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

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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*

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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–