CAN THE ENVIRONMENTAL PROTECTION AGENCY'S AIR QUALITY INDEX (AQI) BE USED TO PREDICT PEDIATRIC EMERGENCY ROOM ADMISSIONS FOR ASTHMA?

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BY

MARTHA TREBER, B.S., R.R.T.-N.P.S., C.P.F.T., A.E.-C.

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DENTON, TEXAS

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Date

To the Dean of the Graduate School:

I am submitting herewith a thesis written by Martha Treber entitled "Can The Environmental Protection Agency's Air Quality Index (AQI) Be Used To Predict Pediatric Emergency Room Admissions For Asthma?" I have examined this thesis for form and content and recommend that it be accepted in partial fulfillment of the requirements for the degree of Master of Science with a major in Health Studies.

Kristin Wiginton
Dr. Kristin Wiginton, Major Professor

We have read this thesis and recommend its acceptance:

Jody S. Ooman

Susan Ward
Department Chair

Accepted:

Jennifer Martin
Dean of the Graduate School

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ABSTRACT

MARTHA TREBER, B.S., R.R.T.-N.P.S., A.E.-C.

CAN THE ENIRONMENTAL PROTECTION AGENCY'S AIR QUALITY INDEX (AQI) BE USED TO PREDICT PEDIATRIC EMERGENCY ROOM ADMISSIONS FOR ASTHMA?

MAY 2004

In 2000, the EPA implemented a color-coded system for easier understanding by the general public to be forewarned of unhealthy air quality days. This study reviewed daily ER admissions at an urban pediatric hospital in Dallas, Texas for the months of May through September 2000, in an effort to predict increased admissions for asthma during high ozone episodes. If the color-coded AQI could be used to predict admissions for asthma, then the hospital would be able to better prepare for that increase. From using a regression analysis with a one-day lag and a two-day lag, no correlation was found between the high ozone days and ER admissions for asthma. Future research should adjust for other variables, such as weather conditions and days of the week, use of controller medications and will compare other health care facilities' asthma admissions for the same days.

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CHAPTER I

INTRODUCTION

Rationale

Despite the numerous advances in asthma therapy during the last few decades, asthma remains a major public health problem. In the United States, an estimated twenty million people have been diagnosed with asthma sometime during their lifetime and five thousand die each year from an asthma exacerbation (Respiratory Institute, 2003). From 1980 to 1996, the incidence of asthma more than doubled, with children less than five years of age experiencing the greatest increase. With this increase, asthma has become the leading cause of childhood morbidity (American Lung Association [ALA], 2003d). There are approximately two million emergency room (ER) visits for asthma each year and nearly 500,000 hospitalizations. Additionally, asthma accounts for 14.5 million missed workdays and 10 million missed school days each year. Annually, 14 billion dollars are spent on direct and indirect asthma expenses (Centers for Disease Control [CDC], 2001).

In susceptible persons, asthma may be triggered by a variety of factors: respiratory tract infections, environmental agents (i.e., airborne pollens and molds), allergenic foods, household dusts, inhalant allergens, sudden changes in the weather, and industrial air pollutants. Evidence indicates that asthma exacerbations are an outcome of these triggers, especially exposure to industrial air pollutants (National Heart Lung Blood Institute [NHLBI], 1997). The American Lung Association (ALA) and the Environmental

Protection Agency (EPA) recently published a meta-analysis that evaluated the health effects of ozone air pollution. The EPA created a panel of medical experts to review the pertinent studies conducted over the course of two and a half years. Over 3,000 studies were reviewed with the advisory panel adopting 1,000 studies on particulate pollution and adopting over 150 studies on ozone to support the new standards for permissible ozone levels. Many of those studies implicated ozone as a significant factor in rising asthma morbidity and mortality (ALA, 2003c). Ozone at ground level develops from pollution reacting chemically with sunlight. High levels of ozone can accumulate during muggy, breezeless summer days and can irritate the throat and chest, reduce lung function and inflame the lining of the lungs, possibly causing damage (Gibbons, 2001).

Harvard Medical School's epidemiological studies of asthma mortality rates in six cities found a clear link between ozone and other air particulates with enhanced allergic response (Gibbons, 2001). Results from these studies indicated that when persons with asthma spent time outdoors on days with high ozone levels, their asthma symptoms were exacerbated. Negative ions from ozone pollution seem to irritate and exert a direct toxic effect on the lungs' bronco-epithelial cells, making it easier for other allergens to get through the epithelium and enhance the asthmatic response (Gibbons, 2001).

In 2000, the EPA developed and implemented a new air quality rating system for ozone (EPA, 1999). The color-coded system rates the Air Quality Index (AQI) as Green-good, Yellow-moderate, Orange-unhealthy for sensitive groups, Red-unhealthy and Purple-very unhealthy. These air quality forecasts are available daily from the EPA and many television and radio stations will announce when the AQI reaches a high level.

People with asthma can use the system to plan their day and help them avoid contact with dangerous levels of ozone.

PURPOSE OF THE STUDY

The purpose of this study is to determine if the EPA's color-coded Air Quality Index rating system is a significant predictor of pediatric emergency room admissions for asthma. Based on the results of this study, emergency room personnel could potentially predict and prepare for increased admissions for asthma exacerbations due to high ozone levels.

HYPOTHESES

This study has the following null hypotheses:

1. The AQI rating for ozone (PPB) for the previous day is not a significant predictor of pediatric emergency room admissions for asthma.
2. The AQI rating for ozone (PPB) for two days prior to admission is not a significant predictor of pediatric emergency room admissions for asthma.

DEFINITION OF TERMS

Asthma-from the Greek word for panting; a chronic, inflammatory disease of the lungs.

Ozone-a blue, gaseous allotrope of oxygen, O_3, derived or formed naturally from diatomic oxygen by electric discharge or exposure to ultraviolet radiation; measured in "parts per billion (PPB)".

Asthma exacerbation-also known as "asthma attack", when the lungs of those with the disease have increased inflammation, mucus production and bronchospasm, making it difficult to breathe.

Environmental Protection Agency- (EPA) government agency developed to protect human health and to safeguard the natural environment.

LIMITATIONS

The following limitations were recognized for this study:

1. Emergency room admissions will be collected from only one hospital in a downtown area.
2. Data is secondary data collected over the course of one year.
3. Sample size is small, due to the lack of data from other hospitals in the surrounding communities.

DELIMITATIONS

The delimitations for this study are as follows:

1. The patients admitted to the hospital emergency room were residents from the surrounding communities of the city hospital.
2. This study will use daily emergency room admissions at an urban pediatric hospital for the ozone season from May to September.

ASSUMPTIONS

1. All daily emergency room admissions for an asthma exacerbation have been counted.
2. Only those with a severe asthma exacerbation are brought to the emergency room.

CHAPTER II

REVIEW OF LITERATURE

The present review includes statistical information on asthma, a historical perspective of asthma, descriptions of studies on the association of air pollution and asthma exacerbations and deaths, and results from studies on the effects of ozone on prevalence of pediatric asthma. Due to the nature of this study, findings related to other asthma triggers and adult asthma will not be discussed.

Statistical Information on Asthma

The U.S. Department of Health and Human Services (USDHHS) has identified asthma as one of the three national health priorities. Asthma was selected because it is one of the most prevalent, costly, and yet, preventable health problems. According to the American Lung Association (ALA), there are approximately twenty million (73.4/1,000 population) people in the United States affected by asthma and five million of those are children under the age of 18. The highest prevalence rate is in the 5-17 year age group (98.1 per 1,000 population), with prevalence rates decreasing with age (Respiratory Institute, 2003). More than 31 million Americans have been diagnosed with asthma at some point in their lifetime and, if left untreated, asthma can lead to a long-term decline in lung functions (Respiratory Institute, 2003). During 2001, twelve million Americans (four million children under age 18) experienced an asthma attack. This represents 59% of the population that currently has asthma (ALA, 2003d). Each year, there are two million emergency room visits, half a million hospitalizations, fourteen million missed

work-days, ten million missed school days, and fourteen billion dollars spent on direct and indirect asthma care (Respiratory Institute, 2003).

The American Lung Association (ALA) is a premier source for trends in asthma mortality and morbidity. The ALA has used information gathered from the National Health Interview Survey (NHIS) as a principal source of asthma prevalence data since 1979 (ALA, 2003b). The organization also utilizes data collected from its own Epidemiology and Statistics Unit. Overall, ALA sponsored research has been able to prove that, within the United States, the burden of asthma has increased over the past 20 years. Within the past few years, death and hospitalization rates due to asthma have plateaued which indicates disease management is taking place. In 2000, there were 4,487 deaths due to asthma and 65% of those deaths were among women, with black women having the highest morality rate (4.2 per 100,000). Of the 292 deaths among Hispanics, women had the highest mortality rate (1.5 per 100,000). Asthma deaths are rare among children and highest among those over age 85. In 2000, 223 children, aged 0-17 (0.3 per 100,000), died from asthma compared with 707 adults over age 85 (16.5 per 100,000) (ALA, 2003d).

In 2001, the ALA reported that, of those with asthma, 42% were males and 57% were females. By race, 72% were white, 14.5% were Non-Hispanic blacks, 10% were Hispanic, and 3.6% were Non-Hispanic other. By age, 38.4% were 18-44 years, 25.6% were 5-17 years, 20.7% were 45-64 years, 9.7% were over age 65, and 5.6% were under age 5 years. Whites had the lowest prevalence rates in those under age 5 and blacks had

the lowest in those over age 65. The highest prevalence rates for both races were among those 5-17 years of age (ALA, 2003d).

Asthma Through the Ages

The word "asthma" is taken from the Greek word for panting. Greek and Roman physicians used the term to describe a shortness of breath (National Library of Medicine [NLM], 1999). It was thought that asthma attacks occurred due to an imbalance of the four body humors that controlled health—yellow bile, black bile, blood, and phlegm. A Greek physician, Aretaeus, who practiced in Rome, first described asthma as the disease known today. He noted symptoms of "heaviness of the chest, difficulty of breathing when running or during other exertions, there is wheezing and hoarseness. The cheeks become ruddy, the eyes protuberant, there is a need for air, there is an incessant and laborious cough and if the symptoms persist, suffocation" (NLM, 1999, pg. 9).

It was not until 1190 that the first treatment for asthma was prescribed by Moses Maimonides, a physician for the Sultan of Egypt who was treating the Sultan's asthmatic son. Maimonides could not cure his patient's asthma, but he did recommend living in a dry climate, keeping calm, living a moderate lifestyle, and eating chicken soup. His was the only practical advise available for the next several hundred years (NLM, 1999).

With the rediscovery of classical Greek thought and improved knowledge of anatomy and pathology, asthma began to be viewed as a specific disease. In 1670, Thomas Willis, an English physician, suggested that asthma was due to a spasm in the airways. The results of his studies led to a better understanding of asthma as a bronchial disease. William Cullen, a Scottish physician born in 1712, suggested that asthma attacks

were caused by extrinsic factors. He recommended avoiding dust, pollens and airborne inhalant allergens (NLM, 1999).

In 1860, an English physician, Henry Hyde Salter prescribed asthma treatments that were widely used into the twentieth century. He believed that various triggers such as animal dander, impure air, hay fever, and foods could initiate an attack. Salter's work led to treatments using ma huang or ephedra. One way of controlling an attack was to have the patient inhale an antispasmodic agent to the lungs, ironically, by smoking it. Another treatment was to drink strong, hot coffee to treat attacks. It is now understood that coffee has the pharmacological property of relaxing spasms (NLM, 1999).

Recent Advances in Asthma Diagnosis and Guidelines

The National Institutes of Health (NIH), through the National, Health, Lung, and Blood Institute (NHLBI), the National Institute of Allergy and Infectious Diseases (NIAID) and the National Institute of Environmental Health Sciences (NIES), has mounted a major effort to discover effective ways to manage and treat asthma. In 1991, a NHLBI expert panel published guidelines for asthma management, establishing the modern definition of asthma as "a chronic inflammatory disorder of the airways in which many cells and cellular elements play a role, in particular, mast cells, eosinophils, T lymphocytes, neutrophils and epithelial cells" (NHBLI, 1997). The guidelines also note that in susceptible individuals, this inflammation causes recurrent episodes of wheezing, breathlessness, chest tightness and cough, particularly at night and in the early morning and that these episodes are associated with widespread, variable airflow obstruction that is reversible either spontaneously or with treatment. The inflammation also causes an

associated increase in the existing bronchial hyperresponsiveness to a variety of stimuli (NHLBI, 1997).

These guidelines have become a standard of care for long-term management. The goals of asthma management are to: (1) prevent chronic and troublesome symptoms, (2) maintain normal pulmonary function, (3) provide optimal pharmacotherapy, (4) maintain normal activity levels, (5) prevent recurrent exacerbations, and (6) meet the needs of the patient and family. The recommendations for the treatment of asthma have been organized into four components for effective management. Component One recommends the use of objective measures of lung function to assess the severity of asthma and to monitor the course of therapy. There are several types of monitoring recommended: signs/symptoms, pulmonary function, quality of life/functional status, history of asthma exacerbations, pharmacotherapy, and patient-provider communication and patient satisfaction (NHLBI, 1997).

Component Two addresses recommendations for environmental control measures to help avoid or eliminate factors that precipitate asthma symptoms or exacerbations. Exposure of sensitive patients to inhalant allergens has been shown to increase airway inflammation, airway hyperresponsiveness, asthma symptoms, need for medication, and mortality due to asthma. Reducing exposures significantly reduces these outcomes. Component Three addresses comprehensive pharmacologic therapy for long-term management. It is designed to reverse and prevent the airway inflammation of asthma as well as manage asthma exacerbations. Lastly, Component Four addresses the teaching of patients and their families in regards to asthma self-management (NHLBI, 1997).

Environmental Effects on Asthma

As the data shows, asthma is a major health concern, considering that asthma exacerbations can be prevented and asthma can be controlled. Significant advances have occurred over the past 15 years in the understanding of asthma. Allergies play a major role in exacerbating asthma. Upper respiratory viral infections are triggers, as well (NHLBI, 1997). Environmental tobacco smoke is an important irritant. Approximately 29 percent of households still expose children to secondhand smoke in the home on a regular basis (President's Task Force, 2000). Other indoor and outdoor pollutants are respiratory irritants as well. Exposure to outdoor air pollutants such as ozone, sulfur dioxide (SO_2), and particulate matter, can exacerbate asthma. Evidence also suggests that exposure to ozone can increase a person's response to inhaled allergens (Peden, 1995). Thus, as noted in the NHLBI guidelines, reducing exposures significantly reduces attacks (NHLBI, 1997).

Air pollution primarily consists of particulate matter (PM), nitrogen dioxide (NO_2) and sulfur dioxide (SO_2). PM is a mixture of substances that includes carbon-based particles, dust, and acid aerosols. A new monitoring system from the EPA tracks fine PM with a diameter of 2.5 microns or less. Exposure to PM can shorten human life and can trigger attacks in sensitive individuals. NO_2 is formed when fuel is burned at high temperatures. On-road vehicles and electric utilities are major sources for NO_2. SO_2 is formed when fuel with sulfur is burned and during metal smelting. Electric utilities and industrial fuel combustion are principle sources of PM (ALA, 2000).

In Texas, over 292 schools are located within a two-mile radius of a major polluter (Public Employees for Environmental Responsibility [PEER], 2003). More than one-third of these schools are within two miles of multiple plants, increasing the potential for exposure to air pollutants. Over 225,000 children attend schools near facilities that are responsible for emitting over 300,000 tons of smog each year. A 1996 report from the Natural Resources Defense Council estimated that Texas power plants emit particulates that are responsible for 2,617 premature deaths of Texans each year (PEER, 2003). In 1990, American industries emitted more than 2.4 billion pounds of toxic pollutants into the air (Methodist Health Care System, 2003).

Ozone Standards

Major sources of NO_2, such as motor vehicle exhaust and industrial emissions, gasoline vapors, and chemical solvents help to form ozone (EPA, 2002). In 1991, 98 American areas exceeded the EPA's recommended levels for ozone (Methodist Health Care System, 2003). In the atmosphere, ozone serves as a protecting shield from the sun's harmful ultraviolet rays, but at ground-level, it interferes with the ability of plants to produce and store food, damages the leaves of trees and other plants, reduces crop and forest yields, and increases plant vulnerability to disease, pests, and harsh weather (EPA, 2002). Sunlight and hot weather cause ground-level ozone to form harmful concentrations in the air (Gibbons, 2001). Thus, peak ozone levels occur during hot, dry, stagnant summertime conditions (EPA, 2002).

In 1997, the EPA set the national ambient air quality standards for ground-level ozone at 0.08 ppm per eight hours (85ppb). This was a change from the one-hour

standard of 0.12 ppm (125ppb) (Texas Natural Resources Conservation Commission [TNRCC], 2002). The EPA has determined that any ozone level over 0.08 ppm averaged for an 8-hour period is a violation (EPA, 2003). During the years 1994-1996, 270 counties in 33 states were in violation (ALA, 2000). High ozone levels can occur anywhere, but certain environmental conditions promote its accumulation. According to the American Academy of Allergy, Asthma, and Immunology, areas with heavy automobile traffic, heat inversions and places that receive a lot of sunlight are more prone to experience high ozone levels (Gibbons, 2001).

Nearly half of the United States population lives in areas with unhealthy levels of ozone (ALA, 2003c). Since 1996, the Texas counties of Collin, Dallas, Denton, and Tarrant have all received an 'F' rating for the high number of days with excessive levels of ozone. Unfortunately, these same counties have a large number of at-risk populations. Of the 2.2 million residents in Dallas County, 10% have asthma, chronic bronchitis or emphysema. Houston and Dallas/Fort Worth are in the top 25 of America's most ozone-polluted cities. By contrast, Laredo and Brownsville are among the least ozone-polluted cities in the U.S. (ALA, 2003c).

In 1976, the EPA established a national uniform air quality index (AQI), called the Pollutant Standard Index (PSI), to be used on a voluntary basis by state and local agencies. This index was established in light of a study conducted by the EPA and the President's Council on Environmental Quality. The results of that study indicated that 55 urban areas in the U.S. and Canada used different indices to report air quality (EPA, 1998).

In 1998, the EPA proposed to change the AQI used by states for daily air quality reporting to the general public (EPA, 1998). The new reporting system included the following categories: "moderate" (51 to 100 ppm), "unhealthy for sensitive groups" (101 to 150 ppm), "generally unhealthy" (151 to 200 ppm), "very unhealthful" (201 to 300 ppm), and "hazardous" (over 301 ppm). These categories were based on a 1996 report that assessed the health risks associated with short-term exposure to tropospheric ozone (EPA, 1998). This report estimated that, for healthy individuals engaged in prolonged moderate exertion, the following risks would occur above a level of 0.12 ppm (8-hour average): 1) approximately 50% would experience temporary moderate lung function impairment, 2) approximately 20% would experience temporary large lung function impairments, and 3) approximately 10 to 15% would experience temporary moderate to severe respiratory symptoms (e.g. chest pain and aggravated cough). Individuals with asthma or other respiratory conditions would be more severely affected than healthy individuals, leading some to increase medication usage and seek medical attention, resulting in increased doctor's visits, emergency room and clinic visits, and hospitalizations (EPA, 1998).

In 1998, the EPA utilized focus groups with the general public to test their understanding of the new categories. From the groups' responses and comments about potential confusion, the EPA added specific colors to be associated with each of the categories. This uniform reporting system allowed for a clearer message to be conveyed to the general public about air quality and associated health effects (EPA, 1998). In August 1999, the EPA published its final rules on AQI Reporting, which allowed for state

and local agencies to report air quality to the general public using the specific color-coded system (EPA, 1999).

The Texas Commission on Environmental Quality (TCEQ, formerly known as the TNRCC) is the ozone forecasting resource for Texas. Ozone Action Day is when agency meteorologists predict the next day's weather conditions to be favorable for high levels of ozone. These predictions are based on a set of criteria from historic meteorological data and ozone measurements to make these predictions. When the TCEQ predicts an ozone day, they contact the National Weather Service, which relays the information to area forecasters, officials in the affected area, local media, governments, businesses and industries. This forecasting is an effort to help the community plan in advance the steps necessary to reduce the pollutants that contribute to ozone formation (TRNCC, 2002). The EPA's color-coded system offers the ozone forecasting at a level the general public can understand. Additionally, by using this system to predict unhealthy ozone levels, hospitals and other health care facilities are better prepared for an increase in patients who might succumb to the ozone's harmful effects.

Physiological Effects of Ozone and Other Pollutants

The ALA identifies sensitive groups as children, the elderly, individuals with lung disease, outdoor workers, and healthy adults who exercise outdoors. When a sensitive individual breathes in ozone, it chemically reacts with internal body tissues and the lungs become red and inflamed (ALA, 2001). The person begins to cough and experience shortness of breath and a burning sensation, which is due to the oxidation that is occurring in the lungs from contact with the ozone. Prolonged exposure (6.5 hours) to

low levels of ozone can reduce lung functions and cause lung inflammation (ALA, 2001). Results from one study indicated that long-time residents of Los Angeles had higher than expected loss of lung function over time (ALA, 2000a). In another study from Sweden, results indicated that, among those diagnosed with asthma who were exposed to ozone, there was a significant increase in Il-4 and Il-5 cytokines, causing inflammation of the airways (Bosson, et al. 2003).

Ozone has other damaging effects on healthy individuals as well. In one study, healthy men who exercised, suffered significant symptoms such as shortness of breath, chest tightness, and wheezing at low ozone concentrations (Brunekreef, Hoek, Breugelmans, and Leentvaar, 1994). In another study healthy young adults developed significant lung function reductions, additional coughing and breathing pains and increased airway reaction to irritants when exposed to ozone levels between 80 and 120 ppb during moderate exercise for five hours. The exercises utilized in this study were designed to mimic the activities of construction workers (Devlin et al. 1991).

From 1993-1994, the Small Area Variations in Air pollution and Health (SAVIAH) study was conducted at the following four centers: Huddersfield, England; Prague, Czech Republic; Poznan, Poland; and Amsterdam, Netherlands. Researchers collected individual data, small geographical data, and air pollution data for 7-10 year old children who lived in the study areas. Parents completed a questionnaire covering socio-economic circumstances, housing, cooking, heating, and parental smoking. The questionnaire also asked about family health history, atopy, health of the child and presence of asthma. Geographical data included information on area population, levels of

education, housing, crowding, water and gas supplied at home, indoor plumbing, car ownership and traffic flows. The study measured NO_2 and SO_2 concentrations in three two-week campaigns (October 1993, February 1994, May 1994). Amazingly, 3,680 out of 4,176 questionnaires were returned. After analysis, researchers found that wheezing prevalence was higher in boys with atopic history and dampness in the home and lower among children with higher educated mothers. They also found an association between NO_2, SO_2 and wheezing despite socio-economic differences (Pikhart et al. 2000).

Researchers in West Germany conducted an investigation of the relationship between traffic related air pollution and atopic sensitization. They enrolled 317 9-year-old children living near major roads in two urban and one suburban cities in West Germany. They wanted to measure the health effects of traffic pollution on children living in urban and suburban areas by measuring individual outdoor NO_2 and personal NO_2. Outdoor NO_2 was highest in Friedrichstadt and Dusseltal, which were the urban areas and lowest in Hellerhof, the suburban area. Results indicated a relationship between atopy and outdoor NO_2, which strengthened the theory that traffic-related air pollution leads to an increased prevalence of atopic sensitizations, allergic symptoms, and diseases (Kramer, Koch, Ranft, Ring, & Behrendt, 2000).

In another study, researchers assessed 8-13 year old children in the Los Angeles and Pasadena areas by having them complete a questionnaire and keep daily diaries of medication use. Daily data for ambient air pollution, weather conditions, pollens, and molds were collected as well. Results indicated the LA participants had higher daily averages of cough, shortness of breath and wheezing (15.1% to 11.5%) than those

participants in Pasadena, and found ambient air pollution significantly affected African American asthmatic children. It was determined that asthma exacerbations were associated with particulate matter and mold counts. Particulate matter and ozone levels were also associated with increases in reports of shortness of breath (Ostro, Lipsett, Mann, Braxton-Owens, & White, 2001).

Researchers at the University of California at Los Angeles reported that non-smoking men and women living in areas with relatively high levels of ozone and other air pollutants had approximately one-half to three-quarters the lung function damage of a one-pack-a-day smoker (Tashkin et al. 1994). Results from another study indicated that ozone causes inflammation in the upper airways of normal, healthy children at concentrations well below the current health standard (Frischer et al. 1993).

In a prospective cohort study conducted by a team from Keck School of Medicine at the University of Southern California, 3535 children from 12 communities in southern California were followed for up to five years (McConnell et al. 2002). None of the children were asthmatics at the onset of the investigation. The communities were chosen partly on the basis of their level of ozone concentration: six had high levels and six had low levels. In the follow-up, 265 of the children had been diagnosed with asthma. Those children who exercised heavily in areas of high ozone were 3.3 times more likely to develop asthma in comparison with children in the low ozone areas. The results also indicated that children who played heavily in low ozone areas were at no greater risk from developing asthma than those who did not participate in sports in the same environment (McConnell et al. 2002).

When individuals with asthma breathe in allergens after exposure to ozone, they suffer a more pronounced allergic response than if they were exposed to the allergen alone (Peden, Setzer, & Devlin, 1995) Dr. Janneane Gent of Yale University School of Medicine observed a significant link between ozone and respiratory symptoms. For every 50 ppb increase in 1-hour ozone level, the likelihood of wheezing and chest tightness increased by 35% among children using maintenance medications (Gent, 2003).

In 2002, researchers in the Department of Preventive Medicine, College of Medicine at Ewha Women's University in Seoul Korea, conducted a study and reported a significant increase in school absenteeism on high ozone days (Park et al. 2002). Another study conducted by researchers from the Keck School of Medicine in southern California found that ozone levels had an impact on respiratory illness-related school absences in children (Gilliland, et al. 2000).

Emergency Room Visits and Admissions

ALA studies have indicated that air pollution has a significant potential to harm children with asthma and elderly individuals. On high ozone days, children with asthma were 40% more likely to suffer asthma attacks compared with days with average ozone levels. Another ALA study found that when air pollution increases, ER visits from the elderly increase (ALA, 2003a). Results from a study in Houston indicated that a person with asthma had a 60% higher probability of having an attack for every 100 ppb increase in the maximum one-hour ozone concentration (Holguin, et al. 1985). When sensitive individuals have an asthma attack, they head for the emergency room. Researchers at the Robert Wood Johnson Medical School studied the relationship between ozone levels and

emergency room admissions for asthma. They found that when ozone levels were above 60 ppb, there was a 28% increase in emergency room admissions, compared with admissions when the ozone level was below 60 ppb (Weisel, Cody, & Lioy, 1995). Data from Toronto and Southern Ontario revealed significant increases in hospital admissions due to ozone and acidic air pollution, even at levels below the health standard at the time (Thurston, Ito, Hayes, Bates, & Lippmann, 1994).

During the 1996 summer Olympic Games in Atlanta, there was a reduction in asthma admissions to the ER. A retrospective study determined that the reduction in traffic congestion in downtown Atlanta during the games resulted in less traffic density during the morning rush hour. This reduced ozone pollution and significantly reduced childhood asthma events (Friedman, Powell, Hutwagner, Graham, & Teague, 2001).

Results from a study conducted at the Children's Hospital Medical Center in Cincinnati, Ohio, indicated that exacerbations of asthma severe enough to require visits to the hospital were associated with elevated concentrations of airborne pollens and particulates, with a significant delayed effect (Lierl & Hornung, 2003). At Children's Medical Center of Dallas, asthma is one of the top three admitting diagnoses. The emergency room is considered one of the busiest in the country, with over 8,000 children seen for respiratory illnesses each year and asthma accounts for almost 5,000 of those emergency room visits (CMC, 2003).

A study conducted in Washington state indicated that one in eight emergency room visits for asthma was linked to particulate matter (PM). The main sources of PM are motor vehicles, wood stoves, open burning, and industrial emissions. The health effects

from these combustion sources are primarily from fine particles ($PM_{2.5}$). This research found an association between particulate matter less than 10 microns in size and respiratory hospital admissions in Spokane. Results also revealed that cardiopulmonary emergency room visits and hospitalizations doubled on days with high ozone levels in some areas of the United States (Schwartz & Neas, 2000).

Mortality Studies

Studies have also indicated a link between ozone levels and mortality. A 1991 study reported an increase of approximately two deaths per 1000 persons at a 10% increase above average ozone levels (Kinney & Ozkaynak, 1991). A Canadian study reported that about eight percent of all non-traumatic deaths in 11 Canadian cities were attributable to the combined effects of carbon monoxide, nitrogen dioxide, ground-level ozone and sulfur dioxide, which translates into about 5,000 preventable premature deaths each year (Burnett et al. 1998). In 1997, Dutch scientists, found that chronic exposure to particulate pollution could shorten lives by one to three years (Brunekreef, Hoek, Breugelmans, & Leentvaar, 1997).

Researchers from Johns Hopkins University assessed the effects of five major outdoor-air pollutants on daily mortality rates in the 20 largest metropolitan areas in the United States (1987 to 1994). They measured daily rates of particulate matter, ozone, carbon monoxide, sulfur dioxide and nitrogen dioxide. Results indicated that the level of particulate matter was positively associated with death rates from all causes in most of the 20 cities. This association was not affected by the inclusion of other pollutants (Samet, Dominici, Curriero, Coursac, & Zeger, 2000).

At Harvard Medical School, researchers collected air pollution data from six cities in the United States: Watertown, Massachusetts; Kingston-Harriman, Tennessee; St. Louis, Missouri; Steubenville, Ohio; Portage, Wisconsin; and Topeka, Kansas. They collected for fine particles $PM_{2.5}$ and PM_{10}. These researchers hypothesized that particle size was an important determinant of the site and efficiency of pulmonary deposition. They analyzed the total daily deaths as well as daily deaths from heart disease, pneumonia, and Chronic Obstructive Pulmonary Disease in each of the cities. They were able to conclude that an increase in $PM_{2.5}$ accounted for a 3.4 percent increase in the daily mortality rate (Laden, Neas, Dockery, and Schwartz, 2000).

There have been multiple studies conducted on the associations between day-to-day changes in air pollution and day-to-day changes in death rates in some cities. A researcher at the Environmental Epidemiology Program at Harvard's School of Public Health examined associations between air pollution that lagged for more than one day and daily deaths of persons 65 years of age and older using PM_{10} levels in the following ten U.S. cities: New Haven, Birmingham, Pittsburgh, Canton, Detroit, Chicago, Minneapolis, Colorado Springs, Spokane, and Seattle. The researcher controlled for temperature, humidity, barometric pressure, day of the week, and seasonal patterns. Results from this study indicated that, in each city, the effect of one day's exposure to PM_{10} lasted up to five days (Schwartz, 2000).

In early December, 1952, a sharp drop in temperature and strong winds hit London. In an effort to keep warm, the residents began to use more coal. The cold air trapped the exhaust close to the ground. For over five days, London had the worst air

pollution it had ever seen. Some 4,585 people died from respiratory related illnesses during those five days and an additional 8,000 died in the following months. Currently, researchers are taking lung samples from the victims of the Great Smog of 1952. They have found high concentrations of fine particulate matter in the lungs of 16 people whose deaths were attributed to the smog (Nagourney, 2003).

Similar air pollution disasters have occurred elsewhere: Meuse Valley in Belguim in 1930, and Donoroa, Pennsylvania in 1948. The air pollution acted primarily to exacerbate preexisting conditions such as pneumonia and chronic conditions such as heart disease (Schwartz, 1994). A report from the British Lung Foundation revealed that over a third of the population is particularly susceptible to the effects of pollution. More than 80% of people in the UK live in urban areas where transport pollution tends to be concentrated (British Lung Foundation, 1998). Today, an estimated 12,000 people die every year in Britain from exposure to pollution and 9,000 are hospitalized for pollution-related respiratory illnesses (Derbyshire, 2002).

Ozone and Development of Asthma

Research is being conducted to uncover the relationship between the development of asthma and exposure to ozone. In 2000, results from a study at the University of California at Davis indicated that occasional exposure to ozone can change how the lungs of young rhesus monkeys develop and can lead to a disease similar to childhood asthma in humans (Fell, 2000). In this study, monkeys of different ages were exposed to ozone for five days, followed by nine days of clean air. The cycle was repeated every two weeks for five months, simulating the effect of ozone exposure as it occurs in the Sacramento

area. Some of the monkeys were also exposed to dust mite allergen. The adult monkeys exposed to the allergen developed lung disease equal to human asthma. Their disease became worse when they were also exposed to ozone. When young monkeys were exposed to this cycle of simulated ozone conditions soon after birth, they showed changes in lung development. The young monkeys' lungs had fewer branches and there were other structural and cellular changes similar to what would be found in human asthmatics (Fell, 2000).

Dr. Rob McConnell has been conducting research for ten years with children and their exposure to ozone. He enrolled 3,500 children who lived in 12 southern California cities. Six were clean air counties and six had high ozone periods. During the 10 years, two hundred and sixty-five new cases of asthma were reported among the children. Children who lived in areas with high ozone levels and played several team sports tripled their risk of developing asthma. This study provided evidence that ozone is involved in the development of new-onset of asthma in children who exercise heavily (McConnell, et al. 2002).

CHAPTER III

RESEARCH METHODOLOGY

Procedures

This study utilized secondary data collected at Children's Medical Center (CMC). ICD-9 codes for asthma were reviewed for daily emergency room (ER) admissions during the months of May through September of 2000. Daily ozone levels for the same time period were also collected. These levels were defined as 0-100 PPM for low ozone days were and 101 to 300 PPM for high ozone days. These levels were in accordance to the Environmental Protection Agency's (EPA) standards as indicated on the Air Quality Index (AQI) Scale. Multiple regression analyses were completed to establish whether the AQI ranking for ozone was a significant predictor of pediatric ER admissions for asthma.

Children's Medical Center is located near the downtown area of a large metropolitan city. With a population of over one million people, pollution from cars and industries is a daily occurrence. From the months of May through September, high temperatures and low wind velocities are common and create increases in ozone levels. CMC's location and high asthma admissions made it an ideal facility for this study's purpose which was to determine if ER admissions for asthma could be predicted on high ozone days.

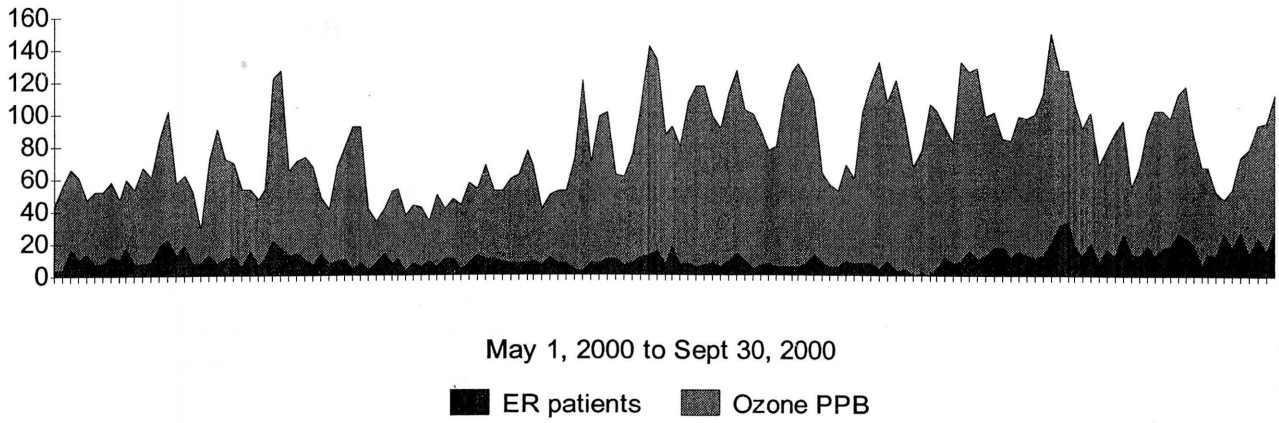
For these reasons, only days from May to September were utilized. ER admissions during low ozone days were compared to ER admissions during high ozone days. The numbers of patients admitted to CMC's ER for an asthma exacerbation were

treated in the ER during that time period. The TCEQ (formerly the TNRCC) was contacted to collect ozone data for the Dallas/Fort Worth area for that same time period. From May to September of 2000, ozone levels in Dallas ranged from 21 ppb to 130 ppb.

CHAPTER IV

RESULTS

The numbers of patients admitted to CMC's ER for an asthma exacerbation were collected from May 1, 2000 to September 30, 2000. A total of 1640 asthma patients were treated in the ER during that time period. The TCEQ (formerly the TNRCC) was contacted to collect ozone data for the Dallas/Fort Worth area for that same time period. From May to September of 2000, ozone levels in Dallas ranged from 21 ppb to 130 ppb.



SPSS was used to conduct a time series regression analysis of the data, with ER admissions as the dependent variable and ozone level as the independent variable. Initially, to determine significance, was conducted. When using time series data, a problem that sometimes occurs involves a correlation of the errors of prediction. This type of correlation is called auto correlation. If the errors of prediction are positively correlated over time, least-squares regression will underestimate the actual standard error

of the regression formula. A Durbin-Watson test determines if the auto correlation is large enough to use another procedure caller the generalized least squares. A Durbin-Watson of .866 indicated an auto correlation that was not significant (p=.643).

Variables Entered/Removed [b]

Model	Variables Entered	Variables Removed	Method
1	PPB [a]		Enter

a. All requested variables entered.
b. Dependent Variable: ERC

Model Summary [b]

Model	R	R Square	Adjusted R Square	Std. Error of the Estimate	Durbin-Watson
1	.038 [a]	0.001	-0.005	6.226	0.866

a. Predictors : (Constant), PPB
b. Dependent Variable: ERC

ANOVA [b]

Model		Sum of Squares	Df	Mean Square	F	Sig.
1	Regression	8.36	1	8.36	0.216	0.643
	Residual	5853.758	151	38.767		
	Total	5862.118	152			

Coefficients [a]

Model		Unstandardized Coefficients		Standardized Coefficients	t	Sig.
		B	Std. Error	Beta		
1	(Constant)	10.258	1.396		7.347	.000
	PPB	8.70E-03	0.019	0.038	0.464	0.643

a. Dependent Variable: ERC

A simple regression of the two variables with a one-day lag revealed no significant correlation with a Durbin-Watson of .884 showing an auto correlation (p=.785).

Variables Entered/Removed [b]

Model	Variables Entered	Variables Removed	Method
1	F3 [a]		Enter

a. All requested variables entered.
b. Dependent Variable: F2

Model Summary [b]

Model	R	R Square	Adjusted R Square	Std. Error of the Estimate	Durbin-Watson
1	.022 [a]	.000	-0.006	6.071	0.844

a. Predictors : (Constant), F3
b. Dependent Variable: F2

ANOVA [b]

Model		Sum of Squares	df	Mean Square	F	Sig.
1	Regression	2.744	1	2.744	0.074	.785 [a]
	Residual	5528.25	150	36.855		
	Total	5530.993	151			

a. Predictors: (Constant), F3
b. Dependent Variable: F2

Coefficients [a]

Model		Unstandardzied Coefficients		Standardized Coefficients		
		B	Std. Error	Beta	t	Sig.
1	(Constant)	11.092	1.37		8.097	.000
	F3	-5.00E-03	0.018	-0.022	-0.273	0.785

a. Dependent Variable: F2

(F2=ERC, F3= PPB, with one-day lag between)

An additional analysis was conducted to remove any residuals. The generalized least squares, one-day lag revealed a Durbin-Watson of 2.312 and no auto correlation, however the F test was not significant (p=.914). Therefore, the first null hypothesis was not rejected.

Model Summary [b]

Model	R	R Square	Adjusted R Square	Std. Error of the estimate	Durbin-Watson
1	.009[a]	0	-.007	5.5451	2.312

a Predictors: (Constant), GPPB
b Dependent Variable: GER

ANOVA[b]

Model		Sum of Squares	Df	Mean Squares	F	Sig.
1	Regression	.296	1	.296	.012	.914[a]
	Residual	3806.670	149	25.548		
	Total	3806.966	150			

a Predictors (Constant), GPPB
b Dependent Vaeriable GER

Coefficients[a]

		Unstandardized Coefficients		Standardized Coefficients	t	Sig.
Model		B	Std. Error	Beta		
1	(Constant)	4.601	.792		5.808	.000
	GPPB	2.386E-03	.022	.009	.108	.914

a Dependent Variable: GER

The process was repeated to addressed the second null hypothesis of two days difference between ER visits and levels of ozone. A simple regression, with two days lag between ER and levels of ozone showed an auto correlation of .855 with a p value of .758 which was not significant.

Model Summary[b]

Model	R	R Square	Adjusted R Square	Std Error of the Estimate	Durbin-Watson
1	.025[a]	.001	-.006	6.219	.855

a Predictors: (Constant), PPBDA2
b Dependent Variable: ER

ANOVA[b]

Model		Sum of Squares	df	Mean Square	F	Sig
1	Regression	3.676	1	3.676	.095	.758[a]
	Residual	5763.000	149	38.678		
	Total	5766.675	150			

a Predictors: (Constant), PPBDA2
b Dependent Variable: ER

Coefficients[a]

		Unstandardized Coefficients		Standardized Coefficients	t	Sig.
Model		B	Std. Error	Beta		
1	(Constant)	10.549	1.408		7.493	.000
	PPBDA2	5.798E-03	.019	.025	.308	.758

a Dependent Variable: GER

To correct for the auto correlation, a generalized least squares analysis was performed. The Durbin-Watson of 2.332 showed that the auto correlation was removed, however, it was not significant (p=.746).

Variables Entered/Removed [b]

Model	Variables Entered	Variables Removed	Method
1	GPPBDA2[a]		Enter

a. All requested variables entered.
b. Dependent Variable:GERDA2

Model Summary [b]

Model	R	R Square	Adjusted R Square	Std. Error of the Estimate	Durbin-Watson
1	.027 [a]	.001	-0.006	5.02456	2.332

a. Predictors : (Constant), GPPBDA2
b. Dependent Variable: GERDA2

ANOVA [b]

Model		Sum of Squares	df	Mean Square	F	Sig.
1	Regression	2.655	1	2.655	0.105	.746 [a]
	Residual	3736.443	148	25.246		
	Total	3739.098	149			

a. Predictors: (Constant), GPPBDA2
b. Dependent Variable: GERDA2

Coefficients [a]

Model		Unstandardzied Coefficients		Standardized Coefficients		
		B	Std. Error	Beta	t	Sig.
1	(Constant)	4.605	0.8		5.755	.000
	GPPBDA2	7.12E-03	0.022	0.027	0.324	0.0746

a. Dependent Variable: GERDA2

(GERDA2=ERC, GPPBDA2= PPB, generalized least squares regression on 2-day lag between)

Even though the auto correlations were corrected through the use of generalized least squares, the results did not show any significance. The second hypothesis was not rejected.

CHAPTER V

CONCLUSIONS AND RECOMMENDATIONS

There have been multiple studies that have proven the harmful effects of ozone and air pollution. For decades the EPA and environmental groups have lobbied for cleaner air and for industries from car manufacturers to power plants to reduce the pollutants pumped into the air. In the meantime, the public has to protect itself during episodes of high air pollution. The EPA and the public media have created a warning system to make the public more aware of the potential hazards that occurs when heat and pollution combine. This warning system is not just for the lay public, but can be useful for health care providers. Ozone is most prevalent during the hotter times of the summer. This is typically a time when hospitals and other health care facilities are at a low census. Many hospitals reduce staff on duty and allow for time-off and vacations.

At Children's Medical Center, summer is when schedules are often spent on education, training and improving/refining skills. This is also a time when Dallas has the highest number of ozone alert days. Countless hours are spent each week for planning staff schedules. Managers use the current census, surgery schedules and admission trends to plan for staffing. Could the ozone alert system be a tool to warn of potential increases in admissions? Theoretically, if an orange or red alert is given, staff planners would be able to plan, such as not canceling ER staff and verifying any on-call staff. If an increase in asthma-related admissions could be predicted for a day or two after an ozone alert day, hospital planners and staff could better prepare for that increase.

For May to September 2000, daily emergency room admissions for all asthma-related ICD-9 codes were collected, as well as ozone levels for each day. Analysis of the data did not indicate any statistical significance. In reviewing the process, there are several possible reasons for this. Ozone and air pollution warnings are for the entire county. CMC treats pediatric patients from all over Dallas County. The patients treated should be a reasonable randomization of the affected population from the county. For that reason, no other hospitals or health care facilities were included. Adding those facilities that treat pediatrics and adults may have shown significance.

Several conditions can affect ozone levels, including wind, humidity, rain, cloud coverage, traffic patterns, weekdays, weekends or holidays. These were not included in the data analysis. Adjusting for these conditions within the analysis could change the results.

The EPA has been using local media to warn the public about ozone levels. The color-coded system was first used during 2000. Thus, a very positive theory could be that the public paid attention to the warnings and avoided situations which could exacerbate their symptoms. Future analysis should compare the 2000 or 2001 data to 1999 or 1998, to determine differences in ER admissions prior to and after the implementation of the ozone alert system.

Additionally, research studies on asthma and air pollution indicate that ozone alone may not be the culprit. Joel Schwartz's research focuses on size of particulate matter. By adding data on additional air pollutants and their particle size, the results of this study might have been different.

Dallas/Fort Worth is directly north of a town called Midlothian, which is where most of the area's cement factories are located. When winds are blowing from the south, emissions from those factories head north and add to the pollution for D/FW. Most of these plants are "fired up" at five in the morning and the pollution reaches downtown by morning rush hour. Plume maps may have been utilized in this study to add an additional variable to further explain the findings.

The annual meeting of the American Academy of Allergy, Asthma and Immunology was recently held in San Francisco. In attendance was Dr. David Peden, Professor of Pediatrics at The University of North Carolina. He has done many studies involving asthma and air pollution. He sited several reasons why the results of this study were not statistically significant. Dr. Peden stated that the sample size was adequate, however, there could be many subsets of patients within the sample population with multiple factors contributing to their emergency room admissions. Asthma severity, age, use of controller medication, location of home, ethnicity are all risk factors that should be investigated further. Surveying the sample population for allergies and Glutathione S-Transferase M1 gene production may show a subset of patients that are more susceptible to increase in airway irritability and hyperreactivity when exposed to ozone. Future investigations and research should focus on particle size, use of controller medications, allergic components as well as weather conditions and sources of pollutants.

Despite the lack of significance in the results, there is still an overwhelming amount of significant evidence that indicates air pollution is hazardous, especially to those with asthma. Also, those with sensitive lungs and other health conditions are at

greater risk. The EPA's AQI warning system for the public should be taken seriously. It was developed for the specific purpose to forewarn the population of a potential health hazard. When those with sensitive lungs and other chronic lung problems heed the warnings, it is possible to reduce the number of exacerbations and potentially reduce the number of visits to the ER.

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