	6	1.48	1.03	0.9505				
		Myeloma	Male	>10	344	304	5.03	1.00			0.111
				8-10	60	53	6.28	1.04	0.8132		
6-8	62			62	7.60	1.29	0.0733				
4-6	71			84	8.72	1.40	0.0105				
2-4	77			69	6.08	1.07	0.6517				
0-2	35			29	6.85	1.16	0.4786				
Female	>10		344	310	4.84	1.00					
	8-10		60	43	4.81	0.77	0.1122				
	6-8		62	58	6.85	1.09	0.5622				
	4-6		71	83	8.46	1.19	0.1884				
	2-4		77	66	5.83	0.89	0.4139				
	0-2		35	18	4.43	0.62	0.0608				

*Rate Ratios adjusted for age, ethnicity, and socioeconomic status

Table 5. Crude annual incidence rates and adjusted rate ratios for association between ambient 1,3-Butadiene levels and cancer incidence among adults (aged ≥20)

Cancer Type	Gender	1,3-Butadiene Level*	# Census Tracts	# Cases	Crude IR (per 100,000)	Rate Ratio**	p-value	p-value for trend
Hodgkin's Disease	Male	1	148	89	3.31	1.00		0.222
		2	158	81	3.52	1.12	0.475	
		3	137	64	3.12	1.06	0.741	
		4	206	81	2.54	0.84	0.307	
	Female	1	148	55	1.92	1.00		0.238
		2	158	48	1.95	1.04	0.842	
		3	137	53	2.67	1.57	0.025	
		4	206	75	2.23	1.19	0.369	
non-Hodgkin's Lymphoma	Male	1	148	487	18.11	1.00		0.010
		2	158	464	20.14	1.08	0.250	
		3	137	413	20.14	1.29	0.015	
		4	206	618	19.41	1.20	0.014	
	Female	1	148	444	15.54	1.00		0.226
		2	158	435	17.71	0.97	0.645	
		3	137	364	18.36	1.09	0.327	
		4	206	508	15.10	0.91	0.168	
All Leukemia	Male	1	148	312	11.60	1.00		0.296
		2	158	303	13.15	0.97	0.702	
		3	137	248	12.09	0.96	0.669	
		4	206	360	11.31	0.91	0.296	
	Female	1	148	236	8.26	1.00		0.358
		2	158	254	10.34	1.06	0.583	
		3	137	212	10.70	1.21	0.112	
		4	206	320	9.51	1.09	0.400	
Acute Lymphocytic Leukemia	Male	1	148	23	0.86	1.00		0.843
		2	158	19	0.82	0.88	0.662	
		3	137	15	0.73	0.76	0.445	
		4	206	32	1.01	1.03	0.922	
	Female	1	148	14	0.49	1.00		0.600
		2	158	15	0.61	1.09	0.813	
		3	137	13	0.66	1.10	0.831	
		4	206	23	0.68	1.22	0.597	

Table 5. (cont.) Crude annual incidence rates and adjusted rate ratios for association between ambient 1,3-Butadiene levels and cancer incidence among adults (aged ≥20)

Cancer Type	Gender	1,3-Butadiene Level*	# Census Tracts	# Cases	Crude IR (per 100,000)	Rate Ratio**	p-value	p-value for trend
Acute Myeloid Leukemia	Male	1	148	74	2.75	1.00		0.470
		2	158	89	3.86	1.24	0.143	
		3	137	54	2.63	0.92	0.670	
		4	206	106	3.33	1.20	0.240	
	Female	1	148	81	2.83	1.00		0.884
		2	158	81	3.30	1.03	0.849	
		3	137	68	3.43	1.23	0.243	
		4	206	98	2.91	1.01	0.955	
Chronic Lymphocytic Leukemia	Male	1	148	105	3.90	1.00		0.085
		2	158	110	4.77	1.01	0.962	
		3	137	85	4.15	0.95	0.754	
		4	206	106	3.33	0.79	0.119	
	Female	1	148	61	2.13	1.00		0.136
		2	158	78	3.17	1.24	0.214	
		3	137	63	3.18	1.47	0.054	
		4	206	95	2.82	1.31	0.117	
Chronic Myeloid Leukemia	Male	1	148	53	1.97	1.00		0.956
		2	158	34	1.48	0.68	0.093	
		3	137	48	2.34	1.07	0.791	
		4	206	57	1.79	0.88	0.601	
	Female	1	148	39	1.36	1.00		0.458
		2	158	34	1.38	0.90	0.650	
		3	137	28	1.41	0.99	0.983	
		4	206	39	1.16	0.83	0.410	
Myeloma	Male	1	148	125	4.65	1.00		0.988
		2	158	161	6.99	1.18	0.177	
		3	137	128	6.24	1.08	0.582	
		4	206	187	5.87	1.06	0.641	
	Female	1	148	119	4.16	1.00		0.200
		2	158	132	5.37	0.92	0.521	
		3	137	118	5.95	0.99	0.921	
		4	206	209	6.21	1.11	0.405	

*1,3-Butadiene levels correspond to: level 1 = <0.266 ppbV; level 2 = 0.266 - 0.381 ppbV; level 3 = 0.382-1.14 ppbV; and level 4 ≥1.15 ppbV

**Rate Ratios Adjusted for Age, Ethnicity, and Socioeconomic Status

Table 6. Crude annual incidence rates and adjusted rate ratios for association between ambient Benzene levels and cancer Incidence among adults (aged ≥20)

Cancer Type	Gender	Benzene Level*	# Census Tracts	# Cases	Crude Incidence Rate (per 100,000)	Rate Ratio**	p-value	p-value for trend
Hodgkin's Disease	Male	1	543	280	3.20	1.00		0.392
		2	79	25	2.41	0.85	0.518	
		3	27	10	2.33	0.81	0.492	
	Female	1	543	199	2.17	1.00		
		2	79	21	2.01	1.05	0.841	
		3	27	11	2.47	1.22	0.551	
non-Hodgkin's Lymphoma	Male	1	543	1728	19.73	1.00		0.585
		2	79	185	17.80	0.98	0.826	
		3	27	69	16.04	0.93	0.588	
	Female	1	543	1487	16.22	1.00		
		2	79	191	18.25	1.11	0.178	
		3	27	73	16.37	1.16	0.528	
All Leukemia	Male	1	543	1038	11.85	1.00		0.861
		2	79	137	13.18	1.05	0.665	
		3	27	48	11.16	1.00	1.000	
	Female	1	543	884	9.64	1.00		
		2	79	102	9.75	0.98	0.871	
		3	27	36	8.07	0.92	0.566	
Acute Lymphocytic Leukemia	Male	1	543	75	0.86	1.00		0.650
		2	79	12	1.15	1.34	0.454	
		3	27	2	0.47	0.51	0.284	
	Female	1	543	55	0.60	1.00		
		2	79	8	0.76	1.02	0.957	
		3	27	2	0.45	0.66	0.566	
Acute Myeloid Leukemia	Male	1	543	287	3.28	1.00		0.118
		2	79	25	2.41	0.65	0.052	
		3	27	11	2.56	0.80	0.402	
	Female	1	543	284	3.10	1.00		
		2	79	31	2.96	1.04	0.844	
		3	27	13	2.92	1.12	0.660	
Chronic Lymphocytic Leukemia	Male	1	543	345	3.94	1.00		0.839
		2	79	44	4.23	0.99	0.940	
		3	27	17	3.95	1.08	0.763	
	Female	1	543	262	2.86	1.00		
		2	79	28	2.68	0.93	0.769	
		3	27	7	1.57	0.64	0.256	
Chronic Myeloid Leukemia	Male	1	543	155	1.77	1.00		0.589
		2	79	32	3.08	1.71	0.037	
		3	27	5	1.16	0.71	0.500	
	Female	1	543	120	1.31	1.00		
		2	79	14	1.34	1.04	0.895	
		3	27	6	1.35	1.12	0.759	
Myeloma	Male	1	543	501	5.72	1.00		0.908
		2	79	70	6.73	0.91	0.516	
		3	27	30	6.98	1.11	0.595	
	Female	1	543	483	5.27	1.00		
		2	79	71	6.78	0.94	0.675	
		3	27	24	5.38	0.89	0.584	

*Benzene Levels correspond to: level 1 = <1.07 ppbV; level 2 = 1.07- 1.19 ppbV and level 3 = ≥1.20 ppbV

**Rate Ratios Adjusted for Age , Ethnicity, and Socioeconomic Status

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1,3-Butadiene Trend Analyses Houston East End

Milby Park Monitor

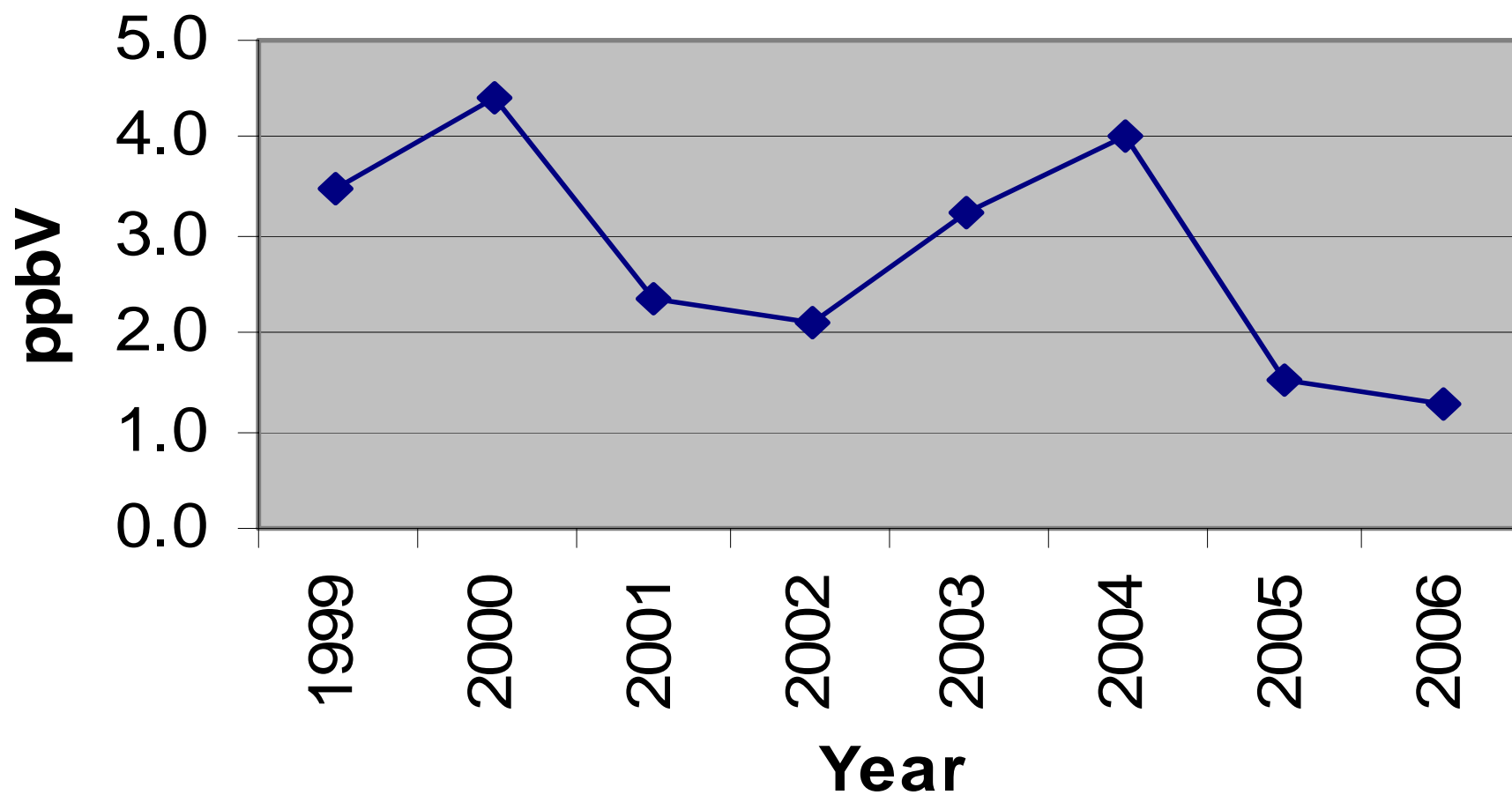
Cesar Chavez High School Monitor

Clinton Monitor

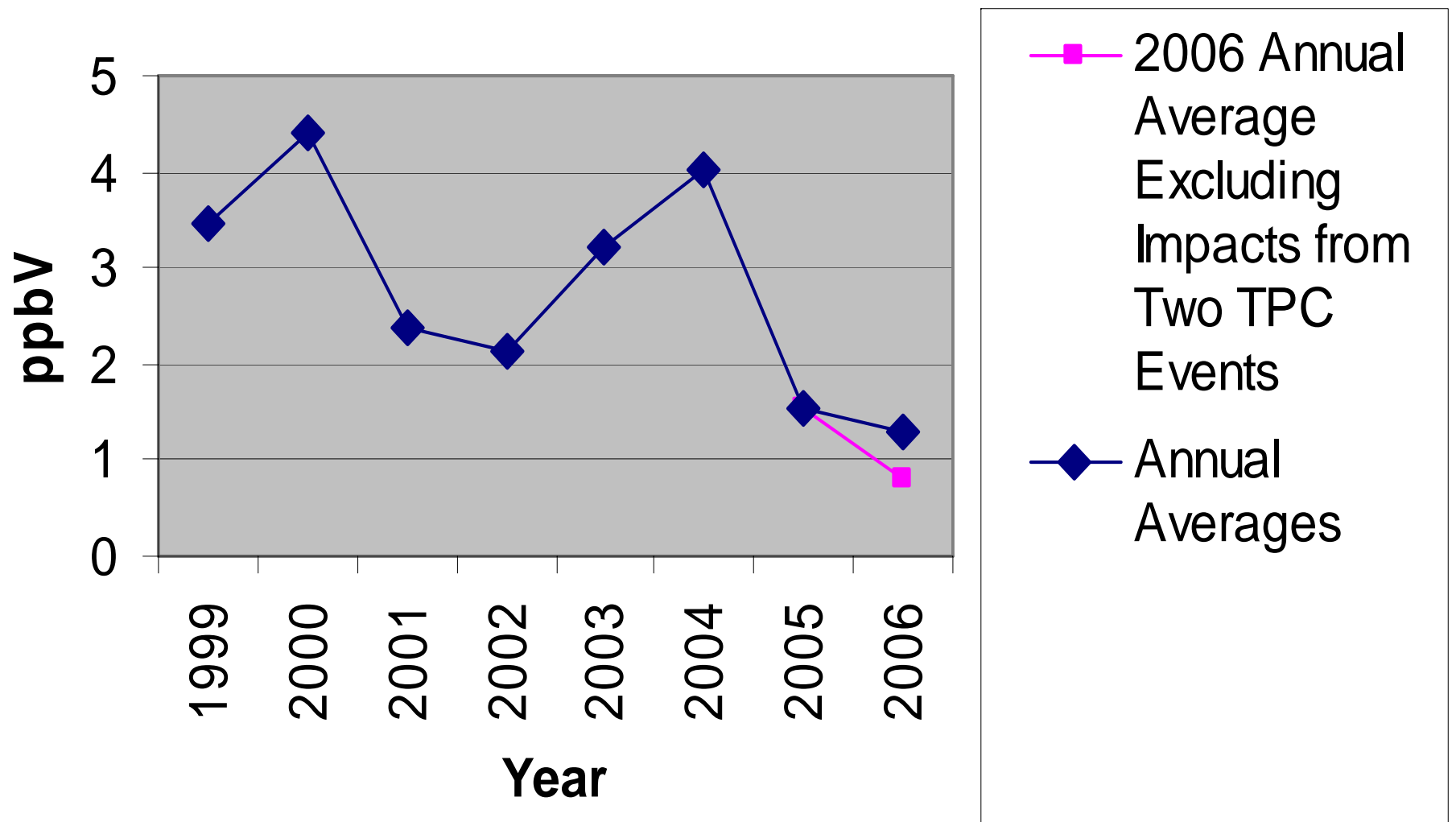
Milby Park Monitor

- TPC is located approximately 1/3 of a mile Southeast of the Milby Park Monitor.
- The Milby Park Monitor utilized a canister, every sixth day from April of 1999 until December of 2004.
- The Milby Park Monitor has utilized an Automated Gas Chromatograph since February 2005

Milby Park Monitor 1,3-Butadiene Annual Averages

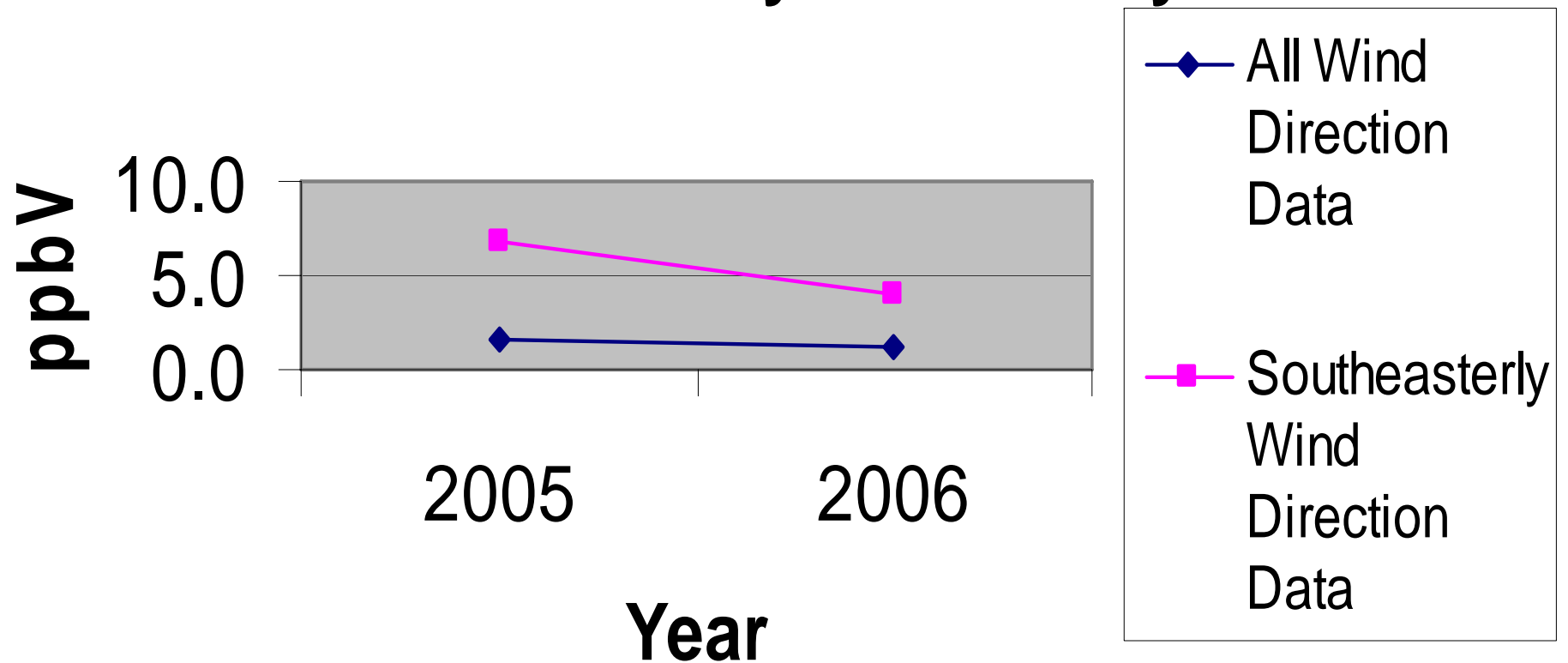


Milby Park Monitor 1,3-Butadiene Annual Average Concentrations



Milby Park Annual Average 1,3-Butadiene Concentrations

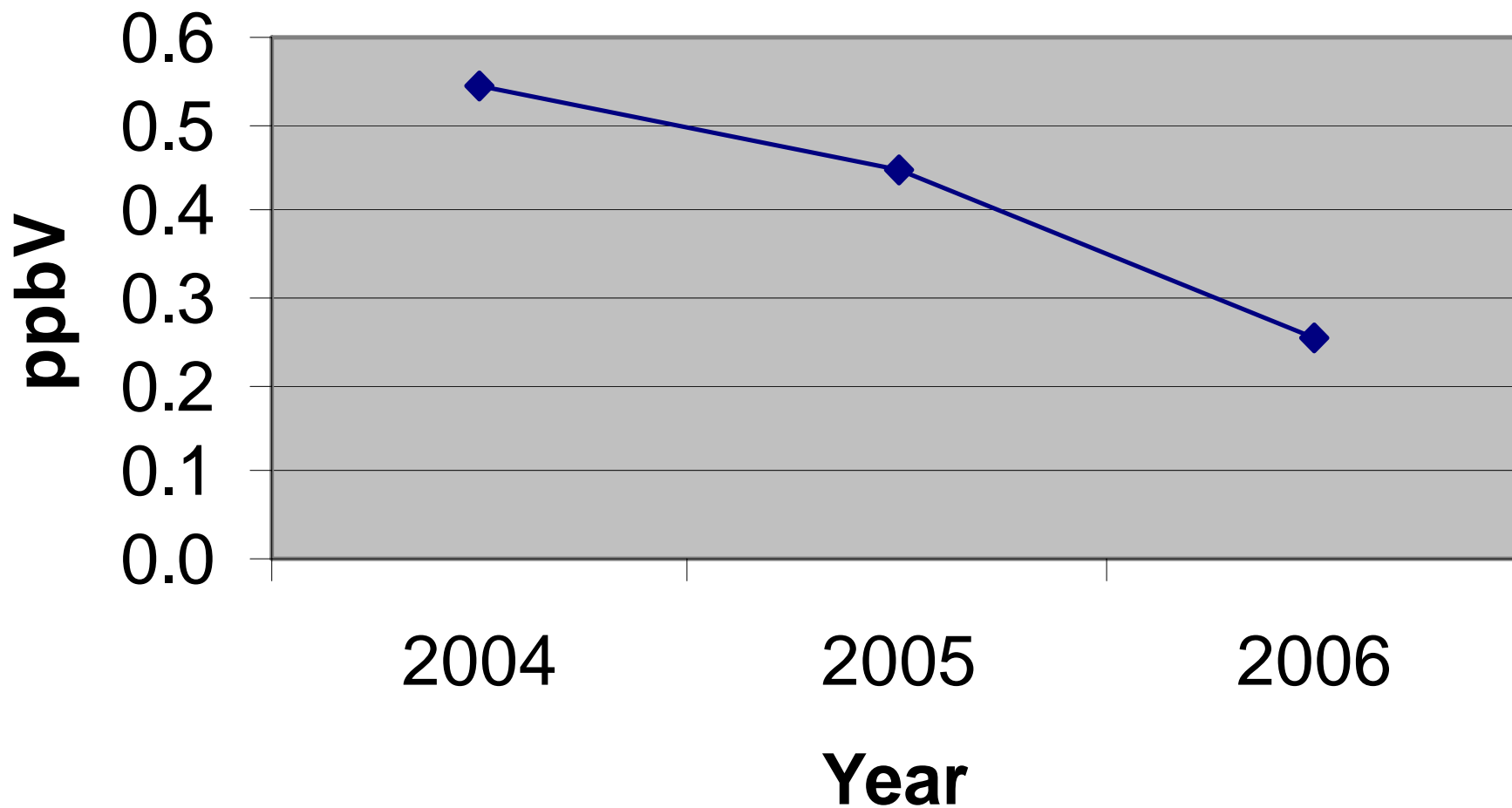
All Wind Directions Compared to Southeasterly Winds Only



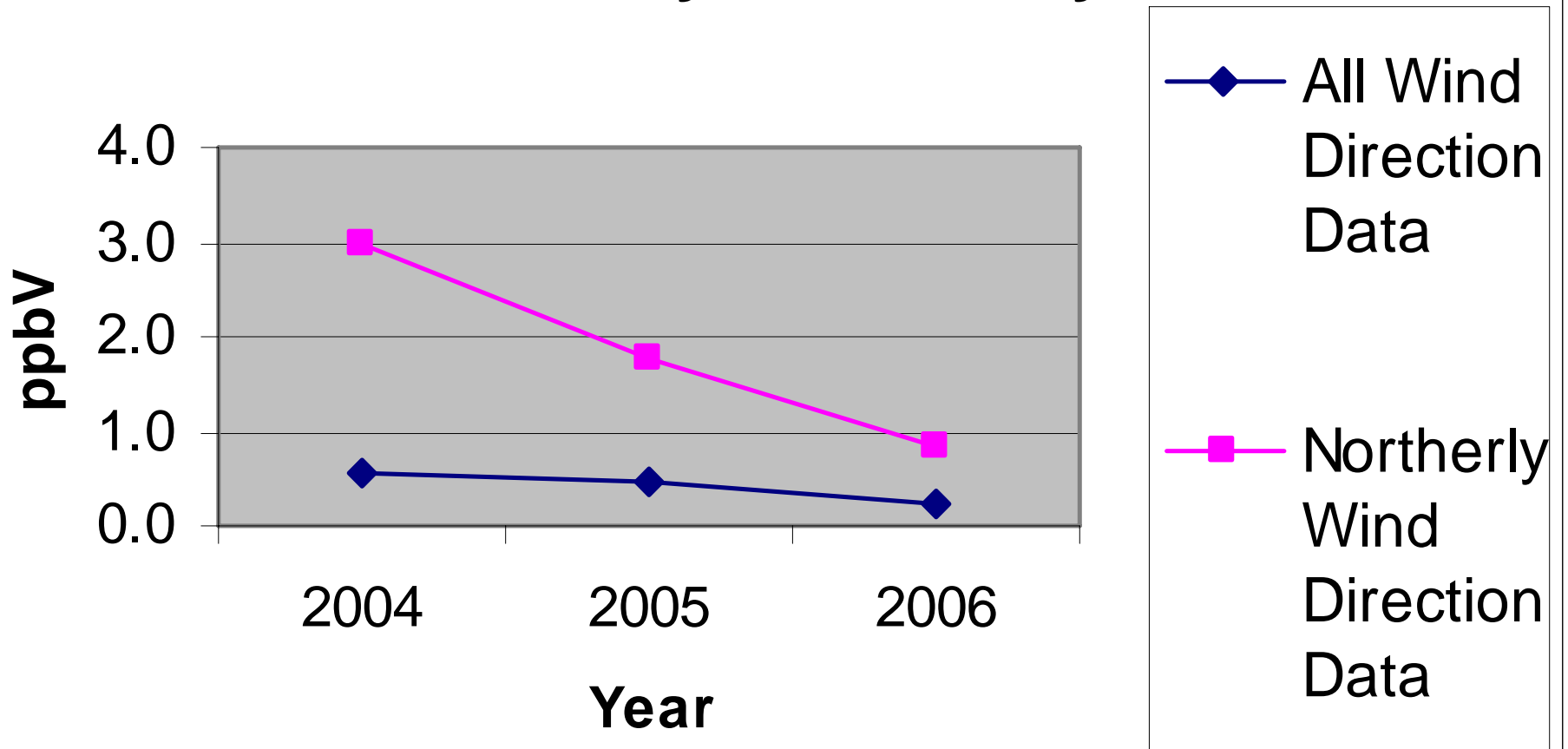
Cesar Chavez High School Monitor

- TPC is located approximately 2/3 of a mile North of the Cesar Chavez High School Monitor.
- The Cesar Chavez High School Monitor has utilized an Automated Gas Chromatograph since April 2004.

Cesar Chavez High School 1,3-Butadiene Annual Averages



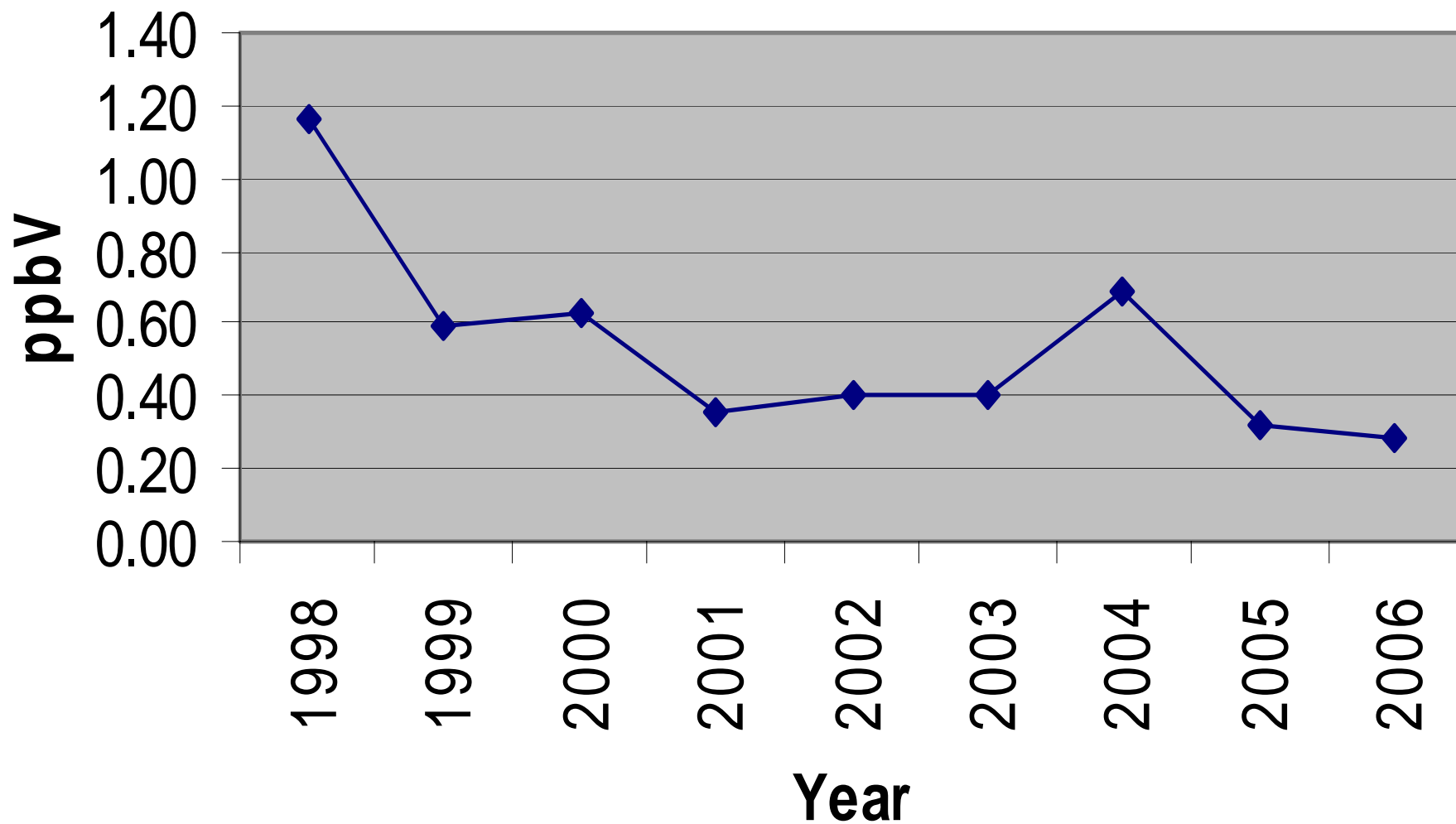
Cesar Chavez High School 1,3- Butadiene Annual Averages All Wind Directions Compared to Northerly Winds Only



Clinton Monitor

- TPC is located approximately 2 ¼ miles South of the Clinton Monitor.
- The Clinton Monitor has utilized an Automated Gas Chromatograph since before 1998.

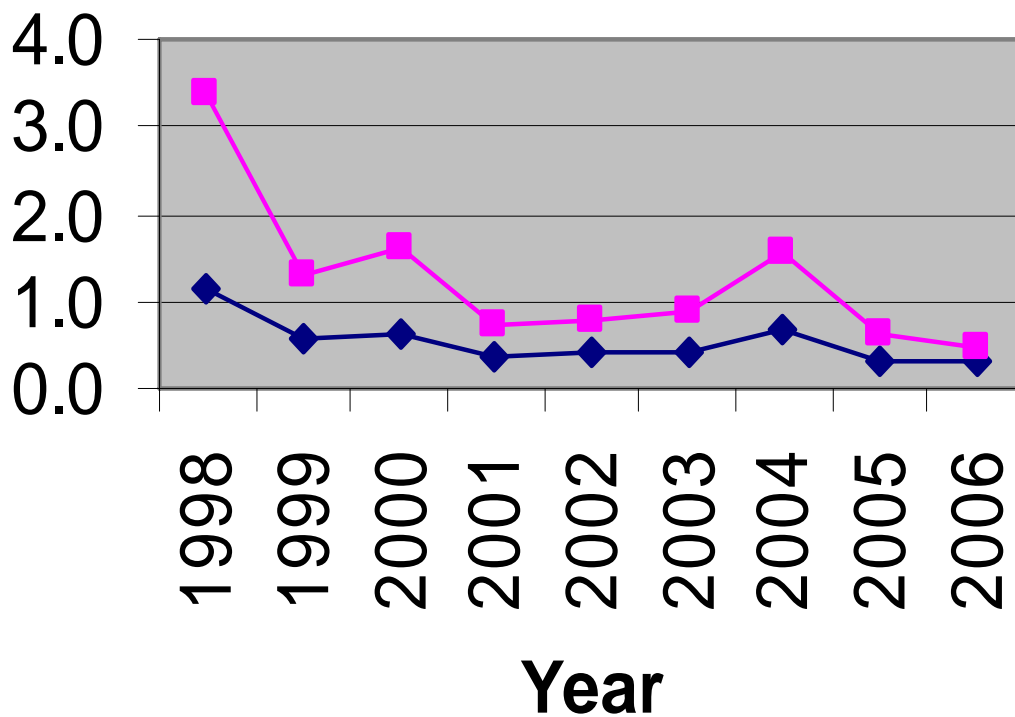
Clinton 1,3-Butadiene Annual Averages



Clinton 1,3-Butadiene Annual Averages

All Wind Directions Compared to Southerly Winds Only

ppbV



◆ All Wind Direction Data

■ Southerly Wind Direction Data

Conclusions

- The City began working with TPC on butadiene reductions in February 2005, followed by the TCEQ in the Spring of 2005, resulting in 1,3-Butadiene (BD) emissions reduction agreements with TPC in December of 2005.
- The annual BD concentrations have decreased significantly since 2004. Two major emissions events at TPC caused the improvements from 2005 to 2006 to be less significant.
- The difference between the annual BD concentrations at each site and the annual BD concentrations when the wind blows from the direction of TPC has decreased from 2004 to 2006, indicating that the BD impact from TPC has decreased from 2004 to 2006.