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Associations Of Traffic Related Air Pollution With Physical Activity And Cardiorespiratory Health Outcomes In At-Risk Populations From El Paso, Texas

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ASSOCIATIONS OF TRAFFIC RELATED AIR POLLUTION WITH PHYSICAL ACTIVITY AND CARDIORESPIRATORY HEALTH OUTCOMES IN AT-RISK POPULATIONS FROM EL PASO, TEXAS

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EPIGRAPH

To my beloved parents, siblings, and future family

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by

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DISSERTATION

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ABSTRACT

Exposure to air pollution from traffic-related emissions is a preventable cause of respiratory and cardiovascular diseases. However, the impacts on at-risk populations, such as children with asthma and low-income residents, are yet to be fully understood in the border city of El Paso, TX. This dissertation focused on the most common traffic-related pollutants which include particulate matter ($PM_{2.5}$ and PM_{10}), nitrogen dioxide (NO_2), and ozone (O_3). The research described in this work provides an overview of air pollution measurements and shares insights from three different studies in our region.

People with asthma are more likely adversely affected by traffic emissions, particularly young children. Previous studies showed regular exercise reduces asthma exacerbation and improves lung function. However, few studies have looked at the physical activity and air quality relationship. We found inverse associations of air pollution and time spent in physical activity by children with asthma attending an elementary school near a heavy traffic road.

Through secondary data analyses, we also linked short term effects of traffic-related pollutants with respiratory outcomes such as airway inflammation and lung function. Furthermore, we found associations between air pollution and metabolic syndrome in our region. Our investigations included measured concentrations of traffic-related pollutants and land use regression models using geographic information system (GIS) measures. As expected, we found associations between air pollution and respiratory outcomes, but also unexpected associations with obesity from both short-term and long-term exposure to air pollutants. We expect future studies to consider statistical models that combine geographic information systems with more health outcomes to elucidate further negative health effects caused by exposure to air pollution in vulnerable populations.

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CHAPTER 1 INTRODUCTION

This dissertation is focused on traffic-related air pollution and the negative health impact it has on physical activity and cardiorespiratory health outcomes such as lung function and inflammation, as well as risk factors for metabolic syndrome. This research provides data to support mitigation strategies that would reduce the impact of air pollution exposure in at-risk environments such as schools and low income communities.

Pollutants in the atmosphere can cause harm to living organisms and the natural environment. Even at relatively low concentrations, the health effects of air pollution are of great concern (Kim, Kabir, & Kabir, 2015). Air pollutants contribute to various health problems including heart or lung disease, irregular heartbeat, aggravated asthma, decreased lung function, and increased respiratory symptoms (Atkinson, Fuller, Anderson, Harrison, & Armstrong, 2010a; Cadelis, Tourres, & Molinie, 2014a; Andrew W. Correia et al., 2013). Furthermore, several reviews summarize the associations of ambient (outdoor) air pollution with diabetes (Eze et al., 2015), asthma emergency room visits (Zheng et al., 2015), blood pressure (Giorgini et al., 2016), and cardiovascular disease (Franklin, Brook, & Pope III, 2015).

Outdoor air pollution leads to 3.3 million premature deaths per year worldwide with important contributions in the U.S from power generation and traffic. (Lelieveld, Evans, Fnais, Giannadaki, & Pozzer, 2015). In an urban environment, traffic air pollutants originate from the emissions of motor vehicles, wear of vehicle components such as brakes and tires, and suspension of road dust (Kok, Driece, Hogervorst, & Briedé, 2006). Those who are more likely to be affected by excessive levels of air pollutants include people with asthma, lower income, and those living close to heavy traffic roads (Alexis et al., 2014; Xianglu Han & Luke P. Naeher, 2006; Makri & Stilianakis, 2008).

Recent studies have found significant associations of traffic-related pollutants with airway inflammation and lung function in children with asthma (Provost, Madhloum, Int Panis, Boever, & Nawrot, 2015). In elementary school children, those living 30-300 meters from a major roadway were found to have increased arterial stiffness, carotid intima-media thickness, higher absenteeism, and asthma symptoms, as well as decreased academic performance (Armijos et al., 2015). In addition, people with lower incomes are considered at risk since they are more likely to live in neighborhoods with higher pollution levels, including traffic-related air pollution (Cushing, Morello-Frosch, Wander, & Pastor, 2015) making this an environmental justice issue.

The focus of this dissertation is to investigate associations of negative cardiorespiratory health effects and air pollutants in at-risk populations (children with asthma, low-income residents, and people living near heavy traffic roads). In addition, the findings of this work will be used to inform stakeholders regarding mitigation strategies that could decrease the exposure of traffic-related pollutants in these populations.

1.1 Structure of the dissertation

The first chapter of this dissertation will provide an overview of air pollution and how it is measured, cardiorespiratory outcomes associated with air pollution, as well as outline the specific aims, concepts, and theoretical framework of this research. The second chapter will describe a research study investigating the association of air pollution with time spent in physical activity by children with asthma attending an elementary school near a heavy traffic road. The third and fourth chapters will focus on secondary data analyses exploring the association between air pollution levels and cardiorespiratory health outcomes in low-income communities. The final chapter will include a summary of the findings, recommendations about air pollution mitigation strategies, suggestions for future research, and conclusions.

1.2 Measures of interest

The research presented in this dissertation used several methods of assessing air quality. These methods will allow explorations of how air quality is associated with physical activity and cardiorespiratory outcomes in our selected populations.

1.2.1 AIR QUALITY MEASUREMENTS

Currently used methods for assessing air quality include the use of ground-level monitors, continuous ambient monitoring stations (CAMS), and emission-based air quality models (El-Harbawi, 2013; Steinle, Reis, & Sabel, 2013). In addition, air quality models can be improved with the use of traffic-related data which can provide information through spatial modeling of traffic volume and density.

Ground-level air pollution monitors placed at sites of interest provide the most accurate data compared to that monitored at locations away from the site and models. Another advantage of ground-level monitors is the possibility to determine their location and ensure they represent an accurate estimate of the air pollution exposure at a selected site. However, purchasing and maintaining the monitoring equipment can be expensive which makes this method less feasible for obtaining air quality data in large studies (Engel-Cox, Oanh, van Donkelaar, Martin, & Zell, 2013; O'Neill et al., 2003).

A more common approach for acquiring air quality data is to retrieve verified data from centralized, state-operated CAMS; traffic-related pollutant data obtained from these stations can be generalized for the communities surrounding them (Gonzales et al., 2012; Sayeed et al., 2020; Staniswalis, Yang, Li, & Kelly, 2009). Using CAMS data provide a cost-effective method to assess air pollution exposure, and many U.S. cities rely on them for air quality monitoring and compliance. However, when compared with on-site monitoring, the distance from the monitoring

station and their spatial variability limit the accuracy of this method. Nevertheless, some regional studies have found success with this method by comparing on-site measurements with CAMS data (Raysoni, Stock, Sarnat, Sosa, et al., 2013; Sandoval, 2012).

Emission-based air quality models that use geographic information system (GIS) measures are another way to assess traffic-related air pollution at exposure sites. For example, dispersion models can predict air pollution near roadways when emission factors, meteorological conditions, and traffic data are available near roadways of interest (D. Wen et al., 2017). Furthermore, traffic density within an impact zone, population density, distance to a major roadway, and percent urbanization have been used in research linking prenatal traffic-related air pollution exposure and birth weight (Lakshmanan et al., 2015). Other traffic-related variables such as length of street around address, census block size, and traffic counts may act as a surrogate exposure indicator for elevated levels of air pollution.

1.2.2 PHYSICAL ACTIVITY

Physical activity is essential for overall health and regular outdoor activities can lead to a significantly lower risk of cardiovascular and other chronic diseases (M. Chen et al., 2013; Janssen & LeBlanc, 2010). Since self-reported data is not very reliable, researchers should use objective measures to accurately assess the impact of physical activity on health outcomes (Gorber & Tremblay, 2016; McCormack et al., 2004). As an objective measure, accelerometers are a standard tool used for an individualized assessment of physical activity. These devices measure acceleration along three axes and use algorithms to categorize movement into time spent in sedentary, light, moderate, and/or vigorous activity (Troiano et al., 2008).

1.2.3 CARDIORESPIRATORY OUTCOMES

The health outcomes of interest for this study which are associated with exposures to traffic air pollution are lung inflammation, lung function, and metabolic syndrome.

1.2.3.1 Lung inflammation

Exhaled Nitric Oxide (eNO) is considered a biomarker of inflammation present in the respiratory tract and lungs; this is an important indicator of symptom exacerbation in asthma and other lung diseases (Pendharkar & Mehta, 2008). An elevated eNO value indicates airway inflammation which could be due to an increased inflammatory response and can trigger symptoms in people with asthma (Holguin, 2008). Assessments of eNO have been used in large epidemiological studies to elucidate the negative impacts of air pollution in children with asthma due to respiratory tract and lung inflammation (Delfino et al., 2006; Holguin, 2008), but also in adults with an without asthma (Tunnicliffe, Harrison, Kelly, Dunster, & Ayres, 2003).

1.2.3.2 Lung function

Lung function measurements are assessed by using a spirometry device. For this test, the participant is required to blow as hard as possible through tubing connected to a device that will record the volume of air expired. The results are expressed in terms of forced vital capacity (FVC), forced expiratory volume in one second (FEV_1), and peak expiratory flow (PEF). The alterations in these measures could indicate if a participant has an increased likelihood of respiratory disease and have been used in assessments of traffic and air pollution exposure (Hankinson, Odencrantz, & Fedan, 1999; Holguin, 2008).

1.2.3.3 Metabolic syndrome

Metabolic syndrome (MetS) is a cluster of associated risk factors for cardiovascular disease, type 2 diabetes, hypertension, and lipid disorders. MetS is linked to insulin resistance, which increases the risk of developing some of the mentioned conditions (Expert Panel on Detection & Treatment of High Blood Cholesterol in, 2001). Currently, MetS integrates the risk of related diseases by considering the presence of at least three out of five risk factors. The five risk factors for MetS are: large waistline, high blood pressure, high triglyceride level, low HDL-cholesterol level, and high fasting blood glucose (Figure 1.1) (Rice et al., 2015).

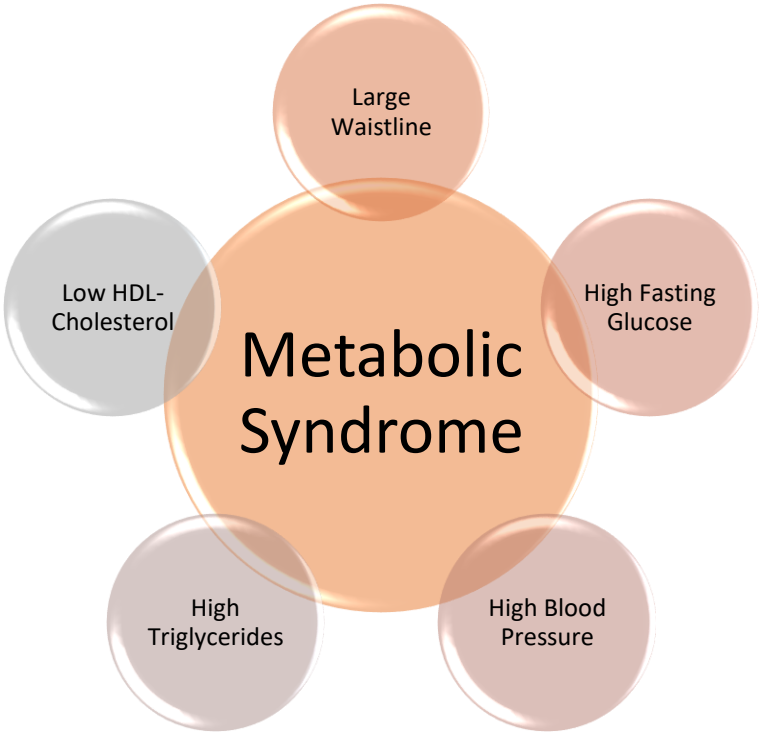


Figure 1.1: Risk factors for metabolic syndrome

In the U.S., the cutoff values for MetS have been established by the NIH guidelines and are based on the National Cholesterol Education Program (NCEP) Adult Treatment Panel III

recommendations. These guidelines are summarized in Table 1.1. (Expert Panel on Detection & Treatment of High Blood Cholesterol in, 2001; Grundy, Hansen, Smith, Cleeman, & Kahn, 2004).

Table 1.1: Diagnostic criteria for metabolic syndrome

Risk Factor	Cutoff values
Large waistline	≥ 102 cm (≥ 40 inches) in men ≥ 88cm (≥ 35 inches) in women
High blood pressure	≥ 130mmHg systolic or ≥ 85mmHg diastolic
High triglycerides	≥ 150mg/dL
Low HDL-cholesterol	< 40mg/dL in men < 50mg/dL in women
High fasting glucose	≥ 100 mg/dL

1.3 Description of relevant research studies

1.3.1 Healthy Living and Traffic-Related Air Pollution in an Underserved Community Study

The second chapter described in this dissertation is part of a larger project that quantifies traffic-related air pollution and the associated respiratory health and physical activity of at-risk school children living in a near-road, underserved community. This study aimed to develop related health guidelines for a selected school district and community to reduce the burden of air pollution on children's health. The project was funded through the Center for Advancing Research in Transportation Emissions, Energy, and Health (CARTEEH). Specific project activities included the collection of children's on-campus school physical activity data and on-site measurements of traffic-related air pollutants (particulate matter, nitrogen dioxide, and ozone). The selected population on which this study focused was a cohort of twelve children with asthma attending an elementary school near a heavy traffic road measured over a period of ten weeks.

1.3.2 Evidence-based Screening for Obesity, Cardiorespiratory Disease, and Environmental Exposures in Low-income El Paso Households Study

The third and fourth chapters in this dissertation analyze the health outcome data from a project funded through the City of El Paso's Department of Public Health (EPDPH). Overall, this project aims to evaluate the health status of participants who are uninsured or have a low-income status by using a questionnaire and health screenings to assess their overall health status.

For the first year of the EPDPH project, the health screenings conducted included blood pressure, anthropometric measurements (height, weight, and waist), spirometry, exhaled nitric oxide, a lipid profile (triglycerides, total cholesterol, HDL, LDL) and fasting glucose measurements. After the first year, nitric oxide and spirometry assessments were not performed due to high testing costs and efforts were redirected into collecting data related to metabolic syndrome and its components which are known risk factors for cardiovascular disease. As of January 2020, this project has served approximately 5,000 participants.

1.4 Specific study aims

The specific aims for this dissertation include the following:

Aim 1: Investigate the longitudinal relationship between traffic-related air pollutants and physical activity in children with asthma attending a school near a heavy traffic road in El Paso, TX.

- Objective 1.1: Assess the relationship between percent time spent in physical activity levels (%time spent in moderate to vigorous physical activity (MVPA), %time spent in light physical activity, and %time spent in sedentary activity) and traffic-related air pollutants ($PM_{2.5}$, PM_{10}, NO_2, and O_3).

Aim 2: Investigate the short-term association between cardiorespiratory health outcomes (MetS risk factors, lung function, and inflammation) and traffic-related air pollutants (PM, NO_2, O_3) in residents of low-income communities of El Paso, TX.

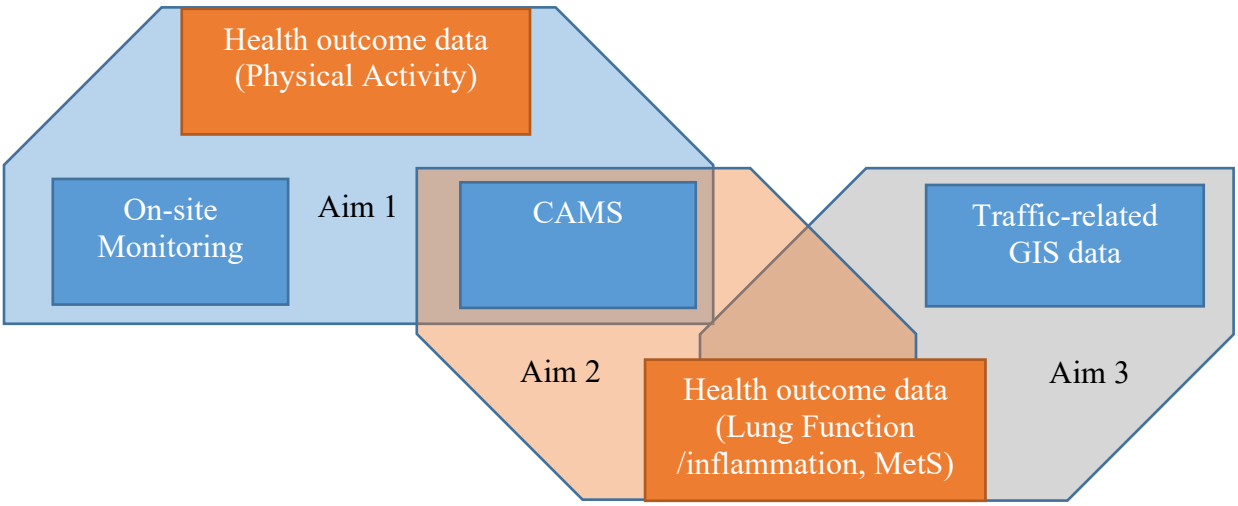
- Objective 2.1: Assess the relationship between individual cardiovascular health outcomes of MetS (BMI, waist circumference, blood pressure, triglycerides, HDL-cholesterol, and glucose) and traffic-related air pollutants ($PM_{2.5}$, PM_{10}, NO_2, and O_3).
- Objective 2.2: Determine if MetS classification (binary) is related to traffic-related air pollutants ($PM_{2.5}$, PM_{10}, NO_2, and O_3).
- Objective 2.3: Determine relationships between respiratory health outcome measures for lung function and inflammation (exhaled nitric oxide, FVC, FEV_1, and PEF) and traffic-related air pollutants ($PM_{2.5,}$ PM_{10}, NO_2, and O_3) using data from the first year of the study that contained respiratory outcome measurements.

- Aim 3: Investigate the long-term association between individual health outcomes (lung function, inflammation, MetS risk factors) and traffic-related GIS data for residents of low-income communities of El Paso, TX.

- Objective 3.1: Assess the relationship between individual cardiovascular health outcomes of MetS (BMI, waist circumference, blood pressure, triglycerides, HDL-cholesterol, and glucose) and traffic-related GIS data (distance to nearest major traffic road, traffic counts, total length of street within a 500m and 1000m radius, distance to nearest port of entry).
- Objective 3.2: Determine if MetS classification (binary) is related to spatial transportation data.

- Objective 3.3: Determine the relationships between respiratory health outcome measures for lung function and inflammation (exhaled nitric oxide, FVC, FEV_1, and PEF) and the spatially distributed traffic-related data among a subset of the data (the first year of the study with respiratory outcome measurements).

The outline and relationship between outcome variables and measurements of air pollution exposure are outlined below (Figure 1.2) while Table 1.2 describes in more detail the variables to be considered by aim and population.



CAMS: Continuous Ambient Monitoring Station
GIS: Geographical Information System
MetS: Metabolic Syndrome

Figure 1.2: Specific aims outline diagram

Table 1.2: Measures to be explored for each specific aim

Specific Aim	Air Pollution	Outcome	Population
Aim 1	• $PM_{2.5}$ (Continuous) • PM_{10} (Continuous) • NO_2 (Continuous) • O_3 (Continuous)	• % time spent in moderate to vigorous physical activity (MVPA) • % time spent in light physical activity • % time spent in sedentary activity	Children with asthma attending an elementary school near a heavy traffic road (2017) (N=12)
Aim 2 Short term exposure	• $PM_{2.5}$ (Continuous) • PM_{10} (Continuous) • NO_2 (Continuous) • O_3 (Continuous)	• BMI (Continuous) • Waist Circumference (Continuous) • Mean Blood Pressure (Continuous) • Triglycerides (Continuous) • HDL (Continuous) • Glucose (Continuous)	Residents from housing authority communities (2014-2015) (N=662)
		• Metabolic syndrome (Categorical)	
		• eNO (Continuous) • FEV_1 (Continuous) • FVC (Continuous)	
Aim 3 Long term exposure	• Distance to nearest high traffic road • Traffic count (VMT) within 500m & 1000m radius (traffic MPO data) • Total length of street within 500m and 1000m (Census data) • Distance to port of entry	• BMI (Continuous) • Waist Circumference (Continuous) • Mean Blood Pressure (Continuous) • Triglycerides (Continuous) • HDL (Continuous) • Glucose (Continuous)	Low-income residents from El Paso County (2014-2020) (N=4,959)
		• Metabolic syndrome (Categorical)	
		• eNO (Continuous) • FEV_1 (Continuous) • FVC (Continuous)	Residents from housing authority communities (2014-2015) (N=662)

1.5 Research questions

The main research question of this dissertation explores if there is an association between traffic-related air pollution and the health-related outcomes (physical activity, lung function and inflammation, and metabolic syndrome risk factors). The initial step was formulating a plan for data collection and defining any specific subgroups to sample. The objectives included collecting or retrieving health data from participants at their school/neighborhoods or at a convenient location. Air quality data were obtained through on-site monitoring and from the Texas Air Monitoring Information System (TAMIS) database for CAMS maintained by the Texas Commission of Environmental Quality (TCEQ), in the case of air pollutant concentrations; traffic-related data were calculated using data available through the Census.gov website, the El Paso Metropolitan Planning Organization (MPO) TransCAD model, and the Department of Transportation (DOT) GIS mapping. Health-related data were obtained through the relevant studies, the *Healthy Living and Traffic-Related Air Pollution in an Underserved Community* study and the *Evidence-Based Screening for Obesity, Cardiorespiratory Disease, and Environmental Exposures in Low-Income El Paso Households* study.

1.6 Theoretical framework

1.6.1 TRAFFIC-RELATED AIR POLLUTANTS

Air pollutants can originate from anthropogenic (man-made) sources or natural activities. In urban areas, the main sources of anthropogenic emissions include those associated with motor vehicle traffic (exhaust emissions; wear of tires, brakes, and roads), industrial activities (power plants, oil refineries, chemical facilities), housing (food cooking and heating), building (excavations, demolitions), as well as cigarette smoking and aerosol canisters. The work of this dissertation will explore the most common traffic-related air pollutants (PM, NO_2, and O_3).

The Environmental Protection Agency (EPA) classifies particulate matter (PM) in different size categories which determines their transport and depth of penetration into the respiratory and cardiovascular system; the categories include coarse particles (PM_{10}) which have an aerodynamic diameter of 10μm or smaller; fine particulates ($PM_{2.5}$) which have a diameter of 2.5μm or smaller; and ultrafine particles which have a diameter less than 0.1μm (Esworthy, 2013). Motor vehicle emissions are a major source of particulate matter (PM) in urban environments and can have a wide range (14 to 50%) of the total fine particle mass (Hailin et al., 2008; Sheesley, Schauer, Chowdhury, Cass, & Simoneit, 2003; Yu et al., 2013). Besides fuel emissions, PM is also generated by the wearing and breakdown of vehicle components such as brakes and tires, as well as suspension of road dust (de Kok, Driece, Hogervorst, & Briedé, 2006). Exposure to particulate matter is associated with heart and lung disease, irregular heartbeat, aggravated asthma, and decreased lung function (Atkinson et al., 2010a; Cadelis et al., 2014a; Andrew W. Correia et al., 2013).

Nitrogen dioxide (NO_2) is one of a group of highly reactive gases known as oxides of nitrogen or nitrogen oxides ($NOx = NO + NO_2$) and it is a primary indicator of emissions from cars, trucks and buses, and off-road equipment. Breathing air with a high level of NO_2 can irritate the respiratory airways leading to inflammation and aggravate respiratory diseases, particularly asthma. This airway inflammation might lead to respiratory symptoms (such as coughing, wheezing or difficulty breathing), hospital admissions, and visits to emergency rooms (Alving, Weitzberg, & Lundberg, 1993). Furthermore, longer exposures to elevated concentrations of NO_2 may contribute to the development of asthma and potentially increase susceptibility to respiratory infections (Khreis et al., 2017).

Ozone (O_3) is composed of three oxygen atoms and at ground level is formed through photolysis of NO_2 in the presence of solar radiation and other precursor compounds such as nitrous compounds and various volatile organic compounds (VOC). Ozone is considered a highly reactive gas and its release into the environment depends on the presence of the precursor pollutants and the conditions promoting the transformations (heat and sunlight) (Krzyzanowski, 1997). When inhaled, ozone chemically reacts in the respiratory tract, leading to a number of adverse health effects (Nuvolone, Petri, & Voller, 2018).

Because of the influence of traffic-related pollutants on human health, there has been a rise in studying their short-term and long-term impact (Abraído-Lanza, Echeverría, & Flórez, 2016; Shima, 2017). However, the differences in measurement methods make it difficult to generalize and compare findings between air pollution exposure studies (Xianglu Han & Luke P Naeher, 2006). Nevertheless, increasing the amount of research by conducting air pollution exposure studies in our region adds knowledge about the impact of traffic-related pollutants which can be used to influence local policy changes.

1.6.2 IMPACTS OF AIR QUALITY ON PHYSICAL ACTIVITY

In an air polluted environment, people who engage in outdoor physical activity are likely to have increased exposure to air pollutants compared to those who have a more sedentary lifestyle, which could be counterproductive to the promotion of physical activity, Nevertheless, the benefits of physical activity are essential for overall health; at least a mean of 30 minutes per day of light physical activity is recommended and moderate to vigorous physical activity should be incorporated when possible (Janssen & LeBlanc, 2010).

Under controlled conditions, exposure to air pollutants during exercise showed reductions in exercise performance (Cutrufello, Rundell, Smoliga, & Stylianides, 2011) and lung function

(Cutrufello, Smoliga, & Rundell, 2012). Furthermore, increased levels of air pollutants have been associated with self-reported physical inactivity (Roberts, Voss, & Knight, 2014; X.-J. Wen, Balluz, Shire, Mokdad, & Kohl III, 2009). In summary, exposure to air-polluted environments might lead to negative health effects due to airway exposure to air pollutants and lack of physical activity.

Although, regular outdoor activities can significantly lower risk of metabolic syndrome (M. Chen et al., 2013), increased respiratory demand during physical activity may lead to higher deposition of air pollutants in the lungs (Giles & Koehle, 2014), which can lead to respiratory and cardiovascular problems resulting from exposure to air pollutants during outdoor physical activity (Le Tertre et al., 2002; Pope et al., 2015; Shah et al., 2013; Sharman, Cockcroft, & Coombes, 2004). Therefore, there may exist a contrast of negative and positive health effects when performing physical activity in areas with higher air pollution.

Health risks of air pollution are thought to increase linearly with increased exposure from low to moderate levels of air pollution. Still, the benefits of physical activity (PA) also follow a linearly increased curve with increasing dose (Kelly et al., 2014). This paradox exposes the need to explore correlations between air pollutant exposure and other factors that impact health. Some studies have estimated the health benefits and risks of physically active traveling (e.g. cycling, walking) in different areas (Doorley, Pakrashi, & Ghosh, 2015; Gröning, 2004; Tainio et al., 2016). In these studies, the health benefits due to PA from increased active traveling were significantly larger than the health risks caused by increases in exposure to air pollution. Furthermore, Tainio and collaborators (2016) showed that promoting cycling and walking is justified in the vast majority of settings, and only in cities with a very high level of PM the risk might outweigh the benefit.

Yet, the question remains whether a dose-response relationship between physical activity and air pollution applies to different types of activities and how it affects vulnerable groups. Other studies have shown that people with asthma (or even those with mild asthmatic symptoms) may have reduced physical activity and avoid aerobic fitness and leisure-time energy expenditure due to concerns about triggering asthma symptoms (Garfinkel, Kesten, Chapman, & Rebuck, 1992; Mälkiä & Impivaara, 1998). Given that asthma affects children at a young age, when they are likely to establish their health habits, it is important to emphasize physical activity with asthma patients (Mancuso et al., 2006). The U.S. management guidelines for asthma state that most patients can be controlled well enough to perform physical activity and that additional therapy options can be made available to them (Busse et al., 2007; Education, Program, Lung, & Asthma, 1997). Therefore, it is in the best interest of those who have asthma to achieve a balance between having a healthy amount of physical activity and controlling their respiratory symptoms.

Unfortunately, the impact of air pollution on people with asthma often prevents people from achieving a physically active lifestyle. Participants with asthma who performed exercise in an environment that had high levels of pollution were at a higher risk of having an asthma attack (Sharman et al., 2004) and lung diseases (Giles & Koehle, 2014). Also, children with asthma living in low income communities are likely to have increased clinical asthma symptoms when they are exposed to short-term increases in air pollutants (Wendt, Symanski, Stock, Chan, & Du, 2014).

In conclusion, the importance of promoting physical activity for overall health conflicts with the negative consequences of physical activity in environments with high levels of air pollutants. Therefore, further research and improvement of exposure models to air pollutants are needed to compare physical activity but also other health outcomes to improve recommendations.

1.6.3 ASSOCIATION BETWEEN AIR QUALITY AND LUNG FUNCTION AND INFLAMMATION

Expiratory function measured by spirometry is used as a marker for respiratory health. Regarding children's respiratory health, a meta-analysis explored associations between primary traffic air pollutants (NO_2, NO, NO_x, $PM_{2.5}$) and lung function in almost 5,000 children (Barone-Adesi et al., 2015). The study showed an inverse association between most of the pollutants and both FEV_1 and FVC. Furthermore, other studies have documented the association between residential air pollution exposure and reduced lung function in children with asthma (Delfino et al., 2008) and relationships with ozone and nitrogen dioxide levels (Ierodiakonou et al., 2016).

Particulate matter can affect gas exchange within the lungs and can even penetrate the lung, Smaller particles behave similarly to gas molecules which allows them to reach into the circulatory system, eventually, these particles in the bloodstream can cause significant health problems (Shah et al., 2013). Also, research suggests associations between changing PM levels and acute-phase reactants, endothelial dysfunction, and altered autonomic control of the heart (Sun, Hong, & Wold, 2010). This exchange between small particles and the circulatory system affects cardiovascular outcomes possibly due to oxidative stress and inflammation (Møller & Loft, 2010). Furthermore, a study by Hoffman and collaborators showed that residential exposure to traffic is associated with coronary heart disease (Hoffmann et al., 2007).

1.6.4 ASSOCIATION BETWEEN AIR QUALITY AND METABOLIC SYNDROME

Metabolic Syndrome is one of the major medical and public health problems in the U.S. affecting about 22.5% of adults in the U.S. (Beltrán-Sánchez, Harhay, Harhay, & McElligott, 2013; Ford, 2005) and about one-quarter of the world population (Cameron, Shaw, & Zimmet, 2004; Grundy, 2015; Saklayen, 2018). Evidence suggests exposure to air pollutants can alter biochemical pathways that control adipose tissue, increase the number of fat cells, alter food intake

and metabolism, influence release of inflammatory mediators, and affect glucose metabolism (Rao, Patel, Puett, & Rajagopalan, 2015; Wellen & Hotamisligil, 2003; Xu et al., 2003). Since the mentioned biochemical pathways contribute in the development of Mets risk factors (obesity, high blood pressure, altered lipids, and high blood glucose) air pollution contributes to the burden of MetS.

In addition, air pollution may lead to type 2 diabetes and promote the development of several cardiovascular risk factors (such as elevated lipid profiles and blood pressure) (Bowe et al., 2018; Pope et al., 2015; Rao et al., 2015). Possible risks for this association include exposure to ambient pollutants from vehicles and industrial emissions; however, the link between inflammation and long-term air pollution exposure in humans is still lacking (J.-C. Chen & Schwartz, 2008). If such links were demonstrated, they might provide insights into the high prevalence of type-2 diabetes and cardiovascular disease.

The goals of our proposed research are to better understand the relationship between health outcomes and air pollutants from anthropogenic sources related to traffic. The research scope of the presented studies will explore air pollution exposure in children with asthma and low-income residents with risk factors related to cardiorespiratory diseases.

A theoretical framework illustrating relationships between air pollution and cardiorespiratory outcomes is provided in Figure 1.3.

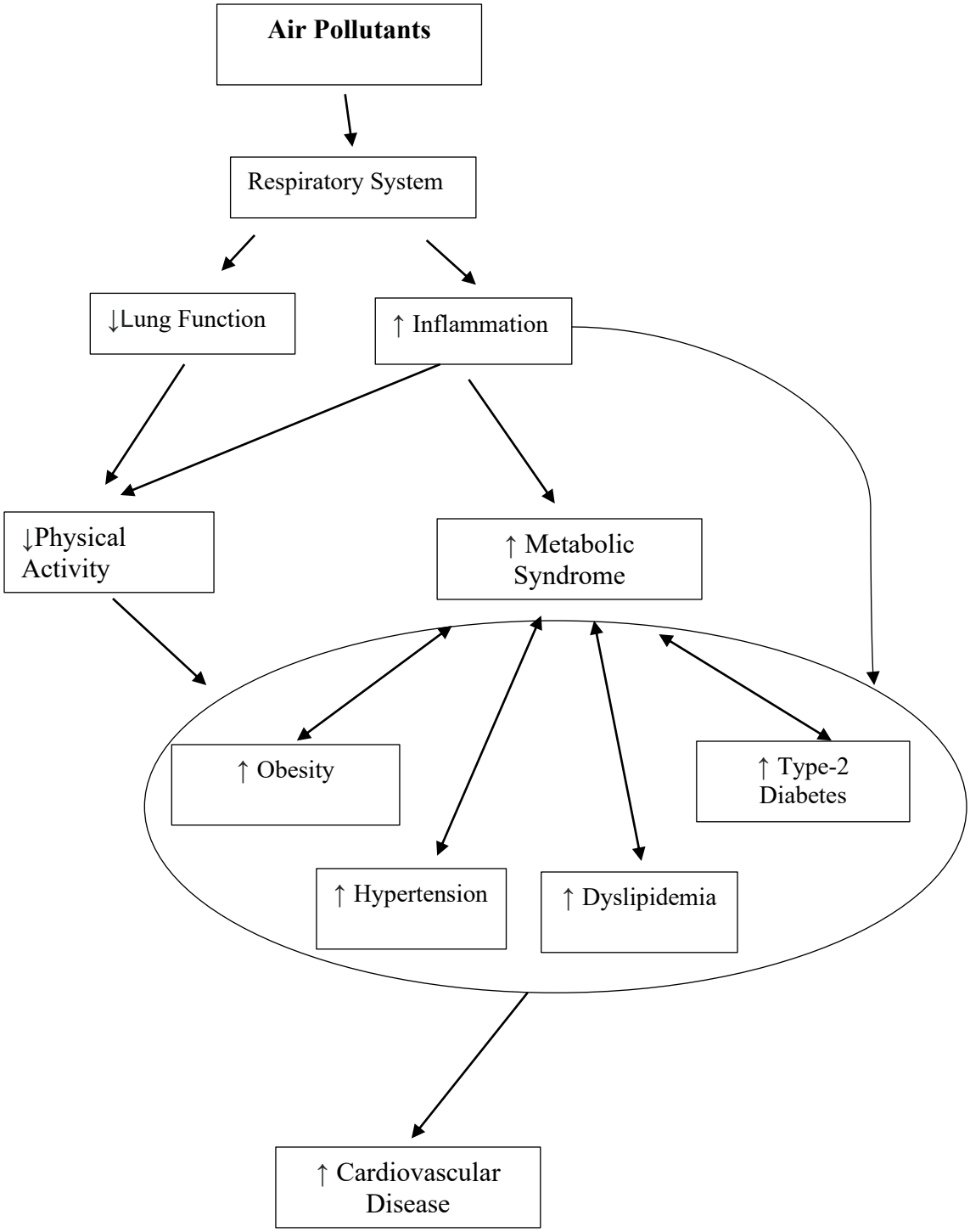


Figure 1.3: Theoretical framework

1.7 Air pollution in El Paso

El Paso, Texas usually meets National Ambient Air Quality Standards for nitrogen dioxide, $PM_{2.5}$, and ozone. However, its high desert location makes attainment of lower levels of coarse particles PM_{10} difficult. Previous studies have determined how anthropogenic sources, meteorological conditions, and topography cause variation on the concentration of air pollutants in the El Paso region. For example, traffic emissions from the El Paso-Ciudad Juarez international border crossings make up a sizable portion of the anthropogenic emissions in El Paso.

Li and collaborators characterized the temporal and spatial variations, along with the composition of particulate matter (PM_{10} and $PM_{2.5}$), and found their concentrations increase during the winter (W.-W. Li et al., 2001) . Also, a 2010 study across four schools determined that PM_{10} concentration was greater in the area encompassed by the I-10 Freeway and the El Paso/Ciudad Juarez border highway (Raysoni et al., 2011). Furthermore, weekday traffic during peak hours contributes to increases in all traffic-related pollutants (Noble et al., 2003).

Nitrogen dioxide has been found to predominate in central El Paso with lower values in the East and West sides of the city. In addition, a winter study showed significant variability in NO_2 concentrations, which decreased as ground elevation increased in the city (Gonzales, Qualls, Hudgens, & Neas, 2005). This was further studied using GIS with elevation, population density, distance to border crossing and distance to a petroleum facility as useful predictive variables of NO_2 concentration (Smith et al., 2006). Furthermore using land use regression showed that concentrations of NO_2 increase during wintertime and in the central regions of the city (Gonzales et al., 2012).

In contrast, ozone concentration seems to be consistent throughout the El Paso region. Although the ozone level has been declining when compared to other years, the 8-hour mean values

where exceeded at three CAMS in 2017. A combination of high surface temperatures, strong sunlight, and high concentrations of precursor gases could be the causes of high ozone concentrations (MacDonald et al., 2001).

1.8 Cardiorespiratory health issues in El Paso

Health issues of high prevalence in El Paso and its border region include asthma and chronic diseases. At El Paso Children's Hospital, asthma without any further complications is the most prevalent General Pediatrics diagnosis, and in the Pediatric Intensive Care Unit, bronchitis and asthma with complications is the most prevalent diagnosis, followed by bronchitis and asthma without complications and diabetes without complications (McGladrey, 2014)

Regarding cardiovascular risk factors, according to the Conduent Healthy Communities Institute, nearly 35% of El Paso County adults reported not having a cholesterol check in five or more years. Also, the prevalence rates of high blood pressure (25%) and type 2 diabetes (15.1%) are higher than state and U.S. rates (Healthy Paso Del Norte, 2017). Furthermore, a 2016 study determined the prevalence of MetS in Hispanic residents of low-income communities and found it affects more than 50% of their selected population (Aguilera, 2016).

1.9 Healthy People 2020

Healthy People 2020 outlines several objectives that are addressed in this research. Regarding air pollution, the third objective under the Environmental Health topic (EH-3) aims to reduce air toxic emissions to decrease the risk of adverse health effects caused by mobile, area, and major sources of airborne toxics.

In terms of physical activity (PA), PA-3 aims to increase the proportion of adolescents who meet current Federal physical activity guidelines for aerobic physical activity and for muscle-

strengthening activity and PA-4 aims to increase the proportion of the Nation's public and private schools that require daily physical education for all students

MetS is a risk factor for cardiovascular disease and under the Heart Disease and Stroke (HDS) topics there are several related objectives. HDS-1 is to increase the overall CVD health among the U.S. population and HSD-2 is to reduce coronary heart disease death. Some of the MetS risk factors are addressed as well by the HSD objectives. HSD-4 aims to increase the proportion of adults who have had their blood pressure measured within the preceding 2 years and can state whether their blood pressure was normal or high and HDS-6 aims to increase the proportion of adults who have had their blood cholesterol checked within the preceding 5 years.

Another topic outlined in Healthy People 2020 related to the outcome variables explored in this dissertation is Nutrition and Weight Status (NSW). NSW-8 aims to increase the proportion of adults who are at a healthy weight. NWS-9 aims to reduce the proportion of adults who have obesity. Lastly, under the topic of Social Determinants of Health, AHS-1 is to increase the proportion of persons with health insurance (Health & Services, 2013; HealthyPeople, 2020)

1.10 Ethical aspects

Awareness by researchers of bioethical concepts was crucial to deliver a better experience for our participants. We followed the National Institutes of Health (NIH) provided guidelines for the inclusion of women, children, and minorities in research studies. The data from human participants used in this dissertation came from studies that have been approved by an institutional review board (IRB). The presented research also followed the same steps to ensure we do not violate anyone's rights, ensure the relevance of the research proposed, and position the researchers as the appropriate team to conduct the study.

Participants' autonomy needs to be respected; therefore, all of our participants went through a consent (and assent when applicable) process in which they became aware of their risks and benefits and were assured they could decide whether to participate or not. In the relevant studies, following the principle of non-maleficence, we guaranteed no risk or minimal risk. Regarding beneficence, being part of the presented studies study might not have a direct effect on a participant but did allow us to understand a topic for the better of the community. Finally, following the principle of justice, we did our best ensure everyone had equal rights to be included as participants in each of the studies (Beauchamp & Childress, 2009).

1.11 Practice model

This dissertation evaluated the association and detrimental health effects of air pollution on health and implications regarding physical activity and cardiorespiratory diseases. Dissemination of the findings of this research will be brought first to the community through local news media and health networks which include the Joint Advisory Committee (JAC) that serves as the local community-based organization overseeing the process to achieve cleaner air for the Paso del Norte region. In addition, presenting this research will further enhance our existing relationships with other community advocates, program coordinators, and service providers to facilitate policy change within the El Paso community. Furthermore, results will be shared through regional and national conferences. Mitigation strategies to reduce the impact of air pollutants in the community will be disseminated separately and presented as a policy brief. A practice model is provided in Figure 1.4.

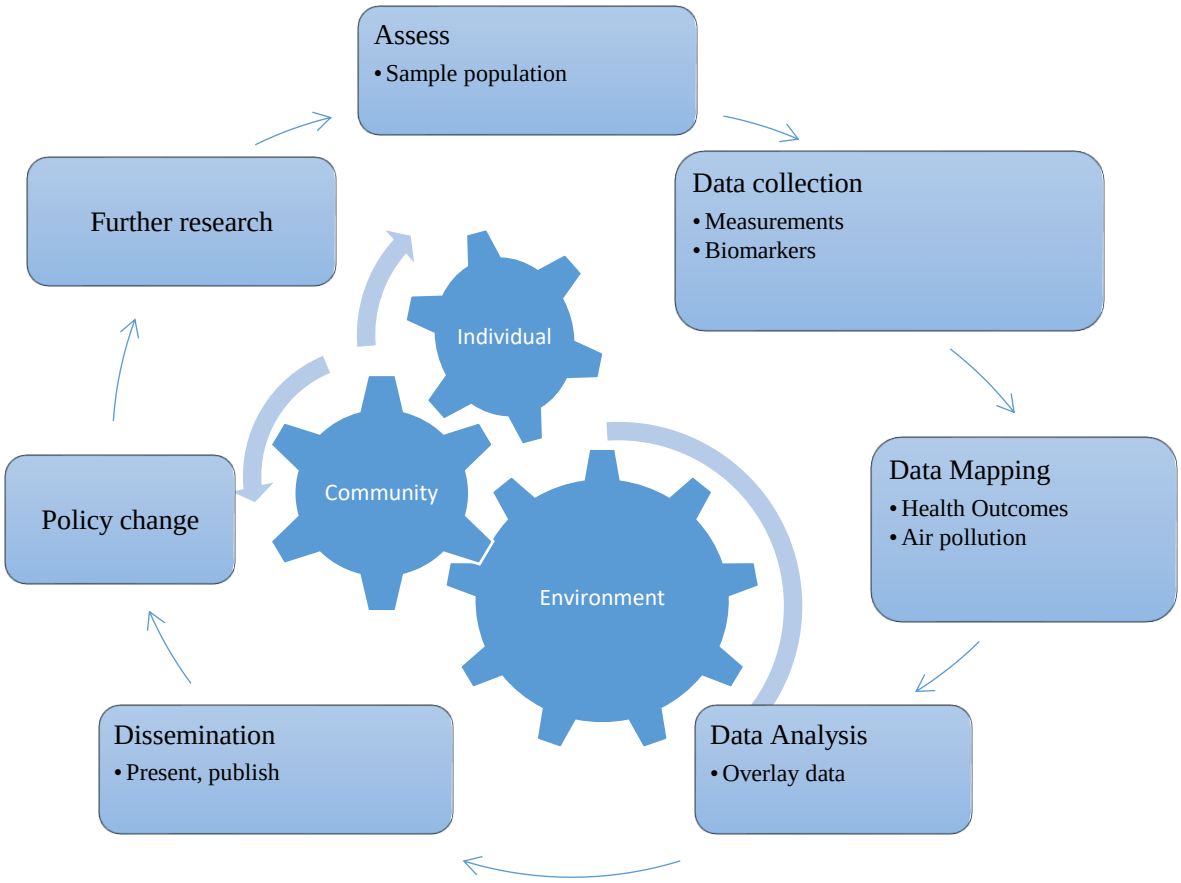


Figure 1.4: Practice model

CHAPTER 2 MODERATE TO VIGOROUS PHYSICAL ACTIVITY LEVELS NEGATIVELY CORRELATE WITH TRAFFIC RELATED AIR POLLUTANTS IN CHILDREN WITH ASTHMA ATTENDING A SCHOOL NEAR A FREEWAY

2.1 Introduction

In a polluted environment, people who engage in outdoor physical activity are likely to have increased health risk compared to those who have a more sedentary lifestyle, which could be counterproductive to the promotion of physical activity

2.1.1 EXPOSURE TO AIR POLLUTANTS AND PHYSICAL ACTIVITY

Physical activity is essential for overall health (Janssen & LeBlanc, 2010). Regular outdoor activities, like walking, jogging, or dancing, can lead to a significantly lower risk of cardiovascular disease and metabolic syndrome (M. Chen et al., 2013). However, outdoor physical activity also exposes people to air pollutants which might lead to adverse health problems such as cardiovascular (Le Tertre et al., 2002; Sharman et al., 2004) or respiratory diseases (Pope III, Ezzati, & Dockery, 2009; Shah et al., 2013).

During physical activity, a higher deposition of air pollutants in the lungs may occur due to increased respiratory intake (Giles & Koehle, 2014). In controlled studies, the exposure to air pollutants during exercise has led to a reduction in performance (Kenneth W. Rundell, Slee, Caviston, & Hollenbach, 2008) and inhalation of airborne particles during exercise has been associated with reduction in lung function (Cutrufello et al., 2012). Also, increased levels of air pollutants are associated with self-reported inactivity (Roberts et al., 2014; X.-J. Wen et al., 2009). For this reason, exposures to an environment with an increased level of air pollution might lead to adverse health effects due to airway exposure to airborne pollutants and lack of physical activity.

2.1.2 AIR POLLUTANTS IN THE SCHOOL ENVIRONMENT

Research suggests that spending time in an environment near heavy traffic is particularly harmful to children. Children attending elementary school spend about 6-8 hours per day in various school microenvironments. Outdoor activities are relatively common in elementary schools due to the lack of indoor playgrounds. In many countries, severe conditions of air pollution frequently require the cancellation of physical or sport activities in elementary schools, which may lead to an increase in sedentary behavior and contribute to the overweight and obesity epidemic (Giles & Koehle, 2014). This is particularly important for schools located near busy traffic intersections or freeways where children may be exposed to an even higher level of traffic pollution. Among the traffic-related air pollutants that children of roadside communities are commonly exposed are coarse particulate matter (PM_{10} or particles less than 10μm in aerodynamic diameter), fine particulate patter ($PM_{2.5}$ or particles less than 2.5μm in aerodynamic diameter), nitrogen dioxide (NO_2), and ozone (O_3).

2.1.3 PHYSICAL ACTIVITY IN THOSE WHO HAVE ASTHMA

People with asthma (or even those with mild asthma symptoms) may engage less in physical activity, avoid aerobic fitness, and reduce leisure-time energy expenditure due to concerns of triggering asthma symptoms (Garfinkel et al., 1992; Mälkiä & Impivaara, 1998). Given that asthma affects children at a young age when they are likely to establish their health habits, it is important to emphasize physical activity with asthma patients (Mancuso et al., 2006). National management guidelines for asthma state that the majority of patients can be controlled well enough to perform physical activity and that additional therapy options can be made available to them (Busse et al., 2007; Education et al., 1997). Therefore, it is in the best interest of those who have

asthma to achieve a balance between having a healthy amount of physical activity and controlling their respiratory symptoms.

However, the impact of air pollution on people with asthma often prevents them from achieving a physically active lifestyle. In controlled studies, among the groups exposed to higher concentrations of air pollutants, there was a higher risk of having an asthma attack (Sharman et al., 2004) and lung diseases (Giles & Koehle, 2014). Children with asthma living in low- income communities are likely to have increased clinical asthma symptoms when they are exposed to short-term increases in air pollutants (Wendt et al., 2014).

In summary, the importance of promoting physical activity for overall health benefits conflicts with the negative consequences of physical activity in environments with high levels of air pollutants. Although air quality is a concern in school environments, since studies have documented that air pollutants are inhaled into lungs during exercise and that people with asthma may reduce or avoid physical activity cardiovascular (Garfinkel et al., 1992; Le Tertre et al., 2002; Mälkiä & Impivaara, 1998; Pope III et al., 2009; Shah et al., 2013; Sharman et al., 2004), there are no studies that assess changes in air quality over time and how those changes correlate with objectively measured physical activity in children with asthma in a school setting. The findings of this study are expected to fill this gap and inform the implementation of policies and health recommendations for communities to reduce the adverse impact of air pollution on physical activity in school settings.

2.2 Methods

2.2.1 SETTING, POPULATION, AND SAMPLE

This study was conducted in El Paso, Texas from October to December 2017 at an elementary school located within 50 ft of a heavy traffic freeway. Air pollutants and concurrent

meteorological data were continuously monitored through the duration of the study. Physical activity was assessed weekly during school hours. The Institutional Review Board (IRB) of The University of Texas at El Paso approved the protocol for this study prior to participant recruitment and data collection.

Children with asthma were recruited by contacting the school nurse and disseminating flyers to each student. The participant's parent or legal guardian provided written consent and children provided assent. English and Spanish versions of consent and assent forms were available for the participants and their parents or guardians. The selection criteria for the study included children between 6 and 12 years with a physician diagnosis of asthma and no other lung disease, no major illness, and living in a non-smoking household. Twelve children satisfied the eligibility requirements and participated in the study.

At the start of the study, parents were asked to answer a baseline questionnaire that provided information on health status, current allergies, insurance status, medication usage, household characteristics, symptoms and activity limitation due to symptoms, emergency room visits, and hospital admissions. In addition, each Friday during the study, the participants answered questions about symptoms and medication use using the Asthma Control Questionnaire (ACQ) (Juniper, Gruffydd-Jones, Ward, & Svensson, 2010). English and Spanish versions were available.

Physical activity rates categorized by activity intensity rated as moderate to vigorous (MVPA), light, and sedentary were measured using an accelerometer (wGT3X-BT; ActiGraph). During the Friday data collection visits, the accelerometer was placed on the wrist of the participants and kept during school hours. Physical activity rates were calculated using the ActiLife (V.6.13.3) software using the children algorithm (Pamty Freedson, 2005). The software

allowed determination of a participant's percentage of time in either sedentary, light, or MVPA during a specific time window (9:00 AM to 2:00 PM).

Air pollutants were continuously measured throughout the study in an outdoor environment close to the school. Air monitors were placed in a fenced area immediately adjacent to Highway I-375 within the school premises. The analysis included measurements for PM_{10}, $PM_{2.5}$, NO_2, and O_3. $PM_{2.5}$ and PM_{10} mass concentrations were measured using GRIMM Technologies Aerosol Spectrometer 11-A. NO_2 measurements were obtained using 2B Technologies Model 405 NO_2/NO/NO_x. Ozone (O_3) was measured using 2B Technologies Model 202. Temperature and relative humidity were collected from the weather station located at El Paso International Airport. Air pollution data recorded at the Texas Commission on Environmental Quality (TCEQ) operated continuous ambient monitoring stations (CAMS) at Chamizal were used for comparison of site-specific $PM_{2.5}$, PM_{10}, and O_3 data. Another CAMS site at Ascarate Park was used to compare NO_2 (Figure 2.1). Hourly measurements were averaged to calculate values for 96, 72, 48, and 24-hours prior to the physical activity measurements.

Figure 2.1: Location of school and CAMS stations

2.2.2 DATA ANALYSIS

Descriptive statistics were calculated to assess air pollution metrics and physical activity (MVPA/light/sedentary) status. Correlation analyses using Spearman correlation were conducted to explore relationships between physical activity, and outdoor pollutant concentrations. Summary statistics of subject demographic information and air pollution metrics were calculated. Physical activity outcomes between the subject-specific factor groups (%time spent in sedentary, light, or moderate to vigorous physical activity) were compared using Kruskal-Wallis test.

Longitudinal associations between MVPA/sedentary physical activity measures and air pollution metrics were examined using generalized estimating equations (GEE) approach (Liang & Zeger, 1986). We assumed the subject-specific cluster and exchangeable correlation structure for the repeated measures of the physical activity data.

Separate models were run for each pollutant variable of interest (PM concentrations, NO_2, or O_3) with various exposure periods (previous 24-hr, 48-hr, 72-hr, or 96-hr means). Meteorological variables such as temperature and relative humidity, were averaged over the same periods of time. We expanded our analysis to include up to 96-hr averages of pollution prior to the physical measurements since an effect of air pollutants on physical activity might require more exposure time to manifest a change in time spent in physical activity

96-hr means of temperature and relative humidity showed strongest associations with response outcome, and as a priori fixed covariates in all models, we controlled for the 96-hr temperature and relative humidity. Effect estimates for each measurement are presented as the percent change in rate of physical activity per increase in pollutant concentrations. A $p < 0.05$ was considered statistically significant. All statistical analyses were performed using R version 3.2.2.

2.3 Results

We considered various exposure windows for the outdoor pollutants. Hourly measurements were aggregated into 24-hr, 48-hr, 72-hr, and 96-hr means. Hourly concentrations measured at the nearest CAMS location were also averaged over the same periods for comparisons. Descriptive statistics for air pollutant concentrations are listed in Table 2.1. Table 2.1 also compares the site-specific and CAMS ambient concentrations, averaged for the 96-hr exposure time. The mean concentrations at CAMS monitoring site were lower than the school measurements, with a tendency for larger variations than those at the school.

Table 2.1: Summary statistics for school and ambient pollutant metrics: mean, standard deviation (SD), median, interquartile range (IQR), minimum (min), and maximum (max)

Pollutant	**24-hr**	**48-hr**	**72-hr**	**96-hr**	**96-hr (CAMS)**
$PM_{2.5}$	$\mu g/m^3$	$\mu g/m^3$	$\mu g/m^3$	$\mu g/m^3$	$\mu g/m^3$
Mean	12.52	11.73	11.48	12.16	10.17
SD	3.71	2.40	1.88	2.80	5.25
Median	13.15	11.13	11.35	11.27	9.75
IQR	4.91	4.14	3.12	4.07	5.22
Max	18.86	15.65	14.33	17.58	18.69
Min	6.33	8.98	8.60	8.61	3.40
PM_{10}	$\mu g/m^3$	$\mu g/m^3$	$\mu g/m^3$	$\mu g/m^3$	$\mu g/m^3$
Mean	45.30	43.05	42.55	44.94	36.89
SD	17.36	12.47	8.70	9.13	12.43
Median	40.30	38.47	40.32	45.84	38.67
IQR	24.57	19.06	11.93	9.56	16.84
Max	74.14	62.31	56.99	60.10	51.61
Min	24.49	25.87	31.36	28.54	13.84
NO_2	ppb	ppb	ppb	ppb	ppb
Mean	17.63	18.20	18.40	18.94	17.90
SD	6.06	3.25	3.06	3.72	5.11
Median	19.22	18.59	18.47	19.04	16.33
IQR	7.81	4.76	2.76	4.96	5.20
Max	26.17	22.16	22.70	23.64	27.13
Min	7.21	12.20	12.17	11.62	13.02
O_3	ppb	ppb	ppb	ppb	ppb
Mean	21.41	20.37	21.75	20.35	19.85
SD	10.51	6.66	7.25	5.47	5.08
Median	19.60	18.94	19.37	18.29	18.85
IQR	18.09	11.69	12.32	8.57	7.51
Max	38.90	31.13	34.52	29.71	28.43
Min	9.16	12.52	13.86	15.59	14.81

Table 2.2 includes summary statistics of participants' age, anthropometric measures, and physical activity rates by level (MVPA, light, and sedentary). The mean age for the participants was 8.3 years (SD=1.5) and mean body mass index (BMI) was 17.9 (SD=5.0). The mean BMI-for-age percentile was 49.8±41.2. The mean (±SD) physical activity levels for MVPA, light, and sedentary activity were 63.4% (±8.2%), 10.1% (±1.7%), and 26.5% (±0.079%), respectively. A

pairwise *t*-test indicated the three activity levels were significantly different from each other (all *p*-values <0.001, with Bonferroni adjustment).

Table 2.2: Summary of subject demographics and physical activity information

Variable	**mean ± SD**	**range**
Age (yrs)	8.3 ± 1.5	(6-10)
Height (in)	54.3 ± 4.4	(46.3-70.0)
Weight (lb)	76.3 ± 27.3	(45.8-134)
BMI (kg/m^2)	17.9 ± 5.0	(12.3-27.8)
BMI (%)	49.8 ± 41.2	(0-99.4)
Physical Activity (%, N=102)		
MVPA	63.4 ± 8.2	(30.4-77.7)
Light	10.1 ± 1.7	(7.1-14.4)
Sedentary	26.5 ± 7.9	(13.7-61.7)

The subject-specific factors including medication information are characterized in Table 2.3. Rates of MVPA and sedentary activities by their factor levels were compared using Kruskal-Wallis test to examine whether the mean rates between factor levels were statistically different. The test results showed significantly different rates for some factors (gender, BMI category, father with asthma status, siblings with asthma, having eczema, health insurance, smoking status) and medications (*Leukotrieneblockers* [LB], *Long-acting bronchodilators and inhaled corticosteroids* [LABAIC], and *Nasal corticosteroids* [NC]) with both MVPA and sedentary activities (see bold *p* in Table 2.3). For example, types of insurance, i.e., Medicaid vs. private, was a significant factor ($p = 0.003$) to have different rates in the MVPA, participants with Medicaid spent more time in MVPA (0.665) than those with private insurance (0.612). Conversely, participants with Medicaid spent less time in sedentary activities (0.239) than those with private insurance (0.279, $p = 0.039$).

Table 2.3: Summary Statistics of subject specific factors and physical activity rates per factor level.

Subject-specific Factor	**Frequency,%**		**Physical activity**			
	(N=12)		MVPA	*p**	Sedentary	*p**
Sex						
Male	7	58%	65.8%	**0.001**	24.2%	**0.001**
Female	5	42%	60.0%		29.2%	
BMI category						
Underweight & Normal	8	67%	61.9%	**0.010**	28.4%	**< 0.001**
Overweight & Obesity	4	33%	66.5%		22.6%	
Mother with Asthma	5	42%	63.2%	0.895	26.1%	0.503
No	7	58%	63.6%		26.7%	
Father with Asthma	3	25%	60.9%	**0.041**	28.8%	**0.032**
No	9	75%	64.3%		25.7%	
Mother with Hay Fever	8	67%	63.4%	0.944	26.3%	0.595
No	4	33%	63.5%		26.8%	
Father with Hay Fever	8	67%	62.7%	0.305	26.9%	0.511
No	4	33%	64.8%		25.6%	
Siblings with Asthma	6	50%	61.2%	**0.005**	28.8%	**0.001**
No	6	50%	65.6%		24.1%	
Siblings with Hay Fever	8	67%	63.0%	0.602	27.2%	0.169
No	4	33%	64.2%		25.1%	
Having Eczema	3	25%	66.8%	**0.012**	23.2%	**0.011**
No	9	75%	62.2%		27.7%	
Allergic Phenotype (Aeroallergens)	8	67%	63.1%	0.597	26.7%	0.794
No	4	33%	64.1%		26.0%	
Allergic Phenotype (Food)	3	25%	61.8%	0.143	27.4%	0.366
No	9	75%	64.1%		26.1%	
Caretaker Education						
Less than or Equal to High School	6	50%	63.8%	0.997	26.3%	0.771
Greater than High School	6	50%	63.1%		26.6%	
Health Insurance Coverage (N=11)						
Medicaid	6	55%	66.5%	**0.003**	23.9%	**0.039**
Private	5	45%	61.2%		27.9%	
Smoking (outside of household)	2	17%	59.9%	**0.013**	29.9%	**0.010**
No	10	83%	64.2%		25.7%	
Cooking Fuel						
Electric	1	8%	68.7%	**0.035**	22.7%	0.127
Gas	11	92%	62.9%		26.8%	
*Leukotrieneblockers (LB)***	7	58%	66.4%	**< 0.001**	23.7%	**< 0.001**
No	5	42%	59.4%		30.3%	
Short-acting bronchodilators (SABA)	7	58%	62.8%	0.155	27.3%	0.065

Subject-specific Factor	Frequency,%		Physical activity			
No	5	42%	64.4%		25.2%	
Inhaled corticosteroids (IC)	6	50%	63.2%	0.894	26.1%	0.493
No	6	50%	63.6%		26.8%	
Long-acting bronchodilators and inhaled corticosteroids (LABAIC)	2	17%	68.1%	**0.012**	22.0%	**0.013**
No	10	83%	62.6%		27.2%	
Nasal corticosteroids (NC)	4	33%	66.8%	**0.003**	23.4%	**0.007**
No	8	67%	61.7%		28.0%	
Systemic corticosteroids (SC)	2	17%	64.6%	0.641	25.3%	0.791
No	10	83%	63.2%		26.7%	

*p for mean difference in physical activity between factor levels using Kruskal-Wallis test.
** All medications are expressed in italic.

2.3.1 MODELS PREDICTING PHYSICAL ACTIVITY DATA

Table 2.4 presents effect estimates using GEE models, 95% confidence intervals, and corresponding *p*. We scaled the effects to interquartile range (IQR) increases in pollutant metrics to compare the magnitude of effect across different scales of the pollutant concentrations. The 96-hr school pollutant concentrations ($PM_{2.5}$, $PM_{10,}$ and NO_2) were negatively associated with moderate to vigorous physical activity (p<0.001 for PM; p =0.036 for NO_2), whereas they were positively associated with sedentary activity (p<0.001 for PM; p=0.019 for NO_2). The relationship between 96-hr O_3 and moderate to vigorous activity was not significant (p=0.661). However, the 72-hr maximum ozone data were associated with a decreased rate in moderate to vigorous activity (p=0.001).

The 96-hr mean ambient PM and NO_2 concentrations at the Ascarate CAMS were significantly associated with physical activity levels, showing consistent patterns of association with 96-hr school concentrations. The largest percent time spent in MVPA per school pollutant increase in IQR was observed in the association between 96-hr $PM_{2.5}$; 3.45% decrease in MVPA (95% CI: -5%, -1.9%). We have a similar amount of percent change in sedentary activity [3.43% increase (95% CI: 1.78%, 5.09%)] as the IQR in $PM_{2.5}$ increases.

Table 2.4: Overall associations between moderate to vigorous (MVPA) and sedentary physical activity and pollutant metrics.

Pollutant		IQR	MVPA				Sedentary			
			Change in rate per IQR	95% CI lower	95% CI upper	*p*	Change in rate per IQR	95% CI lower	95% CI upper	*p*
$PM_{2.5}$	24-hr	4.91	0.47%	-0.54%	1.48%	0.365	-0.96%	-1.92%	0.01%	0.051
	48-hr	4.13	0.80%	-0.37%	1.96%	0.180	-1.53%	-2.75%	-0.31%	**0.014**
	72-hr	3.11	-1.71%	-2.95%	-0.46%	**0.007**	1.43%	0.24%	2.61%	**0.018**
	96-hr	4.07	-3.45%	-5.00%	-1.90%	< **0.001**	3.43%	1.78%	5.09%	< **0.001**
	96-hr CAMS	5.22	-3.86%	-6.12%	-1.59%	**0.001**	4.04%	1.71%	6.37%	**0.001**
PM_{10}	24-hr	24.57	-0.43%	-1.50%	0.64%	0.427	-0.06%	-0.99%	0.87%	0.902
	48-hr	19.05	-0.58%	-1.66%	0.50%	0.293	-0.17%	-1.18%	0.83%	0.735
	72-hr	11.93	-1.32%	-2.24%	-0.39%	**0.005**	1.00%	0.09%	1.91%	**0.031**
	96-hr	9.56	-1.59%	-2.37%	-0.81%	< **0.001**	1.51%	0.69%	2.34%	< **0.001**
	96-hr CAMS	16.84	-2.87%	-4.65%	-1.08%	**0.002**	3.07%	1.19%	4.95%	**0.001**
NO_2	24-hr	7.81	-0.45%	-1.71%	0.82%	0.489	0.43%	-0.62%	1.47%	0.424
	48-hr	4.76	-0.28%	-1.41%	0.85%	0.626	0.29%	-0.72%	1.30%	0.574
	72-hr	2.76	-0.60%	-1.30%	0.11%	0.098	0.66%	-0.06%	1.38%	0.075
	96-hr	4.96	-1.35%	-2.62%	-0.09%	**0.036**	1.52%	0.25%	2.79%	**0.019**
	96-hr CAMS	5.19	-0.78%	-1.53%	-0.04%	**0.040**	0.63%	-0.12%	1.38%	0.099
O_3	72-hr $MaxO_3$8hr	9.94	-3.99%	-6.35%	-1.63%	**0.001**	4.62%	2.15%	7.08%	< **0.001**
	24-hr	18.10	-0.25%	-3.51%	3.01%	0.881	1.16%	-2.10%	4.43%	0.486
	48-hr	11.69	-1.31%	-4.01%	1.40%	0.344	2.07%	-0.85%	4.98%	0.164
	72-hr	12.32	-0.66%	-2.33%	1.01%	0.437	1.41%	-0.37%	3.19%	0.120
	96-hr	8.57	-0.33%	-1.81%	1.15%	0.661	0.49%	-1.05%	2.04%	0.530
	96-hr CAMS	7.50	-0.04%	-1.51%	1.43%	0.955	0.24%	-1.34%	1.82%	0.766

2.4 Discussion

2.4.1 PRINCIPAL FINDINGS

Measurements at the school showed that the mean 96-hr average concentration for each of the pollutants was higher than what was reported by the reference CAMS stations (Table 2.1). This would indicate that a higher exposure to air pollutants took place at this site compared to the "central-site", which is typically reported at a publicly operated CAMS location for the region. The proximity to a major freeway could potentially lead to adverse health outcomes for children attending the elementary school and participating in outdoor activities. In addition, as observed

from the pollutant concentrations, we can infer that the larger time windows considered (72/96-hr) provide a better representation of the current air pollutant exposure for physical activity at the study site.

We found negative correlations between the 96-hr means of $PM_{2.5}$, PM_{10}, and NO_2 at the schools and the amount of time spent in MVPA during school hours. In contrast, sedentary activity was positively correlated with air pollutant concentrations. We could not find previous studies that directly observed the effects of pollutants on physical activity. However, some studies have demonstrated adverse health effects related to physical activity. For example, in healthy males, inhalation of particulate matter during exercise leads to adverse respiratory health related to reduced lung function (Kenneth W. Rundell et al., 2008). Furthermore, a study conducted in California noted a positive association between wheezing and increase levels of NO_2 pollutants (Peters et al., 1999).

In addition, meteorological parameters (humidity and temperature) were also controlled for the approach we took in this study. We initially found positive correlations with O_3 and physical activity, possibly because high O_3 days imply more sunshine (less cloud cover) and increased outdoor temperatures. Consequently, the outdoor environment is more inviting for outdoor activities during winter months. Once the statistical approach considered meteorology factors, associations with O_3 were in the same direction as the other pollutants but were not significant. However, the use of maximum 8-hr mean values of O_3 did yield a significant association. Some studies that have looked at O_3 exposure showed that a high daytime O_3 concentration was consistent with an increased likelihood of new-onset of asthma or exacerbation of undiagnosed asthma in physically active children (McConnell et al., 2002). This could mean that O_3 levels affect differently, or that the effects might be more significant if the values reach a certain threshold.

2.4.2 COMPARISON WITH OTHER STUDIES

We noticed differences in physical activity rates between sexes which are consistent with other published values (Trojano, 2008) but not with BMI. In this study, children with overweight

and obesity were more physically active than underweight and children with normal weight. We found correlations between health insurance and physical activity rates which could be related to the asthma severity and more frequent visits in the Medicare setting when compared to those in the private setting. A study among children with asthma aged 3 to 17 showed that those enrolled in Medicaid were more likely to have a preventive care visit during the last year, and about half of them did receive a clinician's advice about physical activity (Perry & Kenney, 2007).

Having a father or a sibling with asthma (but not a mother) was significantly correlated with more time spent in sedentary behavior and less time spent in MVPA. This is somewhat consistent with a study in Canada which found that having a parent with asthma increased the odds of asthma and wheezing outcomes (Barry et al., 2014). This same study found increased odds of symptom severity if a mother was a previous smoker but did not report any data on having either a father or a sibling with asthma.

The treatment options for children with asthma depend on the severity of their condition (Masoli, Fabian, Holt, Beasley, & Program, 2004). Those with persistent asthma are recommended to take inhaled corticosteroids (ICS) in order to control airway inflammation. The addition of long-acting β_2-agonist (LABA) for patients is an option for those who remain symptomatic with ICS treatment only (Partridge, van der Molen, Myrseth, & Busse, 2006). Higher levels of MVPA in children using some medications could be a result of increased control over asthma symptoms. Furthermore, in a study in healthy adults, pre-treatment with an LB (*Montelukast*) before exercise attenuated the effects of PM inhalation in endothelial dysfunction (a cardiovascular health marker) (Kenneth W Rundell, Steigerwald, & Fisk, 2010).

Regarding physical activity, a study looking into perceptions of health benefit vs. detriment of exercise, researchers found participants with a more severe asthma condition were more likely

to believe exercise was not good for their asthma (Mancuso et al., 2006). In another study that included 27 adults with mild to moderate asthma, exercise participation was rated only 1.6 in a 4-point physical activity scale (Garfinkel et al., 1992). Among children with asthma, the severity of the disease and parental beliefs about physical activity and asthma predicted the activity level, although this was based on self-reported data (Lang, Butz, Duggan, & Serwint, 2004).

2.4.3 STRENGTHS AND LIMITATIONS

Measuring physical activity in children is difficult. Children tend to have short bursts of activities that are more difficult to measure when compared to adults (van Gent et al., 2007). The gold standard for assessing physical activity is the double-labeled water method (Westerterp, 2009). However, this method does not provide data about activity patterns and is expensive and more logistically challenging. Accelerometers record movement of the specific part of the body to which they are attached and thus differences in types of physical activities are mostly accurate (van Gent et al., 2007) and correlate reasonably with the gold standard (Plasqui & Westerterp, 2007).

The sample size was small given the few number of students that have an asthma diagnosis attending the school. However, during the ten weeks of the study, the children followed the study protocol, and we managed to obtain a sizeable number of repeated measurements (N=102). Also, GEE models allowed us to account for individual factors which further validates the longitudinal associations with the mentioned traffic-related air pollutants.

Although this study was longitudinal (repeated measures within individuals over time), there might be latent variables that affect children with asthma; therefore, cause and effect cannot be inferred from the results. Further research is recommended regarding the effect of air pollution

and the physical activity of children with asthma. In future work, expanding this framework to include children without asthma (control group) could strengthen findings.

2.5 Conclusion

To our knowledge, this is the first study to characterize the effects of traffic-related air pollutants in children with asthma using objective measures of physical activity. Our findings suggest that school-based monitoring of air pollutants is an indicator of the health risk of children's exposures and the impact on their physical activity. A higher concentration of traffic-related pollutants over 72 and 96-hour exposures seems to have a greater impact on time spent on physical activity in children with asthma.

During physical activity, changes in the frequency of breathing patterns as well as a switch to predominantly oral respiration and bypass of nasal filtration could exacerbate the effects of air pollutants. Assuming the adverse health effects are related to the amount of pollutants inhaled, in children with asthma this might indicate a decrease in time spent in MVPA with a subsequent increase in sedentary behavior in an outdoor environment.

This research work will also aid in the formulation of healthy living recommendations in this border region such as placement of natural barriers (shade trees, shrubs, natural vegetation, green roofs) at the school to mitigate the effects of air pollutants.

CHAPTER 3 SHORT TERM EFFECTS OF TRAFFIC RELATED AIR POLLUTION ON CARDIORESPIRATORY OUTCOMES AMONG LOW INCOME RESIDENTS FROM EL PASO

3.1 Introduction

3.1.1 EFFECTS OF AIR POLLUTANTS ON CARDIORESPIRATORY HEALTH

People living in areas with higher exposure to air pollution, compared with those in less polluted areas, have higher mortality rates and stronger associations with cardiorespiratory disease (Dockery et al., 1993; Pope et al., 1995). Also, those living in areas with high air pollution have increased likelihood of negative health outcomes like heart and lung disease, irregular heartbeat, aggravated asthma, and decreased lung function (Atkinson, Fuller, Anderson, Harrison, & Armstrong, 2010b; Cadelis, Tourres, & Molinie, 2014b; Andrew W Correia et al., 2013). In recent decades, many cardiorespiratory biomarkers have been identified and studied in relation to air pollution exposure (Rom, Boushey, & Caplan, 2013).

Cardiorespiratory biomarkers can be considered valuable indices of a change in disease risk of air pollution exposure, even if not all of them are in the causal pathway for development of a disease (Thurston et al., 2017). For example, exhaled Nitric Oxide (eNO) is a biomarker of airway inflammation, which is an important determinant of respiratory outcomes and disease (Trachsel et al., 2008). On the other hand, lung function measured by spirometry offers respiratory markers that are affected by exposure to air pollutants in healthy adults and those with a preexisting lung disease (Paulin & Hansel, 2016). Both airway inflammation and decreased lung function have also been linked to the effects of traffic-related pollutants in children (Barraza-Villarreal et al., 2008; Holguin et al., 2007).

There is also evidence of relationships of air pollutants and negative cardiovascular outcomes. A couple of studies showed that yearly mean concentrations of particulate matter have been associated with higher hospitalization risks, congestive heart failure, and recurrent heart attack among patients with previous myocardial infarction (Zanobetti & Schwartz, 2005, 2007). Additional studies have looked at the effects of traffic-related air pollutants together with components related to metabolic syndrome, a predictor of cardiovascular disease, which includes waist circumference, blood pressure, triglycerides, HDL-cholesterol, fasting glucose LDL-cholesterol, and HbA1c (Clementi et al., 2019). Furthermore, a recent meta-analysis suggested that short-term exposure to some air pollutants such as particulate matter, NO_2, and O_3 may increase the risk of hypertension (Cai et al., 2016).

3.1.2 SHORT TERM EXPOSURE ASSESSMENTS

Exposures to traffic-related pollutants include particulate matter ($PM_{2.5}$ and PM_{10}) nitrogen dioxide (NO_2) and ozone (O_3) are considered a major preventable cause of respiratory disease (Laumbach & Kipen, 2012). Research about the short-term effects of exposure to the mentioned air pollutants has linked them with cardio-respiratory mortality as well (Rückerl, Schneider, Breitner, Cyrys, & Peters, 2011). Common study designs have used time-series or cross-sectional analyses to report associations between elevated air pollutant concentrations over short periods of time (one or several days) and increased cardiovascular mortality and morbidity (Panis et al., 2017; Pope III & Dockery, 2006). However, the precise window of exposure for some biomarkers is not clearly defined and differs by study.

Chuang and collaborators (2010) applied mixed models to examine the associations between air pollutants and metabolic markers (systolic blood pressure [SBP], diastolic blood pressure [DBP], HDL-cholesterol [HDL], LDL-cholesterol [LDL] , fasting blood glucose [FBG],

and HbA1c). The exposure variables included levels of PM, NO_2, and O_3 on the same day (24-hr mean) and 48 to 144-hr means before the day of the health measurements (Chuang, Yan, & Cheng, 2010). In 2017, Bell and colleagues estimated exposure to ambient $PM_{2.5}$ based on residential addresses using short-term averaging periods on the day of blood draw, the day before, and a moving mean of the previous 5 days with HDL-cholesterol (Bell et al., 2017). The MESA study, a large epidemiological research, assigned a daily exposure measure from the continuous air monitoring station (CAMS) nearest to the participant's residence with available data for a given day and constructed five exposure measures: PM2 the day before measurement, and mean concentrations over the two, seven, 30, and 60 days prior to measurement using metabolic syndrome as a modifying factor (Park et al., 2010).

Statistical models that associate air pollution exposure with cardiorespiratory outcomes also vary according to the study. A study among patients with type 2 diabetes in China considered spline and multiple linear regressions between short-term exposure to PM_{10}, SO_2, and NO_2 with total cholesterol (TC), triglycerides (TG), LDL-cholesterol, and high-HDL-cholesterol (Wang et al., 2018). A study among Mexican Americans in Southern California used short-term exposure considering up to 58 days of cumulative daily means of $PM_{2.5}$ to find associations with lower insulin sensitivity, HDL-to-LDL ratio, and higher fasting glucose and insulin, total cholesterol, and LDL-cholesterol using log transformations (Z. Chen et al., 2016).

This study aimed to determine the short-term effects on cardiorespiratory outcomes using markers for airway inflammation, lung function, and metabolic syndrome (MetS) from a large epidemiological study and considered 24 to 96-hr mean concentrations of traffic-related pollutants before the health assessments using air pollutant data retrieved from CAMS data near a participant's residential address.

3.2 Methods

3.2.1 SETTING, POPULATION, AND SAMPLE

The research conducted a secondary data analysis using health outcome measurements collected from an epidemiological study entitled "Evidence-based Screening for Obesity, Cardiorespiratory Disease, and Environmental Exposures in Low-income El Paso Households," funded by the City of El Paso's Department of Public Health, which included low-income participants from El Paso, TX measured between September 2014 and March 2019. Participants included in the epidemiological study were residents living within El Paso county recruited from low-income communities. The purpose of the epidemiological study was to obtain biometric and biomarker data from participants and it was approved by the Institutional Review Board (IRB) under the project numbers 590300-4 and 1249235-3. The secondary data analysis in the present study was further approved by the IRB under project number 1611345-1.

3.2.2 MEASURES

The methodology of the epidemiological study is described elsewhere (Aguilera, 2016), but briefly it includes measures for height and weight (to calculate BMI), waist circumference, blood pressure, a lipid profile (TG, TC, HDL, LDL), and fasting glucose. These measures were used to determine the rate of metabolic syndrome among the participants. Also, participants were measured for airway inflammation using a NiOX device (Aerocrine) to determine exhaled nitric oxide (eNO) and lung function was assessed by spirometry (Carefusion Micro Direct MicroLoop Spirometer) which measures forced vital capacity (FVC), forced expiratory volume in one second (FEV_1), and peak expiratory flow (PEF), as well as the %predicted value and best measure for each lung function marker.

Traffic-related air pollutant data for PM_{10}, $PM_{2.5}$, NO_2, and O_3 were extracted using publicly

available datasets from CAMS maintained by the Texas Commission on Environmental Quality (TCEQ). Each participant was assigned to the most representative CAMS station based on their residential address (Figure 3.1), if a participant lived in an area near a CAMS station with no data it was excluded from the analysis. Short term exposures refer to the mean pollutant concentrations averaged over the 24-, 48-, 72-, and 96-hour periods prior to the date of examination for each participant.

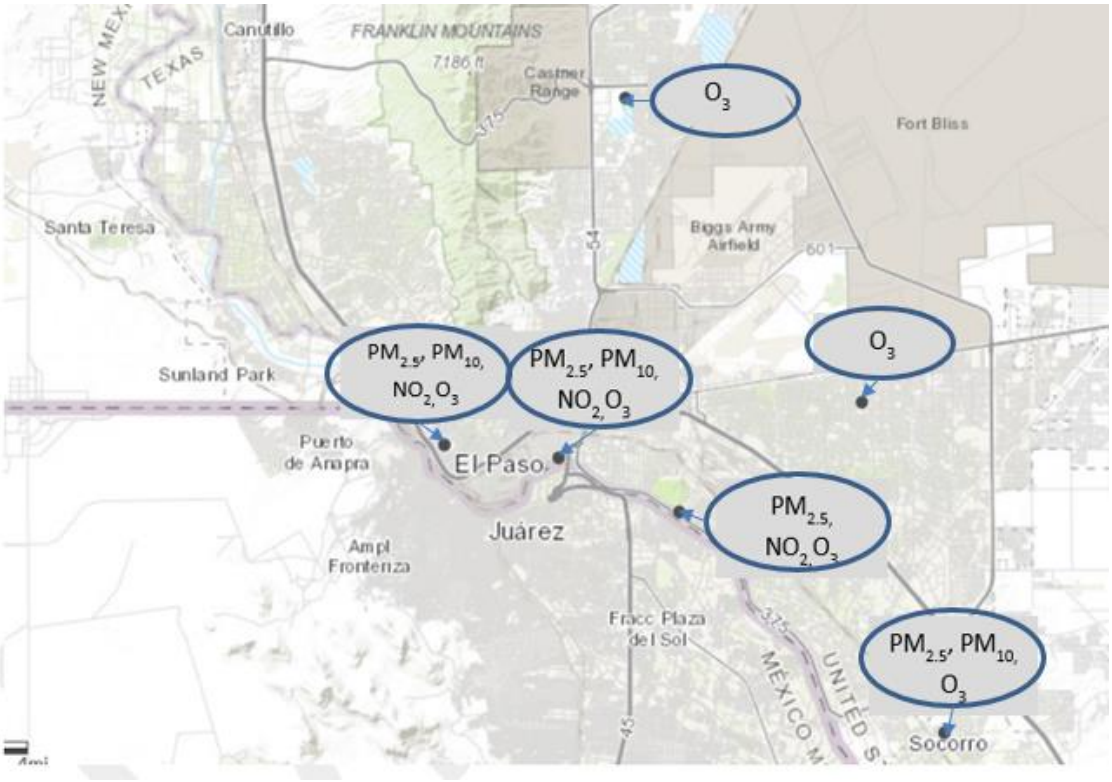


Figure 3.1: Location of CAMS stations in El Paso, TX for selected air pollutants

3.2.3 DATA ANALYSIS

The continuous variables in this study include respiratory markers eNO, FVC, FEV_1, PEF, %predicted FVC, %predicted FEV_1, %predicted PEF; cardiovascular risk markers waist circumference, SBP, DBP, TG, HDL, and FBG as well as pulse pressure (PBP), total cholesterol (TC), LDL, and TC/HDL. For further statistical analysis, waist circumference, SBP, DBP, TG,

HDL, and FBG were coded as binary variables (high or low) to determine whether a participant has metabolic syndrome (MetS) following the current diagnostic criteria (having three out five risk factors) (Expert Panel on Detection & Treatment of High Blood Cholesterol in, 2001).

Initially, summary statistics of participant demographic information and characteristics were calculated. Correlation analyses using Spearmen correlation were conducted to explore relationships between outcome variables and outdoor pollutant concentrations. The associations between pollutant metrics and various health outcomes were analyzed using linear regression. Before the correlation and regression analyses, Box-Cox transformation was applied to the variables TG, TC/HDL, and FBG to account for the skewness in the distribution, and different power exponents were selected to transform the data; we also used the log-transformation for the eNO and %predicted FVC and the exponent of -0.1 for the %predicted FEV_1 values. The square root transformation was applied to the %predicted PEF to improve the distribution of the right-skewed PEF data.

Logistic regression analyses were used to examine the relationship between categorical variables for the specified outcome (presence or absence of Mets and each risk factor) and concentration levels of pollutant variables. Regression models were conducted separately for each pollutant of interest. The level of statistical significance was set at $p < 0.05$ for all tests. We used the statistical software R (version 3.6.2) to perform the statistical analyses.

3.3 RESULTS

3.3.1 DEMOGRAPHICS

Summary statistics of subject demographic information and health characteristics are shown in Table 3.1. Subjects (N=662) participated in the study during the period from September 2014 to May 2015. Most of the participants were female (84.4%) and Hispanic (98.2%), and subjects had a mean age of 47.8 (±13.8) years with a range of 6-89 years old. The BMI was a mean of 30.6 (±6.6) kg/m^2, which ranges from 12.66 to 67.65 kg/m^2, and 81.1% of participants had overweight (35.2%) or obesity (45.9%), whereas 100 participants (15.1%) had a healthy BMI.

Table 3.1: Descriptive of the demographic information for subjects (N=662).

Variable		Frequency	%*
Sex	Female	559	84.4
	Male	103	15.6
Education	Middle School	162	24.5
	Elementary School	148	22.4
	High School, no diploma	130	19.6
	High School graduate	86	13.0
	Some college, not completed	54	8.2
	Associate degree	26	3.9
	Bachelor's degree	23	3.5
	Never attended or Kindergarten only	14	2.1
	Masters, Doctoral, or Professional degree	2	0.3
Language	Spanish	506	76.4
	Both	126	19.0
	English	21	3.2
Employed	Homemaker	211	31.9
	Employed-Part time	146	22.1
	Employed-Full time	81	12.2
	Not employed for more than 1 year	56	8.5
	Not employed for less than 1 year	51	7.7
	Self-Employed	32	4.8
	Student	28	4.2
	Retired	24	3.6
	Unable to work	18	2.7

Variable		Frequency	%*
Income	$0 - $19,999	559	84.4
	$20,000 - $29,999	50	7.6
	$30,000 - $39,999	9	1.4
	$40,000 - $49,999	3	0.5
	$50,000 - $69,999	2	0.3
	$70,000 - $99,999	2	0.3
Marital Status	Married	232	35.0
	Never Married	136	20.5
	Separated	105	15.9
	Divorced	88	13.3
	Widowed	50	7.6
	A member of an unmarried couple	28	4.2
	Civil Union	14	2.1
Ethnicity	Hispanic	650	98.2
	Non-Hispanic	8	1.2
Race	White	600	90.6
	Black or African American	10	1.5
	American Indian or Alaska Native	3	0.5
	Asian	2	0.3
Health	Good	253	38.2
	Fair	236	35.6
	Poor	79	11.9
	Very Good	50	7.6
	Excellent	21	3.2
Obesity	Obesity	304	45.9
	Overweight	233	35.2
	Healthy	100	15.1

*Distribution might not add to 100% due to participants not answering all the questions

Table 3.2: Summary statistics of participant's characteristics

Variable	Min	Q1	Median	Mean	Q3	Max	SD	IQR
Age (years)	6	40	49	47.8	57	89	13.8	17
WEIGHT (kg)	18.1	66.0	76.0	77.4	87.0	164.0	17.9	21.0
HEIGHT (cm)	115.0	154.0	158.0	159.0	163.5	185.0	8.2	9.5
BMI (kg/m^2)	12.7	26.5	29.7	30.6	34.6	67.7	6.6	8.0

3.3.2 AIR POLLUTION MEASUREMENTS

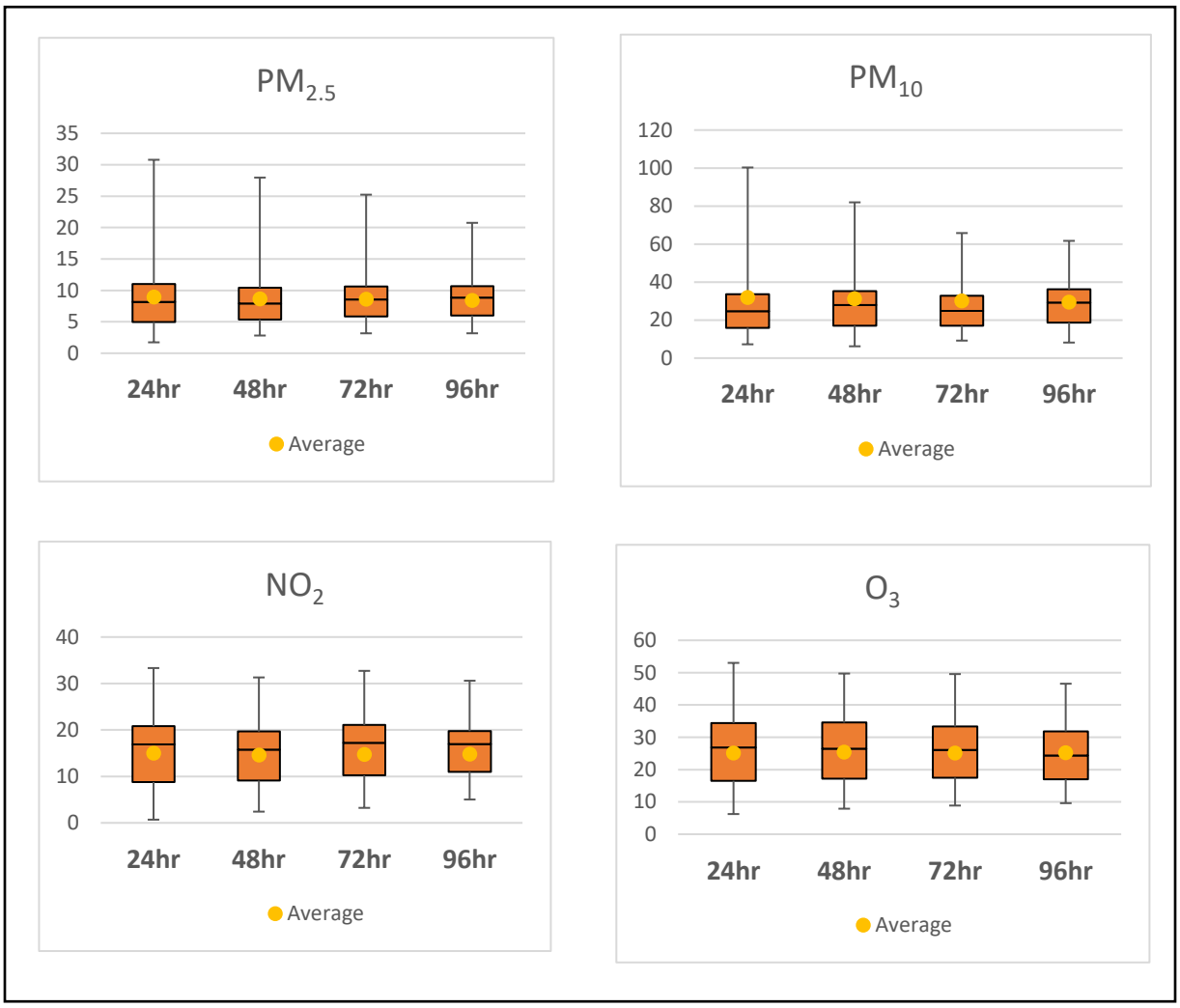
Hourly concentrations at the nearest CAMS locations nearest to subject's residential address (Table 3.3) were averaged over 24-hr, 48-hr, 72-hr, and 96-hr exposure window periods for comparisons. The Chamizal station had the highest frequency as the nearest CAMS station relative to the participant's residential address. Not all pollutants are measured at all CAMS locations. The means were aggregated to represent prior pollutant exposure until 10 AM of the day when health outcomes are measured. Table 3.4 and Figure 3.2 summarize the descriptive statistics for the pollutant measurements for the study participants.

Table 3.3: Spatial distribution of subjects to the nearest CAMS stations

Nearest CAMS		Frequency	%
$PM_{2.5}$	Chamizal	298	45.0
	Ascarate	136	20.5
	UTEP	121	18.3
	Socorro	107	16.2
PM_{10}	Chamizal	391	59.1
	Socorro	147	22.2
	UTEP	124	18.7
NO_2	Chamizal	296	44.7
	Ascarate	242	36.6
	UTEP	124	18.7
O_3	Chamizal	194	29.3
	UTEP	115	17.4
	Skyline	111	16.8
	Ascarate	87	13.1
	Socorro	82	12.4
	Ivanhoe	73	11.0

Table 3.4: Summary statistics for pollutant measurements over various window exposures.

Pollutant		Min	Q1	Median	Mean	Q3	Max	SD	IQR
$PM_{2.5}$ ($\mu g/m^3$)	24hr	1.7	5.0	7.8	8.9	11.1	30.9	5.6	6.1
	48hr	2.8	5.4	7.8	8.6	9.7	27.3	4.9	4.4
	72hr	3.2	5.9	7.9	8.6	9.5	24.1	4.0	3.6
	96hr	3.2	6.0	7.8	8.4	10.0	20.1	3.4	4.0
PM_{10} ($\mu g/m^3$)	24hr	7.3	16.0	24.9	31.8	35.2	102.0	24.2	19.3
	48hr	6.2	17.1	24.4	31.3	37.9	84.7	20.0	20.8
	72hr	9.2	17.0	25.0	30.1	39.0	72.0	16.0	21.9
	96hr	8.2	18.7	25.6	29.4	39.4	65.0	13.6	20.7
NO_2 (ppb)	24hr	0.7	8.8	12.7	15.0	21.4	34.0	8.2	12.6
	48hr	2.4	9.1	13.0	14.6	19.5	31.1	7.0	10.4
	72hr	3.2	10.2	14.1	14.7	18.0	29.7	6.0	7.8
	96hr	5.0	11.0	13.8	14.8	18.8	29.7	5.2	7.8
O_3 (ppb)	24hr	6.3	16.5	24.2	25.2	33.1	51.7	10.7	16.6
	48hr	7.9	17.2	25.3	25.4	31.8	47.0	9.8	14.6
	72hr	8.9	17.5	24.7	25.1	32.4	48.6	9.5	14.9
	96hr	9.6	17.0	24.5	25.2	32.7	47.4	9.3	15.6



*The box represents the first to the third quartile. The vertical line is the median. The yellow dot is the mean concentration. PM_{10} and $PM_{2.5}$ are expressed in $\mu g/m^3$, NO^2 and O^3 are expressed in ppb.

Figure 3.2: Summary boxplots of air pollution concentrations

3.3.3 RESPIRATORY ASSOCIATIONS

Descriptive statistics for exhaled nitric oxide (eNO) and spirometry measurements are summarized in Table 3.5. Range for eNO was from 4.9 to 113 with a mean of 21.37 (±14) ppb. The forced exhaled volume during the first second of expiration ranged from 0.76 to 4.86 L with a mean of 2.4 (±0.6) L, the forced vital capacity ranged from 0.82 to 6 L with a mean of 2.65 (±0.7) L, the peak expiratory flow (PEF) ranged from 1.59 to 11.48 L/min with a mean of 5.29 (±1.7) L/min.

Table 3.5: Descriptive statistics for eNO, FEV_1, FVC, and PEF metrics.

Variable	Min	Q1	Median	Mean	Q3	Max	SD	IQR
eNO (ppb)	4.9	13.0	18.0	21.4	24.0	113.0	14.0	11.0
FEV_1 (L)	0.8	2.0	2.3	2.4	2.7	4.9	0.6	0.7
FVC (L)	0.8	2.2	2.6	2.6	3.0	6.0	0.7	0.8
PEF (L/min)	1.6	4.2	5.1	5.3	6.2	11.5	1.7	2.0
FEV_1 %Pred	18	83	92	96	101	360	31	18
FVC %Pred	16	73	82	85	91	266	24	18
PEF %Pred	14	81	95	96	110	267	27	29
FEV_1/FVC	0.6	0.9	0.9	0.9	1.0	1.0	0.1	0.1
FEV_1Best (L)	0.4	2.1	2.4	2.5	2.8	5.1	0.6	0.7
FVC Best (L)	0.5	2.3	2.7	2.8	3.2	6.0	0.8	0.9
PEF Best (L/min)	0.8	5.1	6.1	6.1	7.1	12.2	1.7	2.0

Table 3.6 presents pollutant effect estimates on respiratory outcomes using linear regression models and corresponding *p*. Regression analysis showed that short-term pollutant concentrations of $PM_{2.5}$ were negatively associated with spirometry measures such as FEV_1; $\beta_1 = -0.011$ for 24-hr $PM_{2.5}$ ($p = 0.038$), $\beta_1 = -0.014$ for 48-hr $PM_{2.5}$ ($p = 0.018$), and $\beta_1 = -0.017$ for 96-hr $PM_{2.5}$ ($p = 0.032$). FEV_1Best value showed similar associations with 24- and 48-hr $PM_{2.5}$; $\beta_1 = -0.011$ for 24-hr $PM_{2.5}$ ($p = 0.043$), and $\beta_1 = -0.013$ for 48-hr $PM_{2.5}$ ($p = 0.034$).

PEF was also negatively correlated with $PM_{2.5}$ for all time exposure periods ($\beta_1 = -0.048$ for 24-hr $PM_{2.5}$, $\beta_1 = -0.058$ for 48-hr $PM_{2.5}$, $\beta_1 = -0.054$ for 72-hr $PM_{2.5}$, $\beta_1 = -0.068$ for 96-hr $PM_{2.5}$; $p < 0.01$). We found that the longer the exposure to $PM_{2.5}$ concentrations, the greater the decrease in lung function, represented by PEF. The 24-, 48- and 96-hr means NO_2 had negative association with PEF; $\beta_1 = -0.023$ for 24-hr NO_2 ($p = 0.013$), $\beta_1 = -0.028$ for 48-hr NO_2 ($p = 0.011$), and $\beta_1 = -0.028$ for 96-hr NO_2 ($p = 0.047$). Only 48-hr PM_{10} showed relevance to the PEF Best value with $\beta_1 = -0.008$ ($p = 0.043$). The log-transformed exhaled NO, FVC, %predicted values in FEV_1, FVC, and PEF did not show any significant relationship with pollutant measurements.

Our finding was robust in the relationship of FEV_1/FVC with pollutant measurements. Using generalized linear regression modeling, we observed negative association between FEV_1/FVC and 96-hr $PM_{2.5}$ ($\beta_1 = -0.023$, $p = 0.040$). The ratio was also negatively associated with 24-hr NO_2 ($\beta_1 = -0.011$, $p = 0.020$) and 96-hr NO_2 ($\beta_1 = -0.019$, $p = 0.011$). However, 24-hr ozone data showed a positive correlation with FEV_1/FVC value ($\beta_1 = 0.008$, $p = 0.040$) which could be due to a negative correlation between NO_2 and O_3.

Table 3.6: Association between respiratory outcome and pollutant metrics.

Respiratory Outcome	**Pollutant**		**Estimate**	**Std. Error**	**t value**	***p***
log(eNO)	**$PM_{2.5}$**	24hr	-0.003	0.004	-0.678	0.498
	(ug/m^3)	48hr	-0.002	0.005	-0.382	0.702
		72hr	-0.001	0.006	-0.216	0.829
		96hr	0.001	0.007	0.159	0.873
	PM_{10}	24hr	0.000	0.001	-0.100	0.920
	(ug/m^3)	48hr	0.001	0.001	0.485	0.628
		72hr	0.001	0.001	0.920	0.358
		96hr	0.002	0.002	1.165	0.244
	NO_2	24hr	-0.005	0.003	-1.693	0.091
	(ppb)	48hr	-0.003	0.003	-0.879	0.380
		72hr	-0.002	0.004	-0.502	0.616
		96hr	-0.001	0.004	-0.339	0.735
	O_3	24hr	0.002	0.002	0.937	0.349
	(ppb)	48hr	0.001	0.002	0.294	0.769
		72hr	0.000	0.002	0.031	0.975
		96hr	0.000	0.002	0.021	0.983
FEV_1	**$PM_{2.5}$**	**24hr**	-0.011	0.005	-2.080	***0.038**
	(ug/m^3)	**48hr**	-0.014	0.006	-2.381	***0.018**
		72hr	-0.012	0.007	-1.725	0.085
		96hr	-0.017	0.008	-2.148	***0.032**
	PM_{10}	24hr	-0.001	0.001	-1.205	0.229
	(ug/m^3)	48hr	-0.001	0.001	-1.047	0.295
		72hr	-0.001	0.002	-0.743	0.458
		96hr	-0.002	0.002	-1.088	0.277
	NO_2	24hr	-0.002	0.003	-0.551	0.582
	(ppb)	48hr	-0.006	0.004	-1.577	0.115
		72hr	-0.006	0.005	-1.190	0.235
		96hr	-0.009	0.005	-1.758	0.079
	O_3	24hr	0.000	0.003	0.101	0.920
	(ppb)	48hr	0.002	0.003	0.863	0.389
		72hr	0.001	0.003	0.293	0.770
		96hr	0.001	0.003	0.449	0.654
FVC	**$PM_{2.5}$**	24hr	-0.010	0.006	-1.594	0.111
	(ug/m^3)	48hr	-0.013	0.007	-1.860	0.064

Respiratory Outcome	Pollutant		Estimate	Std. Error	t value	*p*
		72hr	-0.008	0.008	-0.969	0.333
		96hr	-0.011	0.010	-1.147	0.252
	PM_{10}	24hr	-0.002	0.001	-1.150	0.251
	(ug/m^3)	48hr	-0.001	0.002	-0.693	0.489
		72hr	-0.001	0.002	-0.261	0.794
		96hr	-0.001	0.002	-0.366	0.715
	NO_2	24hr	0.001	0.004	0.364	0.716
	(ppb)	48hr	-0.005	0.005	-1.017	0.310
		72hr	-0.003	0.006	-0.454	0.650
		96hr	-0.005	0.006	-0.788	0.431
	O_3	24hr	-0.002	0.003	-0.642	0.521
	(ppb)	48hr	0.001	0.003	0.251	0.802
		72hr	-0.001	0.003	-0.325	0.746
		96hr	-0.001	0.003	-0.236	0.813
PEF	**$PM_{2.5}$**	**24hr**	-0.048	0.015	-3.289	***0.001**
	(ug/m^3)	**48hr**	-0.058	0.016	-3.555	***0.000**
		72hr	-0.054	0.019	-2.883	***0.004**
		96hr	-0.068	0.022	-3.120	***0.002**
	PM_{10}	24hr	-0.005	0.003	-1.509	0.132
	(ug/m^3)	48hr	-0.007	0.004	-1.964	0.050
		72hr	-0.007	0.005	-1.522	0.129
		96hr	-0.009	0.005	-1.637	0.102
	NO_2	**24hr**	-0.023	0.009	-2.496	***0.013**
	(ppb)	**48hr**	-0.028	0.011	-2.561	***0.011**
		72hr	-0.023	0.013	-1.787	0.075
		96hr	-0.028	0.014	-1.987	***0.047**
	O_3	24hr	0.007	0.007	0.961	0.337
	(ppb)	48hr	0.009	0.008	1.148	0.251
		72hr	0.004	0.008	0.475	0.635
		96hr	0.004	0.008	0.534	0.593
Transformed	**$PM_{2.5}$**	24hr	0.000	0.001	-0.072	0.943
FEV_1 %Pred	**(ug/m^3)**	48hr	0.000	0.002	-0.081	0.935
		72hr	0.001	0.002	0.519	0.604
		96hr	0.001	0.002	0.248	0.804
	PM_{10}	24hr	0.000	0.000	0.705	0.481
	(ug/m^3)	48hr	0.000	0.000	0.129	0.897
		72hr	0.000	0.000	0.283	0.778
		96hr	0.000	0.001	0.262	0.794
	NO_2	24hr	0.000	0.001	0.099	0.922
	(ppb)	48hr	0.000	0.001	0.451	0.652
		72hr	0.000	0.001	0.374	0.708
		96hr	0.000	0.001	0.029	0.977
	O_3	24hr	-0.001	0.001	-1.415	0.158
	(ppb)	48hr	-0.001	0.001	-1.564	0.118
		72hr	-0.001	0.001	-1.630	0.104
		96hr	-0.001	0.001	-1.462	0.144
Transformed	**$PM_{2.5}$**	24hr	0.000	0.002	0.216	0.829
FVC %Pred	**(ug/m^3)**	48hr	0.000	0.002	0.179	0.858

Respiratory Outcome	Pollutant		Estimate	Std. Error	t value	*p*
		72hr	0.003	0.003	0.953	0.341
		96hr	0.002	0.003	0.722	0.470
	PM_{10}	24hr	0.000	0.000	0.355	0.723
	(ug/m^3)	48hr	0.000	0.001	0.269	0.788
		72hr	0.000	0.001	0.547	0.585
		96hr	0.000	0.001	0.525	0.600
	NO_2	24hr	0.001	0.001	1.078	0.281
	(ppb)	48hr	0.001	0.002	0.800	0.424
		72hr	0.002	0.002	0.917	0.360
		96hr	0.001	0.002	0.669	0.504
	O_3	24hr	-0.002	0.001	-1.861	0.063
	(ppb)	48hr	-0.002	0.001	-1.619	0.106
		72hr	-0.002	0.001	-1.704	0.089
		96hr	-0.002	0.001	-1.569	0.117
Transformed	**$PM_{2.5}$**	24hr	-0.357	0.237	-1.508	0.132
PEF %Pred	**(ug/m^3)**	48hr	-0.444	0.262	-1.694	0.091
		72hr	-0.354	0.304	-1.167	0.244
		96hr	-0.522	0.350	-1.491	0.136
	PM_{10}	24hr	-0.001	0.049	-0.011	0.991
	(ug/m^3)	48hr	-0.054	0.059	-0.911	0.363
		72hr	-0.054	0.073	-0.744	0.457
		96hr	-0.068	0.086	-0.799	0.424
	NO_2	24hr	-0.214	0.147	-1.453	0.147
	(ppb)	48hr	-0.124	0.176	-0.706	0.480
		72hr	-0.095	0.204	-0.468	0.640
		96hr	-0.106	0.225	-0.469	0.639
	O_3	24hr	-0.014	0.111	-0.124	0.901
	(ppb)	48hr	-0.073	0.124	-0.589	0.556
		72hr	-0.101	0.127	-0.797	0.426
		96hr	-0.101	0.128	-0.791	0.429
FEV_1/FVC	**$PM_{2.5}$**	24hr	-0.006	0.008	-0.765	0.445
	(ug/m^3)	48hr	-0.007	0.009	-0.877	0.381
		72hr	-0.015	0.010	-1.563	0.119
		96hr	-0.023	0.011	-2.062	***0.040**
	PM_{10}	24hr	0.002	0.002	1.148	0.251
	(ug/m^3)	48hr	0.000	0.002	0.056	0.955
		72hr	-0.001	0.002	-0.379	0.705
		96hr	-0.002	0.003	-0.837	0.403
	NO_2	**24hr**	-0.011	0.005	-2.342	***0.020**
	(ppb)	48hr	-0.007	0.006	-1.170	0.243
		72hr	-0.013	0.007	-1.963	0.050
		96hr	-0.019	0.007	-2.540	***0.011**
	O_3	**24hr**	0.008	0.004	2.057	***0.040**
	(ppb)	48hr	0.005	0.004	1.242	0.215
		72hr	0.006	0.004	1.338	0.182
		96hr	0.006	0.004	1.453	0.147
FEV_1 Best	**$PM_{2.5}$**	**24hr**	-0.011	0.006	-2.027	***0.043**
	(ug/m^3)	**48hr**	-0.013	0.006	-2.123	***0.034**

Respiratory Outcome	Pollutant		Estimate	Std. Error	t value	*p*
		72hr	-0.011	0.007	-1.521	0.129
		96hr	-0.015	0.008	-1.774	0.077
	PM_{10}	24hr	-0.001	0.001	-1.254	0.210
	(ug/m^3)	48hr	-0.002	0.001	-1.082	0.280
		72hr	-0.001	0.002	-0.806	0.421
		96hr	-0.002	0.002	-0.962	0.336
	NO_2	24hr	-0.002	0.004	-0.526	0.599
	(ppb)	48hr	-0.006	0.004	-1.442	0.150
		72hr	-0.005	0.005	-1.079	0.281
		96hr	-0.008	0.005	-1.564	0.118
	O_3	24hr	0.000	0.003	0.106	0.915
	(ppb)	48hr	0.003	0.003	0.852	0.395
		72hr	0.001	0.003	0.462	0.644
		96hr	0.002	0.003	0.613	0.540
FVC Best	**$PM_{2.5}$**	24hr	-0.012	0.007	-1.755	0.080
	(ug/m^3)	48hr	-0.014	0.007	-1.858	0.064
		72hr	-0.009	0.009	-1.046	0.296
		96hr	-0.011	0.010	-1.077	0.282
	PM_{10}	24hr	-0.002	0.001	-1.497	0.135
	(ug/m^3)	48hr	-0.002	0.002	-1.019	0.309
		72hr	-0.001	0.002	-0.599	0.550
		96hr	-0.001	0.002	-0.568	0.570
	NO_2	24hr	0.001	0.004	0.240	0.811
	(ppb)	48hr	-0.005	0.005	-1.061	0.289
		72hr	-0.003	0.006	-0.460	0.646
		96hr	-0.004	0.006	-0.692	0.489
	O_3	24hr	-0.002	0.003	-0.560	0.576
	(ppb)	48hr	0.001	0.004	0.390	0.696
		72hr	0.000	0.004	-0.019	0.985
		96hr	0.000	0.004	0.078	0.938
PEF Best	**$PM_{2.5}$**	**24hr**	-0.048	0.015	-3.154	***0.002**
	(ug/m^3)	**48hr**	-0.055	0.017	-3.317	***0.001**
		72hr	-0.050	0.019	-2.583	***0.010**
		96hr	-0.062	0.022	-2.752	***0.006**
	PM_{10}	24hr	-0.005	0.003	-1.614	0.107
	(ug/m^3)	**48hr**	-0.008	0.004	-2.026	***0.043**
		72hr	-0.008	0.005	-1.623	0.105
		96hr	-0.009	0.005	-1.562	0.119
	NO_2	**24hr**	-0.020	0.009	-2.104	***0.036**
	(ppb)	**48hr**	-0.024	0.011	-2.169	***0.031**
		72hr	-0.018	0.013	-1.372	0.171
		96hr	-0.021	0.014	-1.434	0.152
	O_3	24hr	0.005	0.007	0.658	0.511
	(ppb)	48hr	0.007	0.008	0.844	0.399
		72hr	0.002	0.008	0.227	0.821
		96hr	0.002	0.008	0.212	0.832

*All significant pollutant time exposures and corresponding *p* are expressed in bold.

3.3.4 CARDIOVASCULAR ASSOCIATIONS

Descriptive statistics for cardiovascular measurements are presented in Table 3.7. The mean for BMI was 30.6 (±6.6) kg/m^2 and 95.5 cm for waist. Blood pressure on average was 128/76 (±20/11) mmHg with a differential blood pressure of 52 (±14) mmHg. The lipid profile measures indicated a mean total cholesterol of 190 (±38.7) mg/dL, mean triglycerides of 186 (±114.7) mg/dL, mean HDL of 49 (±14.6) mg/dL, mean LDL of 106 (±31.9) mg/dL a fasting blood glucose mean of 108.7 (±46.5) mg/dL.

Table 3.7: Descriptive statistics for cardiovascular syndrome risk factors.

Variable	Min	Q1	Median	Mean	Q3	Max	SD	IQR
BMI (kg/m^2)	12.66	26.52	29.69	30.56	34.56	67.65	6.58	8.04
Waist (cm)	49	86	94	95.46	104	151	14.41	18
SBP (mmHg)	74	113	125	127.77	140.25	211	20.63	27.25
DBP (mmHg)	35	69	75	76.18	82	128	11.41	13
PBP (mmHg)	6	42	49	51.59	59	107	14.48	17
TC (mg/dL)	99.9	161	187.5	189.95	215	350	38.77	54
TG (mg/dL)	44.9	107.25	161	186.04	224	650.1	114.66	116.75
HDL (mg/dL)	14.9	40	48	49.71	58	100.1	14.58	18
LDL (mg/dL)	12	84	102	106.09	127	220	31.90	43
TC/HDL	1.4	3.1	3.8	4.17	4.8	22	1.71	1.7
FBG (mg/dL)	49.9	86.25	94.5	108.68	108	477	46.48	21.75

Correlation and regression analyses showed that the continuous variable measuring metabolic syndrome risk factors (e.g. waist, HDL, and fasting blood glucose), were associated with pollutant measurements. Detailed results of the correlation and regression analyses are shown in Tables 3.8 and 3.9. Further analyses of waist by gender and blood pressure (SBP and DBP) using clinical cutoff values where explored as well. The waist circumference, for females, showed a strong relationship with most of the pollutants; positive correlation with $PM_{2.5}$ and NO_2 for all exposure periods ($p < 0.005$), while negative correlations with all ozone for all exposure periods ($p < 0.050$). The relationship between waist and $PM_{2.5}$ may be due to a strong correlation observed

between BMI and waist (correlation coefficient of 0.870 for females and 0.893 for males). The 72-hr $PM_{2.5}$ concentration was found to be positively associated with BMI ($\beta_1 = 0.132$, $p = 0.042$).

We observed a significant relationship between 96-hr mean ozone and HDL, showing positive correlation with $\beta_1 = 0.136$ ($p = 0.028$). The increase in 24-/48-hr $PM_{2.5}$ and PM_{10} were significantly associated with an increase in the box-cox transformed fasting blood glucose ($p < 0.05$), but not for the original scale of fasting blood glucose. The transformation of fasting blood glucose was suitable to find linear relationships with air pollution measurement.

Table 3.8: Correlation analysis

Variable	Pollutant[3]	24hr	48hr	72hr	96hr
BMI	**$PM_{2.5}$**	0.070	0.064	**0.080**	0.069
(kg/m^2)	**PM_{10}**	0.022	0.018	0.017	0.009
	NO_2	0.048	0.065	0.065	0.052
	O_3	-0.010	-0.018	-0.024	-0.015
Waist	**$PM_{2.5}$**	**0.113**	**0.121**	**0.134**	**0.129**
(cm)	**PM_{10}**	0.031	0.043	0.045	0.045
	NO_2	**0.126**	**0.149**	**0.158**	**0.142**
	O_3	**-0.098**	**-0.112**	**-0.117**	**-0.107**
Female (N=559)	**$PM_{2.5}$**	**0.148**	**0.161**	**0.179**	**0.171**
	PM_{10}	0.050	0.068	0.076	0.077
	NO_2	**0.126**	**0.157**	**0.164**	**0.141**
	O_3	**-0.100**	**-0.118**	**-0.123**	**-0.108**
Male (N=103)	**$PM_{2.5}$**	-0.036	-0.060	-0.084	-0.063
	PM_{10}	-0.056	-0.086	-0.127	-0.124
	NO_2	0.150	0.126	0.130	0.155
	O_3	-0.118	-0.109	-0.112	-0.128
SBP	**$PM_{2.5}$**	-0.053	-0.053	-0.034	-0.030
(mmHg)	**PM_{10}**	-0.049	-0.065	-0.053	-0.037
	NO_2	-0.030	0.003	0.008	0.022
	O_3	0.021	0.013	0.013	0.010
SBP < 130 (N=377)	**$PM_{2.5}$**	**-0.112**	-0.090	-0.025	-0.015
	PM_{10}	**-0.106**	**-0.124**	-0.076	-0.062
	NO_2	**-0.106**	-0.062	-0.020	0.011
	O_3	0.026	0.002	-0.023	-0.044
SBP >= 130 (N=263)	**$PM_{2.5}$**	-0.033	-0.087	-0.102	-0.120
	PM_{10}	-0.028	-0.055	-0.066	-0.053
	NO_2	-0.006	-0.021	-0.049	-0.078
	O_3	0.039	0.070	0.087	0.094
DBP	**$PM_{2.5}$**	-0.075	-0.076	-0.069	-0.069
(mmHg)	**PM_{10}**	-0.051	-0.050	-0.047	-0.045
	NO_2	-0.015	-0.022	-0.032	-0.021
	O_3	0.054	0.059	0.063	0.060

Variable	Pollutant[3]	24hr	48hr	72hr	96hr
DBP < 85 (N=509)	**$PM_{2.5}$**	-0.062	-0.042	-0.009	0.017
	PM_{10}	-0.052	-0.048	-0.026	0.000
	NO_2	-0.034	-0.029	-0.014	0.020
	O_3	0.044	0.033	0.016	0.007
DBP >= 85 (N=131)	**$PM_{2.5}$**	0.016	-0.040	-0.054	-0.070
	PM_{10}	-0.032	-0.036	-0.046	-0.057
	NO_2	0.002	-0.062	-0.120	-0.126
	O_3	-0.080	-0.015	0.015	0.011
PBP	**$PM_{2.5}$**	-0.016	-0.016	0.006	0.012
(mmHg)	**PM_{10}**	-0.029	-0.054	-0.038	-0.018
	NO_2	-0.031	0.021	0.036	0.047
	O_3	-0.013	-0.028	-0.031	-0.033
TC	**$PM_{2.5}$**	-0.042	-0.009	-0.005	0.000
(mg/dL)	**PM_{10}**	-0.011	0.005	0.020	0.033
	NO_2	-0.039	-0.034	-0.020	-0.017
	O_3	-0.004	-0.006	-0.010	-0.009
TG	**$PM_{2.5}$**	-0.006	-0.008	0.002	0.009
(mg/dL)	**PM_{10}**	-0.033	-0.046	-0.040	-0.033
	NO_2	0.004	-0.005	0.007	0.027
	O_3	-0.065	-0.062	-0.053	-0.053
log.TG	**$PM_{2.5}$**	-0.004	-0.005	0.016	0.021
	PM_{10}	-0.026	-0.039	-0.027	-0.020
	NO_2	0.009	0.011	0.027	0.046
	O_3	-0.059	-0.063	-0.061	-0.062
HDL	**$PM_{2.5}$**	0.038	0.040	0.026	0.023
(mg/dL)	**PM_{10}**	0.049	0.042	0.043	0.045
	NO_2	-0.031	-0.024	-0.037	-0.047
	O_3	0.072	0.069	0.077	**0.087**
LDL	**$PM_{2.5}$**	-0.066	-0.016	-0.026	-0.019
(mg/dL)	**PM_{10}**	-0.048	-0.009	-0.011	0.000
	NO_2	-0.028	-0.004	0.000	-0.006
	O_3	0.008	-0.006	-0.013	-0.015
TC/HDL	**$PM_{2.5}$**	-0.064	-0.038	-0.028	-0.016
	PM_{10}	-0.044	-0.033	-0.027	-0.025
	NO_2	-0.005	0.015	0.027	0.027
	O_3	-0.013	-0.012	-0.011	-0.009
log.TC/HDL	**$PM_{2.5}$**	-0.075	-0.042	-0.027	-0.018
	PM_{10}	-0.060	-0.041	-0.033	-0.029
	NO_2	0.006	0.024	0.039	0.042
	O_3	-0.037	-0.039	-0.042	-0.044
bc.TC/HDL[1]	**$PM_{2.5}$**	-0.076	-0.041	-0.024	-0.016
	PM_{10}	-0.065	-0.043	-0.035	-0.030
	NO_2	0.012	0.029	0.045	0.049
	O_3	-0.044	-0.048	-0.053	-0.055
FBG	**$PM_{2.5}$**	0.019	0.024	-0.002	-0.012
(mg/dL)	**PM_{10}**	0.024	0.027	0.012	0.016
	NO_2	0.004	0.006	-0.005	-0.017
	O_3	0.008	-0.005	0.003	0.011

Variable	Pollutant[3]	24hr	48hr	72hr	96hr
log.FBG	**$PM_{2.5}$**	0.048	0.050	0.022	0.012
	PM_{10}	0.051	0.050	0.032	0.036
	NO_2	0.027	0.033	0.018	0.005
	O_3	-0.011	-0.030	-0.023	-0.015
bc.FBG[2]	**$PM_{2.5}$**	**0.087**	**0.087**	0.065	0.059
	PM_{10}	**0.091**	**0.084**	0.070	0.075
	NO_2	0.054	0.064	0.052	0.043
	O_3	-0.029	-0.052	-0.051	-0.045

All significant correlations are expressed in bold.

[1)] Box-Cox Transformation: bc.TC/HDL = [(TC/HDL)^(-0.5)-1]/(-0.5).

[2)] Box-Cox Transformation: bc.FBG = [FBG^(-2)-1]/(-2).

[3)] PM_{10} and $PM_{2.5}$ are expressed in $\mu g/m^3$, NO_2 and O^3 are expressed in ppb.

Table 3.9: Association between cardiovascular outcomes and pollutant metrics.

Variable	Pollutant		Estimate	Std. Error	t value	p value
BMI	**$PM_{2.5}$**	24hr	0.086	0.048	1.792	0.074
(kg/m^2)	**(ug/m^3)**	48hr	0.089	0.055	1.631	0.103
		72hr	0.132	0.065	2.036	***0.042**
		96hr	0.135	0.077	1.756	0.080
	PM_{10}	24hr	0.006	0.011	0.564	0.573
	(ug/m^3)	48hr	0.006	0.013	0.449	0.654
		72hr	0.007	0.016	0.431	0.666
		96hr	0.004	0.019	0.218	0.828
	NO_2	24hr	0.040	0.032	1.231	0.219
	(ppb)	48hr	0.062	0.037	1.654	0.099
		72hr	0.072	0.044	1.660	0.097
		96hr	0.066	0.050	1.323	0.186
	O_3	24hr	-0.006	0.024	-0.254	0.799
	(ppb)	48hr	-0.012	0.027	-0.469	0.639
		72hr	-0.017	0.027	-0.605	0.545
		96hr	-0.011	0.028	-0.384	0.701
Waist (cm)	**$PM_{2.5}$**	**24hr**	0.301	0.104	2.901	***0.004**
(Overall)	**(ug/m^3)**	**48hr**	0.365	0.117	3.114	***0.002**
		72hr	0.486	0.141	3.459	***0.001**
		96hr	0.554	0.166	3.332	***0.001**
	PM_{10}	24hr	0.019	0.024	0.783	0.434
	(ug/m^3)	48hr	0.031	0.028	1.100	0.272
		72hr	0.041	0.035	1.154	0.249
		96hr	0.048	0.042	1.153	0.249
	NO_2	**24hr**	0.225	0.070	3.238	***0.001**
	(ppb)	**48hr**	0.309	0.081	3.833	***0.000**
		72hr	0.382	0.094	4.060	***0.000**
		96hr	0.393	0.108	3.636	***0.000**
	O_3	**24hr**	-0.132	0.052	-2.527	***0.012**
	(ppb)	**48hr**	-0.166	0.058	-2.870	***0.004**
		72hr	-0.179	0.059	-3.014	***0.003**
		96hr	-0.167	0.061	-2.754	***0.006**

Variable	Pollutant		Estimate	Std. Error	t value	p value
Waist (cm)	**$PM_{2.5}$**	**24hr**	0.386	0.110	3.508	***0.000**
(Female, N=559)	**(ug/m^3)**	**48hr**	0.473	0.124	3.820	***0.000**
		72hr	0.625	0.147	4.262	***0.000**
		96hr	0.712	0.175	4.077	***0.000**
	PM_{10}	24hr	0.029	0.025	1.172	0.242
	(ug/m^3)	48hr	0.048	0.030	1.595	0.111
		72hr	0.067	0.037	1.799	0.073
		96hr	0.079	0.044	1.805	0.072
	NO_2	**24hr**	0.221	0.075	2.953	***0.003**
	(ppb)	**48hr**	0.321	0.087	3.702	***0.000**
		72hr	0.392	0.101	3.868	***0.000**
		96hr	0.388	0.116	3.331	***0.001**
	O_3	**24hr**	-0.132	0.056	-2.351	***0.019**
	(ppb)	**48hr**	-0.171	0.061	-2.781	***0.006**
		72hr	-0.184	0.063	-2.900	***0.004**
		96hr	-0.166	0.065	-2.558	***0.011**
Waist (cm)	**$PM_{2.5}$**	24hr	-0.104	0.287	-0.361	0.719
(Male, N=103)	**(ug/m^3)**	48hr	-0.202	0.336	-0.602	0.549
		72hr	-0.368	0.434	-0.848	0.399
		96hr	-0.316	0.498	-0.635	0.527
	PM_{10}	24hr	-0.040	0.071	-0.566	0.573
	(ug/m^3)	48hr	-0.075	0.086	-0.868	0.387
		72hr	-0.137	0.107	-1.284	0.202
		96hr	-0.150	0.119	-1.258	0.211
	NO_2	24hr	0.273	0.180	1.517	0.132
	(ppb)	48hr	0.270	0.212	1.275	0.205
		72hr	0.318	0.243	1.307	0.194
		96hr	0.438	0.280	1.566	0.121
	O_3	24hr	-0.164	0.137	-1.194	0.235
	(ppb)	48hr	-0.176	0.161	-1.099	0.274
		72hr	-0.186	0.163	-1.136	0.259
		96hr	-0.215	0.165	-1.301	0.196
SBP (mmHg)	**$PM_{2.5}$**	24hr	-0.205	0.154	-1.331	0.184
(Overall)	**(ug/m^3)**	48hr	-0.234	0.175	-1.337	0.182
		72hr	-0.180	0.209	-0.862	0.389
		96hr	-0.185	0.246	-0.752	0.452
	PM_{10}	24hr	-0.043	0.035	-1.228	0.220
	(ug/m^3)	48hr	-0.069	0.042	-1.651	0.099
		72hr	-0.069	0.052	-1.334	0.183
		96hr	-0.057	0.060	-0.942	0.347
	NO_2	24hr	-0.077	0.102	-0.752	0.453
	(ppb)	48hr	0.008	0.119	0.072	0.943
		72hr	0.028	0.138	0.202	0.840
		96hr	0.086	0.157	0.548	0.584
	O_3	24hr	0.040	0.076	0.523	0.601
	(ppb)	48hr	0.028	0.084	0.331	0.741
		72hr	0.029	0.086	0.332	0.740

Variable	Pollutant		Estimate	Std. Error	t value	p value
		96hr	0.022	0.088	0.245	0.807
SBP (mmHg)	**$PM_{2.5}$**	**24hr**	-0.222	0.102	-2.167	***0.031**
(<130, N=377)	**(ug/m^3)**	48hr	-0.203	0.115	-1.756	0.080
		72hr	-0.067	0.139	-0.482	0.630
		96hr	-0.048	0.161	-0.297	0.766
	PM_{10}	**24hr**	-0.047	0.023	-2.039	***0.042**
	(ug/m^3)	**48hr**	-0.066	0.028	-2.410	***0.016**
		72hr	-0.050	0.034	-1.468	0.143
		96hr	-0.048	0.040	-1.197	0.232
	NO_2	**24hr**	-0.140	0.068	-2.059	***0.040**
	(ppb)	48hr	-0.098	0.081	-1.204	0.229
		72hr	-0.035	0.094	-0.377	0.706
		96hr	0.022	0.105	0.210	0.834
	O_3	24hr	0.026	0.051	0.497	0.620
	(ppb)	48hr	0.002	0.057	0.043	0.966
		72hr	-0.026	0.058	-0.441	0.659
		96hr	-0.051	0.060	-0.857	0.392
SBP (mmHg)	**$PM_{2.5}$**	24hr	-0.092	0.173	-0.532	0.595
(>=130, N=263)	**(ug/m^3)**	48hr	-0.283	0.201	-1.410	0.160
		72hr	-0.396	0.238	-1.664	0.097
		96hr	-0.553	0.284	-1.947	0.053
	PM_{10}	24hr	-0.018	0.040	-0.454	0.650
	(ug/m^3)	48hr	-0.042	0.048	-0.882	0.379
		72hr	-0.063	0.059	-1.064	0.288
		96hr	-0.060	0.070	-0.856	0.393
	NO_2	24hr	-0.010	0.115	-0.091	0.928
	(ppb)	48hr	-0.044	0.130	-0.340	0.734
		72hr	-0.119	0.152	-0.782	0.435
		96hr	-0.221	0.176	-1.257	0.210
	O_3	24hr	0.053	0.084	0.628	0.531
	(ppb)	48hr	0.106	0.093	1.141	0.255
		72hr	0.134	0.095	1.413	0.159
		96hr	0.147	0.097	1.517	0.130
DBP (mmHg)	**$PM_{2.5}$**	24hr	-0.162	0.085	-1.906	0.057
	(ug/m^3)	48hr	-0.185	0.097	-1.913	0.056
		72hr	-0.202	0.116	-1.745	0.082
		96hr	-0.239	0.136	-1.758	0.079
	PM_{10}	24hr	-0.025	0.019	-1.276	0.203
	(ug/m^3)	48hr	-0.029	0.023	-1.264	0.207
		72hr	-0.034	0.029	-1.197	0.232
		96hr	-0.038	0.033	-1.129	0.259
	NO_2	24hr	-0.021	0.056	-0.376	0.707
	(ppb)	48hr	-0.035	0.065	-0.541	0.588
		72hr	-0.061	0.076	-0.800	0.424
		96hr	-0.045	0.086	-0.525	0.600
	O_3	24hr	0.058	0.042	1.374	0.170
	(ppb)	48hr	0.070	0.046	1.504	0.133

Variable	Pollutant		Estimate	Std. Error	t value	p value
		72hr	0.076	0.048	1.591	0.112
		96hr	0.074	0.049	1.518	0.130
DBP (mmHg)	**$PM_{2.5}$**	24hr	-0.088	0.063	-1.394	0.164
(<85, N=509)	**(ug/m^3)**	48hr	-0.068	0.072	-0.944	0.345
		72hr	-0.018	0.087	-0.210	0.834
		96hr	0.039	0.103	0.381	0.703
	PM_{10}	24hr	-0.017	0.015	-1.171	0.242
	(ug/m^3)	48hr	-0.019	0.018	-1.088	0.277
		72hr	-0.013	0.022	-0.576	0.565
		96hr	0.000	0.026	0.007	0.995
	NO_2	24hr	-0.033	0.044	-0.759	0.448
	(ppb)	48hr	-0.033	0.051	-0.646	0.518
		72hr	-0.019	0.060	-0.309	0.757
		96hr	0.031	0.067	0.456	0.648
	O_3	24hr	0.033	0.033	1.000	0.318
	(ppb)	48hr	0.027	0.036	0.742	0.458
		72hr	0.013	0.037	0.360	0.719
		96hr	0.006	0.038	0.160	0.873
DBP (mmHg)	**$PM_{2.5}$**	24hr	0.028	0.161	0.176	0.861
(>=85, N=131)	**(ug/m^3)**	48hr	-0.087	0.190	-0.456	0.649
		72hr	-0.130	0.213	-0.611	0.542
		96hr	-0.193	0.240	-0.801	0.425
	PM_{10}	24hr	-0.012	0.032	-0.364	0.716
	(ug/m^3)	48hr	-0.015	0.037	-0.409	0.683
		72hr	-0.024	0.046	-0.520	0.604
		96hr	-0.034	0.053	-0.644	0.521
	NO_2	24hr	0.002	0.082	0.023	0.982
	(ppb)	48hr	-0.065	0.093	-0.702	0.484
		72hr	-0.146	0.106	-1.367	0.174
		96hr	-0.180	0.126	-1.426	0.156
	O_3	24hr	-0.055	0.061	-0.915	0.362
	(ppb)	48hr	-0.012	0.068	-0.173	0.863
		72hr	0.012	0.070	0.167	0.868
		96hr	0.009	0.073	0.120	0.904
PBP (mmHg)	**$PM_{2.5}$**	24hr	-0.042	0.108	-0.393	0.695
	(ug/m^3)	48hr	-0.049	0.123	-0.399	0.690
		72hr	0.021	0.147	0.145	0.885
		96hr	0.054	0.173	0.311	0.756
	PM_{10}	24hr	-0.018	0.024	-0.741	0.459
	(ug/m^3)	48hr	-0.040	0.029	-1.356	0.176
		72hr	-0.035	0.036	-0.956	0.339
		96hr	-0.019	0.042	-0.452	0.652
	NO_2	24hr	-0.056	0.072	-0.775	0.439
	(ppb)	48hr	0.044	0.083	0.527	0.599
		72hr	0.089	0.097	0.915	0.360
		96hr	0.131	0.110	1.193	0.233
	O_3	24hr	-0.018	0.053	-0.336	0.737

Variable	Pollutant		Estimate	Std. Error	t value	p value
	(ppb)	48hr	-0.042	0.059	-0.712	0.477
		72hr	-0.047	0.061	-0.778	0.437
		96hr	-0.052	0.062	-0.846	0.398
TC (mg/dL)	**$PM_{2.5}$**	24hr	-0.298	0.280	-1.064	0.288
	(ug/m^3)	48hr	-0.069	0.318	-0.217	0.828
		72hr	-0.052	0.382	-0.135	0.893
		96hr	0.004	0.452	0.008	0.994
	PM_{10}	24hr	-0.018	0.064	-0.287	0.775
	(ug/m^3)	48hr	0.010	0.076	0.135	0.893
		72hr	0.049	0.095	0.518	0.604
		96hr	0.093	0.112	0.831	0.406
	NO_2	24hr	-0.184	0.188	-0.981	0.327
	(ppb)	48hr	-0.189	0.219	-0.864	0.388
		72hr	-0.132	0.255	-0.515	0.607
		96hr	-0.128	0.293	-0.439	0.661
	O_3	24hr	-0.015	0.141	-0.108	0.914
	(ppb)	48hr	-0.023	0.156	-0.147	0.883
		72hr	-0.040	0.160	-0.247	0.805
		96hr	-0.038	0.163	-0.230	0.819
TG (mg/dL)	**$PM_{2.5}$**	24hr	-0.136	0.829	-0.165	0.869
	(ug/m^3)	48hr	-0.197	0.939	-0.210	0.834
		72hr	0.072	1.129	0.064	0.949
		96hr	0.302	1.337	0.226	0.821
	PM_{10}	24hr	-0.159	0.189	-0.845	0.399
	(ug/m^3)	48hr	-0.264	0.226	-1.171	0.242
		72hr	-0.287	0.280	-1.022	0.307
		96hr	-0.280	0.330	-0.849	0.396
	NO_2	24hr	0.053	0.557	0.096	0.924
	(ppb)	48hr	-0.075	0.648	-0.116	0.907
		72hr	0.126	0.756	0.167	0.868
		96hr	0.600	0.866	0.693	0.489
	O_3	24hr	-0.697	0.417	-1.673	0.095
	(ppb)	48hr	-0.732	0.460	-1.594	0.111
		72hr	-0.642	0.473	-1.359	0.175
		96hr	-0.658	0.483	-1.362	0.174
log.TG	**$PM_{2.5}$**	24hr	0.000	0.004	-0.110	0.913
	(ug/m^3)	48hr	-0.001	0.005	-0.117	0.907
		72hr	0.002	0.006	0.402	0.688
		96hr	0.003	0.007	0.530	0.597
	PM_{10}	24hr	-0.001	0.001	-0.655	0.513
	(ug/m^3)	48hr	-0.001	0.001	-0.997	0.319
		72hr	-0.001	0.001	-0.678	0.498
		96hr	-0.001	0.002	-0.508	0.611
	NO_2	24hr	0.001	0.003	0.216	0.829
	(ppb)	48hr	0.001	0.003	0.275	0.783
		72hr	0.002	0.004	0.674	0.501
		96hr	0.005	0.004	1.161	0.246

Variable	Pollutant		Estimate	Std. Error	t value	p value
	O_3	24hr	-0.003	0.002	-1.511	0.131
	(ppb)	48hr	-0.004	0.002	-1.611	0.108
		72hr	-0.004	0.002	-1.550	0.122
		96hr	-0.004	0.002	-1.594	0.111
HDL (mg/dL)	**$PM_{2.5}$**	24hr	0.102	0.106	0.962	0.336
	(ug/m^3)	48hr	0.121	0.120	1.008	0.314
		72hr	0.094	0.144	0.651	0.515
		96hr	0.101	0.171	0.589	0.556
	PM_{10}	24hr	0.030	0.024	1.233	0.218
	(ug/m^3)	48hr	0.031	0.029	1.062	0.289
		72hr	0.039	0.036	1.098	0.273
		96hr	0.048	0.042	1.129	0.259
	NO_2	24hr	-0.056	0.071	-0.792	0.428
	(ppb)	48hr	-0.050	0.083	-0.610	0.542
		72hr	-0.090	0.096	-0.938	0.349
		96hr	-0.129	0.110	-1.173	0.241
	O_3	24hr	0.098	0.053	1.838	0.066
	(ppb)	48hr	0.104	0.059	1.763	0.078
		72hr	0.117	0.060	1.946	0.052
		96hr	0.136	0.062	2.206	***0.028**
LDL (mg/dL)	**$PM_{2.5}$**	24hr	-0.391	0.241	-1.622	0.105
	(ug/m^3)	48hr	-0.109	0.273	-0.399	0.690
		72hr	-0.203	0.326	-0.624	0.533
		96hr	-0.181	0.386	-0.469	0.639
	PM_{10}	24hr	-0.062	0.054	-1.157	0.248
	(ug/m^3)	48hr	-0.014	0.065	-0.218	0.827
		72hr	-0.022	0.080	-0.273	0.785
		96hr	-0.001	0.095	-0.010	0.992
	NO_2	24hr	-0.110	0.161	-0.680	0.497
	(ppb)	48hr	-0.021	0.188	-0.109	0.913
		72hr	-0.002	0.219	-0.009	0.992
		96hr	-0.034	0.251	-0.137	0.891
	O_3	24hr	0.024	0.121	0.200	0.842
	(ppb)	48hr	-0.021	0.134	-0.159	0.874
		72hr	-0.042	0.137	-0.308	0.758
		96hr	-0.050	0.140	-0.355	0.723
TC/HDL	**$PM_{2.5}$**	24hr	-0.020	0.013	-1.591	0.112
	(ug/m^3)	48hr	-0.014	0.014	-0.953	0.341
		72hr	-0.012	0.017	-0.707	0.480
		96hr	-0.008	0.020	-0.389	0.697
	PM_{10}	24hr	-0.003	0.003	-1.096	0.274
	(ug/m^3)	48hr	-0.003	0.003	-0.823	0.411
		72hr	-0.003	0.004	-0.676	0.499
		96hr	-0.003	0.005	-0.621	0.535
	NO_2	24hr	-0.001	0.008	-0.124	0.902
	(ppb)	48hr	0.004	0.010	0.368	0.713
		72hr	0.008	0.011	0.662	0.508

Variable	Pollutant		Estimate	Std. Error	t value	p value
		96hr	0.009	0.013	0.672	0.502
	O_3	24hr	-0.002	0.006	-0.338	0.735
	(ppb)	48hr	-0.002	0.007	-0.308	0.758
		72hr	-0.002	0.007	-0.288	0.774
		96hr	-0.002	0.007	-0.234	0.815
log.TC/HDL	$PM_{2.5}$	24hr	-0.005	0.003	-1.873	0.062
	(ug/m^3)	48hr	-0.003	0.003	-1.059	0.290
		72hr	-0.002	0.003	-0.681	0.496
		96hr	-0.002	0.004	-0.452	0.651
	PM_{10}	24hr	-0.001	0.001	-1.501	0.134
	(ug/m^3)	48hr	-0.001	0.001	-1.016	0.310
		72hr	-0.001	0.001	-0.835	0.404
		96hr	-0.001	0.001	-0.717	0.473
	NO_2	24hr	0.000	0.002	0.152	0.879
	(ppb)	48hr	0.001	0.002	0.596	0.552
		72hr	0.002	0.002	0.963	0.336
		96hr	0.003	0.003	1.059	0.290
	O_3	24hr	-0.001	0.001	-0.918	0.359
	(ppb)	48hr	-0.001	0.001	-0.977	0.329
		72hr	-0.002	0.001	-1.057	0.291
		96hr	-0.002	0.001	-1.109	0.268
bc.TC/HDL[1]	$PM_{2.5}$	24hr	-0.002	0.001	-1.904	0.057
	(ug/m^3)	48hr	-0.001	0.001	-1.037	0.300
		72hr	-0.001	0.002	-0.606	0.545
		96hr	-0.001	0.002	-0.409	0.683
	PM_{10}	24hr	0.000	0.000	-1.616	0.107
	(ug/m^3)	48hr	0.000	0.000	-1.067	0.286
		72hr	0.000	0.000	-0.870	0.384
		96hr	0.000	0.000	-0.743	0.458
	NO_2	24hr	0.000	0.001	0.292	0.770
	(ppb)	48hr	0.001	0.001	0.728	0.467
		72hr	0.001	0.001	1.126	0.261
		96hr	0.002	0.001	1.228	0.220
	O_3	24hr	-0.001	0.001	-1.115	0.265
	(ppb)	48hr	-0.001	0.001	-1.209	0.227
		72hr	-0.001	0.001	-1.323	0.186
		96hr	-0.001	0.001	-1.394	0.164
FBG (mg/dL)	$PM_{2.5}$	24hr	0.166	0.336	0.495	0.621
	(ug/m^3)	48hr	0.237	0.381	0.622	0.534
		72hr	-0.025	0.458	-0.054	0.957
		96hr	-0.163	0.542	-0.300	0.764
	PM_{10}	24hr	0.047	0.076	0.614	0.539
	(ug/m^3)	48hr	0.064	0.092	0.697	0.486
		72hr	0.034	0.114	0.298	0.766
		96hr	0.053	0.134	0.398	0.690
	NO_2	24hr	0.024	0.225	0.105	0.917
	(ppb)	48hr	0.042	0.262	0.160	0.873

Variable	Pollutant		Estimate	Std. Error	t value	p value
		72hr	-0.040	0.306	-0.132	0.895
		96hr	-0.153	0.350	-0.438	0.662
	O_3	24hr	0.034	0.169	0.200	0.841
	(ppb)	48hr	-0.025	0.187	-0.135	0.893
		72hr	0.012	0.192	0.064	0.949
		96hr	0.054	0.196	0.274	0.784
log.FBG	**$PM_{2.5}$**	24hr	0.003	0.002	1.211	0.226
	(ug/m^3)	48hr	0.003	0.002	1.273	0.203
		72hr	0.002	0.003	0.557	0.578
		96hr	0.001	0.003	0.313	0.754
	PM_{10}	24hr	0.001	0.000	1.294	0.196
	(ug/m^3)	48hr	0.001	0.001	1.269	0.205
		72hr	0.001	0.001	0.809	0.419
		96hr	0.001	0.001	0.920	0.358
	NO_2	24hr	0.001	0.001	0.678	0.498
	(ppb)	48hr	0.001	0.002	0.832	0.406
		72hr	0.001	0.002	0.450	0.653
		96hr	0.000	0.002	0.122	0.903
	O_3	24hr	0.000	0.001	-0.280	0.780
	(ppb)	48hr	-0.001	0.001	-0.754	0.451
		72hr	-0.001	0.001	-0.585	0.558
		96hr	0.000	0.001	-0.375	0.708
bc.FBG[2]	**$PM_{2.5}$**	**24hr**	0.000	0.000	2.220	***0.027**
	(ug/m^3)	**48hr**	0.000	0.000	2.225	***0.026**
		72hr	0.000	0.000	1.673	0.095
		96hr	0.000	0.000	1.517	0.130
	PM_{10}	**24hr**	0.000	0.000	2.324	***0.020**
	(ug/m^3)	**48hr**	0.000	0.000	2.162	***0.031**
		72hr	0.000	0.000	1.794	0.073
		96hr	0.000	0.000	1.924	0.055
	NO_2	24hr	0.000	0.000	1.369	0.171
	(ppb)	48hr	0.000	0.000	1.632	0.103
		72hr	0.000	0.000	1.330	0.184
		96hr	0.000	0.000	1.097	0.273
	O_3	24hr	0.000	0.000	-0.750	0.453
	(ppb)	48hr	0.000	0.000	-1.318	0.188
		72hr	0.000	0.000	-1.294	0.196
		96hr	0.000	0.000	-1.146	0.252

*All significant pollutant time exposures and corresponding *p* are expressed in bold.
[1)] Box-Cox Transformation: bc.TC/HDL = [(TC/HDL)^(-0.5)-1]/(-0.5).
[2)] Box-Cox Transformation: bc.FBG = [FBG^(-2)-1]/(-2).

The classification of metabolic syndrome risk factors (binary outcomes) based on current guidelines is presented in Table 3.10. Associations between classification of metabolic syndrome factors and pollutant metrics are summarized in Table 3.11. Table 3.11 shows effect estimates

using logistic regression models, corresponding *p*, odds ratios, and 95% confidence intervals of the odds ratio. In logistic regression modeling, we also found that increasing $PM_{2.5}$ and NO_2 concentration was associated with increasing likelihoods of having a high waist ($p < 0.05$ for 48-, 72- and 96-hr $PM_{2.5}$; $p < 0.01$ for all windows of NO_2 concentration). However, the odds of having a high waist decreases as the ozone level increases ($p < 0.05$ for 24/48/72/96-hr O_3). The ozone increase was also associated with less likelihood of having low HDL status ($p < 0.05$ for 24-/48-/72-/96-hr O_3), and more exposures to ozone led to a lower odds ratio of having low HDL (0.983 for 24hr O_3, 0.980 for 48- and 72-hr O_3, and 0.976 for 96-hr O_3).

More likelihood of having high glucose was associated with increased PM concentrations; 1.034 and 1.037 times higher odds ratio as 1 $\mu g/m^3$ increase in 24- and 48-hr $PM_{2.5}$, respectively ($p < 0.05$) which is important considering our range for $PM_{2.5}$ from 5 to 31 ug/m3. One $\mu g/m^3$ change in both 24- and 48-hr PM_{10} results in 1.008 times higher in the odds ratio of high glucose ($p < 0.05$). The 72-hr NO_2 concentration was also a significant factor in prediction of the high glucose status, showing an increased likelihood of having high glucose as NO_2 increases (odds ratio = 1.027; $p = 0.048$).

Metabolic syndrome showed significant associations with $PM_{2.5}$, NO_2, and O_3. The odds of having MetS is 1.051 times higher with 1 $\mu g/m^3$ increase in 96-hr $PM_{2.5}$ ($p = 0.043$). The associations of MetS classification with NO_2 concentrations are also positive, showing the increased odds ratio of 1.027 ($p = 0.021$), 1.040 ($p = 0.004$), and 1.056 ($p = 0.001$) for 48-, 72-, and 96-hr exposures, respectively. However, the increased ozone was correlated with a decreased likelihood of having MetS ($p < 0.05$ for all time exposures).

Table 3.10: Summary of metabolic syndrome risk factors.

Variables		Frequency	%*
High Waist	Yes	411	62.1
	No	244	36.9
High BP	Yes	277	41.8
	No	363	54.8
High TG	Yes	363	54.8
	No	291	44.0
Low HDL	Yes	329	49.7
	No	315	47.6
High FBG	Yes	231	34.9
	No	423	63.9
Metabolic Syndrome	Yes	336	50.8
	No	307	46.4

*Percentages might not add to 100% due to missing values

Table 3.11: Associations between metabolic syndrome (MetS) risk factors and MetS classification and pollutant metrics.

Variables	Pollutant		Estimate	Std. Error	z value	*p*	Odds Ratio	Lower 95% CI	Upper 95% CI
High Waist									
	$PM_{2.5}$	24 hr	0.030	0.016	1.902	0.057	1.031	1.000	1.065
	(ug/m^3)	**48 hr**	0.038	0.018	2.084	***0.037**	1.039	1.003	1.078
		72 hr	0.060	0.022	2.697	***0.007**	1.062	1.018	1.112
		96 hr	0.070	0.026	2.697	***0.007**	1.072	1.020	1.130
	PM_{10}	24 hr	0.003	0.003	0.859	0.390	1.003	0.996	1.010
	(ug/m^3)	48 hr	0.004	0.004	0.897	0.370	1.004	0.996	1.012
		72 hr	0.006	0.005	1.105	0.269	1.006	0.996	1.016
		96 hr	0.006	0.006	1.044	0.296	1.006	0.995	1.018
	NO_2	**24 hr**	0.027	0.010	2.641	***0.008**	1.027	1.007	1.048
	(ppb)	**48 hr**	0.036	0.012	3.028	***0.002**	1.037	1.013	1.062
		72 hr	0.048	0.014	3.399	***0.001**	1.050	1.021	1.080
		96 hr	0.055	0.016	3.373	***0.001**	1.056	1.023	1.091
	O_3	**24 hr**	-0.016	0.008	-2.079	***0.038**	0.984	0.970	0.999
	(ppb)	**48 hr**	-0.019	0.008	-2.238	***0.025**	0.981	0.965	0.998
		72 hr	-0.021	0.009	-2.465	***0.014**	0.979	0.963	0.996
		96 hr	-0.022	0.009	-2.485	***0.013**	0.978	0.962	0.995
High BP	**$PM_{2.5}$**	24 hr	-0.012	0.015	-0.781	0.435	0.988	0.958	1.018
	(ug/m^3)	48 hr	-0.007	0.017	-0.410	0.682	0.993	0.959	1.027
		72 hr	-0.009	0.021	-0.417	0.677	0.991	0.952	1.032
		96 hr	-0.007	0.024	-0.300	0.764	0.993	0.946	1.041
	PM_{10}	24 hr	-0.002	0.003	-0.585	0.559	0.998	0.991	1.005
	(ug/m^3)	48 hr	-0.002	0.004	-0.525	0.600	0.998	0.990	1.006
		72 hr	-0.003	0.005	-0.620	0.535	0.997	0.987	1.007

Variables	Pollutant		Estimate	Std. Error	z value	*p*	Odds Ratio	Lower 95% CI	Upper 95% CI
		96 hr	-0.003	0.006	-0.471	0.637	0.997	0.986	1.009
	NO_2	24 hr	-0.002	0.010	-0.213	0.832	0.998	0.979	1.018
	(ppb)	48 hr	0.004	0.012	0.379	0.705	1.004	0.982	1.027
		72 hr	0.004	0.013	0.301	0.763	1.004	0.978	1.031
		96 hr	0.010	0.015	0.649	0.517	1.010	0.980	1.041
	O_3	24 hr	0.005	0.007	0.719	0.472	1.005	0.991	1.020
	(ppb)	48 hr	0.003	0.008	0.331	0.741	1.003	0.987	1.019
		72 hr	0.004	0.008	0.489	0.625	1.004	0.988	1.021
		96 hr	0.004	0.009	0.519	0.603	1.004	0.988	1.022
High TG	**$PM_{2.5}$**	24 hr	0.001	0.015	0.061	0.951	1.001	0.973	1.030
	(ug/m^3)	48 hr	0.000	0.016	0.020	0.984	1.000	0.969	1.033
		72 hr	0.012	0.020	0.615	0.538	1.012	0.974	1.053
		96 hr	0.016	0.024	0.668	0.504	1.016	0.970	1.064
	PM_{10}	24 hr	0.001	0.003	0.378	0.705	1.001	0.995	1.008
	(ug/m^3)	48 hr	-0.001	0.004	-0.181	0.857	0.999	0.992	1.007
		72 hr	0.001	0.005	0.207	0.836	1.001	0.991	1.011
		96 hr	0.002	0.006	0.342	0.732	1.002	0.991	1.013
	NO_2	24 hr	0.005	0.010	0.478	0.633	1.005	0.986	1.024
	(ppb)	48 hr	0.009	0.011	0.796	0.426	1.009	0.987	1.032
		72 hr	0.021	0.013	1.568	0.117	1.021	0.995	1.048
		96 hr	0.027	0.015	1.770	0.077	1.027	0.997	1.059
	O_3	24 hr	-0.011	0.007	-1.435	0.151	0.990	0.975	1.004
	(ppb)	48 hr	-0.014	0.008	-1.690	0.091	0.986	0.971	1.002
		72 hr	-0.016	0.008	-1.874	0.061	0.985	0.968	1.001
		96 hr	-0.016	0.009	-1.919	0.055	0.984	0.967	1.000
Low HDL	**$PM_{2.5}$**	24 hr	-0.012	0.015	-0.837	0.403	0.988	0.960	1.016
	(ug/m^3)	48 hr	-0.006	0.016	-0.364	0.716	0.994	0.962	1.027
		72 hr	0.005	0.020	0.271	0.786	1.005	0.967	1.045
		96 hr	0.007	0.023	0.311	0.756	1.007	0.962	1.055
	PM_{10}	24 hr	-0.003	0.003	-1.057	0.291	0.997	0.990	1.003
	(ug/m^3)	48 hr	-0.003	0.004	-0.651	0.515	0.997	0.990	1.005
		72 hr	-0.004	0.005	-0.747	0.455	0.996	0.987	1.006
		96 hr	-0.005	0.006	-0.807	0.420	0.995	0.984	1.007
	NO_2	24 hr	0.010	0.010	1.066	0.286	1.010	0.991	1.030
	(ppb)	48 hr	0.013	0.011	1.109	0.267	1.013	0.990	1.036
		72 hr	0.017	0.013	1.252	0.211	1.017	0.991	1.044
		96 hr	0.027	0.015	1.765	0.078	1.027	0.997	1.058
	O_3	**24 hr**	-0.017	0.007	-2.317	***0.021**	0.983	0.969	0.997
	(ppb)	**48 hr**	-0.020	0.008	-2.465	***0.014**	0.980	0.964	0.996
		72 hr	-0.021	0.008	-2.462	***0.014**	0.980	0.964	0.996

Variables	Pollutant		Estimate	Std. Error	z value	*p*	Odds Ratio	Lower 95% CI	Upper 95% CI
		96 hr	-0.024	0.009	-2.822	***0.005**	0.976	0.960	0.993
High FBG									
	$PM_{2.5}$	**24 hr**	0.034	0.015	2.269	***0.023**	1.034	1.005	1.065
	(ug/m^3)	**48 hr**	0.037	0.017	2.197	***0.028**	1.037	1.004	1.072
		72 hr	0.030	0.020	1.487	0.137	1.030	0.990	1.072
		96 hr	0.026	0.024	1.073	0.283	1.026	0.979	1.076
	PM_{10}	**24 hr**	0.008	0.003	2.294	***0.022**	1.008	1.001	1.014
	(ug/m^3)	**48 hr**	0.008	0.004	2.011	***0.044**	1.008	1.000	1.016
		72 hr	0.008	0.005	1.661	0.097	1.008	0.998	1.018
		96 hr	0.009	0.006	1.466	0.143	1.009	0.997	1.021
	NO_2	24 hr	0.014	0.010	1.421	0.155	1.014	0.995	1.035
	(ppb)	48 hr	0.023	0.012	1.925	0.054	1.023	1.000	1.047
		72 hr	0.027	0.014	1.975	***0.048**	1.027	1.000	1.055
		96 hr	0.024	0.016	1.551	0.121	1.025	0.994	1.057
	O_3	24 hr	-0.008	0.008	-1.030	0.303	0.992	0.977	1.007
	(ppb)	48 hr	-0.014	0.008	-1.686	0.092	0.986	0.969	1.002
		72 hr	-0.017	0.009	-1.899	0.058	0.983	0.967	1.000
		96 hr	-0.015	0.009	-1.690	0.091	0.985	0.968	1.002
Metabolic Syndrome									
	$PM_{2.5}$	24 hr	0.022	0.015	1.422	0.155	1.022	0.992	1.053
	(ug/m^3)	48 hr	0.029	0.017	1.692	0.091	1.030	0.996	1.066
		72 hr	0.037	0.021	1.772	0.076	1.037	0.997	1.081
		96 hr	0.049	0.024	2.025	***0.043**	1.051	1.002	1.103
	PM_{10}	24 hr	0.003	0.003	0.922	0.357	1.003	0.997	1.010
	(ug/m^3)	48 hr	0.003	0.004	0.746	0.456	1.003	0.995	1.011
		72 hr	0.003	0.005	0.692	0.489	1.003	0.994	1.013
		96 hr	0.005	0.006	0.883	0.377	1.005	0.994	1.017
	NO_2	24 hr	0.019	0.010	1.888	0.059	1.019	0.999	1.039
	(ppb)	**48 hr**	0.027	0.012	2.305	***0.021**	1.027	1.004	1.051
		72 hr	0.039	0.014	2.877	***0.004**	1.040	1.013	1.068
		96 hr	0.054	0.016	3.475	***0.001**	1.056	1.024	1.089
	O_3	**24 hr**	-0.018	0.007	-2.470	***0.014**	0.982	0.968	0.996
	(ppb)	**48 hr**	-0.025	0.008	-3.010	***0.003**	0.975	0.960	0.991
		72 hr	-0.026	0.008	-3.008	***0.003**	0.975	0.959	0.991
		96 hr	-0.027	0.009	-3.079	***0.002**	0.974	0.957	0.990

*All significant pollutant time exposures and corresponding *p* are expressed in bold.

3.4 Discussion

3.4.1 PRINCIPAL FINDINGS

Our study examined the short-term associations (24/48/72/96-hr means) of traffic-related air pollutants ($PM_{2.5}$, PM_{10}, NO_2, and O_3) with biomarkers of respiratory and cardiovascular disease in a group of participants from low-income communities in El Paso, TX. We found associations of short-term air pollutant concentrations with respiratory outcomes which was expected. However, we also found associations with BMI and metabolic syndrome risk factors such as waist and fasting glucose.

The FEV_1 was negatively correlated with mean concentration levels of $PM_{2.5}$ (24/48/96-hr) indicating a relationship between lung function and ambient $PM_{2.5}$ before the measurement. Specifically, this respiratory indicator represents an increase in risk for obstructive respiratory diseases (asthma, chronic obstructive pulmonary disease [COPD]). Furthermore, the PEF, which is also an indicator of increased risk for asthma and COPD, was negatively correlated not only with $PM_{2.5}$, but also NO_2. However, we did not see an influence of PM_{10}, which might indicate significant health effects where caused by smaller particles which affect the lower respiratory tract and can further cause obstructive respiratory diseases (Xing, Xu, Shi, & Lian, 2016). Further analysis using the best results available for respiratory indicators (FEV_1 Best, FVC Best, and PEF Best), as interpreted by the spirometry software (CareFusion Spirometry PC Software™ 36-SPC1000-STK), confirmed the associations with $PM_{2.5}$ air pollutants and also NO_2.

Exhaled nitric oxide (eNO) is a measure of airway inflammation but was not correlated with concentration levels of air pollutants in our population. Even so, eNO is clinically useful in the treatment and control of asthma (Meyts, Proesmans, & De Boeck, 2003). Given that our

inclusion methods did not ask if a participant has asthma, we recommend future studies to consider relationships of eNO with air pollution in participants with asthma.

We also considered the percent predicted values of lung function, but our analyses did not show any significant correlation with air pollutant concentration levels. However, we did find associations with the FEV_1/FVC ratio which can differentiate obstructive from restrictive respiratory diseases. A ratio of 0.7 is indicative of lung obstruction and given the negative correlations found with $PM_{2.5}$ and NO_2 for different exposure periods, we theorize obstructive respiratory diseases to be more prevalent in our population compared to restrictive respiratory diseases (sarcoidosis, lung fibrosis).

Although short-term associations with risk factors related to obesity (BMI and waist circumference) both in linear and logistic models were not expected as part of this study, the relationship was present across most time exposure periods. We also did not expect a causal effect between short-term exposure to air pollution and obesity. However, the decreasing ranges and similar means of short-term air pollution concentrations (shown in Figure 3.2) could be indicative that similar trends could be found with medium or long-term measurements. This can be also representative of the environmental conditions and neighborhoods where participants live. A study focusing on NO_2 had similar concentration trends with increasing windows of time exposure when comparing short-term and long-term effects (Deguen et al., 2015)

We did not find associations with other metabolic outcomes such as blood pressure or lipid profile, but we did find associations with fasting blood glucose both in linear models as well as increased risk among those with high fasting glucose levels. Possible reasons for these associations include oxidative stress and inflammation caused by air pollution exposure (Bowe et al., 2018; Eze et al., 2015; Wolf et al., 2016).

3.4.2 STRENGTHS AND LIMITATIONS

The present study utilized ambient (outdoor) air pollution measured at nearby CAMS stations. However, there could be some variation in the participants' indoor environment related to pollutant exposure. Although research indicates there is a direct relationship between ambient and indoor air pollution which is further confirmed by the literature (Andersen, 1972; Raysoni, Stock, Sarnat, Montoya Sosa, et al., 2013; Zora et al., 2013), the true exposure concentration of a participant can be quite different from the surrogate concentration measured at the CAMS stations.

The measurements of air pollution exposure rely on CAMS with available data. In some cases, the stations where far from certain areas in El Paso County which led to exclusion of some participants from the analysis. Furthermore, not all CAMS had measurements available for every traffic-related pollutant. However, six CAMS measured ozone which was at least two times more than those measuring other pollutants, and yet we still observed similar trends of ozone concentrations compared to the other air pollutants.

3.4.3 COMPARISON WITH OTHER STUDIES

Respiratory outcomes have been associated with air pollution exposure in other epidemiological studies. The Framingham study found that moderate exposure measured by the EPA's Air Quality Index for $PM_{2.5}$, NO_2, and O_3 was associated with lower FEV_1 considering 24 and 48-hr pollutant concentration means before the measurement (Rice et al., 2013). A study among 1,694 female non-smokers from the Wuhan-Zhuhai, China found that in a city at high pollutant levels, the moving mean of $PM_{2.5}$, PM_{10}, NO_2, and O_3 exposures were significantly associated with FEV_1 reductions, but also in the low-level air pollution city PM_{10}, O_3, and $PM_{2.5}$ were significantly associated with reduced FEV_1 (Zhou et al., 2016). The same study also

found associations with FVC; however, we did not find them in our study which might be due to the relatively low levels of exposure, based on the available CAMS data.

Furthermore, a repeated measures study from Belgium found that an increase in PM_{10} on the day of the clinical examination was associated with lower FVC, $FEV_{1,}$ and PEF. Also, an increase in NO_2 was associated with a reduction in PEF on the day of the examination (Panis et al., 2017). In addition, a study of lung function in adults exposed to very low levels of ambient air pollution in Europe did not observe an association between air pollution and longitudinal change in lung function (Adam et al., 2015). However, they observed that an increase in NO_2 exposure was associated with lower levels of FEV_1 and FVC. Moreover, an increase of PM_{10}, but not other PM metrics ($PM_{2.5}$, coarse fraction of PM, PM absorbance), was associated with a lower level of FEV_1. The associations were particularly strong in people with obesity.

Regarding metabolic outcomes, Chuang and collaborators observed increased PM_{10} was marginally (p<0.10) associated with elevated systolic blood pressure (24-hr) and triglycerides (24 to 120-hr), and significantly associated with hemoglobin A1c (72-hr), and reduced HDL (24-hr) (p<0.05). They also reported that ozone was associated with diastolic blood pressure (72 and 120-hr) and hemoglobin A1C (24,72,120-hr) and marginally associated with triglycerides and fasting glucose (Chuang et al., 2010). Unfortunately, their study did not consider $PM_{2.5}$ measurements which showed some associations in our study.

A study conducted in China showed a positive correlation between air pollution (PM_{10}, NO_2, and O_3) and BMI (M. Li et al., 2015), which agrees well with associations from our study, although the time window they considered was based on long term exposures using average concentrations within a 3-year period instead of short term exposure. Furthermore, a 2014 review by Weichenthal, et al., which considered 14 short-term effect studies of air pollutants, suggested

the consistent pattern of associations among participants with obesity suggests that obesity may modify negatively the impact of $PM_{2.5}$ on cardiovascular health (Weichenthal, Hoppin, & Reeves, 2014).

3.5 Conclusions

Short term exposure to traffic-related pollutants were found to be correlated with respiratory outcomes related to pulmonary obstruction in our study. Future studies should consider clinical classifications of obstructive respiratory outcomes such as COPD and asthma while considering the effects on FEV_1 and PEF.

The present study might be the first to find associations of short-term exposure to air pollutants with obesity; we do not expect this to be a causal relationship. However, since it is possible that the short-term data reflect possible similar values of medium to long term data, the associations of air pollution with obesity might be explained by the socio-economic and neighborhood characteristics of participants. In future studies, we recommend considering obesity as a consequence of air pollution exposure and consider extended windows of time (more than 96-hr means) to assess long-term exposure. Furthermore, the use of statistical models that incorporate geographic measures such as distance to nearest pollution sources, street length around neighborhoods, or traffic volumes could further elucidate the relationship between obesity and air pollution relative to the socio-economic characteristics of the neighborhoods surrounding the participants. Lastly, we recommend studies that consider the relationship of air pollution exposure not only with fasting glucose, but also glycated hemoglobin and diabetes diagnosis.

CHAPTER 4 LAND USE REGRESSION OF LONG-TERM TRANSPORTATION DATA ON CARDIORESPIRATORY OUTCOMES OF LOW INCOME RESIDENTS FROM EL PASO, TX

4.1 Introduction

4.1.1 LONG TERM AIR POLLUTION EXPOSURE

Over the last three decades, large cohort studies have found associations of long-term exposures to air pollutants with increased mortality (Dockery et al., 1993; Pope et al., 1995). Highways and roadways are major sources of air pollutants because of vehicle traffic which can negatively affect surrounding communities. People with a lower income are more likely to live in communities with higher pollution levels from traffic-related air pollution, which can be considered an environmental justice issue (Brulle & Pellow, 2006; Cushing et al., 2015).

Examples of traffic-related air pollutants include particulate matter ($PM_{2.5}$ and PM_{10}) nitrogen dioxide (NO_2), and ozone (O_3) which pose a risk for cardiorespiratory diseases. Hoek and collaborators (2013) summarized long-term exposure to particulate matter (PM) and nitrogen dioxide (NO_2) on mortality from cardiovascular and respiratory diseases in epidemiological studies, and concluded participants with lower education and obesity had a larger risk for mortality related to $PM_{2.5}$ (Hoek et al., 2013). There is also increasing evidence of associations between increased long-term exposure to traffic-related air pollutants and lung function decline in children (Barone-Adesi et al., 2015) and adults (Rhee et al., 2019), as well as attenuation of this decline with reductions in air pollution exposure (Downs et al., 2007). Therefore, identifying zones of increased air pollution exposure can help develop strategies to improve the environmental conditions of those living in at-risk areas.

4.1.2 LIMITATIONS OF CENTRAL AIR MONITORING STATIONS

Located in the U.S.-Mexico border, the city of El Paso, TX has twelve central ambient monitoring stations (CAMS) operated by the Texas Commission of Environmental Quality (TCEQ) that measure air pollutants. However, few are equipped to measure all the traffic-related pollutants ($PM_{2.5}$, PM_{10}, NO_2, and O_3) which limits the quantification of air pollutant concentrations in some near-road communities. While previous studies in our region have focused on areas surrounding major highways in the city (Raysoni et al., 2011 & 2013), near-road studies for areas further north from the border are limited.

Large studies have established long-term effects of air pollution exposure to PM_{10}, $PM_{2.5,}$ and NO_2 on respiratory health outcomes including lung function using spirometry measures (Köpf et al., 2017). However, long-term studies that consider metabolic factors related to cardiovascular health are less common and findings remain mixed. A study among Mexican Americans in Southern California was unable to find long-term associations between air pollutants and health outcomes, such as glucose and insulin resistance, using spatial interpolation from air quality monitors (Z. Chen et al., 2016). However, another study that assessed long-term effects of air pollution using land use regression models (LUR) on glucose, insulin, glycated hemoglobin (HbA_{1c}), and C-reactive protein, suggested an association between long-term exposure to air pollution and insulin resistance (Wolf et al., 2016).

4.1.3 INCORPORATING GEOGRAPHICAL INFORMATION TO MODELS

A review of 157 studies using various exposure methods concluded that future research would benefit from hybrid models combining the strengths of air pollution exposure assessments and geospatial information system (GIS) technologies (Zou, Wilson, Zhan, & Zeng, 2009). Some studies have shown consistent associations between near-roadway air pollution and cardiorespiratory

diseases using traffic density and proximity to roadways (Gan, Koehoorn, et al., 2010; Gan, Tamburic, et al., 2010; Jiang et al., 2016; Kan et al., 2008). Furthermore, Bell and collaborators (2017) used a hierarchical spatiotemporal model considering traffic-related air pollutants seasonal trends, long-term pollutant means, and land use regression. They estimated mean pollutant concentrations at each participant's home location during the year of their baseline exam, as well as three months and two weeks prior to each participant's baseline exam. Furthermore, geographic covariates such as distance to roadway and land use characteristics were used in their models to improve prediction (Bell et al., 2017).

However, none of the mentioned studies considered cardiovascular and respiratory outcomes in socioeconomically disadvantaged communities. In addition, traffic and air quality are believed to be associated with cardiorespiratory factors. A better understanding of the impact of these environmental factors on cardiorespiratory health could help improve overall health in low-income communities. Therefore, we used traffic-related variables to explore relationship with cardiorespiratory health measures collected in our community using LUR models. Our main research objective was to determine if there was an association between the traffic-related variables (such as distance to major arterial roads, ports of entry, surrounding length of street, and traffic) and the cardiorespiratory risk factors and if these data can be used to better predict the risk of exposure to air pollution.

4.2 Methods

4.2.1 SETTING, POPULATION, AND SAMPLE

This project integrated air quality and traffic-related data with an epidemiological study conducted in El Paso, TX. The "*Evidence-Based Screening for Obesity, Cardiorespiratory Disease*

and Diabetes Mellitus in Low-Income El Paso Households" is an ongoing study that collects demographic and health-related data from low income participants in El Paso, TX. A team of health professionals conducts a yearly socio-demographic survey and collects health data on-site at convenient locations for the participants including housing authority communities, faith-based organizations, food distribution events by a local food bank, community health fairs, Mexican Consulate clinic days, and grocery stores. Health-related data collected include metabolic syndrome (MetS), a predictor of cardiovascular risk, which includes waist circumference, blood pressure, triglycerides, HDL-cholesterol, and blood glucose. During the baseline year of the study, data collected also included respiratory measures of airway inflammation (measured by an exhaled nitric oxide test) and lung function (measured by spirometry).

The present study conducted a secondary data analysis using health data collected between 2014 and 2020. The larger study protocol and the amendment for conducting this study were approved by the Institutional Review Board (IRB) under the project number study numbers: 590300-4 and 1249235-3 with a separate IRB for the secondary analysis under study number: 1611345-1.

4.2.2 TRAFFIC-RELATED MEASURES

The city of El Paso is located in far west Texas and borders with Ciudad Juarez, Mexico to the south and New Mexico to the west and north. For this study, we used data from the participant's home address to determine latitude and longitude coordinates and create a data layer using GIS software (Figure 4.1).

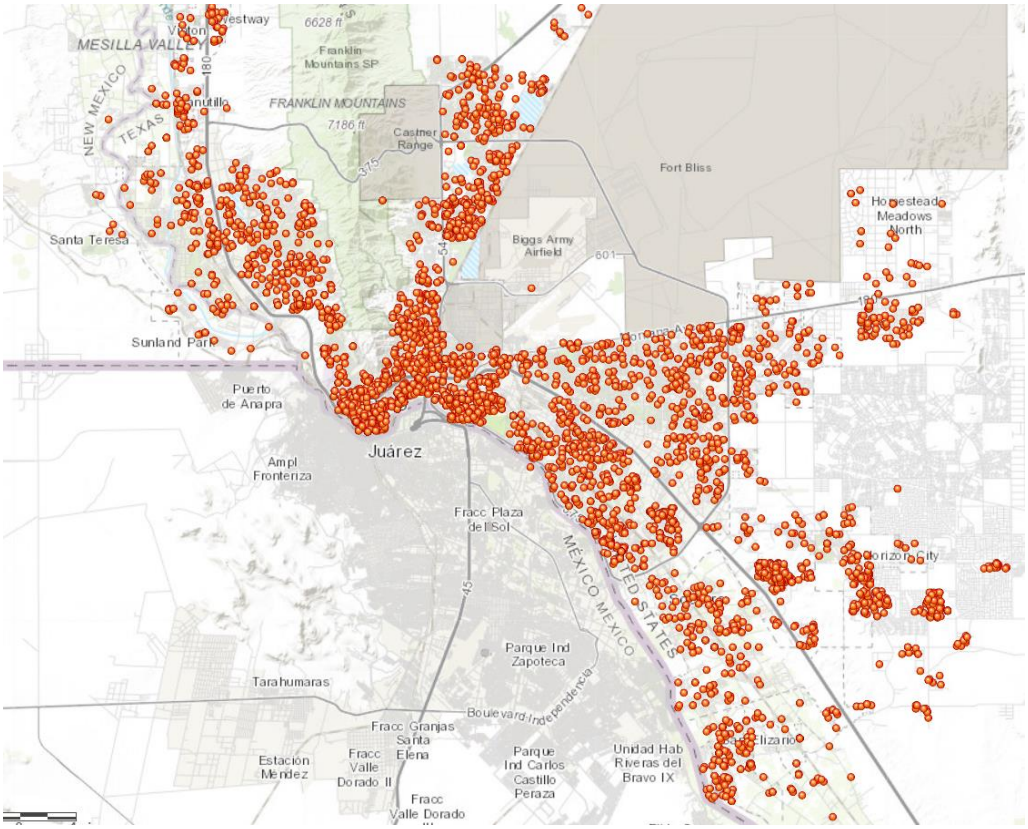


Figure 4.1: Residential addresses of participants from El Paso, TX

We used mapping tools (ArcGIS Pro 2.5) to calculate the distance to the nearest major arterial traffic road (majart) using a GIS layer developed by the Department of Civil Engineering at UTEP in collaboration with the City of El Paso, TX (available at the PDNMAPA website http://gis.elpasotexas.gov/pdnmapajs/) (Figure 4.2).

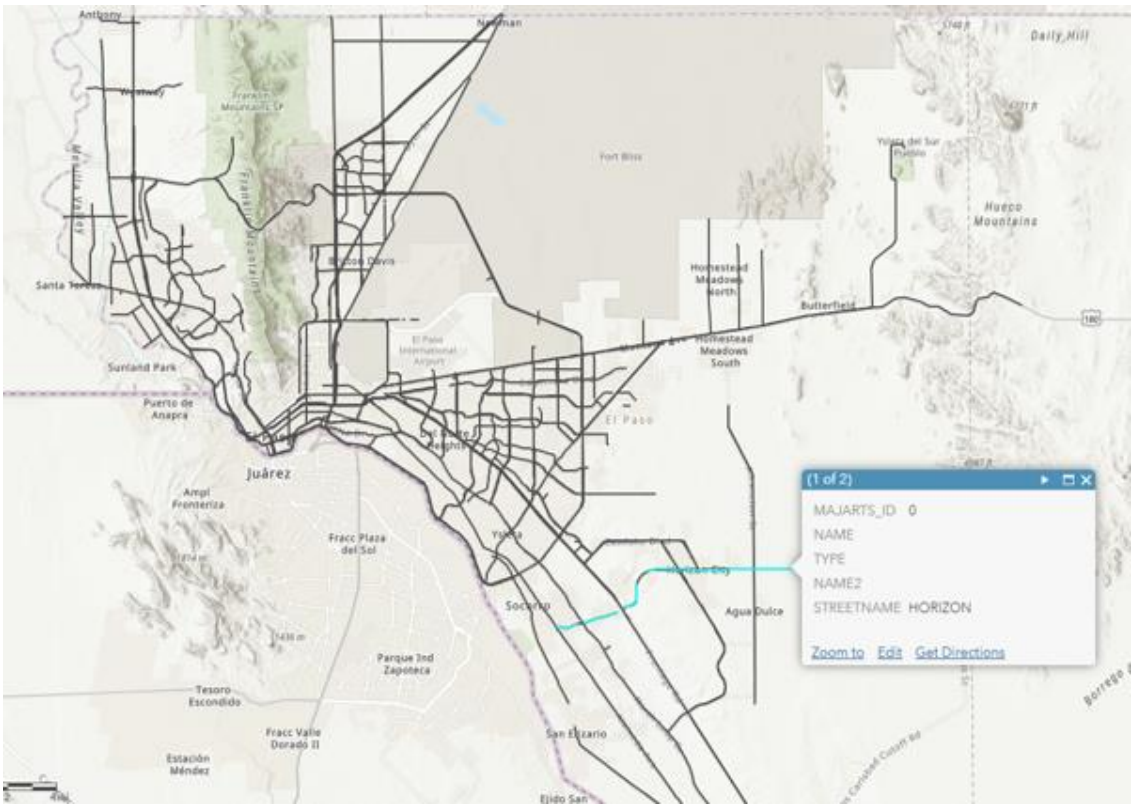


Figure 4.2: Major Arterial Roads Layer

Given its border with Mexico, El Paso has three international bridges which constitute ports of vehicle and pedestrian entry into the U.S. Due to the amount of daily traffic and car idling that occurs at these points of entry, we considered the distance from the participants home address to the nearest international port of entry (POE) as a layer of interest to explore associations of traffic-related air pollution with cardiorespiratory health outcomes (Figure 4.3).

Figure 4.3: Ports of entry in El Paso, TX

To explore the effects of vehicle traffic using GIS tools, we defined impact zones (500m and 1000m) relative to a participant's residential address. We used a GIS layer developed by El Paso's Metropolitan Planning Organization that included traffic counts from the city's major and minor roads address (Figure 4.4). This layer allowed us to calculate the sum of the annual averaged daily vehicle miles traveled (VMT) relative to a participant's residential address.

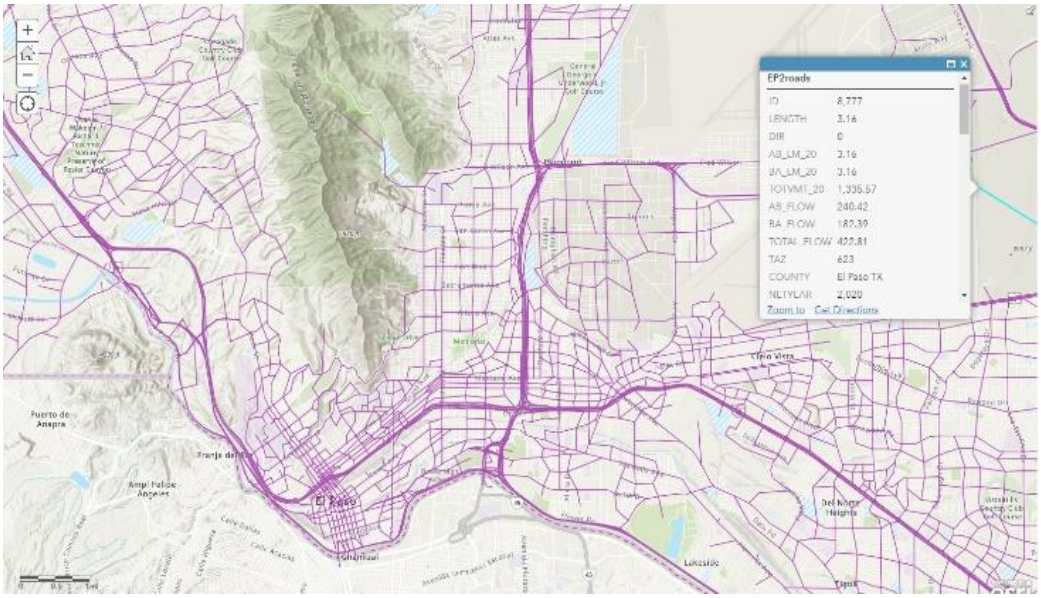


Figure 4.4: Metropolitan Planning Organization traffic layer

Lastly, we used a GIS layer available at the Census.gov website that includes all available streets and roads within El Paso County. This layer allowed us to summarize the length of street road within 500m and 1000m zones for every participant relative to their residential address (Figure 4.5). A land use regression (LUR) model was used to explore associations between the mentioned traffic-related variables with the cardiorespiratory outcomes measured for each participant from the epidemiological study.

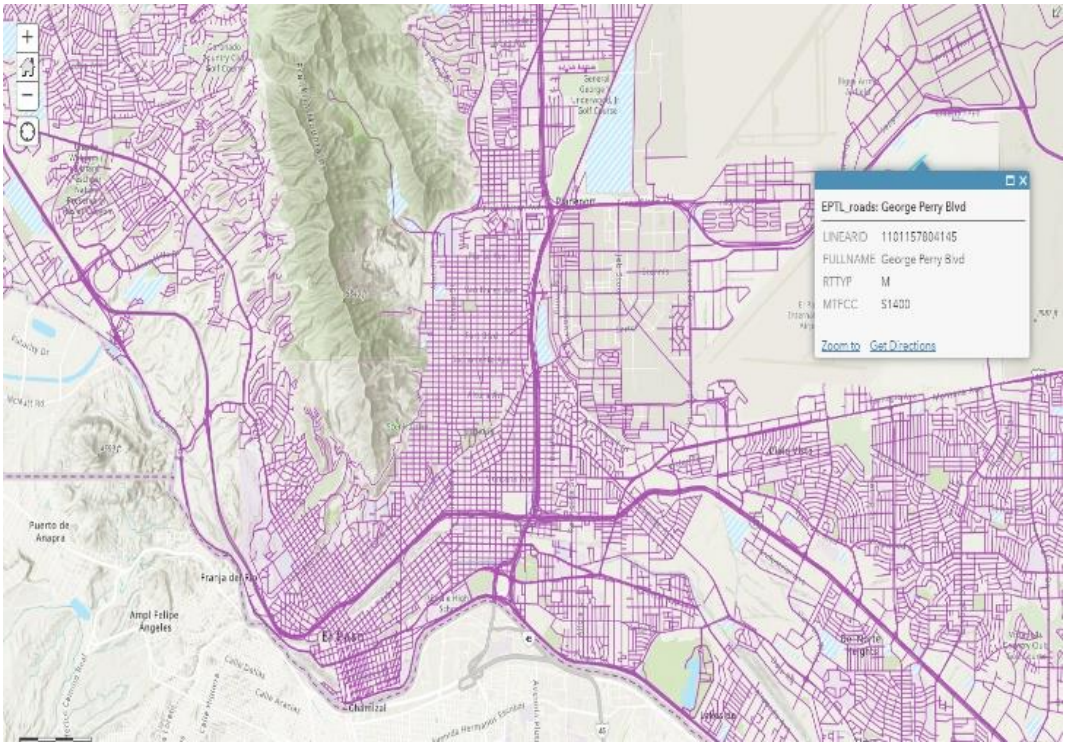


Figure 4.5: U.S. Census street layer and zoomed version

Our analysis established associations between cardiovascular outcome measures using linear models for continuous variables (BMI, waist circumference, blood pressure, triglycerides, HDL-cholesterol, glucose) and logistic models for categorical outcomes (metabolic syndrome [MetS]) with spatial transportation data.

We also used a subset of participants who had respiratory health outcomes (only available for the first year of the epidemiological study) to establish associations between respiratory health outcome measures using hierarchical models for continuous variables (eNO, FVC, FEV_1, PEF) with transportation data. Furthermore, we considered the distribution of the participants that were classified with MetS using the traffic-related variables to determine the geographical areas of a higher probability of MetS classification.

4.2.3 STATISTICAL ANALYSES

Regression models were conducted separately for each independent variable. Box-Cox transformation was applied to the variables %predicted FEV_1, TC/HDL, and glucose to account for the skewness in the distribution, and different power exponents were selected to transform the data; we also used the log-transformation for the eNO and %predicted FVC. The square root transformation was applied to the %predicted PEF to improve the distribution of the right-skewed PEF data. Linear regression considered respiratory as well as cardiovascular risk factors. Cardiovascular risk factors were classified as "high" or "low" based on clinical cut-off values used for the classification of metabolic syndrome (Expert Panel on Detection & Treatment of High Blood Cholesterol in, 2001). Participants that had three or more altered risk factors were categorized as having metabolic syndrome and, in contrast, those who had three or more risk factors within the normal range where categorized as not having metabolic syndrome. Logistic regression analyses were also used to examine the relationship between categorical variables and traffic-related measurements.

We applied the land use regression technique to explore the associations between a set of spatially distributed respiratory factors from 662 participants and metabolic syndrome (MetS) risk factors from 4,959 participants with the traffic and land-use predictors. Lastly, we used a stepwise

selection technique to determine the traffic-related variables associated with metabolic syndrome. The resulting prediction equation from the model was applied to a grid map of the El Paso area to determine zones with higher likelihood of metabolic syndrome. The level of statistical significance was set at p of < 0.05 for all tests. We used the statistical software R (version 3.6.2) to perform the statistical analysis portion of the study.

4.3 Results

4.3.1 GIS MAPPING

The use of GIS mapping tolls allowed us to generate traffic-related data for every participant as a proxy for traffic-related air pollution exposure, Figure 4.6 illustrates a subset of distances to the nearest major arterial traffic road relative to participant's GIS coordinates. In a similar way, we determined distances to the nearest international port of entry (POE) for each participant (not shown).

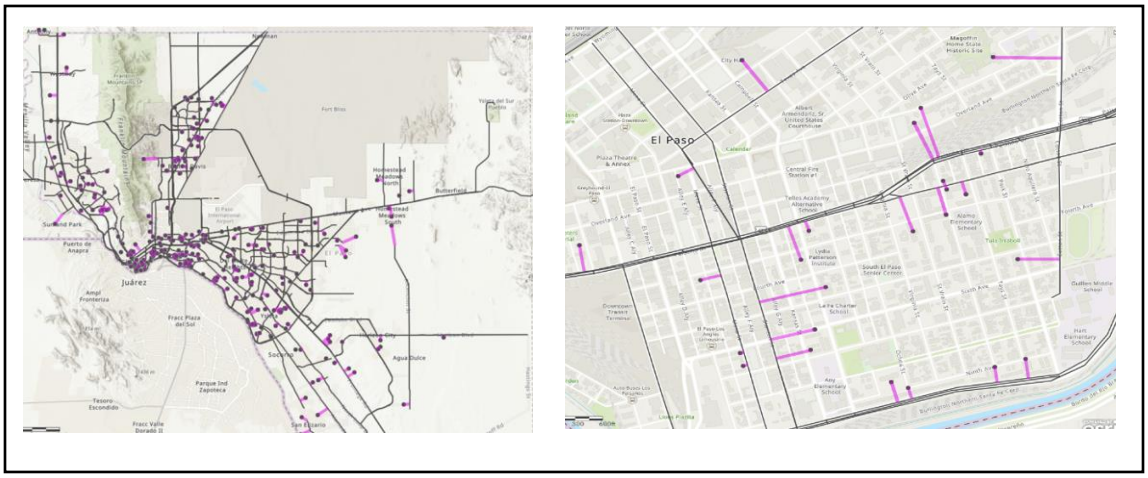


Figure 4.6: Distance to nearest major arterial road layer and layer zoom.

The use of impact zones within 500m and 1000m respective to each participant's residential address was a key component of the analysis. We used these zones to determine the

length of street road as well as the amount of vehicle miles traveled (VMT) using GIS layers from Census.gov and MPO respectively. Figures 4.7 and 4.8 illustrate the calculation of the VMT and the length of street roads within the 500m impact zone. In a similar way, we calculated the variables within a 1000m zone (not shown).

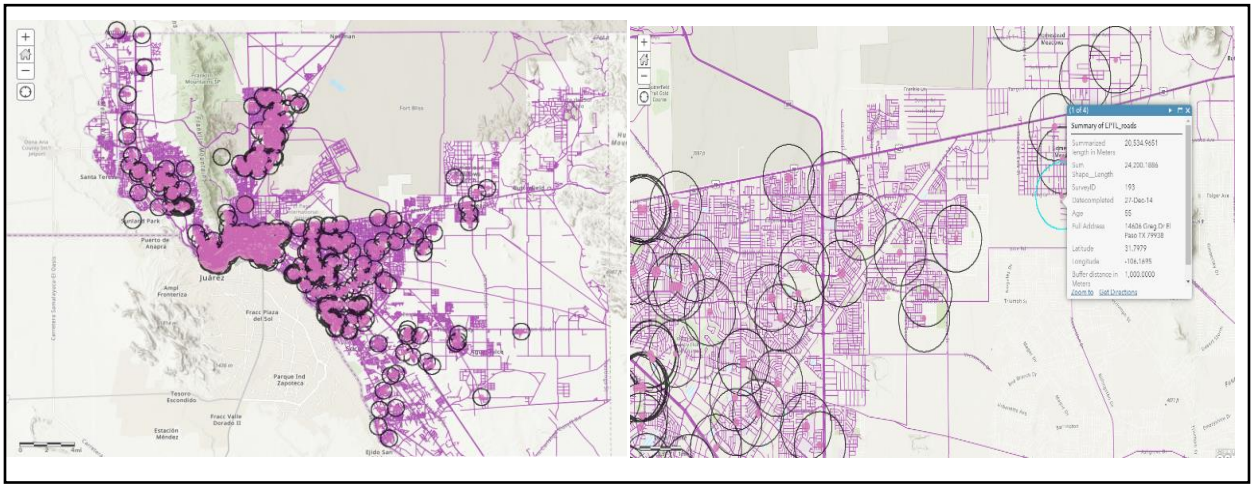


Figure 4.7: Summary of street length within 500m using the Census.gov layer and layer zoom.

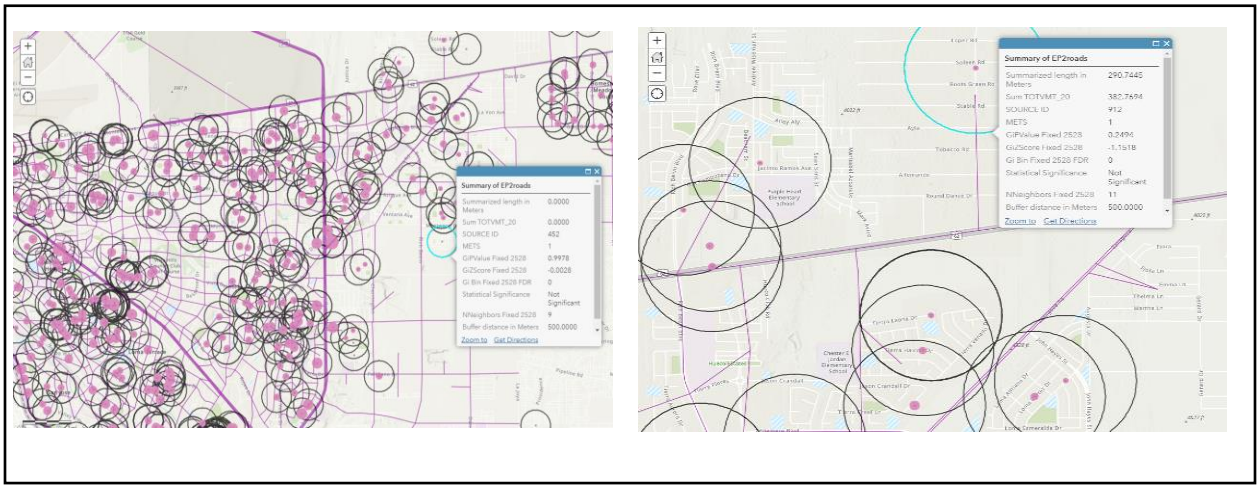


Figure 4.8: Summary of vehicle miles traveled (VMT) within 500m using the MPO layer and layer zoom.

4.3.2 TRAFFIC MEASUREMENTS

Table 4.1 summarizes the descriptive statistics of traffic-related measurements. Distance to the nearest major arterial road (Dist_nearest_Majart), street length within the 500m and 1000m impact zone (Street_Length_500m, Street_Length_1000m), and distance to the nearest port of entry (Distance_nearest_POE) all measured in kilometers. Due to the exponential decay of distance measurements, we also considered the inverse of distance to the nearest port of entry (InvDist_POE) and the inverse of the distance squared (InvSqDis_POE) as alternatives. Traffic counts were calculated from the average daily amount of vehicle miles traveled (VMT) within the 500m and 1000m zone of impact (Traffic_VMT_500m and Traffic_VMT_1000m) and converted to the unit in thousands.

In Figure 4.9, we show the scatterplot matrix for the pairs of traffic variables to explore the distribution of each variable and collinearity between variables. Based on the scatterplot, we decided on the impact zone with a 500m radius to be utilized in multivariate regression models.

Table 4.1: Descriptive statistics of traffic variables (unit: km, in thousands)

Variable	Min	Q1	Median	Mean	Q3	Max	SD	IQR
Distance_nearest_Majart	0.00	0.09	0.20	0.24	0.32	2.26	0.22	0.23
Street_Length_500m	3.04	8.46	10.96	11.48	14.16	24.85	3.97	5.70
Street_Length_1000m	14.09	34.32	44.42	43.60	50.36	81.15	12.76	16.04
Distance_nearest_POE	0.25	2.16	6.60	6.85	11.15	25.36	5.12	8.99
InvDist_POE	0.04	0.09	0.15	0.35	0.46	4.05	0.42	0.37
InvSqDist_POE	0.00	0.01	0.02	0.29	0.21	16.44	1.09	0.21
Traffic_VMT_500m	0.00	13.82	21.98	26.56	33.57	152.94	21.99	19.75
Traffic_VMT_1000m	0.31	61.73	110.53	126.45	164.79	412.10	85.01	103.06

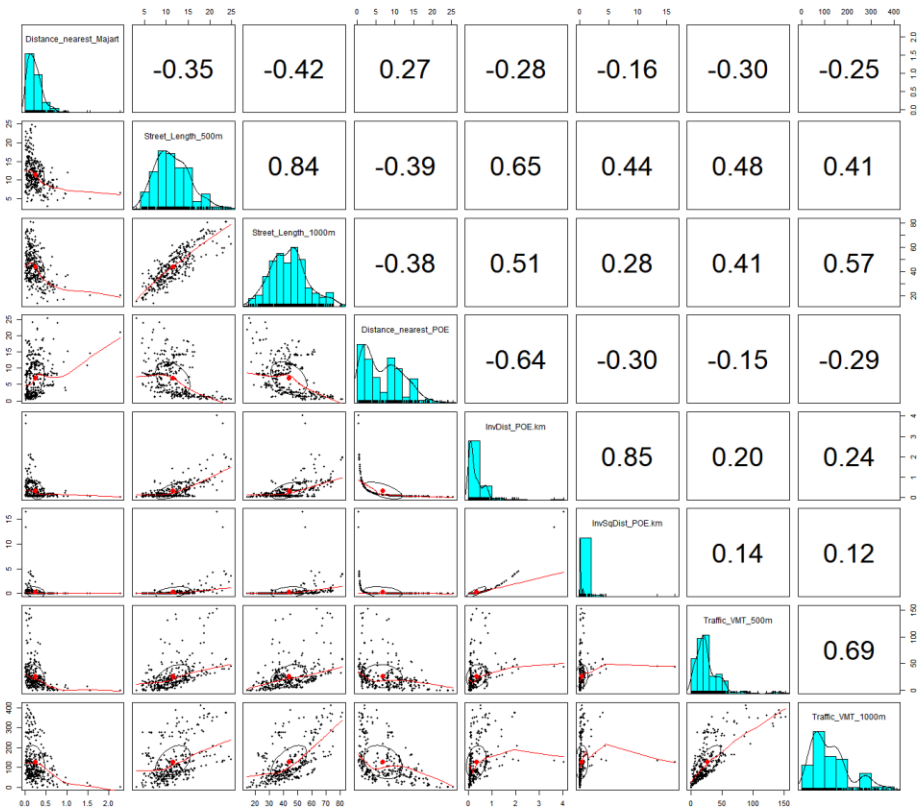


Figure 4.9: Scatterplot matrix of pairs of traffic variables (N=662).

4.3.3 Respiratory Associations

Descriptive statistics for exhaled nitric oxide (eNO) and spirometry measurements are summarized in Table 4.2 (N=662). The range for eNO was from 4.9 to 113.0 ppb with a mean of 21.4 ppb. The forced exhaled volume during the first second of expiration ranged from 0.76 to 4.86 L with a mean of 2.4 L, the forced vital capacity ranged from 0.82 to 6 L with a mean of 2.65 L, the peak expiratory flow (PEF) ranged from 1.59 to 11.48 L/min with a mean of 5.29 L/min.

Table 4.2: Descriptive statistics for eNO, FEV_1, FVC and PEF metrics.

Variable	Min	Q1	Median	Mean	Q3	Max	SD	IQR
eNO (ppb)	4.9	13.0	18.0	21.4	24.0	113.0	14.0	11.0
FEV_1 (L)	0.8	2.0	2.3	2.4	2.8	4.9	0.6	0.7
FVC (L)	0.8	2.2	2.6	2.7	3.0	6.0	0.7	0.8
PEF (L/min)	1.6	4.2	5.1	5.3	6.2	11.5	1.7	2.1
FEV_1 %Pred	18	83	92	96	101	360	31	18

Variable	Min	Q1	Median	Mean	Q3	Max	SD	IQR
FVC %Pred	16	73	82	85	91	266	24	18
PEF %Pred	14	81	95	96	110	267	27	29
FEV_1/FVC	0.6	0.9	0.9	0.9	1.0	1.0	0.1	0.1
FEV_1Best (L)	0.4	2.1	2.4	2.5	2.8	5.1	0.6	0.7
FVCBest (L)	0.5	2.3	2.7	2.8	3.2	6.0	0.8	0.9
PEFBest (L/min)	0.8	5.1	6.1	6.1	7.1	12.2	1.7	2.0

In the correlation analysis and univariate linear regression modeling, the length of the street and VMT have shown to be important traffic predictors to find relationships with lung function (see Table 4.3). Increases in length of the street within 500m radius zone were associated with decreased in lung function; $\beta_1 = -0.017$ for FEV_1 ($p = 0.016$), $\beta_1 = -0.017$ for FVC ($p = 0.045$), $\beta_1 = -0.049$ for PEF ($p = 0.011$), $\beta_1 = -0.046$ for PEF Best ($p = 0.021$). The finding was similar in the relationships between FEV_1/FVC/PEF/PEF Best and street length within a bigger zone of 1000m radius.

Traffic density within 500m impact zone were also negatively correlated with most of spirometry measures; $\beta_1 = -0.004$ for FEV_1 ($p = 0.005$), $\beta_1 = -0.004$ for FVC (p = 0.01), $\beta_1 = -0.008$ for PEF ($p = 0.03$), $\beta_1 = -0.003$ for FEV_1 Best ($p = 0.01$), $\beta_1 = -0.004$ for FVC Best ($p = 0.03$), $\beta_1 = -0.008$ for PEF Best ($p = 0.03$). The traffic amount within 1000m zone, in contrast, did not correlate with any respiratory measures. In addition to street length and VMT variables, distance to the nearest major road was another significant predictor showing a positive correlation with PEF ($p= 0.09$, $\beta_1 = 0.663$; $p = 0.045$).

For the LUR modeling, we applied multivariate linear regression including five traffic variables; distance to the nearest major arterial road, street length within 500m impact zone, distance to the nearest port of entry, inverse of the distance to the port of entry squared, and traffic

vehicle miles traveled within 500m zone. As with findings from the univariate regression, street length within 500m zone was a significant traffic variable in modeling of PEF ($\beta_1 = -0.056$, p=0.026) and PEF Best ($\beta_1 = -0.057$, p=value=0.025). Traffic volume (Traffic_VMT_500m) had no significant relationship FEV_1 Best.

Table 4.3: Correlation analysis between respiratory outcome and traffic variables (unit: km, in thousands).

Variable	Distance_ nearest_ Majart	Street_ Length_ 500m	Street_ Length_ 1000m	Distance_ nearest_ POE	InvDist_ POE	InvSqDist _POE	Traffic_ VMT_ 500m	Traffic_ VMT_ 1000m
Log.eNO (ppb)	0.008	0.036	0.003	-0.052	0.025	-0.007	0.034	0.013
FEV_1 (L)	0.071	**-0.108**	**-0.108**	0.021	0.010	0.055	**-0.125**	-0.048
FVC (L)	0.072	**-0.090**	**-0.091**	0.047	-0.014	0.032	**-0.116**	-0.051
PEF (L/min)	**0.090**	**-0.114**	**-0.141**	-0.006	-0.006	0.027	**-0.097**	-0.017
FEV_1 %Pred	0.031	0.010	0.009	**-0.105**	**0.134**	**0.118**	-0.054	0.001
bc.FEV_1 %Pred[1)]	0.040	0.010	0.006	**-0.115**	**0.118**	**0.095**	-0.052	0.006
FVC %Pred	0.041	0.007	0.013	-0.077	**0.101**	**0.093**	-0.066	-0.009
log.FVC %Pred	0.038	0.006	0.012	-0.082	**0.088**	0.069	-0.062	-0.002
PEF %Pred	0.047	-0.046	-0.063	**-0.108**	0.075	0.065	-0.060	0.013
sqrt.PEF %Pred	0.053	-0.050	-0.066	**-0.112**	0.072	0.063	-0.053	0.024
FEV_1/FVC	-0.001	-0.024	-0.033	-0.086	0.066	0.061	-0.019	-0.001
FEV_1 Best (L)	0.048	-0.079	-0.075	0.005	0.025	0.055	**-0.116**	-0.046
FVC Best (L)	0.051	-0.068	-0.060	0.039	-0.006	0.027	**-0.100**	-0.043
PEF Best (L/min)	0.064	**-0.103**	**-0.127**	-0.016	0.010	0.045	**-0.096**	-0.015

All significant correlations are expressed in bold.

[1)] Box-Cox Transformation: bc.FEV_1.%Pred = [(FEV_1.%Pred)^(-0.1)-1]/(-0.1)

Table 4.4: Summary and parameter estimates of multivariate regression models for respiratory outcomes.

Respiratory Variable	Traffic Variable	Estimate	Std. Error	t value	Pr(>|t|)
log.eNO	(Intercept)	2.913	0.023	128.220	0.000
	Distance_nearest_Majart	0.043	0.139	0.308	0.758
	Street_Length_500m	0.003	0.008	0.368	0.713
	Distance_nearest_POE	-0.005	0.005	-1.124	0.261
	InvSqDist_POE	-0.014	0.022	-0.645	0.519
	Traffic_VMT_500m	0.001	0.001	0.525	0.599
FEV_1	(Intercept)	2.396	0.028	86.197	0.000
	Distance_nearest_Majart	0.110	0.134	0.820	0.413
	Street_Length_500m	-0.017	0.009	-1.843	0.066
	Distance_nearest_POE	0.000	0.006	-0.014	0.989

Respiratory Variable	Traffic Variable	Estimate	Std. Error	t value	Pr(>|t|)
	InvSqDist_POE	0.059	0.026	2.302	**0.022**
	Traffic_VMT_500m	-0.002	0.001	-1.584	0.114
FVC	(Intercept)	2.642	0.033	80.533	0.000
	Distance_nearest_Majart	0.147	0.158	0.926	0.355
	Street_Length_500m	-0.012	0.011	-1.074	0.283
	Distance_nearest_POE	0.004	0.007	0.614	0.540
	InvSqDist_POE	0.050	0.030	1.665	0.097
	Traffic_VMT_500m	-0.003	0.002	-1.633	0.103
PEF	(Intercept)	5.279	0.075	69.972	0.000
	Distance_nearest_Majart	0.399	0.364	1.096	0.274
	Street_Length_500m	-0.056	0.025	-2.235	**0.026**
	Distance_nearest_POE	-0.015	0.016	-0.914	0.361
	InvSqDist_POE	0.113	0.070	1.623	0.105
	Traffic_VMT_500m	-0.003	0.004	-0.724	0.470
FEV_1%Pred	(Intercept)	96.021	1.370	70.071	0.000
	Distance_nearest_Majart	6.931	6.617	1.048	0.295
	Street_Length_500m	-0.250	0.452	-0.553	0.580
	Distance_nearest_POE	-0.611	0.290	-2.103	**0.036**
	InvSqDist_POE	2.997	1.265	2.369	**0.018**
	Traffic_VMT_500m	-0.076	0.072	-1.044	0.297
bc.FEV_1%Pred	(Intercept)	3.641	0.007	510.238	0.000
	Distance_nearest_Majart	0.044	0.034	1.285	0.199
	Street_Length_500m	-0.001	0.002	-0.394	0.693
	Distance_nearest_POE	-0.004	0.002	-2.476	**0.014**
	InvSqDist_POE	0.012	0.007	1.749	0.081
	Traffic_VMT_500m	0.000	0.000	-0.976	0.330
FVC%Pred	(Intercept)	84.733	1.093	77.489	0.000
	Distance_nearest_Majart	6.554	5.280	1.241	0.215
	Street_Length_500m	-0.034	0.361	-0.094	0.925
	Distance_nearest_POE	-0.362	0.232	-1.561	0.119
	InvSqDist_POE	1.869	1.009	1.852	0.065
	Traffic_VMT_500m	-0.078	0.058	-1.348	0.178
log.FVC%Pred	(Intercept)	4.407	0.011	391.189	0.000
	Distance_nearest_Majart	0.064	0.054	1.171	0.242
	Street_Length_500m	0.000	0.004	0.017	0.987
	Distance_nearest_POE	-0.004	0.002	-1.772	0.077
	InvSqDist_POE	0.013	0.010	1.243	0.214
	Traffic_VMT_500m	-0.001	0.001	-1.297	0.195
PEF%Pred	(Intercept)	95.949	1.204	79.693	0.000
	Distance_nearest_Majart	5.796	5.814	0.997	0.319
	Street_Length_500m	-0.652	0.397	-1.641	0.101

Respiratory Variable	Traffic Variable	Estimate	Std. Error	t value	Pr(>|t|)
	Distance_nearest_POE	-0.700	0.255	-2.743	**0.006**
	InvSqDist_POE	1.770	1.111	1.593	0.112
	Traffic_VMT_500m	-0.035	0.064	-0.557	0.578
sqrt.PEF%Pred	(Intercept)	9.702	0.060	160.660	0.000
	Distance_nearest_Majart	0.338	0.292	1.160	0.247
	Street_Length_500m	-0.036	0.020	-1.812	0.071
	Distance_nearest_POE	-0.037	0.013	-2.915	**0.004**
	InvSqDist_POE	0.088	0.056	1.575	0.116
	Traffic_VMT_500m	-0.001	0.003	-0.303	0.762
FEV_1/FVC	(Intercept)	0.915	0.003	290.795	0.000
	Distance_nearest_Majart	-0.002	0.015	-0.137	0.891
	Street_Length_500m	-0.002	0.001	-1.518	0.130
	Distance_nearest_POE	-0.001	0.001	-1.966	**0.050**
	InvSqDist_POE	0.004	0.003	1.402	0.162
	Traffic_VMT_500m	0.000	0.000	0.035	0.972
FEV_1Best	(Intercept)	2.505	0.029	87.041	0.000
	Distance_nearest_Majart	0.064	0.139	0.461	0.645
	Street_Length_500m	-0.012	0.009	-1.269	0.205
	Distance_nearest_POE	-0.001	0.006	-0.166	0.868
	InvSqDist_POE	0.052	0.027	1.964	0.050
	Traffic_VMT_500m	-0.003	0.002	-1.765	0.078
FVCBest	(Intercept)	2.762	0.035	79.642	0.000
	Distance_nearest_Majart	0.100	0.167	0.595	0.552
	Street_Length_500m	-0.008	0.011	-0.680	0.497
	Distance_nearest_POE	0.004	0.007	0.570	0.569
	InvSqDist_POE	0.041	0.032	1.289	0.198
	Traffic_VMT_500m	-0.003	0.002	-1.549	0.122
PEFBest	(Intercept)	6.099	0.077	78.796	0.000
	Distance_nearest_Majart	0.220	0.374	0.588	0.557
	Street_Length_500m	-0.057	0.026	-2.241	**0.025**
	Distance_nearest_POE	-0.016	0.016	-0.945	0.345
	InvSqDist_POE	0.137	0.071	1.920	0.055
	Traffic_VMT_500m	-0.004	0.004	-0.868	0.386

All significant predictors and corresponding *p* are expressed in bold.

4.3.4 CARDIOVASCULAR ASSOCIATIONS

Table 4.5 shows the demographic information of the full dataset from the epidemiological which contains data from participants from the last five and a half years (September 2014 to

January 2020, N=4,959). The participants' ages ranged 18-94 years (mean 45.5 years). Most of the participants were female (79.5%) and Hispanic (95.5%), and 54.8% of participants were found to have overweight (23.9%) or obesity (30.9%), whereas 13.7% of participants were found to have a healthy BMI.

Table 4.5: Descriptive of the demographic information for subjects (N=4,959).

Variable	Category	Frequency	%*
Sex	Female	3941	79.5
	Male	954	19.2
Education	Middle School	896	18.1
	High School graduate	832	16.8
	High School, no diploma	723	14.6
	Elementary School	680	13.7
	Some college, not completed	636	12.8
	Bachelor's degree	532	10.7
	Associate degree	319	6.4
	Masters, Doctoral, or Professional degree	119	2.4
	Never attended or Kindergarten only	72	1.5
Language	Spanish	3408	68.7
	Both	1050	21.2
	English	396	8.0
	Other	8	0.2
Employed	Homemaker	1606	32.4
	Employed-Part time	1025	20.7
	Employed-Full time	795	16.0
	Student	313	6.3
	Retired	290	5.8
	Not employed for more than 1 year	232	4.7
	Not employed for less than 1 year	228	4.6
	Self-Employed	197	4.0
	Unable to work	126	2.5
	Seasonal worker	17	0.3
Income	$0 - $19,999	3532	71.2
	$20,000 - $29,999	603	12.2
	$30,000 - $39,999	237	4.8
	$50,000 - $69,999	142	2.9
	$40,000 - $49,999	133	2.7
	$70,000 - $99,999	62	1.3
	$100,000 or more	51	1.0
Marital Status			
	Married	2248	45.3
	Never Married	905	18.2
	Divorced	453	9.1
	Separated	407	8.2
	Single/Never Married	313	6.3
	Widowed	313	6.3

	A member of an unmarried couple	148	3.0
	Civil Union	70	1.4
Ethnicity	Hispanic	4738	95.5
	Non-Hispanic	79	1.6
	White	41	0.8
	Black or African American	9	0.2
	Asian	4	0.1
	American Indian or Alaska Native	3	0.1
	Native Hawaiian	2	0.0
	Other	1	0.0
Race	White	3302	66.6
	Black or African American	40	0.8
	American Indian or Alaska Native	16	0.3
	Asian	14	0.3
	Native Hawaiian	3	0.1
Health	Good	2072	41.8
	Fair	1457	29.4
	Very Good	575	11.6
	Poor	400	8.1
	Excellent	339	6.8
Obesity	Obesity	1534	30.9
	Overweight	1185	23.9
	Healthy	681	13.7

*Distribution might not add to 100% due to participants not answering all the questions

Table 4.6 summarizes the descriptive statistics of traffic-related measurements. In Figure 4.10, the scatterplot matrix presented for the pairs of traffic variables explored the distribution of each variable and collinearity between variables. Based on the scatterplot, we decided the impact zone with a 500m radius to be used in the multivariate regression models.

Table 4.6: Descriptive statistics of traffic variables (N=4,959; unit: km, in thousands)

Variable	Min	Q1	Median	Mean	Q3	Max	SD	IQR
Distance_nearest_Majart	0.00	0.10	0.22	0.33	0.43	3.35	0.34	0.32
Street_Length_500m	0.28	7.84	10.23	10.73	13.18	25.51	4.23	5.34
Street_Length_1000m	0.20	28.88	36.95	39.20	48.29	83.04	15.40	19.41
Distance_nearest_POE	0.16	3.39	8.62	9.48	13.81	37.58	7.00	10.42
InvDist_POE.km	0.03	0.07	0.12	0.28	0.29	6.15	0.46	0.22
InvSqDist_POE.km	0.00	0.01	0.01	0.29	0.09	37.78	1.70	0.08
Traffic_VMT_500m	0.00	6.92	15.49	23.34	27.69	178.54	27.47	20.77
Traffic_VMT_1000m	0.17	33.96	65.65	102.38	136.48	437.44	100.86	102.52

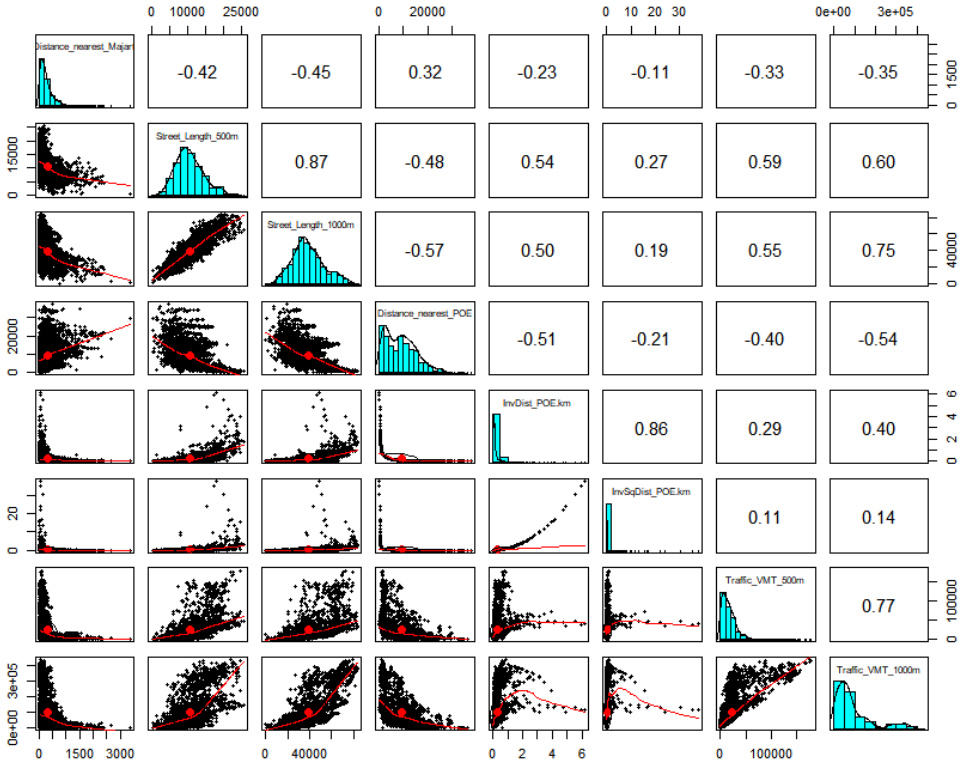


Figure 4.10: Scatterplot matrix of pairs of traffic variables (N=4,959).

Descriptive statistics for cardiovascular risk measurements (metabolic syndrome) using the full five-year dataset are summarized in Table 4.7 (N=4,959). The waist circumference ranged from 56 to 154 cm with a mean of 95 (±14.6) cm. Blood pressure (SBP/DBP) measurements ranged from 71/35 to 232/151 mmHg with a mean of 123/76 (±19/11) mmHg. Triglyceride (TG) levels ranged from 45 to 650 mg/dL with a mean level of 186 (±108) mg/dL. HDL-cholesterol ranged from 15 to 100 mg/dL with a mean of 48.6 (±15) mg/dL. Glucose levels ranged from 50 to 500 mg/dL with a mean value of 113 (±49) mg/dL. Other variables of interest of cardiovascular risk which are not components of metabolic syndrome but could potentially offer more information related to cardiovascular risk included BMI, pulse pressure (a measure of difference between systolic and diastolic blood pressure [PBP]), total cholesterol (TC), TC/HDL ratio and LDL-cholesterol.

Table 4.7: Descriptive statistics for cardiovascular risk factors (N=4,959).

Variable	Min	Q1	Median	Mean	Q3	Max	SD	IQR
BMI (kg/m^2)	15.3	25.7	29.1	29.9	33.2	67.7	6.1	7.5
Waist (cm)	56.0	84.0	93.0	94.0	103.0	154.0	14.6	19.0
SBP (mmHg)	71	110	121	123	134	232	19	24
DBP (mmHg)	35	69	75	76	83	151	11	14
PBP (mmHg)	3	37	45	47	54	133	14	17
TC (mg/dL)	100.0	160.0	186.0	189.6	214.0	500.0	41.9	54.0
TG (mg/dL)	44.9	110.0	164.0	186.6	233.0	650.1	108.2	123.0
HDL (mg/dL)	14.9	38.0	47.0	48.6	57.0	100.1	15.3	19.0
LDL (mg/dL)	14.0	81.0	102.0	105.3	127.0	314.0	34.7	46.0
TC/HDL	1.4	3.1	3.9	4.3	5.0	15.5	1.6	1.9
Glucose (mg/dL)	49.9	90.0	99.0	113.8	116.0	500.0	49.0	26.0

As shown in Table 4.8, linear relationships were found between traffic variables and a few metabolic syndrome risk factors. Waist measurement significantly correlated with the street length within 500m area ($p = 0.045$, $\beta_1 = 0.155$), the inverse of the distance to the nearest POE ($p = 0.046$, $\beta_1 = 1.426$), and inverse squared distance ($p = 0.033$, $\beta_1 = 0.270$). Blood pressure monitoring showed that traffic variables were more associated with pulse pressure (PBP), rather than systolic blood pressure (SBP) or diastolic blood pressure (DBP). The PBP increases related to increase in street length within 500 and 1000m zones ($p = 0.035$, $\beta_1 = 0.115$; $p = 0.050$, $\beta_1 = 0.046$, respectively), decrease in the distance to the nearest POE ($p = -0.059$, $\beta_1 = -0.118$), rise in the inverse of the distance to the POE ($p = 0.040$, $\beta_1 = 1.227$), and increase in traffic amount within 500 and 1000m ($p = 0.051$, $\beta_1 = 0.026$; $p = 0.055$, $\beta_1 = 0.008$, respectively). Both log-transformed and Box-Cox transformed glucose levels also showed similar correlation results.

Log transformed triglycerides were significantly associated with the street length within the 500m zone ($\beta_1 = 0.005$, p=0.036). The fasting glucose showed significant relationships with the POE-related distance variables; negative association with distance to the nearest POE ($p = -0.036$, $\beta_1 = -0.257$), positive associations with the inverse of the distance ($p = 0.064$, $\beta_1 = 6.723$) and the inverse of the distance squared ($p = 0.051$, $\beta_1 = 1.380$).

Table 4.9 summarizes the frequency of the five metabolic syndrome risk factors (binary outcomes) and the MetS classification. The univariate associations between the binary classification of MetS risk factors and traffic variables were examined using logistic regression modeling (see Table 4.10). The risk of low HDL-cholesterol was found to be higher as the street length within the impact zone (odds ratio=1.023, p = 0.006 for the 500m zone) and the inverse distance to the POE increases (odds ratio=1.212, p = 0.012). The effect of street length is more substantial for the smaller region, i.e., 500m zone, rather than 1000m zone. The street length within the 500m impact zone was also an important factor correlated with a higher risk of metabolic syndrome (odds ratio=1.020, 95% C.I.=[1.003, 1.037]). Increase of the inverse distance to the nearest POE, implying a decrease in the distance to POE, was related to a higher risk in metabolic syndrome (β_1 = 0.192, p = 0.012).

Five traffic variables, i.e., distance to the nearest major arterial road, street length within 500m impact zone, distance to the nearest port of entry, the inverse of the distance to the port of entry squared, and traffic vehicle miles traveled within 500m zone, were included in land-use regression modeling for multivariate analyses of the 5-year data. As shown in Table 4.11, the most significant predictor in the LUR models was the total length of the street within a 500m radius. The increase in the street length associated with increasing the MetS factors, in particular, BMI (β_1 = 0.110, p = 0.002), waistline (β_1 = 0.294, p < 0.001), log-transformed triglycerides (β_1 = 0.007, p = 0.025), and Box-Cox transformed fasting glucose (β_1 = 2.218e-07, p = 0.049). However, the fasting glucose and log-transformed glucose showed positive relationships with inverse squared distance to the port of entry (β_1 = 1.156, p = 0.015; β_1 = 0.007, p = 0.023, respectively). In the modeling of PBP, the increase in PBP was associated with the increase in amount of traffic within a 500m radius (β_1 = 0.021, p = 0.048) and the proximity to the nearest port of entry (β_1 = −0.095,

p = 0.013). We also found the effect of traffic volume within the 500m zone on the DBP measurement for the participants whose DBP is less than 85mmHg.

Logistic regression models, including the five traffic predictors, also showed the significance of the length of the street within the 500m impact zone (see Table 4.12). As the total length of the street increases, the risks of a high waist (β_1 = 0.034, p = 0.002), high triglycerides (β_1 = 0.024, p = 0.034), and low HDL-cholesterol (β_1 = 0.032, p = 0.004) were observed. The significance of the street length variable in predicting three metabolic syndrome risk components may have influenced the prediction of metabolic syndrome classification. The increasing likelihood of metabolic syndrome was related to the increased street length within the 500m impact zone (β_1 = 0.038, p = 0.001, odds ratio = 1.039 [1.016, 1.062]).

Table 4.8: Correlation analysis with traffic variables (N=4,959, unit: km, in thousands)

Health Measures	Distance_ nearest_ Majart	Street_ Length_ 500m	Street_ Length_ 1000m	Distance_ nearest_ POE	InvDist_ POE	InvSqDist _POE	Traffic_ VMT_ 500m	Traffic_ VMT_ 1000m
BMI (kg/m^2)	0.022	0.005	-0.010	**0.039**	-0.002	0.000	**-0.043**	**-0.042**
Waist (cm)	-0.001	**0.045**	0.031	-0.002	**0.046**	**0.033**	0.002	0.006
Female	-0.004	**0.059**	**0.046**	-0.009	**0.054**	0.024	0.013	0.017
Male	0.045	-0.018	-0.054	0.065	-0.001	0.028	-0.050	-0.062
SBP (mmHg)	0.012	0.008	0.018	**-0.031**	0.018	0.013	0.028	0.021
SBP < 130	**0.041**	-0.021	-0.015	0.029	-0.011	0.012	0.006	-0.007
SBP >= 130	0.031	-0.001	-0.001	-0.022	0.033	0.031	0.041	0.020
DBP (mmHg)	0.022	-0.029	**-0.032**	0.020	-0.020	-0.006	-0.016	**-0.033**
DBP < 85	0.021	-0.025	-0.025	0.027	**-0.040**	-0.022	0.010	-0.020
DBP >= 85	-0.033	0.030	0.009	0.008	0.013	-0.010	-0.004	-0.018
PBP (mmHg)	-0.002	**0.035**	**0.050**	**-0.059**	**0.040**	0.023	**0.051**	**0.055**
TC (mg/dL)	**0.036**	-0.026	**-0.040**	0.022	-0.015	0.002	-0.012	-0.031
TG (mg/dL)	0.012	0.028	0.006	-0.010	0.023	0.017	0.014	-0.019
log.TG	0.013	**0.035**	0.009	-0.007	0.025	0.019	0.011	-0.027
HDL (mg/dL)	0.016	**-0.046**	**-0.046**	0.001	**-0.041**	-0.027	-0.025	-0.021
LDL (mg/dL)	0.023	-0.026	-0.032	0.026	-0.024	-0.007	-0.011	-0.020
TC/HDL	0.013	0.011	0.000	0.012	0.013	0.018	0.021	-0.003
log.TC/HDL	0.011	0.019	0.009	0.010	0.020	0.024	0.021	-0.003
bc.TC/HDL[1]	0.010	0.021	0.013	0.010	0.022	0.025	0.020	-0.004
Glucose (mg/dL)	0.003	0.032	0.021	**-0.036**	**0.064**	**0.051**	0.018	0.006

Health Measures	Distance_ nearest_ Majart	Street_ Length_ 500m	Street_ Length_ 1000m	Distance_ nearest_ POE	InvDist_ POE	InvSqDist _POE	Traffic_ VMT_ 500m	Traffic_ VMT_ 1000m
log.Glucose	0.001	**0.037**	0.023	**-0.035**	**0.066**	**0.049**	0.017	0.004
bc.Glucose[2]	0.001	**0.043**	0.025	-0.031	**0.061**	**0.040**	0.016	0.001

All significant correlations are expressed in bold.

[1)] Box-Cox Transformation: bc.TC/HDL = [(TC/HDL)^(-0.5)-1]/(-0.5).

[2)] Box-Cox Transformation: bc.Glucose = [Glucose^(-2)-1]/(-2).

Table 4.9: Summary of metabolic syndrome risk factors (N=4,959).

Variables		Frequency	%
High Waist	Yes	2307	46.5
	No	1603	32.3
	NA	1049	21.2
High BP	No	2561	51.6
	Yes	1622	32.7
	NA	776	15.6
High TG	Yes	2047	41.3
	No	1588	32.0
	NA	1324	26.7
Low HDL	Yes	1835	37.0
	No	1750	35.3
	NA	1374	27.7
High Glucose	No	1827	36.8
	Yes	1795	36.2
	NA	1337	27.0
Metabolic Syndrome	Yes	1851	37.3
	No	1626	32.8
	NA	1482	29.9

NA: Not available due to not being measured or being able to determine risk factor

Table 4.10: Univariate associations between metabolic syndrome (MetS) risk factors and MetS classification and traffic variables (N=4,959).

Health Variable	Traffic Variable	Estimate	Std. Error	z value	Pr(>|z|)	Odds Ratio	Lower 95% CI	Upper 95% CI
High Waist	Distance_nearest_Majart	-0.012	0.095	-0.129	0.898	0.988	0.820	1.192
	Street_Length_500m	0.010	0.008	1.276	0.202	1.010	0.995	1.026
	Street_Length_1000m	0.000	0.002	-0.065	0.948	1.000	0.996	1.004
	Distance_nearest_POE	0.009	0.005	1.834	0.067	1.009	0.999	1.018
	InvDist_POE.km	0.014	0.070	0.204	0.838	1.014	0.885	1.166
	InvSqDist_POE.km	0.000	0.019	-0.024	0.981	1.000	0.964	1.038

	Traffic_VMT_500m	0.000	0.001	-0.319	0.750	1.000	0.997	1.002
	Traffic_VMT_1000m	0.000	0.000	-1.370	0.171	1.000	0.999	1.000
High BP	Distance_nearest_Majart	0.005	0.093	0.051	0.959	1.005	0.836	1.205
	Street_Length_500m	0.002	0.008	0.225	0.822	1.002	0.987	1.017
	Street_Length_1000m	0.002	0.002	0.733	0.463	1.002	0.997	1.006
	Distance_nearest_POE	-0.008	0.005	-1.706	0.088	0.992	0.983	1.001
	InvDist_POE.km	0.038	0.068	0.550	0.583	1.038	0.907	1.186
	InvSqDist_POE.km	-0.001	0.019	-0.027	0.978	0.999	0.962	1.036
	Traffic_VMT_500m	0.000	0.001	0.380	0.704	1.000	0.998	1.003
	Traffic_VMT_1000m	0.000	0.000	0.341	0.733	1.000	0.999	1.001
High TG	Distance_nearest_Majart	0.090	0.101	0.894	0.371	1.094	0.899	1.336
	Street_Length_500m	0.011	0.008	1.297	0.195	1.011	0.995	1.027
	Street_Length_1000m	0.001	0.002	0.621	0.534	1.001	0.997	1.006
	Distance_nearest_POE	0.002	0.005	0.467	0.640	1.002	0.993	1.012
	InvDist_POE.km	0.034	0.073	0.468	0.640	1.035	0.898	1.197
	InvSqDist_POE.km	0.004	0.019	0.216	0.829	1.004	0.968	1.044
	Traffic_VMT_500m	0.000	0.001	-0.104	0.917	1.000	0.997	1.002
	Traffic_VMT_1000m	-0.001	0.000	-1.612	0.107	0.999	0.999	1.000
Low HDL	Distance_nearest_Majart	0.002	0.100	0.022	0.982	1.002	0.823	1.221
	Street_Length_500m	0.023	0.008	2.756	**0.006**	1.023	1.007	1.039
	Street_Length_1000m	0.006	0.002	2.696	**0.007**	1.006	1.002	1.011
	Distance_nearest_POE	0.001	0.005	0.244	0.808	1.001	0.992	1.011
	InvDist_POE.km	0.192	0.076	2.523	**0.012**	1.212	1.047	1.413
	InvSqDist_POE.km	0.037	0.021	1.787	0.074	1.038	0.999	1.085
	Traffic_VMT_500m	0.002	0.001	1.248	0.212	1.002	0.999	1.004
	Traffic_VMT_1000m	0.000	0.000	1.152	0.249	1.000	1.000	1.001
High Glucose	Distance_nearest_Majart	0.033	0.100	0.329	0.742	1.033	0.849	1.257
	Street_Length_500m	0.004	0.008	0.478	0.633	1.004	0.988	1.020
	Street_Length_1000m	-0.001	0.002	-0.622	0.534	0.999	0.994	1.003
	Distance_nearest_POE	0.004	0.005	0.866	0.387	1.004	0.995	1.014
	InvDist_POE.km	0.136	0.074	1.843	0.065	1.145	0.993	1.327
	InvSqDist_POE.km	0.025	0.020	1.285	0.199	1.025	0.988	1.068
	Traffic_VMT_500m	0.000	0.001	-0.247	0.805	1.000	0.997	1.002
	Traffic_VMT_1000m	-0.001	0.000	-1.586	0.113	0.999	0.999	1.000
Metabolic Syndrome	Distance_nearest_Majart	-0.047	0.102	-0.460	0.645	0.954	0.781	1.166
	Street_Length_500m	0.020	0.008	2.368	**0.018**	1.020	1.003	1.037
	Street_Length_1000m	0.004	0.002	1.598	0.110	1.004	0.999	1.008
	Distance_nearest_POE	0.002	0.005	0.355	0.722	1.002	0.992	1.012
	InvDist_POE.km	0.150	0.076	1.964	**0.050**	1.162	1.003	1.355
	InvSqDist_POE.km	0.016	0.019	0.802	0.423	1.016	0.979	1.057
	Traffic_VMT_500m	0.000	0.001	-0.052	0.959	1.000	0.997	1.002
	Traffic_VMT_1000m	0.000	0.000	-0.802	0.422	1.000	0.999	1.000

All significant correlations are expressed in bold.

Table 4.11: Summary and parameter estimates of multivariate regression models for continuous MetS risk factors (N=4,959).

Health Variable	Traffic Variables*	Estimate	Std. Error	t value	Pr(>|t|)
BMI	(Intercept)	29.961	0.108	277.530	0.000
(kg/m^2)	Distance_nearest_Majart	0.562	0.394	1.426	0.154
	Street_Length_500m	0.110	0.035	3.122	**0.002**
	Distance_nearest_POE	0.044	0.018	2.382	**0.017**
	InvSqDist_POE.km	0.002	0.061	0.040	0.968
	Traffic_VMT_500m	-0.013	0.005	-2.647	**0.008**
Waist	(Intercept)	93.875	0.241	389.725	0.000
(cm)	Distance_nearest_Majart	0.462	0.878	0.526	0.599
	Street_Length_500m	0.294	0.077	3.793	**0.000**
	Distance_nearest_POE	0.054	0.041	1.329	0.184
	InvSqDist_POE.km	0.176	0.140	1.255	0.210
	Traffic_VMT_500m	-0.020	0.011	-1.801	0.072
Waist (cm)	(Intercept)	92.889	0.267	347.822	0.000
(Female, N=3941)	Distance_nearest_Majart	0.322	0.953	0.338	0.736
	Street_Length_500m	0.351	0.086	4.097	**0.000**
	Distance_nearest_POE	0.049	0.045	1.099	0.272
	InvSqDist_POE.km	0.053	0.196	0.269	0.788
	Traffic_VMT_500m	-0.019	0.012	-1.548	0.122
Waist (cm)	(Intercept)	97.755	0.536	182.392	0.000
(Male, N=954)	Distance_nearest_Majart	2.616	2.139	1.223	0.222
	Street_Length_500m	0.190	0.178	1.065	0.287
	Distance_nearest_POE	0.173	0.098	1.762	0.079
	InvSqDist_POE.km	0.231	0.202	1.145	0.253
	Traffic_VMT_500m	-0.022	0.025	-0.886	0.376
SBP (mmHg)	(Intercept)	123.202	0.308	399.361	0.000
	Distance_nearest_Majart	1.804	1.122	1.608	0.108
	Street_Length_500m	-0.054	0.099	-0.542	0.588
	Distance_nearest_POE	-0.084	0.052	-1.610	0.107
	InvSqDist_POE.km	0.110	0.183	0.600	0.549
	Traffic_VMT_500m	0.021	0.014	1.518	0.129
SBP (mmHg)	(Intercept)	112.606	0.207	542.694	0.000
(<130, N=2801)	**Distance_nearest_Majart**	1.764	0.754	2.340	**0.019**
	Street_Length_500m	-0.027	0.066	-0.402	0.688
	Distance_nearest_POE	0.044	0.034	1.285	0.199
	InvSqDist_POE.km	0.131	0.119	1.094	0.274
	Traffic_VMT_500m	0.015	0.010	1.509	0.131
SBP (mmHg)	(Intercept)	144.678	0.396	365.337	0.000
(>=130, N=1382)	Distance_nearest_Majart	1.123	1.449	0.775	0.438
	Street_Length_500m	-0.143	0.130	-1.102	0.271

Health Variable	Traffic Variables*	Estimate	Std. Error	t value	Pr(>|t|)
	Distance_nearest_POE	-0.069	0.071	-0.979	0.328
	InvSqDist_POE.km	0.294	0.253	1.162	0.246
	Traffic_VMT_500m	0.029	0.018	1.648	0.100
DBP (mmHg)	(Intercept)	76.386	0.183	417.888	0.000
	Distance_nearest_Majart	0.445	0.665	0.669	0.503
	Street_Length_500m	-0.053	0.059	-0.897	0.370
	Distance_nearest_POE	0.011	0.031	0.360	0.719
	InvSqDist_POE.km	0.013	0.108	0.120	0.905
	Traffic_VMT_500m	0.001	0.008	0.108	0.914
DBP (mmHg)	(Intercept)	71.840	0.140	514.876	0.000
(<85, N=3246)	Distance_nearest_Majart	0.644	0.516	1.249	0.212
	Street_Length_500m	-0.046	0.045	-1.019	0.308
	Distance_nearest_POE	0.030	0.024	1.284	0.199
	InvSqDist_POE.km	-0.056	0.085	-0.658	0.511
	Traffic_VMT_500m	0.013	0.006	1.972	**0.049**
DBP (mmHg)	(Intercept)	92.209	0.247	373.154	0.000
(>=85, N=937)	Distance_nearest_Majart	-1.092	0.856	-1.275	0.203
	Street_Length_500m	0.087	0.080	1.077	0.282
	Distance_nearest_POE	0.036	0.042	0.848	0.397
	InvSqDist_POE.km	-0.071	0.135	-0.529	0.597
	Traffic_VMT_500m	-0.008	0.012	-0.723	0.470
PBP (mmHg)	(Intercept)	46.816	0.227	206.456	0.000
	Distance_nearest_Majart	1.359	0.825	1.648	0.099
	Street_Length_500m	-0.001	0.073	-0.014	0.989
	Distance_nearest_POE	-0.095	0.038	-2.481	**0.013**
	InvSqDist_POE.km	0.097	0.135	0.720	0.472
	Traffic_VMT_500m	0.021	0.010	1.978	**0.048**
TC (mg/dL)	(Intercept)	189.327	0.720	263.063	0.000
	Distance_nearest_Majart	2.711	2.649	1.023	0.306
	Street_Length_500m	-0.195	0.233	-0.837	0.403
	Distance_nearest_POE	0.038	0.123	0.308	0.758
	InvSqDist_POE.km	0.236	0.409	0.578	0.564
	Traffic_VMT_500m	0.012	0.033	0.369	0.712
TG (mg/dL)	(Intercept)	187.067	1.867	100.215	0.000
	Distance_nearest_Majart	12.724	6.868	1.853	0.064
	Street_Length_500m	0.901	0.603	1.494	0.135
	Distance_nearest_POE	0.094	0.319	0.295	0.768
	InvSqDist_POE.km	0.730	1.055	0.692	0.489
	Traffic_VMT_500m	0.026	0.085	0.312	0.755
log.TG (mg/dL)	(Intercept)	5.078	0.010	528.973	0.000
	Distance_nearest_Majart	0.068	0.035	1.917	0.055
	Street_Length_500m	0.007	0.003	2.236	**0.025**

Health Variable	Traffic Variables*	Estimate	Std. Error	t value	Pr(>|t|)
	Distance_nearest_POE	0.001	0.002	0.636	0.525
	InvSqDist_POE.km	0.004	0.005	0.734	0.463
	Traffic_VMT_500m	0.000	0.000	-0.151	0.880
HDL (mg/dL)	(Intercept)	48.563	0.263	184.810	0.000
	Distance_nearest_Majart	-0.220	0.971	-0.227	0.821
	Street_Length_500m	-0.222	0.085	-2.616	**0.009**
	Distance_nearest_POE	-0.059	0.045	-1.318	0.188
	InvSqDist_POE.km	-0.151	0.148	-1.024	0.306
	Traffic_VMT_500m	0.000	0.012	0.002	0.998
LDL (mg/dL)	(Intercept)	104.895	0.624	168.048	0.000
	Distance_nearest_Majart	-0.419	2.311	-0.181	0.856
	Street_Length_500m	-0.130	0.202	-0.645	0.519
	Distance_nearest_POE	0.037	0.106	0.346	0.729
	InvSqDist_POE.km	-0.025	0.349	-0.073	0.942
	Traffic_VMT_500m	0.000	0.029	0.015	0.988
TC/HDL	(Intercept)	4.247	0.029	147.650	0.000
	Distance_nearest_Majart	0.077	0.106	0.727	0.467
	Street_Length_500m	0.005	0.009	0.492	0.622
	Distance_nearest_POE	0.005	0.005	1.046	0.296
	InvSqDist_POE.km	0.017	0.016	1.065	0.287
	Traffic_VMT_500m	0.002	0.001	1.198	0.231
log.TC/HDL	(Intercept)	1.382	0.006	224.893	0.000
	Distance_nearest_Majart	0.013	0.023	0.586	0.558
	Street_Length_500m	0.002	0.002	0.950	0.342
	Distance_nearest_POE	0.001	0.001	1.143	0.253
	InvSqDist_POE.km	0.004	0.003	1.284	0.199
	Traffic_VMT_500m	0.000	0.000	0.907	0.364
bc.TC/HDL	(Intercept)	0.982	0.003	320.741	0.000
	Distance_nearest_Majart	0.006	0.011	0.530	0.596
	Street_Length_500m	0.001	0.001	1.148	0.251
	Distance_nearest_POE	0.001	0.001	1.185	0.236
	InvSqDist_POE.km	0.002	0.002	1.330	0.184
	Traffic_VMT_500m	0.000	0.000	0.751	0.453
Glucose (mg/dL)	(Intercept)	113.656	0.841	135.214	0.000
	Distance_nearest_Majart	3.365	3.096	1.087	0.277
	Street_Length_500m	0.261	0.271	0.962	0.336
	Distance_nearest_POE	-0.200	0.144	-1.395	0.163
	InvSqDist_POE.km	1.156	0.474	2.438	**0.015**
	Traffic_VMT_500m	-0.005	0.038	-0.143	0.886
log.Glucose (mg/dL)	(Intercept)	4.678	0.005	908.585	0.000
	Distance_nearest_Majart	0.019	0.019	1.027	0.305
	Street_Length_500m	0.002	0.002	1.395	0.163

Health Variable	Traffic Variables*	Estimate	Std. Error	t value	Pr(>|t|)
	Distance_nearest_POE	-0.001	0.001	-1.161	0.246
	InvSqDist_POE.km	0.007	0.003	2.279	**0.023**
	Traffic_VMT_500m	0.000	0.000	-0.368	0.713
bc.Glucose (mg/dL)	(Intercept)	0.500	0.000	1436139	0.000
	Distance_nearest_Majart	0.000	0.000	1.086	0.278
	Street_Length_500m	0.000	0.000	1.973	**0.049**
	Distance_nearest_POE	0.000	0.000	-0.839	0.401
	InvSqDist_POE.km	0.000	0.000	1.641	0.101
	Traffic_VMT_500m	0.000	0.000	-0.596	0.551

All significant predictors and corresponding *p* are expressed in bold.
*Traffic variable units: km, in thousands

Table 4.12: Summary and parameter estimates of multivariate logistic regression model for binary MetS factors (N=4,959).

Health Variables	Traffic Variables*	Estimate	Std. Error	z value	Pr(>|z|)	Odds Ratio	Lower 95% CI	Upper 95% CI
High Waist	(Intercept)	0.359	0.034	10.612	0.000	1.431	1.340	1.529
	Distance_nearest_Majart	-0.037	0.123	-0.302	0.763	0.964	0.758	1.227
	Street_Length_500m	0.034	0.011	3.157	**0.002**	1.035	1.013	1.058
	Distance_nearest_POE	0.016	0.006	2.677	**0.007**	1.016	1.004	1.027
	InvSqDist_POE.km	-0.006	0.019	-0.291	0.771	0.994	0.957	1.034
	Traffic_VMT_500m	-0.002	0.002	-1.326	0.185	0.998	0.995	1.001
High BP	(Intercept)	-0.459	0.033	-13.944	0.000	0.632	0.592	0.674
	Distance_nearest_Majart	0.106	0.119	0.886	0.376	1.112	0.879	1.403
	Street_Length_500m	0.000	0.011	-0.002	0.998	1.000	0.979	1.021
	Distance_nearest_POE	-0.010	0.006	-1.801	0.072	0.990	0.979	1.001
	InvSqDist_POE.km	-0.006	0.020	-0.311	0.756	0.994	0.954	1.032
	Traffic_VMT_500m	0.000	0.002	-0.133	0.894	1.000	0.997	1.003
High TG	(Intercept)	0.253	0.035	7.291	0.000	1.288	1.203	1.379
	Distance_nearest_Majart	0.146	0.128	1.136	0.256	1.157	0.901	1.491
	Street_Length_500m	0.024	0.011	2.120	**0.034**	1.024	1.002	1.047
	Distance_nearest_POE	0.005	0.006	0.875	0.381	1.005	0.994	1.017
	InvSqDist_POE.km	-0.001	0.020	-0.061	0.951	0.999	0.961	1.039
	Traffic_VMT_500m	-0.001	0.002	-0.763	0.446	0.999	0.996	1.002
Low HDL	(Intercept)	0.049	0.035	1.398	0.162	1.050	0.981	1.124
	Distance_nearest_Majart	0.129	0.128	1.006	0.314	1.138	0.885	1.464
	Street_Length_500m	0.032	0.011	2.862	**0.004**	1.033	1.010	1.056
	Distance_nearest_POE	0.010	0.006	1.645	0.100	1.010	0.998	1.022

Health Variables	Traffic Variables*	Estimate	Std. Error	z value	Pr(>|z|)	Odds Ratio	Lower 95% CI	Upper 95% CI
	InvSqDist_POE.km	0.028	0.021	1.319	0.187	1.028	0.988	1.075
	Traffic_VMT_500m	0.000	0.002	0.039	0.969	1.000	0.997	1.003
High Glucose	(Intercept)	-0.013	0.034	-0.371	0.710	0.987	0.923	1.056
	Distance_nearest_Majart	0.095	0.127	0.744	0.457	1.099	0.857	1.412
	Street_Length_500m	0.009	0.011	0.782	0.434	1.009	0.987	1.031
	Distance_nearest_POE	0.009	0.006	1.490	0.136	1.009	0.997	1.021
	InvSqDist_POE.km	0.028	0.020	1.371	0.170	1.028	0.989	1.073
	Traffic_VMT_500m	0.000	0.002	-0.119	0.906	1.000	0.997	1.003
Metabolic Syndrome	(Intercept)	0.127	0.035	3.589	0.000	1.135	1.059	1.216
	Distance_nearest_Majart	0.022	0.130	0.169	0.866	1.022	0.793	1.319
	Street_Length_500m	0.038	0.011	3.309	**0.001**	1.039	1.016	1.062
	Distance_nearest_POE	0.009	0.006	1.564	0.118	1.009	0.998	1.022
	InvSqDist_POE.km	0.006	0.020	0.283	0.777	1.006	0.968	1.047
	Traffic_VMT_500m	-0.002	0.002	-1.538	0.124	0.998	0.994	1.001

All significant predictors and corresponding *p* are expressed in bold.
*Traffic variable units: km, in thousands

4.3.5 PREDICTIVE PROBABILITY MODEL

The multivariate regression analysis quantified the relationships between different types of traffic variables and risk factors for metabolic syndrome. Using a stepwise selection technique, we built a multivariate logistic regression model which showed the best performance in estimating the likelihood of metabolic syndrome. Based on the modeling, the most relevant variables were the length of street 500m, distance to nearest POE, and traffic VMT 500m (Table 4.13).

Table 4.13: Summary of variable selection for multivariate logistic regression models using a stepwise selection technique.

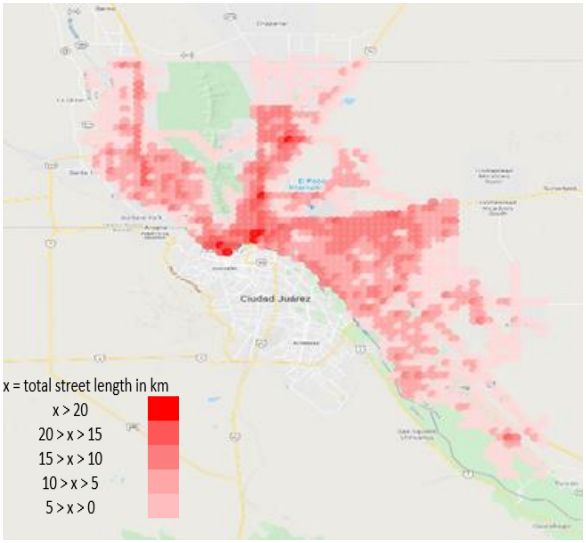
Health Variables	Traffic Variables*	Estimate	Std. Error	z value	Pr(>|z|)	Odds Ratio	Lower 95% CI	Upper 95% CI
Metabolic Syndrome	(Intercept)	0.126	0.035	3.586	0.000	1.134	1.059	1.215
	Street_Length_500m	0.038	0.011	3.459	**0.001**	1.039	1.017	1.062
	Distance_nearest_POE	0.009	0.006	1.569	0.117	1.009	0.998	1.021
	Traffic_VMT_500m	-0.003	0.002	-1.597	0.110	0.997	0.994	1.001

All significant predictors and corresponding *p* are expressed in bold.
*Traffic variable units: km, in thousands

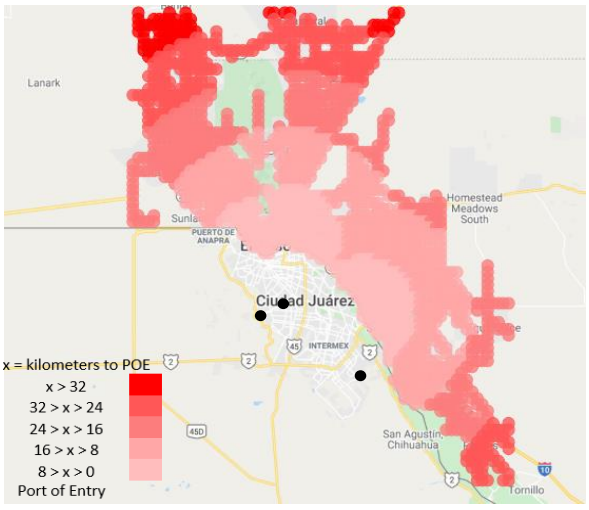
The multivariate regression model calculated the coefficients estimates for the selected traffic variables which were used to predict the probabilities of occurring in El Paso for MetS. The length of street within 500m was positively associated with the likelihood of having Mets ($p=0.001$). Distance to nearest POE was positively associated with the likelihood of Mets, while traffic VTM within 500m was negatively correlated with MetS, both were not significant. Using these estimates, a land use probability map was made for each traffic variable. The land-use maps show the length of street 500m, distance to nearest POE, and traffic VMT 500m with the values grouped into different areas for visual interpretation (Figure 4.11).

The maps show that the areas with the highest amount of street length are located in the central part of the city, while the areas with the most traffic are located in the vicinity of the major freeways. The distance to POE is associated with the outer west, northeast, and far east of the city. Figure 4.11 shows the land-use based on the mentioned variables. Each map provides various forecasting results, especially regarding spatial patterns of street length and traffic.

a)



b)



c)

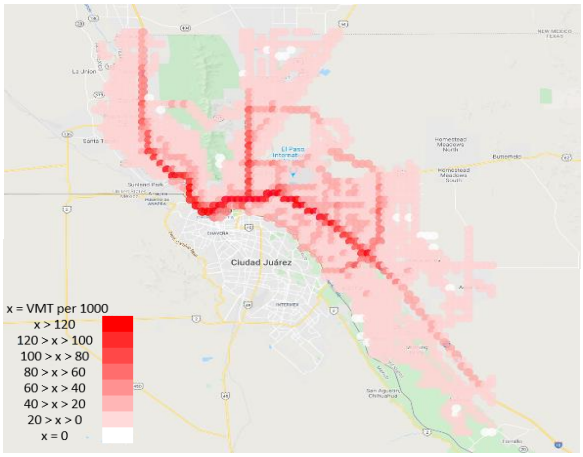


Figure 4.11: Traffic-related variables applied to a city grid for a) street length (500m), b) distance to nearest port of entry (POE), c) vehicle miles traveled (500m)

Lastly, the coefficient estimates of the traffic predictors for the selected model based on the variable selection technique, were applied to calculate a predicted probability equation for MetS:

$$\frac{\exp\{0.126 + 0.038(Street\_Length_{500m} - 10.731) + 0.009(Distance_{POE} - 9.482) - 0.003(VMT_{500m} - 23.337)\}}{1 + \exp\{0.126 + 0.038(Street\_Length_{500m} - 10.731) + 0.009(Distance_{POE} - 9.482) - 0.003(VMT_{500m} - 23.337)\}}$$

The predicted values were applied to a gridded map representative of areas in El Paso, TX in which the resulting layer (Figure 4.2) shows areas of higher and lower probability of metabolic syndrome.

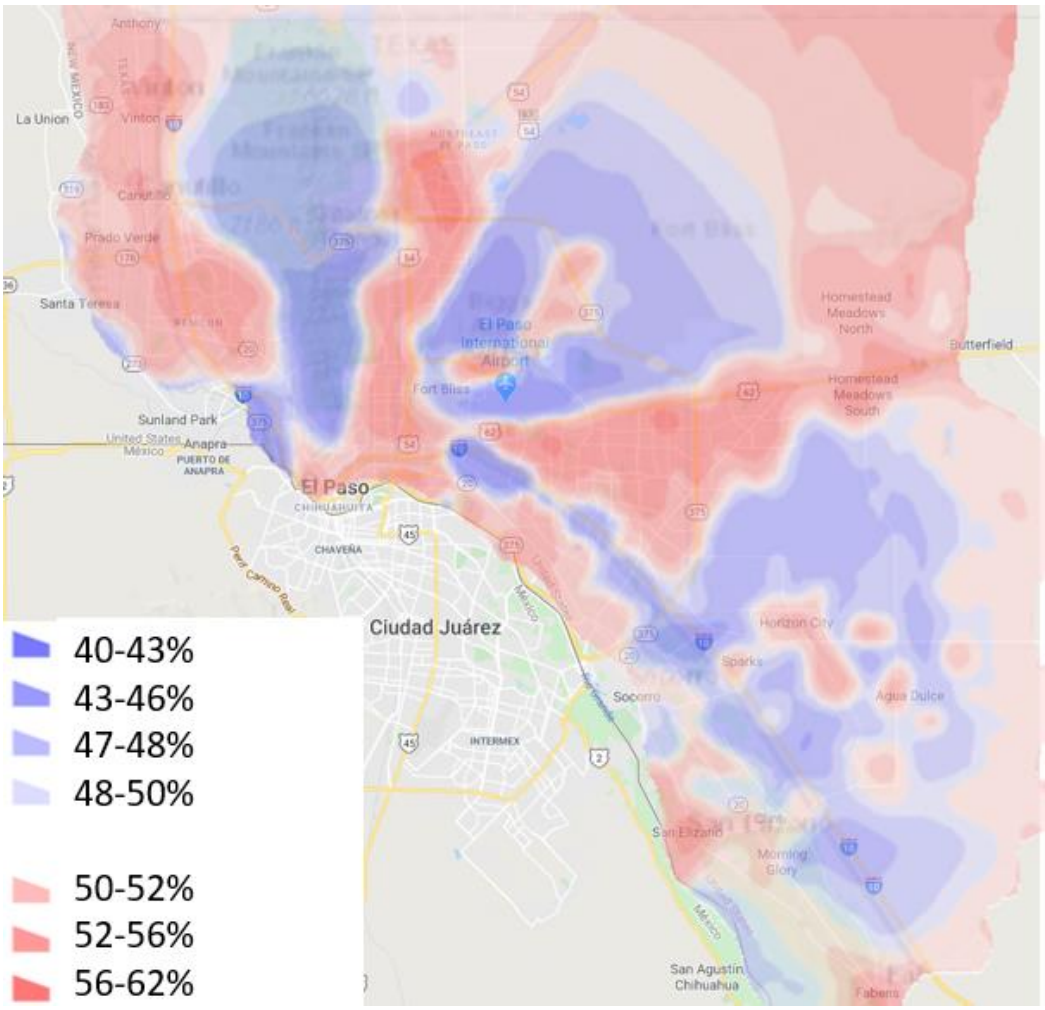


Figure 4.12: Predictive model of higher risk of metabolic syndrome based on the land use regression model

4.4 Discussion

4.4.1 PRINCIPAL FINDINGS

The present study found expected associations with respiratory outcomes caused by long-term exposure to traffic emission using the available data subset from the 1st year of the larger health study. We found correlations of length of street within the 500m & 1000m impact zone as well as the VMT with measures of lung function (FEV_1, FVC, PEF). Furthermore, multivariate models showed length of street within the 500m was an important traffic predictor for lung function based on the peak exploratory flow (PEF & PEF Best).

Regarding the cardiovascular outcomes, the present study found associations with long-term exposure to traffic emission using the available data of a larger health study. The most significant predictor in the LUR models of MetS risk factors was the total length of the street within a 500m radius from a resident's address. The increase in the street length was associated with increasing waist and triglycerides and decreasing HDL in multivariate models. Furthermore, the increase in the inverse of the distance squared to the POE (i.e. a decrease in the distance to POE) was significantly associated with an increase in glucose levels, which suggest an increased likelihood of higher glucose levels in the neighborhoods further away from the city (north and far east areas). In addition, PBP was associated with an increased amount of traffic within a 500m radius and the proximity to the nearest POE.

While we hypothesized that a closer distance to a POE would lead to an increased risk of Mets and its risk factors, the outer regions of the city also included *colonias* (disadvantaged neighborhoods of extreme poverty in the suburban areas) which may convolute our findings with respect to this parameter. Our results were further confirmed in logistic regression models which found that as the total length of street increases, the risks of a high waist, high triglycerides, low

HDL-cholesterol, and MetS (three or more risk factors) increased. Based on the LUR, MetS is higher in the central areas of El Paso where there is likely more street length and traffic. We also identified clusters of high MetS in the outer regions of the city, which matches with low-income communities and *colonias*, compared to areas in the west side and lower valley.

4.4.2 STRENGTHS AND LIMITATIONS

The strength of this study is seen in the large size of sample (N=4,959) of low-income participants from El Paso, TX which are widely distributed in the city. Also, the use of the LUR models allowed direct exploration of several transportation variables on health outcomes instead of using concentration estimates, which were used in other studies, that were developed from central monitors and a fraction of known pollutants which introduced additional uncertainties.

The lack of data regarding indoor air pollutants may result in some complications with our study. Although difference between the indoor/outdoor exposure may exist, studies in the region have shown, in general, that there is a direct relationship between outdoor and indoor air pollution (Andersen, 1972; Raysoni, Stock, Sarnat, Montoya Sosa, et al., 2013; Zora et al., 2013). In addition, the traffic data retrieved from the GIS layers provided by El Paso MPO, Census, and PDNMAPA were limited to be within the U.S. jurisdiction. For participants living within 1,000 m from the border area, our analysis could not include the traffic variables or GIS layers fall on the jurisdiction of Ciudad Juarez, Mexico. However, this lack of partial information did not have much influence on the overall analyses since less than 2% of participants lived within 1,000 m of the border. Future studies would benefit from including information from GIS layers with data from Mexico.

Lastly, it is important to emphasize that the predictions of our models can be further improved by including parameters representing the socioeconomic characteristics of the

population. The use of clinically aligned information in conjunction with spatial traffic-related data would provide additional insights for the border communities.

4.4.3 COMPARISON WITH OTHER STUDIES

In recent decades, studies about long-term exposure to outdoor air pollution have played a crucial role in assessing the impact on different populations (Amini et al., 2017; Hoek et al., 2008). In our region, previous studies have used land use regression to predict concentrations of traffic-related air pollutants. In 1999, a LUR model developed to predict concentrations of NO_2 (and other related pollutants), found the most useful predictive variables were elevation, population density, distance to an international port of entry (POE), and distance to a petroleum facility considering two monitoring sites (Smith et al., 2006). This was further evaluated in 2006-2007 using a series of mixed model LURs which confirmed the mentioned variables as useful predictors of NO_2 even when considering seasonal variation (Gonzales et al., 2012). Distance to an international port of entry (POE) was an important predictor in our model too, however with regards to cardiovascular health residents located further from them have a higher risk.

A study along the interstate highway I-10 in El Paso, comparing field measurements and LUR modeling results, suggested that PM concentrations within a 1,000m buffer zone from the highway are most likely to be impacted by emissions from the interstate highway (Olvera, Jimenez, & Provencio-Vasquez, 2014). In our study, we considered the impact of distance to major traffic roads but did not find any associations with cardiorespiratory health outcomes. Another LUR study, found that adding traffic variables (vehicle miles traveled, speed, traffic demand, and street length) to existing models generated a good set of predictors for air quality estimation (Olvera et al., 2012). In our study we found street length and vehicle miles traveled to be important predictors as well. Most recently, a study compared PM pollution measured at four monitoring sites to those

estimated using surrogate variables (land use, traffic intensity, population density, and property value) for traffic emissions (Alvarez, Myers, Weigel, & Armijos, 2018). The results, however, were reported to be influenced by climate-specific meteorological events and did not yield any findings we could compare.

Finally, it seems only one study has correlated the use of LUR with air pollution and health outcomes in El Paso. A 2015 study tested relationships for residential pest and $PM_{2.5}$ exposures with children's self-reported wheezing severity based on socio-economic factors and a previously developed LUR regression model (Grineski, Collins, & Olvera, 2015). Our study used cardiorespiratory outcomes and found relationship with traffic-related pollutants which adds to knowledge to air pollution research in our region.

4.4 Conclusions

The dissemination of results can lead to decision making and improve policies related to healthy living in communities close to busy roadways. Furthermore, the use of predictive models based on LUR allows identification of communities at risk for cardiorespiratory health challenges. This study offers the transportation professionals a rapid assessment of health risks associated with exposures to traffic emissions and assists communities close to busy roadways a way to understand the health risks posed to them. Future studies should focus on the validation of the model with field air pollution data and integrate the model with additional layers of information such as socio-economic status, population density, time of residence, among others, which could further allow more accurate predictors of cardiorespiratory diseases. Finally, the use of such models can be paired with clinical health outcomes to improve strategies aimed to reduce the effects of air pollution exposure on health and associated diseases.

CHAPTER 5 CONCLUSION

5.1 General findings

Air pollution emissions quickly disperse into the atmosphere; therefore, understanding their characteristics is essential for developing strategies to improve air quality. A large body of work demonstrates a link between the effects of air pollution exposure on respiratory health. However, the analysis of the data used in this dissertation also found links between air pollution exposure and physical activity, obesity, and metabolic syndrome in at-risk populations (children and low-income adults).

As shown in Chapter 2, children with asthma spent less time in moderate to vigorous physical activity when exposed to higher concentrations of traffic-related pollutants while attending their elementary school. Severe conditions of air pollution can require cancellation of physical or sport activities while in school, which may lead to an increase in sedentary behavior and contribute to the overweight and obesity epidemic (Giles & Koehle, 2014). Also, given that children attending school spend about 6-8 hours per day in various school microenvironments, it is recommended to reduce children's air pollution exposure in schools (U.S.EPA, 2015). This is particularly important for schools located near busy traffic intersections or freeways where children may be exposed to an even higher level of traffic pollution.

Chapters 3 and 4 focused on the relationship between short-term and long-term traffic-related air pollution and health outcomes in participants from low-income communities. From previous studies, we know people living in areas with high air pollution have increased likelihood of cardiorespiratory health outcomes like heart and lung disease, irregular heartbeat, aggravated asthma, and decreased lung function (Atkinson et al., 2010b; Cadelis et al., 2014b; Andrew W Correia et al., 2013). This dissertation showed short-term air pollution exposure, based on air

quality data obtained from central monitoring stations, is positively correlated with decreased correlated with decreased lung function, obesity, and higher fasting glucose levels. Furthermore, the use of land use regression modeling allowed us to determine traffic-related variables such as the length of street within 500m, distance to nearest port of entry, and distance to major traffic roads as valuable surrogates to determine long-term exposure to air pollution associations with cardiorespiratory health outcomes.

For the mentioned reasons, we suggest the implementation of mitigation strategies listed in the following section to improve the health of not only at-risk populations, but also the community in general. We aim to reduce the exposure to air pollution and improve the health of our community by providing these recommendations that can be implemented in communities such as schools and low-income housing neighborhoods.

5.2 Mitigation strategies

Mitigation strategies can be divided into three options, each with their own benefits and challenges: reducing the magnitude of air pollution, controlling the emission rate of pollution sources, and controlling the source-receptor pathways (McNabola, Broderick, & Gill, 2008; Tong, Baldauf, Isakov, Deshmukh, & Zhang, 2016). The first can be achieved with measures such as mandatory greenhouse emission reductions, fuel efficiency standards, carbon taxes, introduction of electric mass transit, and climate change goals. (Galinato & Yoder, 2010; Münzel et al., 2017). Examples of emission control include vehicle tax systems that encourage the use of smaller vehicles that produce less pollutants, improvements in vehicle technology, and charging drivers who enter congestion zones (Atkinson et al., 2009; Giblin & McNabola, 2009; Matter, 2011; Styles, O'Brien, & Jones, 2009). However, both reducing concentrations of air pollution and

controlling emission rates require changes in policy or infrastructure which makes them difficult to implement at a community level.

The third option, controlling source-receptor pathways, considers passive control measures, such as solid or porous barriers, which can be implemented near roads or within built environments to decrease pathways by which pollutants disseminate (Gallagher et al., 2015). Particulate matter (PM_{10} and $PM_{2.5}$) can adhere to the surface of plants (Hosker Jr & Lindberg, 1982; Nowak, Crane, & Stevens, 2006), and several studies suggest different types of natural barriers can be useful such as trees (Gromke & Ruck, 2007), hedgerows (Gromke, Jamarkattel, & Ruck, 2016), and green roofs (Perini, Ottelé, Fraaij, Haas, & Raiteri, 2011; Speak, Rothwell, Lindley, & Smith, 2012). Natural barriers lead to the improvement of air quality and overall health of those living in urban environment (Currie & Bass, 2008).

Another option that controls source-receptor pathways includes the use of titanium dioxide (TiO_2) paint, a photocatalytic agent, that reduces NO_2 concentrations (Jeanjean, Gallagher, Monks, & Leigh, 2017; Lasek, Yu, & Wu, 2013). Lastly, the presence of enough tree shade near streets and sidewalks can reduce higher temperatures on asphalt which leads to a decrease in atmospheric O_3 concentration. Example of trees and plants helpful in reducing O_3 include curtain fig, camphor, savin juniper, and Australian laurel (Jim & Chen, 2008).

Mitigation strategies that control source-receptor pathways that include wide vegetation barriers with high leaf density, solid barrier with of photocatalytic agents like TiO_2, or combinations of vegetation and solid barriers could improve the air quality in a region. Furthermore, these mitigation strategies can be implemented in at-risk communities such as schools and housing communities without requiring major changes in policy or infrastructure

5.3 Final remarks

Education is key to improve the health of our communities and children, I feel fortunate to have been a part of programs like the EPA-UTEP Air Quality Internship which allowed graduate students to interact with elementary school children. During school visits, I was able to teach to young students the importance of air quality and how they can contribute to reduce air pollution. This interaction also aimed to inspire future experts in the field which will continue the work and research beyond what we have done.

Next steps include submitting our findings for publication and engaging stakeholders to bring awareness about the impact of air pollution and health. Regarding schools specifically, we will discuss the impact air pollution is having on children with asthma and discuss the implementation of mitigation strategies that would reduce air pollution exposure at schools.

We also aim to expand our land use regression models to account for sociodemographic information and the possible use of low-cost sensors deployed in low-income communities to measure the concentrations of traffic-related pollutants in ambient and indoor environments. We hope this will allow us to create a network that will further expand our knowledge of air pollution in our region and further identify hot spots of high exposure.

Lastly, we hope to reduce cardiorespiratory risk by offering educational materials in collaboration with the ongoing epidemiological study, and collaborate with our local health department, city planners, and school district officials to further find strategies that can allow us to collectively improve the overall quality of life for El Paso residents. To achieve these goals, we will need an interdisciplinary, cross-sector approach which illustrates the value of my training in the Interdisciplinary Health Sciences PhD Program.

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APPENDIX

Cardiovascular associations with traffic-related variables (2014-15 data)

Descriptive statistics for cardiovascular risk measurements (metabolic syndrome) are summarized in Table 4.5 (N=662). The waist circumference ranged from 49 to 151 cm with a mean of 95 cm. Blood pressure (SBP/DBP) measurements ranged from 74/35 to 211/128 with a mean of 127/76 mmHg. Triglyceride (TG) levels ranged from 45 to 650 mg/dL with a mean of 186 mg/dL. HDL-cholesterol ranged from 15 to 100 mg/dL with a mean of 49 mg/dL. Glucose levels ranged from 50 to 477 mg/dL with a mean value of 109 mg/dL. Other variables of interest of cardiovascular risk which are not components of metabolic syndrome but could potentially offer more information related to cardiovascular risk included BMI, pulse pressure (PBP), total cholesterol (TC), and LDL-cholesterol.

Table A.1 Descriptive statistics for metabolic syndrome risk factors.

Risk Factor	Min	Q1	Median	Mean	Q3	Max	SD	IQR
Waist (cm)	49.0	86.0	94.0	95.5	104.0	151.0	14.4	18.0
SBP (mmHg)	74.0	113.0	125.0	127.8	140.3	211.0	20.6	27.3
DBP (mmHg)	35.0	69.0	75.0	76.2	82.0	128.0	11.4	13.0
PBP (mmHg)	6	42	49	52	59	107	14	17
TC (mg/dL)	100	161	188	190	215	350	39	54
TG (mg/dL)	45	107	161	186	224	650	115	117
HDL (mg/dL)	14.9	40.0	48.0	49.7	58.0	100.1	14.6	18.0
LDL (mg/dL)	12.0	84.0	102.0	106.1	127.0	220.0	31.9	43.0
TC/HDL	1.4	3.1	3.8	4.2	4.8	22.0	1.7	1.7
Glucose (mg/dL)	49.9	86.3	94.5	108.7	108.0	477.0	46.5	21.8

Correlation and univariate regression analyses showed that a few metabolic syndrome risk factors were associated with inverse distance and inverse squared distance to the nearest port of entry (see Table 4.6). The inverse of the distance to the nearest port of entry was associated with increases in fasting glucose and triglycerides ($\beta_1 = 17.124$, $p < 0.001$; $\beta_1 = 22.351$, $p = 0.039$,

respectively). The inverse of the distance squared to the port of entry also showed positive correlations with fasting glucose and triglycerides ($\beta_1 = 9.209$, $p < 0.001$; $\beta_1 = 14.086$, $p = 0.001$, respectively), implying that the metabolic risk related to fasting glucose and triglycerides decreases as subjects live farther away from the port of entry.

The classification of metabolic syndrome risk factors (binary outcomes) based on current guidelines is presented in Table 4.7. Separate logistic regression models were run for each traffic variable of interest to evaluate the binary outcome of the MetS factors. The associations between the classification of metabolic syndrome factors and traffic variables are summarized in Table A.2.

The logistic regression models showed that the street length within the 1000m impact zone was also a significant factor related to a higher risk of high-BP (odds ratio=1.013, p=0.048). The increase in the length of the street within the 1000m zone also associated with the risk of high SBP (odds ratio=1.014, p=0.030), and the high value in SBP may play a role to determine the high blood pressure.

The land-use regression model included the five traffic-related variables within the 500m impact zone in a multivariate regression model. The most significant predictor in the LUR models of MetS risk factors was the inverse squared distance to the nearest port of entry (see Table A.3). The increase in the inverse of the distance squared to the port of entry, implying decrease in the distance to POE, were significantly associated increases in total cholesterol ($\beta_1 = 3.689$, $p = 0.019$), triglycerides ($\beta_1 = 15.063$, $p = 0.001$), and fasting glucose ($\beta_1 = 9.805$, $p < 0.001$). In logistic regression modeling, we also found that increasing inverse distance squared to the port of entry was associated with an increased likelihood of high total cholesterol (odds ratio = 1.221; $p = 0.055$). The LUR model was found to have a week correlation between MetS classification and street length within the 500m zone, which implies more likelihood of having metabolic syndrome

with increased street length around the residential area (odds ratio =1.050, p = 0.082). However, as shown in the previous univariate models, the larger impact zone within the 1000m distance may be more appropriate than the 500m zone when modeling binary risk factors of metabolic syndrome.

Table A.2: Correlation Analysis

Variable	Distance_ nearest_ Majart	Street_ Length_ 500m	Street_ Length_ 1000m	Distance_ nearest_ POE	InvDist_ POE	InvSqDist _POE	Traffic_ VMT_ 500m	Traffic_ VMT_ 1000m
SBP	0.023	0.008	0.016	0.028	0.024	0.054	0.038	0.030
SBP < 130	0.086	-0.079	-0.091	**0.102**	-0.053	-0.010	-0.016	-0.019
SBP >= 130	0.107	-0.073	-0.085	0.024	-0.024	-0.012	-0.004	-0.027
DBP	0.007	0.005	0.015	0.069	0.015	0.044	0.048	0.018
DBP < 85	-0.060	-0.048	-0.017	0.051	-0.022	0.010	0.039	0.003
DBP >= 85	0.016	0.122	0.070	0.003	0.031	-0.026	0.087	-0.028
PBP	0.028	0.007	0.012	-0.014	0.023	0.042	0.017	0.028
TC	0.053	-0.035	-0.069	-0.017	0.024	0.070	-0.018	-0.046
TG	**0.078**	0.056	-0.015	0.025	**0.081**	**0.134**	-0.030	**-0.080**
log.TG	**0.087**	0.049	-0.003	0.036	0.053	**0.100**	-0.033	-0.069
HDL	0.004	-0.032	-0.020	-0.026	-0.024	-0.033	-0.028	0.009
LDL	0.013	-0.039	-0.061	-0.032	-0.035	-0.057	0.030	-0.017
TC/HDL	0.015	0.012	0.003	0.006	0.031	0.071	0.016	-0.035
log.TC/HDL	0.042	0.015	-0.003	0.013	0.024	0.060	0.017	-0.031
bc.TC/HDL[2)]	0.053	0.013	-0.006	0.014	0.018	0.049	0.013	-0.027
Glucose	-0.036	0.074	0.054	-0.039	**0.153**	**0.217**	0.010	0.006
log.Glucose	-0.024	0.059	0.058	-0.053	**0.127**	**0.166**	0.003	0.023
bc.Glucose[3)]	-0.006	0.040	0.068	-0.059	**0.084**	**0.095**	-0.004	0.045

All significant correlations are expressed in bold.

[2)] Box-Cox Transformation: bc.TC/HDL = [(TC/HDL)^(-0.5)-1]/(-0.5).

[3)] Box-Cox Transformation: bc.Glucose = [Glucose^(-2)-1]/(-2).

Table A.3: Summary of metabolic syndrome risk factors.

Variable		Frequency	%
High Waist	Yes	411	62.1
	No	244	36.9
High BP	Yes	277	41.8
	No	363	54.8
High TG	Yes	363	54.8
	No	291	44.0
Low HDL	Yes	329	49.7
	No	315	47.6
High Glucose	Yes	231	34.9
	No	423	63.9
MetS	Yes	336	50.8
	No	307	46.4

Table A.4: Univariate associations between metabolic syndrome (MetS) risk factors and MetS classification and traffic variables.

Outcome	Traffic Variable	Estimate	Std. Error	z value	Pr(>|z|)	Odds Ratio	Lower 95% CI	Upper 95% CI
High Waist	Distance_nearest_Majart	-0.220	0.364	-0.605	0.545	0.802	0.391	1.663
	Street_Length_500m	0.009	0.021	0.422	0.673	1.009	0.969	1.051
	Street_Length_1000m	0.007	0.006	1.115	0.265	1.007	0.995	1.020
	Distance_nearest_POE	0.017	0.016	1.031	0.303	1.017	0.985	1.050
	InvDist_POE.km	-0.042	0.193	-0.217	0.828	0.959	0.658	1.417
	InvSqDist_POE.km	0.012	0.076	0.162	0.871	1.012	0.877	1.202
	Traffic_VMT_500m	0.000	0.004	0.054	0.957	1.000	0.993	1.008
	Traffic_VMT_1000m	0.001	0.001	0.844	0.398	1.001	0.999	1.003
High BP	Distance_nearest_Majart	-0.449	0.385	-1.166	0.244	0.638	0.290	1.326
	Street_Length_500m	0.030	0.020	1.495	0.135	1.031	0.991	1.073
	Street_Length_1000m	0.013	0.006	1.977	**0.048**	1.013	1.000	1.025
	Distance_nearest_POE	0.004	0.016	0.266	0.790	1.004	0.974	1.035
	InvDist_POE.km	0.228	0.193	1.182	0.237	1.256	0.862	1.852
	InvSqDist_POE.km	0.146	0.093	1.567	0.117	1.157	0.988	1.445
	Traffic_VMT_500m	0.005	0.004	1.301	0.193	1.005	0.998	1.012
	Traffic_VMT_1000m	0.001	0.001	1.103	0.270	1.001	0.999	1.003
High TG	**Distance_nearest_Majart**	0.919	0.424	2.168	**0.030**	2.507	1.124	5.931
	Street_Length_500m	0.015	0.020	0.762	0.446	1.015	0.976	1.056
	Street_Length_1000m	0.000	0.006	-0.014	0.989	1.000	0.988	1.012
	Distance_nearest_POE	0.021	0.016	1.327	0.185	1.021	0.990	1.053
	InvDist_POE.km	0.107	0.193	0.558	0.577	1.113	0.767	1.646
	InvSqDist_POE.km	0.149	0.106	1.403	0.161	1.161	0.978	1.503

Outcome	Traffic Variable	Estimate	Std. Error	z value	Pr(>|z|)	Odds Ratio	Lower 95% CI	Upper 95% CI
	Traffic_VMT_500m	-0.003	0.004	-0.929	0.353	0.997	0.990	1.004
	Traffic_VMT_1000m	-0.001	0.001	-1.423	0.155	0.999	0.997	1.000
Low HDL	Distance_nearest_Majart	0.055	0.386	0.143	0.886	1.057	0.494	2.276
	Street_Length_500m	0.025	0.020	1.226	0.220	1.025	0.985	1.067
	Street_Length_1000m	0.009	0.006	1.489	0.137	1.009	0.997	1.022
	Distance_nearest_POE	0.009	0.016	0.581	0.561	1.009	0.979	1.041
	InvDist_POE.km	0.123	0.191	0.643	0.520	1.131	0.779	1.664
	InvSqDist_POE.km	0.067	0.079	0.856	0.392	1.070	0.925	1.284
	Traffic_VMT_500m	0.002	0.004	0.640	0.522	1.002	0.995	1.009
	Traffic_VMT_1000m	0.000	0.001	0.389	0.697	1.000	0.999	1.002
High Glucose	Distance_nearest_Majart	0.463	0.394	1.176	0.240	1.589	0.730	3.474
	Street_Length_500m	-0.001	0.021	-0.051	0.960	0.999	0.959	1.040
	Street_Length_1000m	0.002	0.006	0.348	0.728	1.002	0.990	1.015
	Distance_nearest_POE	-0.013	0.016	-0.799	0.424	0.987	0.956	1.019
	InvDist_POE.km	0.170	0.193	0.880	0.379	1.185	0.806	1.734
	InvSqDist_POE.km	0.078	0.074	1.056	0.291	1.081	0.935	1.273
	Traffic_VMT_500m	-0.003	0.004	-0.725	0.468	0.997	0.990	1.005
	Traffic_VMT_1000m	0.000	0.001	0.397	0.692	1.000	0.998	1.002
MetS	Distance_nearest_Majart	0.133	0.387	0.343	0.732	1.142	0.534	2.477
	Street_Length_500m	0.031	0.020	1.541	0.123	1.032	0.992	1.074
	Street_Length_1000m	0.010	0.006	1.629	0.103	1.010	0.998	1.023
	Distance_nearest_POE	0.011	0.016	0.726	0.468	1.011	0.981	1.043
	InvDist_POE.km	0.197	0.196	1.007	0.314	1.218	0.835	1.814
	InvSqDist_POE.km	0.158	0.106	1.488	0.137	1.171	0.986	1.514
	Traffic_VMT_500m	-0.001	0.004	-0.271	0.787	0.999	0.992	1.006
	Traffic_VMT_1000m	0.000	0.001	0.111	0.912	1.000	0.998	1.002

All significant correlations are expressed in bold.

Table A.5: Summary and parameter estimates of multivariate regression models for continuous MetS risk factors.

Health Variables	Traffic Variables	Estimate	Std. Error	t value	Pr(>|t|)
SBP	(Intercept)	127.671	0.816	156.374	0.000
	Distance_nearest_Majart	3.606	4.172	0.864	0.388
	Street_Length_500m	-0.126	0.276	-0.457	0.648
	Distance_nearest_POE	0.157	0.178	0.883	0.378
	InvSqDist_POE.km	1.391	0.836	1.664	0.097
	Traffic_VMT_500m	0.052	0.043	1.208	0.228
SBP	(Intercept)	114.051	0.549	207.899	0.000
(<130, N=377)	Distance_nearest_Majart	2.820	2.841	0.993	0.322

Health Variables	Traffic Variables	Estimate	Std. Error	t value	Pr(>|t|)
	Street_Length_500m	-0.302	0.204	-1.479	0.140
	Distance_nearest_POE	0.212	0.125	1.692	0.092
	InvSqDist_POE.km	2.108	1.385	1.522	0.129
	Traffic_VMT_500m	0.030	0.036	0.848	0.397
SBP	(Intercept)	147.381	0.910	162.039	0.000
(>=130, N=263)	Distance_nearest_Majart	7.348	4.596	1.599	0.111
	Street_Length_500m	-0.345	0.309	-1.118	0.265
	Distance_nearest_POE	-0.080	0.198	-0.404	0.687
	InvSqDist_POE.km	0.310	0.645	0.481	0.631
	Traffic_VMT_500m	0.032	0.039	0.805	0.422
DBP	(Intercept)	76.136	0.449	169.455	0.000
	Distance_nearest_Majart	0.364	2.296	0.159	0.874
	Street_Length_500m	-0.057	0.152	-0.378	0.706
	Distance_nearest_POE	0.198	0.098	2.026	**0.043**
	InvSqDist_POE.km	0.741	0.460	1.611	0.108
	Traffic_VMT_500m	0.032	0.024	1.356	0.176
DBP	(Intercept)	71.994	0.350	205.512	0.000
(<85, N=509)	Distance_nearest_Majart	-3.725	1.943	-1.917	0.056
	Street_Length_500m	-0.250	0.122	-2.053	**0.041**
	Distance_nearest_POE	0.092	0.079	1.162	0.246
	InvSqDist_POE.km	0.651	0.524	1.241	0.215
	Traffic_VMT_500m	0.027	0.020	1.356	0.176
DBP	(Intercept)	92.411	0.668	138.441	0.000
(>=85, N=131)	Distance_nearest_Majart	1.873	2.723	0.688	0.493
	Street_Length_500m	0.365	0.230	1.589	0.115
	Distance_nearest_POE	0.074	0.135	0.547	0.585
	InvSqDist_POE.km	-0.375	0.403	-0.930	0.354
	Traffic_VMT_500m	0.012	0.028	0.415	0.679
PBP	(Intercept)	51.535	0.575	89.652	0.000
	Distance_nearest_Majart	3.242	2.938	1.104	0.270
	Street_Length_500m	-0.069	0.194	-0.353	0.724
	Distance_nearest_POE	-0.041	0.125	-0.329	0.742
	InvSqDist_POE.km	0.650	0.589	1.104	0.270
	Traffic_VMT_500m	0.020	0.030	0.656	0.512
TC	(Intercept)	190.077	1.516	125.367	0.000
	Distance_nearest_Majart	9.553	8.363	1.142	0.254
	Street_Length_500m	-0.834	0.514	-1.622	0.105
	Distance_nearest_POE	-0.222	0.332	-0.670	0.503
	InvSqDist_POE.km	3.689	1.566	2.356	**0.019**
	Traffic_VMT_500m	0.037	0.080	0.465	0.642
TG	(Intercept)	186.352	4.443	41.944	0.000

Health Variables	Traffic Variables	Estimate	Std. Error	t value	Pr(>|t|)
	Distance_nearest_Majart	63.169	24.507	2.578	**0.010**
	Street_Length_500m	2.310	1.506	1.534	0.126
	Distance_nearest_POE	1.491	0.972	1.535	0.125
	InvSqDist_POE.km	15.063	4.589	3.282	**0.001**
	Traffic_VMT_500m	-0.243	0.235	-1.035	0.301
log.TG	(Intercept)	5.068	0.022	232.742	0.000
	Distance_nearest_Majart	0.328	0.120	2.729	**0.007**
	Street_Length_500m	0.013	0.007	1.789	0.074
	Distance_nearest_POE	0.008	0.005	1.595	0.111
	InvSqDist_POE.km	0.054	0.022	2.396	**0.017**
	Traffic_VMT_500m	-0.001	0.001	-1.056	0.291
HDL	(Intercept)	49.662	0.572	86.764	0.000
	Distance_nearest_Majart	-3.169	3.152	-1.005	0.315
	Street_Length_500m	-0.157	0.195	-0.807	0.420
	Distance_nearest_POE	-0.137	0.126	-1.091	0.276
	InvSqDist_POE.km	-0.414	0.588	-0.705	0.481
	Traffic_VMT_500m	-0.012	0.030	-0.404	0.686
LDL	(Intercept)	106.017	1.308	81.072	0.000
	Distance_nearest_Majart	2.814	7.747	0.363	0.717
	Street_Length_500m	-0.501	0.457	-1.096	0.274
	Distance_nearest_POE	-0.451	0.290	-1.557	0.120
	InvSqDist_POE.km	-2.582	2.113	-1.222	0.222
	Traffic_VMT_500m	0.091	0.069	1.329	0.184
TC/HDL	(Intercept)	4.171	0.068	61.072	0.000
	Distance_nearest_Majart	0.212	0.374	0.567	0.571
	Street_Length_500m	-0.010	0.023	-0.428	0.668
	Distance_nearest_POE	0.007	0.015	0.459	0.646
	InvSqDist_POE.km	0.134	0.070	1.929	0.054
	Traffic_VMT_500m	0.002	0.004	0.559	0.576
log.TC/HDL	(Intercept)	1.366	0.014	100.898	0.000
	Distance_nearest_Majart	0.096	0.074	1.298	0.195
	Street_Length_500m	0.000	0.005	-0.046	0.963
	Distance_nearest_POE	0.002	0.003	0.538	0.591
	InvSqDist_POE.km	0.023	0.014	1.634	0.103
	Traffic_VMT_500m	0.000	0.001	0.616	0.538
bc.TC/HDL	(Intercept)	0.976	0.007	145.985	0.000
	Distance_nearest_Majart	0.057	0.037	1.551	0.121
	Street_Length_500m	0.000	0.002	0.066	0.948
	Distance_nearest_POE	0.001	0.001	0.448	0.654
	InvSqDist_POE.km	0.009	0.007	1.358	0.175
	Traffic_VMT_500m	0.000	0.000	0.583	0.560

Health Variables	Traffic Variables	Estimate	Std. Error	t value	Pr(>|t|)
Glucose	(Intercept)	108.802	1.789	60.832	0.000
	Distance_nearest_Majart	-3.955	9.866	-0.401	0.689
	Street_Length_500m	-0.199	0.606	-0.328	0.743
	Distance_nearest_POE	0.230	0.391	0.588	0.556
	InvSqDist_POE.km	9.805	1.847	5.307	**0.000**
	Traffic_VMT_500m	-0.030	0.095	-0.322	0.748
log.Glucose	(Intercept)	4.635	0.011	405.152	0.000
	Distance_nearest_Majart	-0.013	0.063	-0.201	0.841
	Street_Length_500m	-0.001	0.004	-0.290	0.772
	Distance_nearest_POE	-0.001	0.003	-0.200	0.842
	InvSqDist_POE.km	0.046	0.012	3.891	**0.000**
	Traffic_VMT_500m	0.000	0.001	-0.364	0.716
bc.Glucose	(Intercept)	0.500	0.000	652753	0.000
	Distance_nearest_Majart	0.000	0.000	0.061	0.951
	Street_Length_500m	0.000	0.000	-0.082	0.935
	Distance_nearest_POE	0.000	0.000	-0.884	0.377
	InvSqDist_POE.km	0.000	0.000	2.005	**0.045**
	Traffic_VMT_500m	0.000	0.000	-0.383	0.702

All significant predictors and corresponding *p* are expressed in bold.

Table A.6: Summary and parameter estimates of multivariate logistic regression model for binary MetS factors.

Outcome	Traffic Variables	Estimate	Std. Error	z value	P value	Odds Ratio	Lower 95% CI	Upper 95% CI
High Waist	(Intercept)	0.534	0.081	6.563	0.000	1.706	1.456	2.003
	Distance_nearest_Majart	-0.193	0.414	-0.467	0.641	0.824	0.367	1.900
	Street_Length_500m	0.021	0.028	0.745	0.456	1.021	0.967	1.078
	Distance_nearest_POE	0.026	0.018	1.416	0.157	1.026	0.990	1.064
	InvSqDist_POE	0.012	0.087	0.134	0.893	1.012	0.859	1.238
	Traffic_VMT_500m	-0.002	0.004	-0.359	0.719	0.998	0.990	1.007
High BP	(Intercept)	-0.280	0.081	-3.478	0.001	0.756	0.645	0.885
	Distance_nearest_Majart	-0.253	0.418	-0.604	0.546	0.777	0.332	1.743
	Street_Length_500m	0.014	0.027	0.497	0.619	1.014	0.960	1.070
	Distance_nearest_POE	0.021	0.018	1.224	0.221	1.022	0.987	1.058
	InvSqDist_POE	0.138	0.108	1.285	0.199	1.148	0.960	1.504
	Traffic_VMT_500m	0.003	0.004	0.604	0.546	1.003	0.994	1.011
High TC	(Intercept)	-0.518	0.082	-6.337	0.000	0.596	0.507	0.699
	Distance_nearest_Majart	0.358	0.443	0.809	0.419	1.430	0.599	3.457
	Street_Length_500m	-0.057	0.028	-2.034	**0.042**	0.944	0.893	0.998
	Distance_nearest_POE	-0.001	0.018	-0.033	0.974	0.999	0.965	1.035

	InvSqDist_POE	0.200	0.104	1.916	0.055	1.221	1.023	1.577
	Traffic_VMT_500m	0.001	0.004	0.306	0.760	1.001	0.993	1.010
High TG	(Intercept)	0.237	0.081	2.933	0.003	1.267	1.082	1.485
	Distance_nearest_Majart	1.296	0.499	2.598	**0.009**	3.653	1.416	9.979
	Street_Length_500m	0.044	0.028	1.553	0.121	1.045	0.988	1.104
	Distance_nearest_POE	0.033	0.018	1.829	0.067	1.033	0.998	1.071
	InvSqDist_POE	0.204	0.150	1.362	0.173	1.226	0.982	1.785
	Traffic_VMT_500m	-0.004	0.004	-0.956	0.339	0.996	0.988	1.004
Low HDL	(Intercept)	0.046	0.079	0.584	0.559	1.047	0.897	1.224
	Distance_nearest_Majart	0.332	0.443	0.749	0.454	1.393	0.590	3.391
	Street_Length_500m	0.033	0.027	1.218	0.223	1.034	0.980	1.090
	Distance_nearest_POE	0.020	0.017	1.132	0.258	1.020	0.986	1.056
	InvSqDist_POE	0.049	0.088	0.556	0.578	1.050	0.891	1.293
	Traffic_VMT_500m	0.001	0.004	0.138	0.891	1.001	0.992	1.009
High Glucose	(Intercept)	-0.603	0.082	-7.328	0.000	0.547	0.465	0.642
	Distance_nearest_Majart	0.431	0.446	0.966	0.334	1.539	0.636	3.727
	Street_Length_500m	-0.007	0.028	-0.255	0.799	0.993	0.940	1.049
	Distance_nearest_POE	-0.016	0.018	-0.858	0.391	0.985	0.950	1.020
	InvSqDist_POE	0.088	0.084	1.045	0.296	1.092	0.927	1.321
	Traffic_VMT_500m	-0.002	0.004	-0.416	0.677	0.998	0.989	1.007
Metabolic Syndrome	(Intercept)	0.092	0.080	1.150	0.250	1.096	0.937	1.283
	Distance_nearest_Majart	0.383	0.449	0.853	0.394	1.466	0.617	3.623
	Street_Length_500m	0.048	0.028	1.739	0.082	1.050	0.994	1.109
	Distance_nearest_POE	0.029	0.018	1.614	0.106	1.029	0.994	1.066
	InvSqDist_POE	0.142	0.123	1.150	0.250	1.152	0.949	1.583
	Traffic_VMT_500m	-0.004	0.004	-0.980	0.327	0.996	0.988	1.004

All significant predictors and corresponding *p* are expressed in bold.

VITA

Juan Aguilera has an educational background that includes medicine, public health, and interdisciplinary research in the border community of El Paso and Ciudad Juarez. He has a Medical Doctor (MD) degree from the University of Ciudad Juarez (UACJ) and a Master's in Public Health (MPH) from The University of Texas at El Paso (UTEP). His research focuses on assessing risk factors for metabolic syndrome and their complications such as hypertension and diabetes in Hispanic populations. He was awarded the *Paso del Norte Health Foundation Fellowship* and served at The Paso del Norte Institute for Healthy Living, an organization that provided leadership and innovative approaches to support regional community efforts to promote healthy eating, active living, and metabolic health. Concurrently, he coordinated the "Evidence Based Screening for Cardiorespiratory Health Outcomes in Low Income Communities from El Paso", a study funded by the City of El Paso's Department of Public Health. Juan also served as President of the Graduate Student Assembly (2019-20), was recognized as an *Agent of Change*, and received the *2019 Distinguished Leader Award*, the most prestigious student leadership award at UTEP. Juan will continue his professional development as a postdoctoral scholar at Stanford University.

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