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Investigation of an association between childhood leukemia incidences and airports in Texas

Sala N. Senkayi [1], Melanie L. Sattler [2], Nancy Rowe [3], Victoria C.P. Chen [4]

[1] *U.S. Environmental Protection Agency (EPA) Region 6, 1445 Ross Ave., Suite 1200 (6WQ), Dallas, TX, 75202*
[2] *Department of Civil Engineering, The University of Texas at Arlington, Box 19308, Arlington, TX 76019*
[3] *Office of Information Technology, The University of Texas at Arlington, Box 19318, Arlington, TX 76019*
[4] *Department of Industrial and Manufacturing Systems Engineering, The University of Texas at Arlington, Woolf Hall, Room 420, Arlington, TX, 76019*

ABSTRACT

As worldwide demand for air travel increases, emissions from airports will likely also increase. Airport emissions pose a concern due to lack of information about their quantity and impacts on human health and the environment. This research aimed to address the question of whether there is an association between childhood leukemia cases and airport emissions in Texas. Rather than looking at the impacts of a single airport on the surrounding community, this study looks at all airports in the state of Texas, and 2 134 incidences of childhood leukemia (children age 9 and under) state–wide over a 10–year period. The distance to airports of block groups with standardized incidence ratios >100 for childhood leukemia was found to be shorter than the distance to airports for block groups with standardized incidence ratios <100, to a 98% level of confidence. A Poisson regression model was developed to estimate incidences of childhood leukemia, based on county–wide benzene emissions. Benzene emissions from airports were found to be a statistically significant predictor variable. The two analyses provide evidence of an association between airports and incidences of childhood leukemia in Texas.

Keywords: *Airports, emissions, benzene, childhood leukemia, geographic information systems*

Corresponding Author:
Sala N. Senkayi
☎ : *+1-214- 665-6477*
🖷 : *+1-214- 665-7373*
✉ : *senkayi.sala@epa.gov*

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1. Introduction

Emission sources at airports include aircraft engines, ground support equipment (GSE), ground access vehicles, and auxiliary power units (APUs). Emissions from these sources pose a concern due to lack of information about their quantity and impacts on human health and the environment (TRB, 2008). As worldwide demand for air travel increases, these emissions will likely also increase. The Federal Aviation Administration (FAA) forecasts that domestic aircraft operations will increase approximately 1 percent annually through 2020, and that passenger enplanements will increase 3 percent annually (FAA, 2007). Texas currently has two airports ranked among the top 10 U.S. airports in terms of enplanements: Houston ranks 7th, while Dallas ranks 3rd.

Air pollutants associated with airports include criteria pollutants (particulate matter, sulfur dioxide, nitrogen dioxide, carbon monoxide, and ozone precursors), as well as hazardous air pollutants. Among hazardous air pollutants, a number of studies have measured benzene in aircraft exhaust (Anderson et al., 2006; Herndon et al., 2006; Environ International Corporation, 2008; Wood et al., 2008). Most aircraft emissions arise from idling (at the gate and awaiting takeoff), taxiing, taking off, and landing (Kinsey et al., 2012). Benzene has also been measured in the exhaust from aircraft auxiliary power units (Kinsey et al., 2012), as well as in automobile exhaust (Amigou et al., 2011).

Benzene is toxic to hematopoietic stem cells or progenitor cells, from which all leukemias and related disorders arise (Smith et al., 2011). This lowers blood counts, producing hematotoxicity. In adults, consensus clearly shows that benzene causes acute nonlymphocytic leukemia (ANLL), particularly the acute myeloid leukemia (AML) type and precursor myelodisplastic syndromes (MDS), even at relatively low doses (Wilbur et al., 2008; Galbraith et al., 2010; Smith, 2010; IARC, 2012). Evidence has grown that benzene exposure can initiate acute lymphocytic leukemia (ALL) as well; benzene causes chromosomal rearrangements and mutations that are part of the cause of both AML and ALL (Smith, 2010).

A number of studies suggest that benzene exposure also increases the risk of childhood leukemia, based primarily on indirect evidence. Road traffic exhaust is a source of low doses of benzene (Smith and Zhang, 1998; Duarte–Davidson et al., 2001). Vinceti et al. (2012) found that exposure to benzene from motorized traffic appeared to be associated with an excess risk for childhood leukemia (particularly AML) in a northern Italian community. Ghosh et al. (2013) found support for a link between prenatal exposure to traffic exhaust and risk of ALL in Los Angeles County, California, US. The study considered NO_2 levels to represent traffic–related air pollution, but not direct levels of benzene. Amigou et al. (2011) found a significant association between childhood leukemia in France and a high–density of heavy–traffic roads within 500 m of the place of residence, although benzene levels were not specifically quantified. Steffen et al. (2004) found an association between acute childhood leukemia

and dwellings neighboring auto repair garages and gas stations, both sources of benzene emissions. Brosselin et al. (2009) also found an association between living next to a gas station and acute childhood leukemia in France. Whitworth et al. (2008) found that census tracts in Houston, Texas, with the highest benzene levels estimated by U.S. Environmental Protection Agency models had elevated rates of all leukemias.

Exposure of the mother to benzene could be just as important as childhood exposures in producing childhood leukemia (Smith, 2010). A number of studies have shown that childhood ALL and AML are typically initiated in utero because leukemic translocations and other genetic changes are present in blood spots collected at birth (Wiemels et al., 1999; Wiemels et al., 2002; Greaves and Wiemels, 2003; McHale et al., 2003; Eden, 2010). Several studies have reported a positive association between maternal occupational exposure to hydrocarbons and childhood leukemia (Shu et al., 1988; van Duijn et al., 1994; Shu et al., 1999), with particularly strong correlations for benzene. Recent animal studies support the hypothesis that childhood leukemias are initiated in utero (Lau et al., 2009; Badham et al., 2010). Badham et al. (2010) demonstrated that transplacental benzene exposure can induce hepatic and hematopoietic tumors in mice, which may be dependent on fetal benzene metabolism capability. Bonaventure et al. (2012), however, found no association between maternal smoking during pregnancy and AML or ALL.

A number of studies have evaluated cancer risk in the vicinity surrounding airports. Visser et al. (2005) found moderately increased risk of non–Hodgkin lymphoma and acute lymphoblastic leukemia in the area around Amsterdam Airport Schiphol, but this risk could not be explained by higher levels of ambient air pollution in the area. Focusing on benzene, 1,3–butadiene, and benzo [a]pyrene, Zhou and Levy (2009) estimated the emission rates required at 32 airports across the U.S. to exceed a 10^{-6} lifetime cancer risk for the maximally exposed individual (emission thresholds) and estimated the total population risk at these emission rates. A study of air toxics risks from O'Hare International Airport in Chicago, Illinois (ORD) estimated that cancer risks associated with the airport exceeded 10^{-6} for a 1 000 square mile area surrounding the airport, with a maximum individual risk (MIR) of 10^{-4} (Environ International Corporation, 1999). Vanderslice and Fulton (2012) examined lung cancer incidence rates for Warwick, RI, and concluded that airport emissions were one of several factors that could potentially explain geographical distribution of lung cancer cases. Yim et al. (2013) developed an inventory of U.K. airport emissions, and then used an air quality model to assess air quality impacts and early deaths due to cardiopulmonary disease and lung cancer caused by $PM_{2.5}$ exposure.

To evaluate whether airport emissions pose a health risk, some of the previous studies mentioned above used risk assessment (Zhou and Levy, 2009; Yim et al., 2013), based on:

(a) Emission estimates for airport sources, based on measurements or emissions models like Emissions and Dispersion Modeling System (EDMS),

(b) Ambient concentrations of pollutants surrounding the airport, based on measurements or dispersion models like AERMOD,

(c) A human exposure model and coupled dose–response risk assessment model to estimate human risk.

Other studies (Visser et al., 2005; Vanderslice and Fulton, 2012) have used epidemiological approaches that examine statistical correlations between cancer incidence rates (expected and actual) and risk factors, such as proximity to an emission source.

This study takes advantage of the capabilities of Geographic Information Systems (GIS) to analyze large quantities of spatially–based data, in order to expand the geographic scope of the epidemiological analysis. Rather than looking at the impacts of a single airport on the surrounding community, this study looks at all airports in the state of Texas, and incidences of childhood leukemia state–wide, to determine whether an association may exist; this includes evaluation of more than 2 134 childhood leukemia cases over a 10–year period. This is the first study to our knowledge to investigate the geographical association between airport emissions and childhood leukemia incidence. Although previous studies have examined cancer incidences in the proximity of airports, no previous study to our knowledge has examined specifically childhood leukemia incidences in the proximity of airports.

This study thus aims to address the question of whether there may be an association between airport emissions and childhood leukemia in Texas. Specifically, it examines the questions of:

- Whether proximity to airports can explain geographic distribution of childhood leukemia cases in Texas, and
- Whether childhood leukemia incidences county–wide can be correlated with benzene emissions from airports.

2. Methodology

To evaluate whether there may be an association between childhood leukemia cases (children age 9 and under) and airport emissions in Texas, two approaches were used:

(1) Comparison of the distance to airports of census block groups with high standardized childhood leukemia incidence ratios to block groups with low standardized incidence ratios.

(2) Development of a regression model to predict childhood leukemia incidences by county in Texas based on benzene emissions from various sources (airports, railroads, industrial facilities, roads).

Each of these approaches will be discussed in turn.

2.1. Comparison of the distance to airports of census block groups with high childhood leukemia standardized incidence ratios to block groups with low standardized incidence ratios

To calculate standardized incidence ratios, the observed incidence (the observed cancer cases per population in a given geographic area, per time period of interest) was divided by the expected incidence (the expected cancer cases per population in a given geographic area, per time period of interest). Standardized incidence ratios for childhood leukemia were calculated for block groups state–wide. Data used to calculate observed–to–expected–incidence ratios is discussed below.

Block group and census tract data. A shape file containing GIS information for Texas was obtained from SimplyMap (SimplyMap, 2009). Spatial data in the form of polygon shape files and demographic data (race, gender and age) for Texas' 254 counties were obtained from the U.S. Environmental Protection Agency (EPA) Region 6 (R6) GIS Group. In addition, EPA R6 GIS Group also provided demographic data (race, gender and age) at the block group level, which was joined to block group polygon shape files in GIS; this data represented the total population of those 9 years and under for the year 2000 (all other data were not used). Block group and census tract shape files for the state of Texas were obtained from Environmental Systems Research Institute (ESRI), including 14 463 block groups and 4 388 census tracts (ESRI, 2010). A census block group is the smallest geographical unit for which the United States Census Bureau publishes sample data.

Observed cancer incidences. The Department of State Health Services (DSHS) provided 10 years of cancer data (from 1995–

2005) for Texas (DSHS, 2008). The database included 925 781 total observed incidences of all types of cancer. Incidences of childhood leukemia (all kinds) were pulled from the database. Cancer incidences that fell outside of Texas (most likely due to the submission of permanent addresses rather than current residence) and those with inconclusive latitude/longitude values were eliminated. The Cancer Incidence Data contained latitude/longitude values of patient residences at diagnosis, which were used to upload this shape file into GIS.

The block group shape file was joined with the cancer shape file to generate a new count column representing the leukemia incidences within each block group. Since greater numbers of cancer cases are expected to be found in population centers simply due to the larger population, cancer cases in a given geographic area are typically divided by the population of the area to provide an idea of how many incidences occurred within these areas for that timeframe (cases/population). The cancer data spanned 10 years, while the census data was only for the year 2000; thus, the Census 2000 Population Data was multiplied by 10 in order to account for all 10 years of the cancer data.

Block group population numbers range from very small (e.g. 0) to very large (e.g. 4 224) populations. It is important to note that migration patterns of individuals within these block groups could not be accurately tracked for this study since that information was not provided by DSHS; however this could be useful for future studies similar to this one.

Expected leukemia incidences. Expected cancer incidences are cancer incidences that are typically common for a given area during a defined length of time. In order to calculate the expected leukemia incidences, the Census 2000 Population Data (which had been multiplied by 10 years) was multiplied by rates specific for the differing age, race and gender groups, thus adjusting for those inherent differences. The state–wide rates used in this study were obtained from the Texas Department of State Health Services (DSHS) (DSHS, 2005), as shown in Table 1. The Center for Disease Control documents how these rates are obtained (CDC, 2006).

Table 1. *Selected Texas cancer rates (Texas Department of State Health Services)*

Type of Rate		Childhood Leukemia (0–9 years) per 1 000 000
All Races	Male	69.6
	Female	51.7
White Non–Hispanic	Male	66
	Female	51.2
Black	Male	27.7
	Female	27.7
Hispanic	Male	86.2
	Female	59.1
Other Race	Male	69.7
	Female	51

Calculation of standardized incidence ratios. The observed cases were simply divided by the aggregate expected cases (which had already been adjusted for age, race and gender) for each block group within the State. Since calculations were performed at block group levels, which are much smaller than census tracts and counties, we obtained some very large ratios. However, these calculations were very useful in that we were able to notice minute associations between higher observed–to–expected–incidence ratios and visibly closer distances to certain emitters, as discussed below.

Plotting the standardized incidence ratios for each block group vs. distance to airports using GIS. Shapefiles containing locations of all airports in Texas were provided by EPA. A distance from each leukemia incidence to the nearest airport was obtained using the "near to" tool in GIS. For each block group, the standardized incidence ratio was then plotted versus the average distance of the leukemia incidences to airports. Because of the log scale, block groups with observed–to–expected incidence ratios of zero do not appear.

2.2. Development of a regression model to predict childhood leukemia incidences by county in Texas, based on benzene emissions from various sources (airports, railroads, industrial facilities, roads)

Table 2 describes the benzene emissions data obtained from U.S. EPA 1999 National–Scale Air Toxics Assessment (NATA) (U.S. EPA, 1999a). The emissions data in NATA is from EPA's 1999 National Emissions Inventory (U.S. EPA, 1999b). Sources of industrial emissions estimates in the NEI include state/local/tribal governments, Maximum Achievable Control Technology (MACT) emissions rate, Toxics Release Inventory (TRI), industry, and the 1996 NTI. The 1999 NEI nonpoint source estimates were developed by combining emission factors with activity data, from information provided for MACT source categories, and from data and revisions provided by state and local agencies (U.S. EPA, 1999b).

SPSS was used to develop a Poisson regression equation to relate county–wide childhood leukemia incidences to county–wide emissions of benzene from airports, railroads, industrial facilities, and roads, as follows:

$$\ln(L)=\beta_0+\beta_1\ln(P)+\beta_2\ln(B_{airports})+\beta_3\ln(B_{railroads})+\beta_4\ln(B_{industrial\ facilities})+\beta_5\ln(B_{roads}) \quad (1)$$

where, L is the leukemia incidences in children 9 years and under within the county, βs are the parameters to be determined using Poisson regression, P is the population of individuals 9 and under in the county in the year 2000, $B_{airports}$ is county–wide benzene emissions from airports (tons/year), $B_{railroads}$ is the county–wide benzene emissions from airports (tons/year), $B_{industrial\ facilities}$ is the county–wide benzene emissions from industrial facilities (tons/year), and B_{roads} is the county–wide benzene emissions from roads (tons/year).

Table 2. *Data for major benzene emission sources, U.S. Environmental Protection Agency*

Shape File		Year Data Represents	Number (ex: Points, Segments, Polygons, or Items)
Airport benzene emissions		2005	1 966 Points
Toxic Release Inventory (TRI) Facilities – Benzene Emissions		2005	247 Points
Roads	GIS – (Interstate, Major Road, State Highway and US Highways only)	2000	25 131 Segments
	Statistics: County Level Benzene Emissions	2005	254 Items
Railroads	GIS	2000	47 287 Segments
	Statistics: County Level Benzene Emissions	2005	254 Items

Emissions from other potentially significant sources of benzene were included, in addition to airports, because they may also be important in predicting childhood leukemia incidences. Including benzene emissions from various types of sources separately in the model allows us to determine their relative importance. The influence of various sources of benzene emissions on leukemia would be expected to be different, due to the differing distances from various sources to impacted populations, and due to the various release configurations that would influence dispersion (e.g. elevated vertical stack vs. ground–level tailpipe release).

The regression equation was developed using data from 251 of Texas' 254 counties. Data from 3 counties (1.2%) was excluded from the analysis since these items contained zeros and missing values. Logs were taken of the predictor variable values because the data was not normally distributed and did not fit linear assumptions in its raw form (as demonstrated by SPSS scatter plots); thus the data needed to be transformed, increasing normality and ensuring a better linear model.

3. Results and Discussion

3.1. Comparison of the distance to airports of census block groups with high standardized incidence childhood leukemia incidence ratios to block groups with low standardized incidence ratios

Figure 1 shows plots of standardized incidence ratios, or observed-to-expected incidence ratios for each block group vs. average distance of each block group to the nearest airport. A number greater than 1 on the vertical axis indicates that more cases were observed than what was expected. The data points are color coded by standardized incidence ratio as follows:

- >500 – red
- 500>ratio>100 – blue
- 100>ratio>10 – green
- 10>ratio>1 – yellow
- 1>ratio – gray

In order for an association to exist between disease and the source, the points with larger observed–to–expected ratios should be visibly closer to the source, compared to the points of other colors. The red and blue data points appear from visual inspection to be closer on average to the y–axis (or closer to the emission source) than the rest of the data points. To confirm this, a *t*–test was conducted, with results presented in Table 3. The mean distance to the airport for the points in the red and blue groups was significantly shorter than the points in the green, yellow and gray groups, to a level of confidence of 98%. Thus, the block groups with the largest observed–to–expected childhood leukemia incidence ratios were generally closer to airports, with a 98% level of confidence.

3.2. Development of a regression model to predict childhood leukemia incidences by county in Texas, based on benzene emissions from various sources (airports, railroads, industrial facilities, roads)

The regression equation developed is shown below:

$$\ln(L) = -6.664 + 0.440 \ln(P) + 0.497 \ln(B_{roads}) + 0.230 \ln(B_{airports}) \quad (2)$$

or, alternately,

$$L = 0.00128\, P^{0.440}\, B_{roads}^{0.497}\, B_{airports}^{0.230} \quad (3)$$

where, L is the leukemia incidences in children 9 years and under within the county, P is the population of individuals 9 and under in the county in the year 2000, $B_{airports}$ is the county–wide benzene emissions from airports (tons/year), B_{roads} is the county–wide benzene emissions from roads (tons/year).

It should be noted that correlations in Equations (2) and (3), in and of themselves, do not imply causality. Animal studies, cited in the introduction, do lend support to a biological mechanism for benzene to cause leukemia.

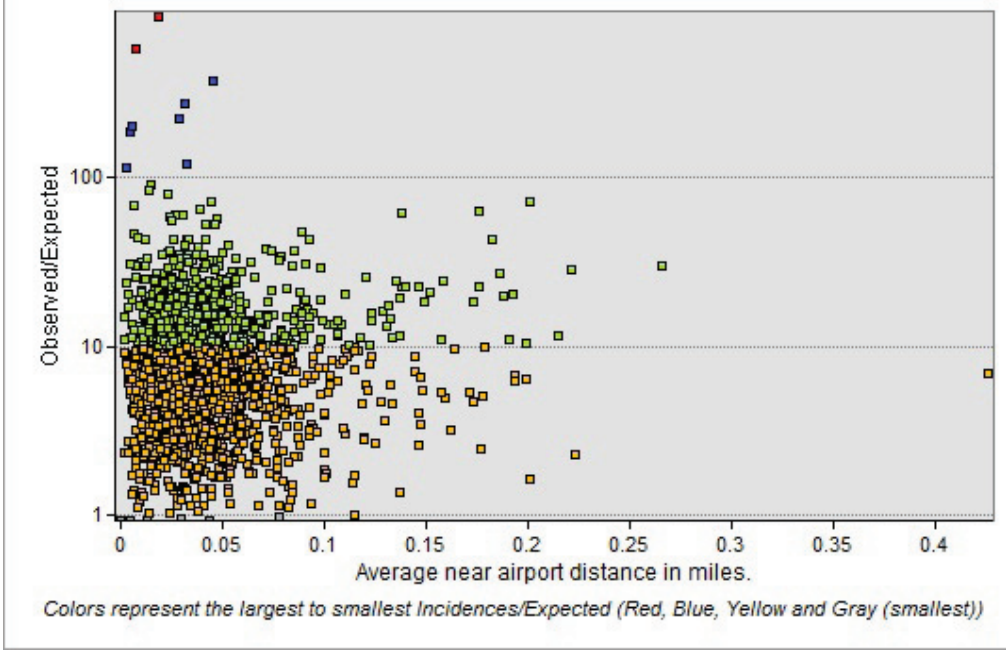

Figure 1. *Scatter plot of observed–to–expected–incidence ratios for leukemia for children age 9 and under, versus average near distance to airports in miles at the block group level.*

Table 3. *t–test comparing mean distance of block groups with standardized leukemia incidence ratios >100 with mean distance of other block groups with incidence ratio <100*

Parameter	Value	
	Incidence Ratio >100 (Red and Blue)	Incidence Ratio <100 (Yellow and Gray)
Mean (mi.)	0.020	0.042
Standard Deviation (mi.)	0.015	0.033
N	9	1 862
df	1 869	
$t_{Calculated}$	–2.056	
$t_{Critical}$ – 99%	2.326	
$t_{Critical}$ – 98%	2.054	
$t_{Critical}$ – 95%	1.645	
Level of confidence to which means are significantly different	98%	

Table 4 provides minimum, maximum, mean, and standard deviation values for the response and predictor variables, which indicate the range of values over which the regression equation applies. Table 5 indicates that population and benzene emissions from roads and airports are statistically significant predictor variables for childhood leukemia incidences, as demonstrated by the beta (β) values. A goodness of fit test was run and yielded a value of 1.949, which indicates a good fit for the model since it is much less than 5 and close to 1. Benzene emissions from U.S. EPA Toxic Release Inventory (TRI) facilities did not demonstrate a strong relationship with childhood leukemia when viewed in an SPSS scatter plot of dependent vs. independent variables; thus benzene emissions from TRI facilities were eliminated as a predictor variable. Railroad benzene emissions were initially included as a potential predictor variable, but its coefficient was not statistically significant at a 95% level of confidence; therefore, it was eliminated from the final model.

Table 4. *Poisson regression model response and predictor variable information*

Variable	N	Min.	Max.	Mean	Std. Deviation
L	251	0.0	408.0	8.49	34.08
ln P	251	8.42	20.13	14.52	1.77
ln B_{roads}	251	–0.09	6.38	2.5	1.2
ln $B_{airports}$	251	–4.73	2.74	–1.82	1.48

In Equation (2), the coefficient (0.497) for the term involving benzene emissions from roads is larger than the coefficient (0.230) for the term involving benzene emissions from airports [In Equation (3), these coefficients become exponents]. The larger coefficient for roads indicates a stronger relationship between childhood leukemia incidences roads: increasing benzene emissions from roads by a given percent per year will cause a larger increase in leukemia incidences than increasing airport emissions by the same percent. This is not surprising given the lesser distance between residences and roads, compared to residences and airports. In addition, road releases occur at ground–level, close to locations of human exposure. Although some airport releases occur near ground–level (auxiliary power units, automobiles, and taxiing aircraft), other emissions from aircraft occur above ground–level, farther from locations of human exposure. Releases above ground–level are generally subject to higher wind speeds, which provide quicker dilution.

3.3. Study strengths and limitations, recommendations for future work

Limiting the analysis to childhood leukemia may have reduced several confounding factors associated with adult exposure history, including smoking and workplace exposure. Exposure to second–hand smoke, particularly from parental smoking, was not ruled out, however. Parental occupational exposures, particularly during pregnancy, could also have confounded study results. Socio–economic status (SES) could be related to proximity of the residence to the airport (Brainard et al., 2004; Ogneva–Himmelberger and Cooperman, 2010; Correia et al., 2013). Those with lower SES could both live closer to airports and have more parental occupational exposures in key time periods than those in with higher SES.

Exposure history linked to other locations (including moving from another city/state or country after having lived there for 50 years or so) is a source of exposure misclassification for adults, which is less of an issue for children (Syracuse Research Corporation, 2007). This study implicitly assumes that the address at diagnosis is an adequate proxy for the address during pregnancy, which may be the relevant window of exposure for childhood leukemias. ALL dominates childhood leukemia, with cases of AML being only 15% of the number of cases of ALL. For ALL, diagnosis occurs at 2–6 years (median 4 years), with a somewhat higher male to female ratio (1.2:1) (Eden, 2010; IARC, 2012). The extent to which residential mobility may have affected the results (likely biasing the relative risk/coefficients towards the null) is not known. Data was not available to track the migration patterns of individuals within the 14 463 block groups included in the study. This is a potentially significant limitation of the study.

Restricting the analysis to childhood cases should have helped to address the issue of uncertainty in cancer latency periods, since the maximum length of latency is clearly defined. For example, for children less than 10 years old with leukemia diagnosed during 1995–2005, their etiologically relevant window of time would likely be from 1985 to 2000, assuming 5–10 year latency. Confounding exposure to pollutants besides benzene is another study limitation.

Indirect standardization was used in calculating the standardized incidence ratios (Boyle and Parkin, 1991; Bains, 2009; NHS Public Health Network, 2013). Indirect standardization is recommended for rare events, like childhood leukemia incidents (Fitzpatrick, 2009). The primary limitation of indirect standardization, however, is that ratios from different areas cannot be directly compared. Since Texas state–wide leukemia rates were used to calculate the expected incidences, block group rates can be compared to Texas state–wide rates but not directly to each other. In addition, the choice of the standardized set of rates for the expected values impacts the resulting ratios (Ghilagaber and Hedlin, 2010).

Table 5. *Poisson regression model parameter estimates*

Parameter	β	Std. Error	95% Wald Confidence Interval		Hypothesis Test		
			Lower	Upper	Wald Chi–Square	df	Sig.
(Intercept)	–6.664	0.6643	–7.967	–5.362	100.631	1	0.000
ln P	0.440	0.0507	0.340	0.539	75.221	1	0.000
ln B_{roads}	0.497	0.0712	0.358	0.637	48.811	1	0.000
ln $B_{airports}$	0.230	0.0414	0.149	0.311	30.813	1	0.000
(Scale)	1.765						

Like all emissions inventory data, the benzene emissions data used to develop the regression model were subject to uncertainty. In addition, only benzene emissions from airports, railroads, roadways, and industrial sources were considered in model development, as these were the cleanest sources of data available; emissions from other sources, such as gas stations, were not considered. Emission rates from the sources considered were assumed to be constant over the 10–year exposure period. The regression equation coefficients were significant to a 95% level of confidence, meaning there is a 5% chance that they were not significant.

A strength of our analysis was the large quantities of data analyzed; a limitation was that specific details associated with each airport were not considered. These specifics include meteorological patterns of wind speed, wind direction, and atmospheric stability that impact dispersion of pollutants from particular airports, and benzene sources within a particular airport, such as aircraft emissions vs. those from auxiliary power units.

Future work should compare the risk of developing childhood leukemia from airport benzene exposure, compared to exposure from roadways and other sources.

4. Conclusions

The two approaches used in this study reinforce the conclusion that an association exists between airport emissions and incidences of childhood leukemia in the State of Texas.

- Block groups with observed–to–expected incidence ratios for childhood leukemia >100 were generally closer to airports than block groups with ratios <100 in Texas, with a 98% level of confidence.

- County–wide airport benzene emissions were a statistically significant predictor of incidences of childhood leukemia county–wide in Texas.

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