THE ASSOCIATION BETWEEN LUNG FUNCTION AND CUMULATIVE EXPOSURE TO PARTICULATE MATTER ($PM_{2.5}$) AND TRAFFIC-RELATED EXPOSURES

by

Anjani R. Parikh

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This thesis was presented

by

Anjani R. Parikh

It was defended on

April 15, 2015

and approved by

Fernando Holguin, MD, MPH
Associate Professor of Medicine
Assistant Director, Asthma Institute
University of Pittsburgh Medical Center

Jane E. Clougherty, ScD
Assistant Professor
Director, Exposure Science
Department of Environmental and Occupational Health
Graduate School of Public Health, University of Pittsburgh

Thesis Director: Mary E. Hawk, DrPH
Assistant Professor
Senior Associate Director, Evaluation Institute
Department of Behavioral and Community Health Sciences
Graduate School of Public Health, University of Pittsburgh

Mary E. Hawk, DrPH

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ABSTRACT

Introduction of Literature: Air pollution has been related to acute and chronic respiratory health effects in asthmatics for a number of years. These chronic exposures to high levels of traffic density and particulate matter (PM 2.5) lead to significant decrements in lung function. Historically, the city of Pittsburgh has been known to have high levels of air pollution, (6^{th} Highest per American Lung Association) – but little to no analysis has been done on this population to document the respiratory health impact of traffic density levels and particulate matter exposure.

Methods: The sample population of this study was comprised of Registry Participants from The Asthma Institute at The University of Pittsburgh Medical Center in Pittsburgh, PA. Patients that have had lung function tests recorded by the Asthma Institute, and also live at residences that were able to be geocoded by the Department of Environmental and Occupational Health at the University of Pittsburgh Graduate School of Public Health were included in the final analysis (n= 452). We used the LUR to create a spatial smoothed surface of PM concentrations across the area, then averaged, for each residence, the 100-m cell centroid predictions from this surface that fell within 300-m of each home. Regulatory data was used to adjust these measures to estimate PM at each location for the month prior to lung function testing. We used linear regression

analysis to determine: A) the linear relationship between exposures and lung function tests; and B) the association between exposures with road density exposures and their respective quartiles. We adjusted for race, age, and sex as confounders.

Limitations: There are some expected limitations to the findings of this study. First, because this is a cross-sectional study, we cannot assume any causation. Additionally, exposures to PM and traffic density are roughly estimated through GIS which may contain errors, and there are no indoor exposure readings available. Moreover, single measurements of lung function tests provide only a snapshot of asthma severity at the time they were recorded.

Results: The univariable analysis found living in areas with higher road density levels is associated with reductions in lung function, which may imply that asthmatics living in the Pittsburgh area that are exposed to higher levels of air pollution experience steeper airway function declines. However, when adjusting for the confounders of race, age, and sex, there was no true association found between the PFT test results and road density or $PM_{2.5}$ exposure.

Public Health Significance: From a public health perspective, this study may help planning committees better understand and recognize the necessity to monitor traffic related air pollution mechanisms in specific areas of Allegheny County. Individuals are exposed to manmade and natural air pollutants at all times of the day throughout the course of their lifetime. An individual that is exposed to larger amounts of air pollution, and has clinical asthma, may benefit from being knowledgeable about the effects of air pollution on his or her body, and how to limit their daily exposures.

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PREFACE

The data source, “Asthma Institute Registry”, was developed as a means to recruit research participants for various studies conducted at the Asthma Institute at the University of Pittsburgh Medical center. To all the brilliant physicians, nurses, project coordinators, and support staff at The Asthma Institute, thank you. This Thesis would not have been possible without your hard work and commitment to expanding the scope of clinical asthma research in Western Pennsylvania. To my mentor, Dr. Fernando Holguin, thank you for your constant support, words of wisdom, and confidence – without you, this project would not have been possible. It was an honor working with you these past two years.

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And finally, to my family, and friends - thank you for your constant love and support on all of my endeavors. This Thesis is dedicated to my late grandfather, Rashiklal Shah – the man who taught me to chase my dreams with integrity.

1.0 INTRODUCTION

Respiratory health is a multi-faceted, complex issue whose improvement often requires knowledge of a patient's medical and environmental history. In the State of Pennsylvania more than 9% of all adults have asthma. While the air in Pittsburgh, Pennsylvania is cleaner than it has been in the past few decades, the American Lung Association ranks the city as the one of the most polluted metropolises in the country. According to the American Lung Association's 2014 State of the Air Report, Pittsburgh is ranked number 6 for year-round particle pollution and short-term particle pollution.(American Lung Association, 2014b) According to the report, although air pollution levels have decreased in recent years, overall air quality has decreased since the 2013 report with increased PM exposure levels – giving the city a rating of F for air quality. In Allegheny County there are 265,000+ adults and children with asthma and the 149,00+ individuals with Chronic Obstructive Pulmonary Disease (COPD), and our current air quality places them at increased risk of further respiratory health complications. Several studies have associated higher levels of PM exposures with acute respiratory illness in people, as demonstrated by increased respiratory symptoms or hospitalizations.

The aim of this study is to examine the respiratory health impact of road density levels and particulate matter exposure by analyzing survey data collected at the Asthma Institute at the University of Pittsburgh Medical Center (UPMC). This thesis will begin with a review of the literature, examining each of the specific areas of interest to the study, that is, fine particulate

matter ($PM_{2.5}$) and road density exposures. The document will then describe survey methodology, study participants, and study measures. It will also describe the data collection processes and methods of analysis. The thesis will report results of the analysis and provide an interpretation of findings. Finally, the conclusion will provide recommendations and implications drawn from the data.

2.0 REVIEW OF LITERATURE

2.1 ASTHMA AND AIR POLLUTION

Asthma is defined as chronic lung disease that narrows and inflames the airways, causing periods of wheezing, chest tightness, shortness of breath, and coughing. It affects people of all ages but is often diagnosed during childhood. In the United States, 25 million people (8% of the overall population) have been diagnosed asthma, and 7 million of these people are children (National Heart Lung Blood Institute, 2014). Although the exact cause of asthma is unknown, genetics, allergies, respiratory infections, and environmental pollutants may play significant roles in adult-onset asthma (American Lung Association, 2014a). When an individual's airways react, the muscles around the airways tighten, narrowing the airways and reducing airflow into the lungs. Asthma attacks are also identified as flare-ups or exacerbations. There is no cure for asthma, but treatment through medication may help individuals with asthma better manage its symptoms, and reduction of outdoor exposures that trigger exacerbations may allow them to live more normal, active lives.

To better interpret results of this study it is important to understand the biological processes that occur when individuals with asthma are exposed to air pollution. In areas that have high concentrations of pollution, direct irritant and inflammatory effects on airway neurological receptors and epithelium have been observed (Guarnieri & Balmes). However, even in areas with

lower concentrations of pollution, such as those in high income countries like the United States, pollutants such as $PM_{2.5}$, nitrogen dioxide, and ozone levels induce airway inflammation and airway hyper-responsiveness. (Kirby, Hargreave, Gleich, & O'Byrne, 1987; Lippmann, 1989). Studies have also found that oxidative stress, a mechanism of severe asthma, has been associated with exposures to the previously identified pollutants (Esposito et al., 2014; Holmstrup et al., 2010; Suhaimi & Jalaludin, 2014) Pollutants can cause oxidative stress on the lungs, and the inability of antioxidant defenses to handle this increased level of oxidative stress after exposure is a major determinant of risk for adverse effects (MacIntyre et al., 2014).The findings of these studies highlight the association between pollutants and exacerbations, but the biological mechanisms that pollutants initiate are not fully understood, and are still under investigation.

The United Kingdom's Committee on the Medical Effects of Air Pollutants identified four main mechanisms as a part of a framework for understanding how air pollution contributes to the exacerbation of asthma. They are: 1) oxidative stress and damage, 2) airway remodeling; 3) inflammatory pathways and immunological responses, and 4) enhancement of respiratory sensitivity.(Crapo, 2003) Additionally, genetic variation, which, in part, regulate these mechanisms, may play a role in increasing an individual's susceptibility to asthma exacerbations related to air pollution exposure (McCunney, 2005).

Certain enzyme genes such as GSTM1, GSTP1, glutathione, and S-transferase, can modify an individual’s risk of responses during increased levels of oxidative stress (Li et al., 2013; Polosukhin et al., 2014). Immune response pathways are also affected by oxidizing pollutants, thus playing a role in the severity of asthma symptoms (Trejo Bittar, Yousem, & Wenzel, 2014). Ambient hydrocarbons and diesel-exhaust particulates significantly affect the epigenetic mechanisms of T-cell (Treg) functions (Gruzieva, Merid, & Melén, 2014; Hew et al.,

2015). Chronic exposure to hydrocarbons or diesel-exhaust particles also leads to the suppression of Treg functionality and increases asthma severity when assessed by lung function testing (Gruzieva et al., 2014). In vitro studies with lab rats have also suggested that allergic inflammation is a result of PM exposure (Saravia et al., 2014).

There is also evidence of pollution exposure effects on inhaled allergen responses in lung function and inflammatory responses to nitrogen dioxide, sulfur dioxide, and diesel-exhaust (Auerbach & Hernandez, 2012; Ezratty et al., 2014; Kodgule & Salvi, 2012). Studies have suggested that different mechanistic pathways may increase the effect of pollutant exposure by increasing the deposition of allergens in the airways when the allergens are carried into the airways by particles, thus increasing epithelial permeability because of oxidative injury (Bernstein, 2012). In conclusion, air pollutants such as $PM_{2.5}$, which may cause inflammation and lung remodeling, may lead to oxidative injury to the airways. These effects are more severe in individuals that may be genetically predisposed to inflammation, making them more susceptible to developing clinical asthma. It is important to note that the combination of atopy and air pollutants may increase the risks associated to inflammatory responses when allergens are inhaled by individuals with asthma (Kaji et al., 2014).

2.2 PARTICULATE MATTER (PM)

Particulate matter (PM) is a multifaceted mixture of small particles and liquid drops made up of “acids such as nitrates and sulfates, organic chemicals, metals, and solid or dust particles”. (United States Environmental Protection Agency, 2013) A particle’s size is directly linked to its potential capacity to cause adverse health problems. Particulate matter is described by its

aerodynamic equivalent diameter or AED. In research, particles are subdivided into AED categories based on how the particles are created and where they deposit in human airways: <10 $\mu g/m^3$ (PM_{10}) or course particles, <2.5 $\mu g/m^3$ ($PM_{2.5}$) or fine particles, and <0.1 $\mu g/m^3$ ($PM_{0.1}$) or ultrafine particles. Particulate matter is produced through both manmade (combustion in mechanical and industrial processes, vehicle emissions, and tobacco smoke) and natural sources (volcanoes, fires, dust storms, and aerosolized sea salt), and is a complex mixture of small particles and liquid droplets made up of acids, organic chemicals, metals, and soil or dust particles (J. O. Anderson, Thundiyil, & Stolbach, 2012).

The first major research based regulatory effort directed at setting limits on emissions and air pollution in the United States was the 1970 Clean Air Act (CAA), which defined the National Ambient Air Quality Standards (NAAQS) that set limits on six primary pollutants found in air: carbon monoxide, lead, nitrogen dioxide, ozone, sulfur dioxide, and particulate matter (Belden, 2001). The World Health Organization (WHO) estimates that exposure to $PM_{2.5}$ concentration particulates contribute to approximately 800,000 premature deaths per year, ranking it as the 13^{th} leading cause of mortality worldwide (J. O. Anderson et al., 2012).

Until the early 1990's there was much disagreement on the type of exposures that affected population health. Novel studies by researchers at Utah Valley, Harvard University, and the American Cancer Society (ACS) set the stage with intervention and cohort studies that presented evidence depicting the negative health impacts such as reduced life expectancy of chronic exposure to particulate matter (Pope III, 1991; Pope III et al., 2002). These studies measured hospitalizations related to respiratory events, lung function testing, use of bronchodilators, and premature mortality. The results of these studies promulgated the

reevaluation of the health effects of particulate matter, and prompted the review of international standards of air quality guidelines.

In the United States, the U.S. Environmental Protection Agency (EPA) issued standards for fine particles after the evaluation of many health studies and an extensive review process (United States Environmental Protection Agency, 2013). The organization established the 1997 annual standard at a level of "15 micrograms per cubic meter ($\mu g/m^3$), based on the 3-year average of annual mean $PM_{2.5}$ concentrations." The 24-hour standard for that same year was "established as a level of 65 $\mu g/m^3$, determined by the 3-year average of the annual 98th percentile concentrations." In September of 2006 the Environmental Protection Agency reevaluated the standards and the "EPA strengthened the 24-hour fine particle standard from the 1997 level of 65 $\mu g/m^3$ to 35$\mu g/m^3$, and retained the annual fine particle standard at 15$\mu g/m^3$." (United States Environmental Protection Agency, 2013) However, these standards are lower than those established by the World Health Organization (WHO), who is a leader in setting the norms and health standards at the international level. The most current WHO standard was set in 2006 for $PM_{2.5}$ of 25 $\mu g/m^3$ for the 24-hour average and 10 $\mu g/m^3$ for the annual average (World Health Organization Regional Office for Europe, 2006).

Evidence supports that long-term exposure to $PM_{2.5}$ leads to negative health outcomes, and also establishes long-term particulate exposure as a cause of cardiovascular mortality and morbidity. Further research is necessary to better understand the biological mechanisms of both short and long term impacts of $PM_{2.5}$ exposure. Although there is minimal evidence indicating that one specific property of PM is responsible for negative health outcomes, epidemiological studies have demonstrated that three different components of particulate matter, black carbon, secondary organic and secondary inorganic and aerosols, all significantly contribute to adverse

health effects as noted by the literature (Mauderly & Chow, 2008). A majority of evidence found that particulate matter from carbon material from traffic has a major impact on health outcomes in cities similar to Pittsburgh, PA. Some studies suggest that road dust, including road, brake, and tire wear generated by traffic contribute to negative health effects (Amato et al., 2014). Additionally, studies have found evidence relating biomass combustion from oil and coal industries to cardiovascular hospital admissions and respiratory episodes (Faustini, Héroux, & Forastiere, 2014).

There is also strong evidence linking short- and long-term exposure to $PM_{2.5}$ to mortality and morbidity events. Experimental studies have found that exposure to particulate matter results in airway remodeling, oxidative stress, and airway hyper-responsiveness when coupled with allergic sensitization, or through independent exposure of only PM when researchers adjusted for co-exposures (Dominici, Greenstone, & Sunstein, 2014). In terms of short-term exposure to ambient $PM_{2.5}$, prospective cohort studies of asthmatic children and adults have found associations with asthma symptoms, especially in the younger cohorts (Jedrychowski et al., 2007; Mirowsky et al., 2013). Long-term exposure to particulate matter has been associated with poor levels of asthma control in addition to lung function decrements in children and adults (Patel, Chillrud, Deepti, Ross, & Kinney, 2013). It is important to note that effects of long-term exposure are more detrimental to health outcomes, and lead not only to exacerbations, but also may be a contributing factor to the development of underlying diseases.

More recent studies provide further evidence for associations between long-term exposure to $PM_{2.5}$ and respiratory symptoms and asthma development. For example, a number of nationwide studies that used data from the National Health Interview Survey reported associations between long-term exposure to $PM_{2.5}$ and respiratory symptoms among children and

adults in terms of respiratory allergy events and frequent ear infections (Bhattacharyya & Shapiro, 2010; Parker, Akinbami, & Woodruff, 2009). A 2010 study observed associations between annual average concentrations of $PM_{2.5}$ and frequent asthma symptoms when they examined long-term exposure to $PM_{2.5}$ and weekly asthma symptoms among participants (Meng et al., 2010). A 2012 community intervention study found a decrease in ambient $PM_{2.5}$ concentration was associated with decreases in wheezing and respiratory infections such as colds, bronchitis, influenza, and throat infections, suggesting that decreases in concentrations are beneficial to health outcomes (Noonan, Ward, Navidi, & Sheppard, 2012). Pulmonary diseases have been associated with exposure to higher levels of particulate matter in air pollution and have found decreased lung function in both children and adults, inhibited lung development in children, and evidence of causation that greater exposure results in increased hospitalization. (Nishimura et al., 2013; Weiss, Gergen, & Wagener, 1993)

Although a number of the studies highlighted have identified associations between the prevalence of asthma to increased exposure to outdoor particulate matter, this finding is not always constant (Gowers et al., 2012). Moreover, these associations may be confounded when PM is correlated with nitrogen dioxide, sulfur dioxide, and ozone levels. In summary, there is substantial evidence that supports the idea that ambient levels of PM contribute to oxidative stress and allergic inflammation, thus exacerbating asthma levels.

2.3 TRAFFIC DENSITY AND RESPIRATORY EFFECTS

Traffic-related air pollution is a gaseous mixture that consists of combinations of elemental or black carbon; road dust, tire wear, and break wear known as non-combustion

sources; and nitrogen oxides, which are categorized as primary gaseous emissions. These emissions generate secondary pollutants such as nitrates, ozone, and organic aerosol. In recent years, modeling of airway mechanisms and exposure pathways has increased researcher's understanding of the role of air pollutants in asthma exacerbations and other disease mechanisms (Laumbach & Kipen, 2012). Reviews from the last five years have investigated the distance from roadways where increased air pollution levels are observed (De Nazelle et al., 2011). In large metropolises such as the City of Pittsburgh, 30-45% of individuals live within 300-500m distance of major highways and roadways (Hazenkamp-von Arx et al., 2011).

Numerous epidemiological studies of traffic-related air pollution have found increases in respiratory symptoms, negative changes in lung function results, and increased healthcare use in children and adults (Chang et al., 2009; Jerrett et al., 2008; Rosenlund et al., 2009; Spira-Cohen, Chen, Kendall, Lall, & Thurston, 2011). Studies have also found dose-response associations between asthma symptoms and exposure to truck traffic (H. R. Anderson, Favarato, & Atkinson, 2013; Asher et al., 2010; Kelly & Fussell, 2011), and that short-term exposure to $PM_{2.5}$, NO_2, and CO leads to increased long term health effects (Delfino et al., 2014). Associations between the reduction of traffic related air pollution and reductions in asthma exacerbation in urban areas suggest the feasibility of decreasing symptomology in individuals with asthma by limiting pollution levels (Boogaard et al., 2012). In terms of lung function, research suggest that 1) long-term exposure is associated with changes in lung function in adolescent and adults; 2) lung function measures are lower in people who live in more polluted areas; and 3) changing residences to a less-polluted area is associated with improvements in lung function (Downs et al., 2007). This increasing body of evidence and knowledge supports the role of traffic-related air

pollution in exacerbating asthma in both adults and children, and suggests the need for further research assessing the effects of these exposures.

2.4 RISK MODIFIERS

Individuals with asthma, especially young children, are very susceptible to the negative health effects of air pollution because they are still in the process of developing their lungs and metabolic pathways. Additionally, they are more likely to spend increased amounts of time outdoors, increasing their overall levels of exposure to air pollution (Pinkerton & Joad, 2006; Urman et al., 2013). In utero exposure may also contribute to narrow airway development, another risk factor of air pollution exposure at an early age (Schildcrout et al., 2006). In terms of sex, asthma exacerbation levels are higher in young boys, and in adults, asthma has a higher prevalence in woman when compared to men (Singh & Busse, 2006). Additionally, older adults are more likely to be at risk for negative health outcomes related to asthma and air pollution.

There is minimal evidence supporting the differences in susceptibility to asthma and air pollution interactions related to ethnicity, but higher rates of asthma have been associated to air pollution in larger and more diverse US cities (Thakur et al., 2013). However, it is important to keep in mind that the difference in effects between different ethnicities may be associated to the low socioeconomic status of individuals in these cities (Thakur et al., 2013). Children living in families within low socioeconomic status areas are more likely to be exposed and affected by air pollution, making them more susceptible to asthma exacerbations (E. Chen et al., 2011; Pittman et al., 2012; Tzivian, 2011). Additionally, factors such as crime rates, food deserts, stress, and diet may contribute to overall susceptibility for individuals both young and old (McConnell et

al., 2010). Poor diets may make individuals more susceptible to the effects of pollutants. This is backed by evidence indicating higher levels of intake of fruits and vegetables are beneficial for strengthening oxidative stress pathways (Giles et al., 2011; Jarjour et al., 2012; Kozyrskyj, Bahreinian, & Azad, 2011). Obesity has also been found to increase an individual's susceptibility to the adverse effects of air pollution (Camargo, Weiss, Zhang, Willett, & Speizer, 1999; Y. Chen, Dales, Tang, & Krewski, 2002; Ford, 2005). Secondhand smoke is also considered to be a modifier of the effects of air pollutants, because it contains a mixture of gasses and particulate matter that adversely affect asthma outcomes (U.S. Department of Health and Human Services, 2004; Von Mutius, 2009).

2.5 MITIGATING IMPACT

A technique that local governments and state health agencies may use to reduce particulate matter exposure for individuals living in large cities with high levels of traffic-related air pollution such as Pittsburgh is to issue alerts for high levels of $PM_{2.5}$ levels, so that citizens can limit their outdoor exposures. According to the United States Environmental Protection Agency (EPA), individuals exposed to $PM_{2.5}$ ranging from >15 - 40 $\mu g/m^3$ are likely to experience respiratory symptoms. Localities may also prioritize making air quality data available to the general public on a daily basis through resources such as AirNow, a government funded website that reports daily air quality index levels for specific areas. Currently, minimal data is available on the effects of advising and educating individuals to avoid outdoor physical activity on days where $PM_{2.5}$ levels are high. However, there is some evidence that suggests individuals that are more susceptible to air pollution are likely to benefit from staying indoors if they have

the proper resources available to them to ensure cleaner indoor air quality levels, as indoor air pollution may be higher (Behndig et al., 2006; Carls, 2010; McCreanor et al., 2007; World Health Organization, 2003).

Individuals that are unusually sensitive to air pollution, such as those with asthma, chronic bronchitis, and emphysema will experience possible aggravation of heart and lung disease, especially those with cardiopulmonary disease, and older adults. Additionally, the EPA recommends that individuals that are unusually sensitive should consider reducing prolonged or heavy exertion related to physical activity (Mintz, 2006). Increased physical activity increases the amount of particulate matter inhaled per minute, thus increasing the overall total inhaled dose for patients. Clinicians should consider advising patients to avoid physical activity on days where air quality is lower as a part of their patients' individualized asthma management plans.

The EPA also recommends that individuals with asthma should live at least 300-meters from major roadways, particularly in areas that are more likely to have heavy truck exposure or higher levels of ozone exposure (Mintz, 2006). Another technique to reduce exposure may be to close windows when traveling roads with heavy traffic and during rush hour (Yang et al., 2015). Lastly, inhaled corticosteroid therapy, in conjunction with behavioral efforts to reduce exposure to PM, may decrease inflammatory responses to pollutants (Croisant & Scott, 2014; Rodrigo, 2014).

3.0 METHODOLOGY

3.1 SURVEY

This thesis is based on a survey developed for the recruitment of research participants at the Asthma Institute at the University of Pittsburgh Medical Center (UPMC). (Appendix B) The baseline registry questionnaire was approved by the IRB on January 16, 2014 and consists of demographic information, medical history, childhood and family history, asthma triggers, past and current medication usage questions, and on-site lung function test results recorded at the Asthma Institute at the University of Pittsburgh Medical Center. Lung function tests, also known as spirometry or pulmonary function tests (PFTs) assess how well an individual's lungs work by determining how much air the lungs can hold, and how quickly air can move in and out of the lungs. They also measure how well the lungs put oxygen into and remove carbon dioxide from the lungs. For the purpose of this study, forced vital capacity (FVC), forced expiratory volume (FEV1), and the ratio of forced vital capacity to forced expiratory volume (FEV1/FVC) were used. FVC measures the amount of air an individual can exhale with force after he inhales as deeply as possible, FEV1 measures the amount of air an individual can exhale with the force of one breath at one second, and FEV1/FVC measures the percentage of the vital capacity which is expired in the first second of maximal expiration. In patients with obstructive lung disease, FEV1/FVC is lower than 70% and can be as low as 20-30% in severe obstructive airway disease.

3.2 EXPOSURE ASSIGNMENT

The coordinate locations of residential addresses from the Asthma Registry were cleaned and standardized (Figure 1) and then geocoded in ArcGIS® using an address locator specifying StreetMap Premium™ reference data (Figure 2). The match rate was 97.6 % for a total of 453 addresses. One additional survey participant was excluded because of missing pulmonary function tests, making the final study sample (n = 452).

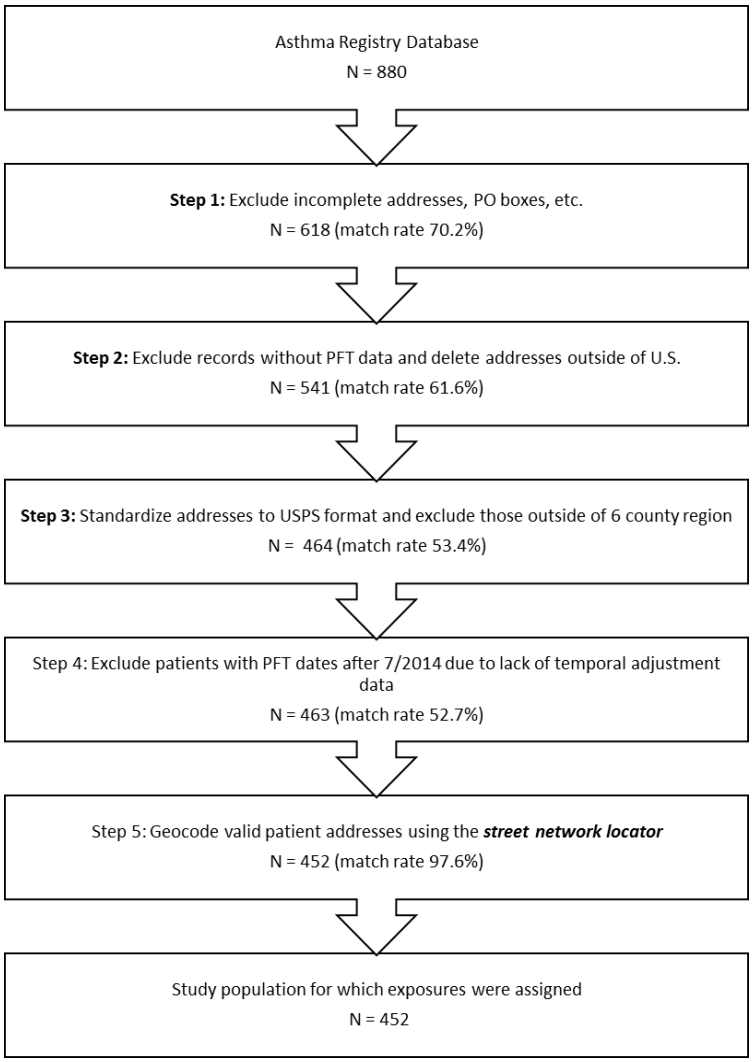

Figure 1: Exclusion Cleaning Methods for Asthma Registry Case Data and Geocoding Methodology

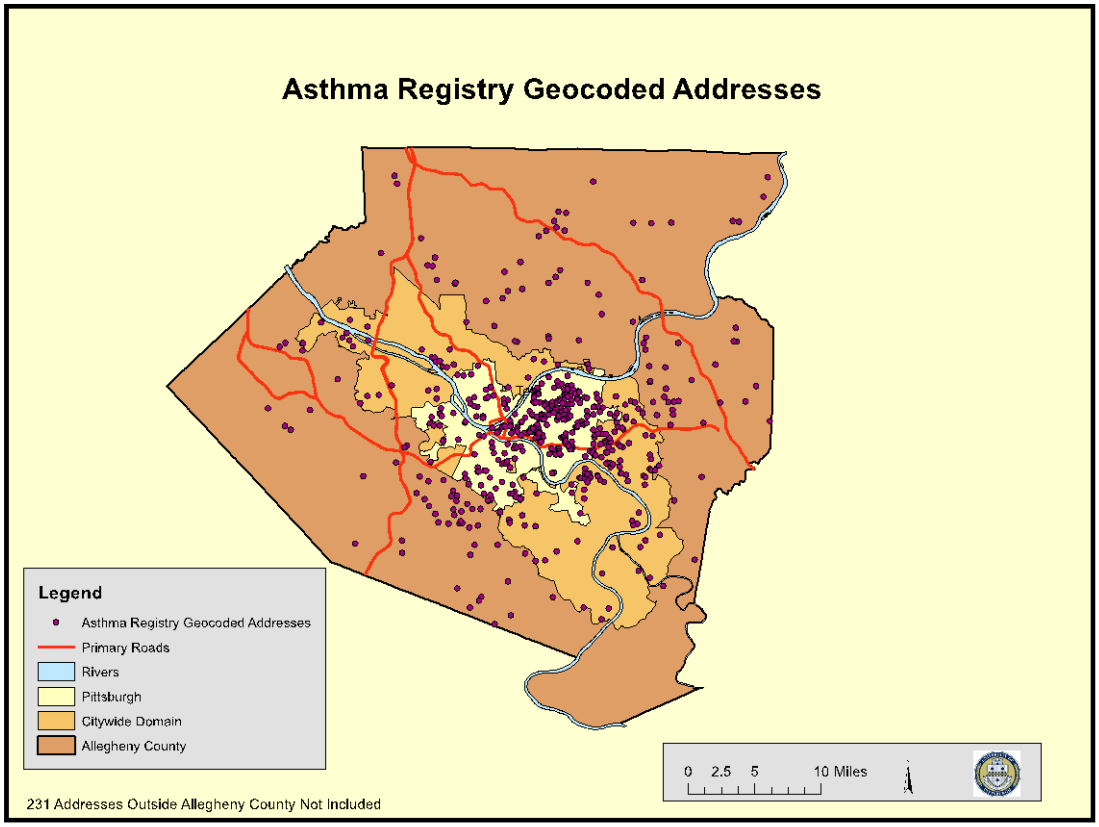

Figure 2: Asthma Registry Geocoded Addresses

3.3 PM2.5

A predicted surface of $PM_{2.5}$ was developed for the six county area surrounding the city of Pittsburgh using a Land Use Regression (LUR) model. This model was modified from one previously developed for a smaller area of Allegheny County (Tunno et al., 2015) (in press). Significant covariates in the LUR model were $PM_{2.5}$ emissions aggregated from the U.S. Environmental Protection Agency's (USEPA) 2011 National Emissions Inventory (NEI) and signaled intersections within a 750-meter buffer. Near-residence $PM_{2.5}$ exposures were assigned by averaging the concentrations within a 300-meter buffer around each residential location.

Exposure estimates were then temporally adjusted using daily data monitoring data from the USEPA Air Quality System (AQS) site in Lawrenceville, PA to obtain exposure for a one month period prior to the month of the lung function test.

3.4 TRAFFIC DENSITY

Traffic data were obtained from the Pennsylvania Department of Transportation, which uses raw traffic counts to calculate statewide traffic volumes along roadway sections. Data includes 2014 annual average daily traffic (AADT) and annual average daily truck traffic (ADTT) for primary and secondary roadways within the study domain.

An interpolated surface of traffic volume (AADT) was derived using a kernel density function in ArcGIS. Kernel density calculates the density of roadways (primary and secondary roads) in the neighborhood of each output raster cell. Conceptually, a smoothed curved surface is fitted over each line feature by applying a Gaussian decay function to traffic volumes on road segments within the study domain. Values are greatest on the line feature and diminish with distance from the road. From this traffic density surface, we calculated mean traffic density within a 100 meter buffer and 300 meter buffer of each residential location.

3.5 DATA ANALYSIS

A total of 452 valid responses were used for the analysis of this study. Valid responses were defined as completing on-site spirometry results, and providing a current address that was

able to be geocoded. The data were analyzed with the statistical program STATA 13 after being entered into a Stata worksheet and cleaned for missing values. We used linear regression analysis to determine: A) the linear relationship between exposures and lung function tests; and B) to determine the association between exposures with road density exposures and their respective quartiles. We adjusted for race, age, and sex as confounders.

4.0 RESULTS

The mean age of respondents was 41.13 (age, 18-82) years of age. 65.49% of participants identified as female, and 87.61% of respondents identified as White (Table 1). According to Body Mass Index, determined by height and weight recordings during the day of the on-site PFT, 29.65% of respondents were overweight, while 41.81% were obese.

Table 1: Demographic Data

Characteristic	Overall (n= 452)
Age (years as a whole number) [a]	41.13±15.80
Height (in.) [a]	66.82±8.46
Sex [b]	
Female	296 (65.49)
Male	152 (33.63)
Unidentified	4 (0.88)
Race [b]	
White	396 (87.61)
Black	47 (10.40)
Asian	3 (0.66)
Native	1 (0.22)
Other	5 (1.11)
Weight Category [bc]	
Underweight	5 (1.11)
Normal	124 (27.43)
Overweight	134 (29.65)
Obese	189 (41.81)
Urban/Suburban	352 (77.88)
Rural	100 (22.12)
PFT [a]	
FEV1	2.72 ± 0.95
FEVPCT	82.66 ± 19.83
FVC	3.63 ± 1.16
FVCPCT	89.33 ± 18.33
Ratio	00.75 ± 00.17

Abbreviations: PFT = Pulmonary Function Tests; FEV1 = Forced Expiratory Volume; FEVPCT = Percent of Forced Expiratory Volume; FVC = Forced Vital Capacity; FVCPCT = Percent of Forced Vital Capacity; Ratio = Ratio of FEV1 over FVC

[a] Mean ± standard deviation

[b] Number of subjects (%)

[c] Weight classifications are determined by Centers for Disease Control and Prevention guidelines for weights (ADD definitions)

Out of the 452 individuals that completed the survey, 180 respondents were diagnosed with asthma in childhood (before the age of 18), whereas 272 individuals were diagnosed with asthma in adulthood (on or after the age of 18). Of the number of individuals that responded to their insurance status (235 responses), 53 (22.5%) use Medicare or Medicaid, 155 (65.9%) have private insurance, and 27 (11.6%) self-pay for their medical expenses. Of the individuals that responded to questions about their smoking history (447 responses), 344 (76.9%) respondents had never smoked, 99 (22.1%) indicated they had smoked at some point in their lives, and 4 (1.1%) respondents indicted having smoked "in moderation: through the course of their lifetimes". 270 (60.6%) individuals indicated that they have ever had an asthma- related ER visit, 165 (37.2%) said they had an asthma related overnight hospital stay, and 57 (12.8%) respondents stated that they have had a asthma related ICU admission (Table 2).

Table 2: Clinical Data

Characteristic	Overall (n=452)
Age of Asthma Onset	
Early	180
Late	272
Insurance (n= 235)	
Medicare/Medicaid	53
Private	155
Self-Pay	27
Ever-Smoked (n=447)	
No	344
Yes	99
In Moderation	4
Asthma Related ER Visits (n=445)	
No	175

Table 2 Continued

Yes	270
Asthma Related Overnight Hospital Stay (n=443)	
No	278
Yes	165
Asthma Related ICU Admissions (n=442)	
No	385
Yes	57
Symptoms	
Coughing (n=446)	
Never	78
Yes/Currently	131
Past Only	237
Sputum (n= 445)	
Never	56
Yes/Currently	152
Past Only	237
Chest Tightness (n=444)	
Never	36
Yes/Currently	178
Past Only	230
Wheezing (n=445)	
Never	30
Yes/Currently	194
Past Only	221
Shortness of Breath (n=445)	
Never	22
Yes/Currently	227
Past Only	196
Nighttime Symptoms (n=445)	
Never	106
Yes/Currently	170
Past Only	169
Asthma Control Medication Use	
Inhaled steroids (n=445)	
No	169
Yes	276
Inhaler beta-agonist (n= 445)	
Never	15
Yes/Currently	340
Past Only	90
Nebulized beta-agonist (n= 443)	
Never	180
Yes/Currently	81
Past Only	182
Oral beta-agonist (n= 437)	

Table 2 Continued

Never	421
Yes/Currently	3
Past Only	13
Long-acting bronchodilator (n= 444)	
Never	174
Yes/Currently	145
Past Only	125
Leukotriene inhibitor (n= 443)	
Never	219
Yes/Currently	116
Past Only	118
Theophyllines (n= 443)	
Never	364
Yes/Currently	13
Past Only	66
Ipratroprium bromide (n=441)	
Never	352
Yes/Currently	17
Past Only	12
Tiotroprium bromide Spiriva (n=440)	
Never	398
Yes/Currently	10
Past Only	32
Injectable Corticosteroids (n=443)	
Never	382
Yes/Currently	10
Past Only	51

a Early Asthma Onset = Before the age of 18; Late Asthma Onset = After the age of 18

Current asthma symptoms in respondents included: Coughing 131 (30.9%), sputum 152 (34.2%) chest tightness 178 (40.1%), wheezing 194 (43.6%), shortness of breath 227 (51%), and nighttime symptoms 170 (38.2%). Current asthma control medication use in respondents included: Inhaled steroids 276 (62.0%), inhaler beta-agonist 340 (76.4%), nebulized beta-agonist 81(18.3%), oral beta agonist 3 (<1%), long acting bronchodilator dilator 145 (32.7%), theophyllines 13 (<5%), ipratroprium bromide 17 (<5%), trioprium bromide 10 (<5)%), and injectable corticosteroids 10 (<5%). (Table 2)

Air pollution ($PM_{2.5}$) and pulmonary function test data are presented in Table 1 and Table 3 by overall values. Overall $PM_{2.5}$ levels ranged between 6.39 and 21.59 $\mu g/m^3$ (IQR= 3.59); FEV1 levels ranged from 0.7 to 5.4 liters/second (IQR= 1.3); FEV1PCT levels ranged from 28% to 139% (IQR = 25.5); FVC levels ranged from .4 to 7.4 liters (IQR= 1.5); FVCPCT levels ranged from 1% to 136% (IQR= 24); Ratio levels ranged from 35% to 325% (IQR) = 13%.

Pulmonary function tests showed associations with traffic-related pollutants. A linear regression analysis was conducted after checking for assumptions of linear regression: 1) variables were normally distributed, and 2) a scatter plot showed a linear relationship between the independent and dependent variables. The researcher conducted a crude analysis for the relationship between PFT test results and $PM_{2.5}$ and road density exposure at the 100-meter and 300-meter levels. A significant association was found between Road 300 meter exposure levels and FEV1 test results $F (3, 448) = 2.41$ $p < .05$; and Road 300 meter exposure levels and FEV1 PCT test results $F (3, 448) = 2.02$ $p < .05$ (Figure 3) (Figure 4). No crude associations were found between $PM_{2.5}$ and FEV1, FEV1PCT, FVC, FVC PCT, FEV/FVC. (Table 3) No crude associations were found between FVC, FVC PCT, FEV/FVC and the Road 100 meter and Road 300 meter exposures (Table 4). When adjusting for the confounders of race, age, and sex, there was no true association found between the PFT test results and road density or $PM_{2.5}$ exposure (Table 5).

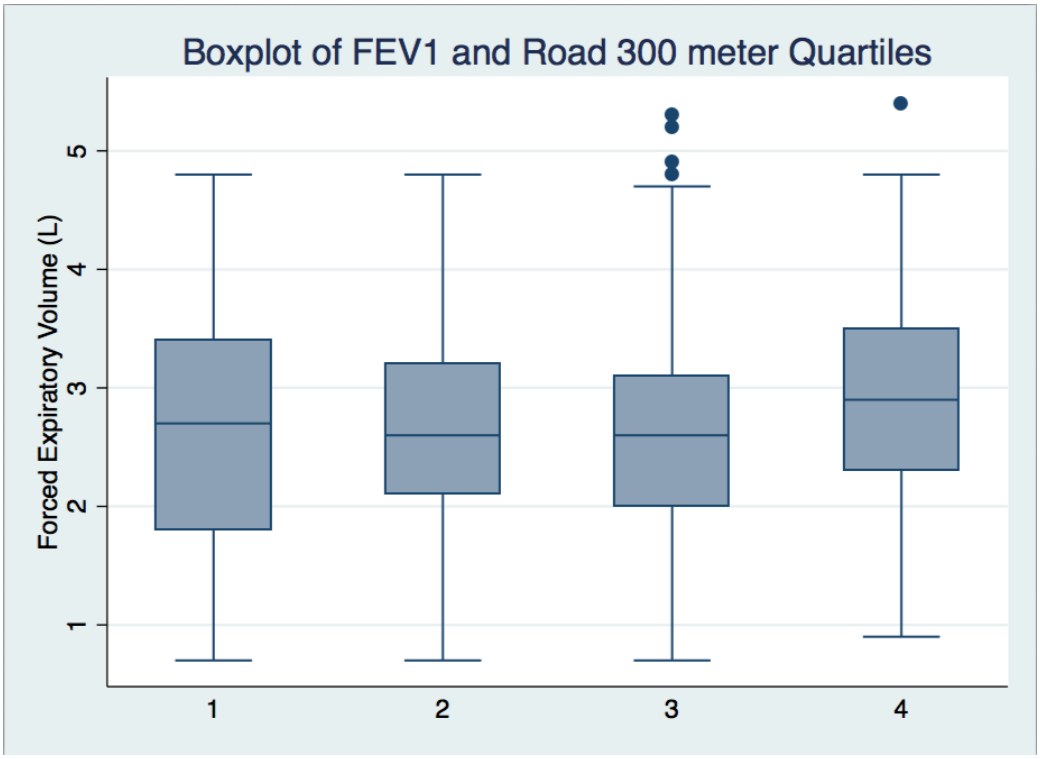

Figure 3: Boxplot of FEV1 and Quartiles of 300-meter Traffic Density Values

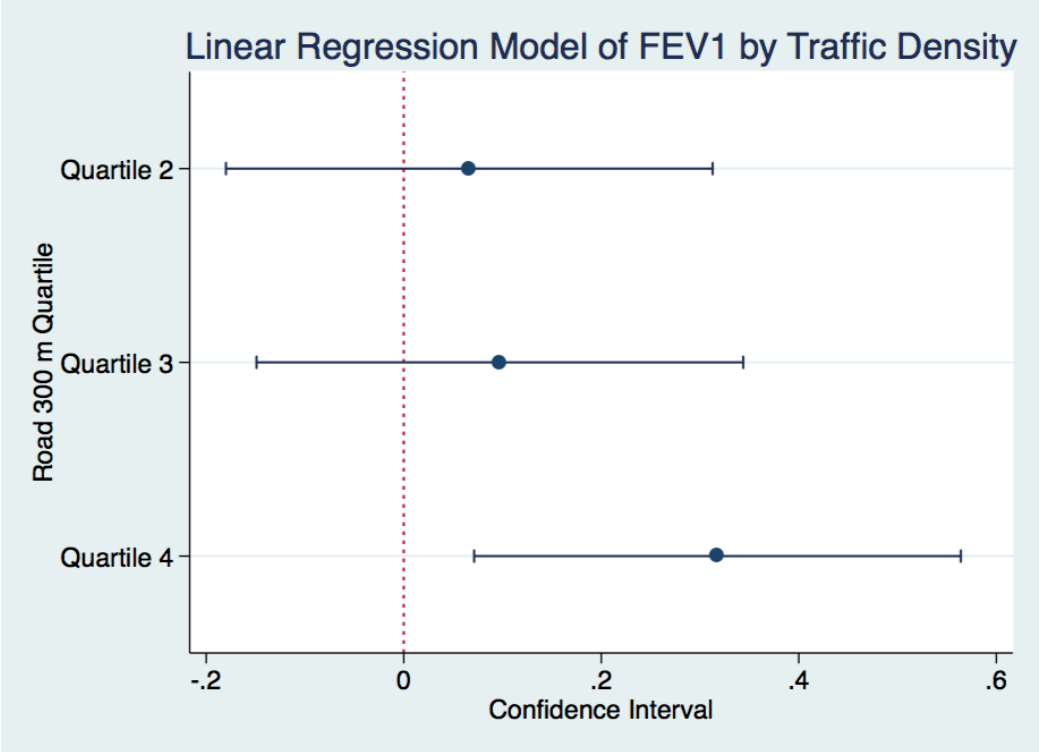

Figure 4: Confidence Interval for the Linear Regression Model of FEV1 and 300-meter Traffic Density

Table 3: Linear Regression Analysis of PM2.5 Exposure and Pulmonary Function Test Results

	PM2.5 β (std. error)	Confidence Interval	P>|t|
FEV	-.0145765 (.0158257)	(-.045678, .0165249)	0.358
FEV PCT	-.4632595 (.3309891)	(-1.113736, .1872167)	0.162
FVC PCT	-.0706959 (.306632)	(-.6733043, .5319125)	0.818
Ratio	-.002065 (.0028271)	(-.007621, .003491)	0.466

Table 4: Linear Regression Analysis of 100-meter and 300-meter Road Density Exposures and Pulmonary Function Tests by Quartile Distribution (Excluding Confounders)

	Road 100-meter	Confidence Interval	P>|t|	Road 300-meter	Confidence Interval	P>|t|
FEV						
Q_1	-	-	-	-	-	-
Q_2	.1699115 (.1260998)	(-.077909, .417732)	0.179	.0663717 (.1253911)	(-.1800561, .3127994)	0.597
Q_3	.0814159 (.1260998)	(-.1664046, .3292364)	0.519	.0973451 (.1253911)	(-.1490826, .3437729)	0.438
Q_4	.1415929 (.1260998)	(-.1062276, .3894134)	0.262	.3176991 (.1253911)	(.0712714, .5641268)	0.012*
FEV PCT						
Q_1	-	-	-	-	-	-
Q_2	-1.017699 (2.646083)	(-6.217976, 4.182578)	0.701	4.672566 (2.629176)	(-.4944826, 9.839615)	0.076
Q_3	-.6725664 (2.646083)	(-5.872843, 4.527711)	0.799	2.699115 (2.629176)	(-2.467934, 7.866164)	0.305
Q_4	-1.274336 (2.646083)	(-6.474613, 3.925941)	0.630	6.088496 (2.629176)	(.9214466, 11.25554)	0.021*
FVC						
Q_1	-	-	-	-	-	-
Q_2	.219469 (.1547044)	(-.0845674, .5235054)	0.157	.0469027 (.1543571)	(-.2564513, .3502566)	0.761
Q_3	.1389381 (.1547044)	(-.1650983, .4429744)	0.370	.1654867 (.1543571)	(-.1378672, .4688407)	0.284
Q_4	.1982301 (.1547044)	(-.1058063, .5022665)	0.201	.2982301 (.1543571)	(-.0051239, .6015841)	0.054
FVC PCT						
Q_1	-	-	-	-	-	-
Q_2	-1.415044 (2.444923)	(-6.219986, 3.389898)	0.563	4.146018 (2.433786)	(-.6370369, 8.929072)	0.089
Q_3	-1.729204 (2.444923)	(-6.534146, 3.075738)	0.480	4.516814 (2.433786)	(-.2662404, 9.299869)	0.064
Q_4	-1.839823 (2.444923)	(-6.644765, 2.965119)	0.452	4.420354 (2.433786)	(-.3627006, 9.203409)	0.070
Ratio						
Q_1	-	-	-	-	-	-
Q_2	.0159292 (.0225435)	(-.028375, .0602334)	0.480	-.0004425 (.0225049)	(-.0446708, .0437858)	0.984
Q_3	-.0048673 (.0225435)	(-.0491715, .039437)	0.829	-.0234513 (.0225049)	(-.0676796, .020777)	0.298
Q_4	-.0048673 (.0225435)	(-.0491715, .039437)	0.829	.0127434 (.022504)	(-.0314849, .0569716)	0.572

*p<0.05

Table 5: Regression Analysis of 100-meter and 300-meter Road Density Exposures and Pulmonary Function Tests by Quartile Distribution (Including Confounders of Race, Age, and Sex)

	Road 100-meter	Confidence Interval	P>|t|	Overall P>|t|	Road 300-meter	Confidence Interval	P>|t|	Overall P>|t|
FEV				0.753				
Q_1								0.173
Q_2	-.0082454 (.0928399)	-(.1907172, .1742263)	0.929		.1011537 (.091122)	(-.0779427, .2802501)	0.268	
Q_3	-.06301 (.093811)	(-.2473905, .1213705)	0.502		.0308091 (.0942028)	(-.1543413, .2159594)	0.744	
Q_4	-.0952707 (.1032438)	(-.2981908, .1076494)	0.357		.200639 (.1012842)	(.0015705, .3997074)	0.048	
FEV PCT								
Q_1				0.791				
Q_2	-1.373221 (2.607455)	(-6.49803, 3.751588)	0.599		3.311116 (2.559223)	(-1.718896, 8.341128)	0.196	0.253
Q_3	-1.235306 (2.63473)	(-6.413722, 3.94311)	0.639		1.567969 (2.645731)	(-3.632071, 6.768009)	0.554	
Q_4	-2.937422 (2.899654)	(-8.636533, 2.761688)	0.312		5.262655 (2.844615)	(-.3282805, 10.85359)	0.065	
FVC								
Q_1				0.899				0.475
Q_2	-.0427108 (.1102037)	(-.2593101, .1738885)	0.699		.1355738 (.1081652)	(-.0770189, .3481665)	0.211	
Q_3	-.0679324 (.1113565)	(-.2867974, .1509326)	0.542		.1069225 (.1118215)	(-.1128564, .3267014)	0.340	
Q_4	-.0881605 (.1225535)	(-.3290326, .1527116)	0.472		.176231 (.1202273)	(-.0600691, .4125311)	0.143	
FVC PCT								
Q_1				0.669				0.364
Q_2	-2.325126 (2.385461)	(-7.013619, 2.363367)	0.330		3.230949 (2.341336)	(-1.370818, 7.832716)	0.168	
Q_3	-2.346879 (2.410414)	(-7.084416, 2.390658)	0.331		3.75034 (2.420479	(-1.006979, 8.507659)	0.122	
Q_4	-2.998181 (2.652783)	(-8.212081, 2.215719)	0.259		3.871713 (2.602431)	(-1.243221, 8.986648)	0.138	
Ratio								
Q_1								0.75
Q_2	.0228858 (.0232932)	(-.0228958, .0686674)	0.326	0.522	-.0044025 (.022862)	(-.0493372, .0405322)	0.847	
Q_3	-.0019461 (.0235369)	(-.0482066, .0443144)	0.934		-.0289919 (.023635)	(-.0754455, 0174618)	0.221	
Q_4	-.010794 (.0259035)	(061706, .040118)	0.677		.0050388 (.0254119)	(-.0449068, .0549844)	0.843	

*p<0.05

5.0 DISCUSSION

This pilot study presents the first analysis of pollution exposure rates and asthma exacerbation data specific to residents living in Alleghany County, PA (Pittsburgh, PA). The main goals of this study were to document the respiratory health impact of road density levels and particulate matter specific to city residents to help guide future research and policy. This study also provided a unique opportunity to assess the effectiveness of data collection strategies related to air-pollution exposure when using the data to analyze individual specific medical outcomes, such as asthma exacerbation rates. The results indicate significant associations between forced expiratory volume test results (FEV1) and the highest quartile mean 300-meter traffic density of each residential location. As highlighted in previous sections, forced expiratory volume flow is the volume of air expired in the first second during maximal expiratory effort. FEV1 levels are generally lower in patients that have both obstructive lung disease because of airway resistance, and restrictive lung disease because of low vital capacity. It is important to note that researchers have previously reported significant associations between increased concentrations of a number of pollutants, specifically reductions in FEV1. (Li et al, 2011)

The lack of statistical significance in the associations of other main models in the current analysis may be due to several factors. The environmental exposure data collected for this study provided a brief snapshot in space and time of a potential exposure-response relationship. It is possible that the pollutant exposure levels and variability occurring during this study period were

not sufficient to produce a statistically detectable response in a clinical testing atmosphere. Moreover, single measurements of lung function tests provide only a snapshot of asthma severity at the time they were recorded, and air pollution data for the purpose of this study was collected during the month prior to the date of lung function testing. These results may attest to a number of factors, including baseline differences in modifying risk factors among the different respondents, as well as limited power to detect subtle changes in daily air quality exposure levels.

Limitations of this study exist related to the analytical methods. First, because this is a cross-sectional study, we cannot assume any form of causation. Additionally, exposures to PM and traffic density are roughly estimated through GIS, which may contain errors, and there are no indoor exposure readings available for those that do have exacerbated asthma symptoms.

Furthermore, although the study sample size (N = 452) was large enough to detect for the expected effects, this study was conducted through the use of a convenience sample that is not truly demographically representative of the Allegheny county population. This sampling strategy further confounds the results of this study. Because race and socioeconomic status have shown to play a significant role in asthma incidence and exacerbation rates, it is likely that a randomized sample may have provided significant results. 87.61% of the sample identified as White and 10.40% identified as Black; however, according to 2013 census data, 79.9% of the Allegheny County population identifies as White and 13.3% identifies as Black.

The main strength of the study is that the population consisted of a well-characterized study population that meets rigorous testing for asthma diagnosis, unlike other epidemiologic studies that rely on self-referred diagnosis. As the registry grows, the study will have power to detect potential effect modifiers, including use of medications, and the response to pollution

according to different phenotypical features (i.e. age of onset, atopy, severity level). Although the analysis is largely non-significant when accounting for other demographic variables, the univariable analysis does suggest that there is a chronic or cumulative effect of $PM_{2.5}$, which is related to lower lung function.

It is recommended that future research studies that focus on racially and socioeconomically diverse cities such as Pittsburgh consider the representativeness of their study population to the target area. It is important to note, however, that in the clinical setting, collecting data of this caliber may be limited in scope because of the demographic populations that are most likely to seek care and treatment at institutions that do have the ability to conduct research studies such as the one presented in this paper. Subsequent studies need to be done in this regional area, using better exposure resolution, to determine more accurately the chronic effects of air pollution on the lung function of asthmatics. Eventually, this line of research could lead to preventive strategies to prevent loss of function and improve quality of life among more susceptible asthmatics exposed to higher ambient pollution concentrations.

APPENDIX: IRB APPROVED REGISTRY

PEMC	Date Completed								Assessor ID#		Subject ID#				

Asthma & Allergic Diseases Registry Questionnaire

Date of Consent: __ __/__ __/__ __ __ __ **Date Blood Drawn:** __ __/__ __/__ __ __ __

Demographics:

Date of Birth __ __/__ __/__ __ __ __ Age ____

Contact Information (for you/your child):

1. First Name: _____ *confname* MI: ___ *conmi* Last: _____
conlname

Maiden/ Other Name(s): _____ *conmdname*

Address: _____ *conaddress*

City: _____ *concity* State: _____ *constate* Zip: _____
conzip

Phone: (H / W / C) ( ) - _ _ _ - _ _ _ _ *conphone1* (H / W / C)( ) - _ _ _ - _ _ _ _ *conphone2*
conphonetype1 *conponetype2*

Email: _____ *conemail*

Secondary Contact Information

2. Name(s): _____ *conname2*

   Address: _____ *conaddress2*

   City: _____ *concity2* State: _____ *constate2* Zip: _____ *conzip2*

   Phone: (H / W / C)( ) - _ _ _ - _ _ _ _ *con2phone1* (H / W / C)( ) - _ _ _ - _ _ _ _ *con2phone2*

   con2phonetype1 *con2phonetype2*

   Relation: _____ *relation*

Provider Information

1. Primary Care Provider:

   Clinic Name: _____ *primcarename*

   Doctor: _____ *primcaredr*

   Address: _____ *primcareaddrs*

   City: _____ *primcarecity* State: _____ *primcarestate* Zip: _____ *primcarezip*

   Phone: ( ) - _ _ _ - _ _ _ _ *primcarephone* Fax: ( ) - _ _ _ - _ _ _ _ *primcarefax*

   Email: _____ *primcareemail*

2. Specialist: Pulmonologist/ Allergist- Immunologist

Clinic Name: _____ *specname*

Doctor: _____ *specdr*

Address: _____ *specaddress*

City: _____ *speccity* State: _____ *specstate* Zip: _____ *speczip*

Phone: ( ) - _ _ _ - _ _ _ _ *specphone* Fax: ( ) - _ _ _ - _ _ _ _ *specfax*

Referring Physician:

1. Chose one: *refermd* 1 ☐ Family Medicine/ Internal Medicine

   2 ☐ Pulmonologist/ Allergist- Immunologist

2. Do we have your permission to request pertinent medical information from these providers? *permitinfo*

   0 ☐ No 1 ☐ Yes 2 ☐ Both 3 ☐ One only

   oneonly 1 ☐ First 2 ☐ Second

General Information About You/Your Child

1. Age: _____ *ycage*

2. Sex: *gender* 1 ☐ Male 2 ☐ Female

3. Race: *race* 1 ☐ White/ Caucasian

   2 ☐ Black/ African American

   3 ☐ Asian/ Pacific

4 ☐ Native American

5 ☐ Other _____ *othrace*

4. Ethnicity: *ethnicity* 1 ☐ Hispanic/ Latino 2 ☐ NOT Hispanic/ Latino

5. Marital Status: 1 ☐ Single, never married *maritalstats*

2 ☐ Married

3 ☐ Remarried

4 ☐ Separated

5 ☐ Divorced

6 ☐ Widowed

6. Are you/your child employed outside of home? *emplyd*

1 ☐ Yes 0 ☐ No

7. Occupation: _____ *occupation*

8. Type of Insurance: 1 ☐ Medicare/Medicaid 2 ☐ Private 3 ☐ Self-pay *insurance*

9. Education: Highest level of school completed by you/your child and either parent (mother or father):

	You/Your Child *educself*	**Either parent** *educparent*
Less than fifth grade	☐	☐
Fifth grade to eighth grade	☐	☐
Junior High School (9th grade)	☐	☐
Partial High School (10-11th grade)	☐	☐
High School graduate	☐	☐
Partial College	☐	☐
Complete College	☐	☐
Graduate School	☐	☐

10. Did you/your child miss school or work due to asthma in the past year? *asthmadays*

1 ☐ Yes 0 ☐ No

10a. If YES, how many days of school or work were missed in the last 30 days (1 month) due to asthma? _____ *nodaysmiss*

Disease Onset

1. When did you first notice asthma symptoms in yourself/your child?

Year: _____ *frstasthmayr* Age: ____ *frstasthmaage*

2. When did a physician first diagnose you/your child as having asthma?

Year: _____ *frstdiagyr* Age: ____ *frstdiagage*

Smoking History

1. Are you/your child exposed to Second Hand smoke during your day? *scndhandsmk*

1 ☐ Yes 0 ☐ No

1a) If **YES**: Location (mark all that apply):

☐ Home *schdhome* ☐ Work *schdwork* ☐ Other *schother*

2. Have you/your child ever smoked cigarettes? *eversmoke*

1 ☐ Yes (if more than 1 yr of smoking an average of 1 pack per day, or 2 years of

½ pack per day)

2 ☐ If less than this, how many cigarettes a day?_____ *cigsaday*

0 ☐No

3. Do you/your child now smoke cigarettes? *cursmoke*

1 ☐ Yes 0 ☐ No

4. How many years have you/your child smoked cigarettes: *yrssmoke*

1 ☐ < 1 year 2 ☐ 1-5 years 3 ☐ <5 years

4 ☐ 5-10 years 5 ☐ 10-20 years 6 ☐ more than 20 years

5. Average packs per day: 1 ☐ less than one 2 ☐ one 3 ☐ more than one *avgpcksday*

6. Have you/your child quit smoking? 0 ☐ No 1 ☐ Yes *quitsmke*

If Yes, ____years *yrsquite*____months *mnthsquite*

Childhood History

	Yes	No	Don't know
1. Did your mother smoke during pregnancy? If answering for your child, did you smoke during your pregnancy with your child? *pregsmk*	1 ☐	0 ☐	888 ☐
2. Did your father, if living together with your mother, smoke in the home during her pregnancy? If answering for your child, did your child's father live with you and smoke during your pregnancy? *fathersmk*	1 ☐	0 ☐	888 ☐
3. Were you/your child breast-fed? *breastfed*	1 ☐	0 ☐	888 ☐

Family History:

	Yes	No
1. Are you/your child adopted? *adopted*	1 ☐	0 ☐
2. Do you/your child have biological Brothers / Sisters or children? *siblings*	1 ☐	0 ☐

If **Yes**: How many Brothers/Sisters? ___ *siblingno* Children? ____

nochildren

3. Do you/your child have non-biological(step-) / adopted Brothers / Sisters or children? 1 ☐ 0 ☐

adoptedsibs

If **Yes**: How many Brother/Sisters? _____ *adptsibno* Children? ____

adptchldrno

To the best of your knowledge, has a physician ever diagnosed your/your child's biological Father / Mother / Brother / Sister or Child with:

	Father	Mother	Brother / Sister or Child (Biological)	Brother / Sister or Child (Adopted)
1) Asthma?	1 ☐ Yes 0 ☐ No 888 ☐ Don't Know *fasthma*	1 ☐ Yes 0 ☐ No 888 ☐ Don't Know *masthma*	1 ☐ Yes 0 ☐ No 888 ☐ Don't Know *sibasthma*	1 ☐ Yes 0 ☐ No 888 ☐ Don't Know *adptsibasthma*
2) Chronic Obstructive Lung Disease? Chronic Bronchitis / Emphysema?	1 ☐ Yes 0 ☐ No 888 ☐ Don't Know *fchrnlung*	1 ☐ Yes 0 ☐ No 888 ☐ Don't Know *mchrnlung*	1 ☐ Yes 0 ☐ No 888 ☐ Don't Know *sibchrnlung*	1 ☐ Yes 0 ☐ No 888 ☐ Don't Know *adptsibchrnlung*
3) Hay Fever (Allergies)?	1 ☐ Yes 0 ☐ No 888 ☐ Don't Know *fhayfvr*	1 ☐ Yes 0 ☐ No 888 ☐ Don't Know *mhayfvr*	1 ☐ Yes 0 ☐ No 888 ☐ Don't Know *sibhayfvr*	1 ☐ Yes 0 ☐ No 888 ☐ Don't Know *adptsibhayfvr*

Health Care Utilization for Asthma Related Illness

				Yes	No
1. Have you/your child ever had any unscheduled visits or phone contacts related to asthma that required a boost in your/your child's medications? *asthmdboost*				1 ☐	0 ☐
mdboostno	**1a**) If **YES**, number of times in the last year:	1 ☐ one	2 ☐ two or more		0 ☐ none
2. Have you/your child ever had any emergency room visits related to asthma? *ervisits*				1 ☐	0 ☐
ervisitno	**2a)** If **YES**, number times in last year:	1 ☐ one	2 ☐ two or more		0 ☐ none
3. Have you/your child ever had any overnight hospitalizations due to asthma? *onhosp*				1 ☐	0 ☐
onhospno	**3a**) If **YES**, number in the last year:	1 ☐ one	2 ☐ two or more		0 ☐ none
4. Have you/your child ever been admitted to the intensive care unit (ICU)? *admiticu*				1 ☐	0 ☐
5. Have you/your child ever had any respiratory episodes that were life threatening (required intubation or associated with loss of consciousness)? *lifethreat*				1 ☐	0 ☐

Triggers

Have any of the following caused you/your child to have asthma symptoms such as coughing, wheezing or shortness of breath?

1. Allergens	**Never**	**Some of the time**	**Most of the time**	**All of the time**	**Don't know**
a. Cats *cats*	0 ☐	1 ☐	2 ☐	3 ☐	888 ☐
b. Dogs *dogs*	0 ☐	1 ☐	2 ☐	3 ☐	888 ☐
c. House dust *housedust*	0 ☐	1 ☐	2 ☐	3 ☐	888 ☐
d. Molds/damp areas *molds*	0 ☐	1 ☐	2 ☐	3 ☐	888 ☐
2. Irritants:					
a. Sprays or perfumes *perfumes*	0 ☐	1 ☐	2 ☐	3 ☐	888 ☐
b. Tobacco smoke *smoke*	0 ☐	1 ☐	2 ☐	3 ☐	888 ☐
3. Daily Physical Activities: *phyact*	0 ☐	1 ☐	2 ☐	3 ☐	888 ☐
4. Exercise: *exercise*	0 ☐	1 ☐	2 ☐	3 ☐	888 ☐
5. Cold/upper Respiratory tract infection: *cold*	0 ☐	1 ☐	2 ☐	3 ☐	888 ☐
6. Emotional factors:	**Never**	**Some of the time**	**Most of the time**	**All of the time**	**Don't know**
a. Stress/anxiety *stress*	0 ☐	1 ☐	2 ☐	3 ☐	888 ☐
b. Anger *anger*	0 ☐	1 ☐	2 ☐	3 ☐	888 ☐
7. Drugs/Medicines					
a. Aspirin, ibuprofen, Motrin, aleve/naprosyn, etc. *aspirin*	0 ☐	1 ☐	2 ☐	3 ☐	888 ☐
b. Penicillin *penicillin*	0 ☐	1 ☐	2 ☐	3 ☐	888 ☐
8. Women/Girls only:					
a. Menstruation *menstruation*	0 ☐	1 ☐	2 ☐	3 ☐	888 ☐
b. Pregnancy *pregnancy*	0 ☐	1 ☐	2 ☐	3 ☐	888 ☐

Seasons

How severe are your/your child's asthma symptoms in each of the seasons of the last year?

	None	Mild	Moderate	Severe
1. Spring *spring*	0 ☐	1 ☐	2 ☐	3 ☐
2. Summer *summer*	0 ☐	1 ☐	2 ☐	3 ☐
3. Fall *fall*	0 ☐	1 ☐	2 ☐	3 ☐
4. Winter *winter*	0 ☐	1 ☐	2 ☐	3 ☐

Additional Respiratory Conditions

Do you/your child have or ever had:

	Never	Currently	Past only	Don't know
1. Seasonal nasal or eye-related allergies *nasallergies*	0 ☐	1 ☐	2 ☐	888 ☐
2. Eczema (itchy, scaley, weepy skin rash without hives)	0 ☐	1 ☐	2 ☐	888 ☐
3. Chronic sinusitis (sinus congestion, post nasal drip)	0 ☐	1 ☐	2 ☐	888 ☐
4. Sinus surgery *sinusurg*	0 ☐	1 ☐	2 ☐	888 ☐
5. Acute sinusitis (episodes of sinus pain/nasal discharge treated with antibiotics) *actsinusitis*	0 ☐	1 ☐	2 ☐	888 ☐

	Never	Currently	Past only	Don't know
6. Nasal Polyps (growths or mass protruding from mucous membrane usually associated with structural abnormality or obstruction of nasal passages)	0 ☐	1 ☐	2 ☐	888 ☐
7. Vocal Cord Dysfunction (spasm of the upper airway throat or voice box)	0 ☐	1 ☐	2 ☐	888 ☐
8. Obstructive Sleep Apnea (breathing problems during sleep that cause low oxygen levels and are usually treated with a machine) *obsslapnea*	0 ☐	1 ☐	2 ☐	888 ☐

9. Supplemental Oxygen *supoxygen*	0 ☐	1 ☐	2 ☐	888 ☐
10. Pneumonia diagnosed by a doctor. *penudiag*	0 ☐ Never	1 ☐Past	888 ☐ Don't know	
10a. Did you/your child get a chest x-ray? *chstxray*	1 ☐ Yes	0 ☐ No	888 ☐ Don't know	
10b. Did you/your child get an antibiotic? *antibiotic*	1 ☐ Yes	0 ☐ No	888 ☐ Don't know	

Non-respiratory Related History

Do you/your child have or ever had:

	Never	Currently	Past only	Don't know
1. Heartburn/Gastroesophageal Reflux Disease (GERD) *gerd*	0 ☐	1 ☐	2 ☐	888 ☐
2. Depression *depression*	0 ☐	1 ☐	2 ☐	888 ☐
3. Anxiety/ Panic disorder *anxiety*	0 ☐	1 ☐	2 ☐	888 ☐
4. Hypertension (high blood pressure) *htn*	0 ☐	1 ☐	2 ☐	888 ☐
5. Osteoporosis (thinning or decreased strength of bones) *osteo*	0 ☐	1 ☐	2 ☐	888 ☐
6. Diabetes/high sugars *diabetes*	0 ☐	1 ☐	2 ☐	888 ☐
7. Cataracts *cataracts*	0 ☐	1 ☐	2 ☐	888 ☐

Women/Girls Only ____N/A *male*

1. You/your child has started *menstr/preg/menp* ☐ Menstruation ☐ Pregnancy ☐ Menopause

Age at which you/your child began menstruation: _____ *menstrage*

2. Contraceptives: 0 ☐ Never 1 ☐ Currently 2 ☐ Past only 888 ☐ Don't know

contracptv

2a) If Yes, Type: *type* 1 ☐ Condom 2 ☐ Oral 3 ☐ Patch 4 ☐ Ring 5 ☐ IUD

3. Have you/your child ever been pregnant: *evrpregnnt* 1 ☐ Yes 0 ☐ No

3a) If YES, During Pregnancy did you/your child's asthma symptoms: *prgnasthma*

1 ☐ Improve 2 ☐ Worsen 3 ☐ Remain Same 4 ☐ Not Applicable

4. Surgery to remove you uterus (womb) (If YES Date completed: __/__/___) *utersrgdt*

utersrg 0 ☐ No 1 ☐ Yes 8888 ☐ Don't know

5. Surgery to remove one or both ovaries (If YES Date completed: __/__/___) *ovaryremvedte*

ovaryremve 0 ☐ No 1 ☐ Yes 8888 ☐ Don't know

6. Hormone Replacement Therapy 0 ☐ Never 1 ☐ Currently 2 ☐ Past only 888 ☐ Don't know

hrt

7. Postmenopausal: *pstmenopause* 0 ☐ No 1 ☐ Yes 2 ☐ Uncertain

7a) If Yes, did the onset occur: *naturalonst* 1 ☐ Naturally 2 ☐ Surgically

General Symptoms of Lung Disease

Do you/your child have or ever had:	Yes/currently	Past only	Never
1. **Cough**: Deep, chest, chronic *cough*	1 ☐	2 ☐	0 ☐
2. **Sputum**: Phlegm or mucus while coughing *sputum*	1 ☐	2 ☐	0 ☐
3. Chest tightness: difficult to breathe deeply/pressure in chest *chesttightness*	1 ☐	2 ☐	0 ☐
4. Wheezy, Whistling or Musical sound in Chest: *wheezy*	1 ☐	2 ☐	0 ☐
5. Shortness of Breath *shortnessbreath*	1 ☐	2 ☐	0 ☐
6. Nighttime Symptoms: waking from sleep, nighttime use of Albuterol, early morning chest tightness *pmsymptoms*	1 ☐	2 ☐	0 ☐

Medications

A. Steroids	Yes/currently	Past only	Never
1. Do/Did you/your child take oral steroids (prednisone or medrol) on a daily basis? *posteroids*	1 ☐	2 ☐	0 ☐

2. What is the total daily dosage? *dose*	1 ☐ 1-5 mg	2 ☐ 6-10mg	3 ☐ 11-20mg	4 ☐ 21+mg
3. How many steroid bursts did you/your child use in the last year? *steroidbursts*	1 ☐ One	2 ☐ 2-3	3 ☐ 4 or more	4 ☐ None
4. Do you/your child currently use inhaled steroids? *inhldsteroids*	0 ☐ No	1 ☐ Yes		
a. If Yes: Which one(s) do you use? (Please select only two)				
Advair Diskus (fluticasone/salmeterol) *advaird*	1 ☐ 100/50	2 ☐ 250/50	3 ☐ 500/50	

Advair HFA (fluticasone/salmeterol) *advairhfa*	1 ☐ 45/21	2 ☐ 115/21	3 ☐230/21
Aerospan HFA (Flunisolide hemihydrate) *aerospanhfa*			☐ 80
Asmanex (mometasone furoate) *asmanex*			☐ 220
Azmacort (triamcinolone acetonide) *azmacort*			☐ 100
Flovent (fluticasone) *flovent*	1 ☐ 44	2 ☐ 110	3 ☐ 220
Pulmicort Turbuhaler (budesonide) *pulmicortth*			☐ 220
Pulmicort Flexhaler (budesonide) *pulmicortfh*	1 ☐ 90	2 ☐ 180	
Pulmicort Respules (budesonide) *pulmicortrp*	1 ☐ 250	2 ☐ 500	
Qvar HFA (beclomethasone dipropionate) *ovarhfa*	1 ☐ 40	2 ☐ 80	
Symbicort (budesonide/formoterol) *symbicort*	1 ☐ 80/4.5	2 ☐ 160/4.5	
Alvesco *alvesco*	1 ☐ 80mcg	2 ☐ 160mcg	

b. What is your current prescribed daily dose? (Total puffs per day) *puffsperdaycur*

1 ☐ 1 2 ☐ 2 3 ☐ 3 4 ☐ 4 5 ☐ 6 6 ☐ 8

B. Other asthma controller medications:

Do you/your child use or have ever used:	**Yes/currently**	**Past use**	**Never taken**
1. Inhaler beta-agonist (Albuterol: ProAir, Proventil, Ventolin, Xopenex)? *betaagonistih*	1 ☐	2 ☐	0 ☐

1a) What is your total puffs per day?

ibapuffs 1 ☐ **1** 2 ☐ **2** 3 ☐ **3** 4 ☐ **4** 5 ☐ **6** 6 ☐ **8**

2. Nebulized beta-agonist (Alupent soln, Proventil, Xopenex)? *nebulizedba*	1 ☐	2 ☐	0 ☐

2a) What is your total nebulized dose per day? *nbadose*

1 ☐ **1** 2 ☐ **2** 3 ☐ **3** 4 ☐ **4** 5 ☐ **6** 6 ☐ **8**

	Yes/currently	Past use	Never taken
3. Oral beta-agonist (Volmax, Repetab) or liquid albuterol? *oralba*	1 ☐	2 ☐	0 ☐
4. Long-acting bronchodilator (Foradil, Serevent, Brovana or Advair)? *lgacbroch*	1 ☐	2 ☐	0 ☐
5. Leukotriene inhibitors (Singular, Accolate, or Zyflo)? *leukotrieneinhb*	1 ☐	2 ☐	0 ☐
6. Theophyllines (Theo-dur, Slobid, Uniphyl)? *theophyllines*	1 ☐	2 ☐	0 ☐
7. Ipratroprium bromide (Atrovent or Combivent)? *atrovent*	1 ☐	2 ☐	0 ☐
8. Tiotroprium bromide (Spiriva)? *spiriva*	1 ☐	2 ☐	0 ☐
9. Injectable Corticosteroids (Kenalog, Decadron, Depomedrol, Solumedrol)? *injcort*	1 ☐	2 ☐	0 ☐
C. Immunotherapy *immunothrpy*	**Yes/currently**	**Past use**	**Never taken**
Have you/your child ever had allergy shots/immunotherapy?	1 ☐	2 ☐	0 ☐
D. Anti-IgE therapy			
Have you/your child ever had Xolair? *xolair*	1 ☐	2 ☐	0 ☐
E. Alternative Medications			
Have you/your child ever tried any alternative medicines or treatments for your asthma such as acupuncture, chiropractor, or herbal teas? *altmeds*	1 ☐	2 ☐	0 ☐

Medications for Other Conditions

	Yes/currently	Past use	Never taken
1. Nasal Steroids (Beconase, Flonase, Fluticasone, Nasocort, Nasonex, Rhinocort, Vancenase, or Veramyst)? *nslstrds*	1 ☐	2 ☐	0 ☐
2. Reflux medications (for GERD) *gerdmds*	1 ☐	2 ☐	0 ☐
2a) Proton pump inhibitors, (Prevacid (lansoprazole); Prilosec (omeprazole); Nexium (esomeprazole) *protpinhb*	1 ☐	2 ☐	0 ☐
2b) H2 blockers (Tagamet (cimetidine); Zantac (ranitidine); Pepcid (famotidine). *h2blockers*	1 ☐	2 ☐	0 ☐
3. Osteoporosis/ Bone Density Medications *bonemeds*	1 ☐	2 ☐	0 ☐
4. **Antidepressants** *antidep*	1 ☐	2 ☐	0 ☐
5. Anti-anxiety medicines (Valium/Ativan) *antianxds*	1 ☐	2 ☐	0 ☐
6. Diabetes Medications (including insulin and pills) *diabtsmds*	1 ☐	2 ☐	0 ☐
7. High blood pressure medications *hbpmeds*	1 ☐	2 ☐	0 ☐

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