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An analysis of cardiovascular and respiratory mortality as a function of ambient ozone and fine particulate matter in the Baton Rouge Air Quality Control Region

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AN ANALYSIS OF CARDIOVASCULAR AND RESPIRATORY MORTALITY AS A FUNCTION OF AMBIENT OZONE AND FINE PARTICULATE MATTER IN THE BATON ROUGE AIR QUALITY CONTROL REGION

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ABSTRACT

The Baton Rouge Air Quality Control Region has come under fire due to difficulties in reaching attainment of one-hour daily ozone limitations and its general perception as being a polluted place to live. Ozone is a reactive oxygen species which has been shown to result in damage to biological molecules and is detrimental to human physiology, especially in regards to cardio-respiratory structure and function. In the last decade, fine particulate matter less than 2.5 microns in mean aerodynamic diameter have been identified as a possible pollutant that is affecting public health at levels lower than the EPA's established limits. $PM_{2.5}$ has also been linked to adverse cardiovascular and respiratory health affects. The intent of this study was to examine the air quality of Baton Rouge and the surrounding five parishes and compare these concentrations to mortality cases for cardiovascular and respiratory causes of death. Poisson regression analysis found that $PM_{2.5}$ had no significant effect on mortality frequency. Ozone was found to have a negative relationship; as ozone levels increased, mortality rates decreased. Multiple regression of both pollutants confirmed the results obtained individually, with no indications of synergistic or antagonistic effects.

1. INTRODUCTION

As one passes through Baton Rouge, Louisiana, high traffic volume is readily apparent. Besides its status as the state capitol, Baton Rouge sits as the hub of the state's highway system. Interstate-10 and -12 bring large volumes of passenger car traffic and commercial trailer shipping over the Mississippi River and through this city of over 227,000 residents to points East and West along the I-10 corridor (U.S. Census, 2000). Additionally, this area is well known as a center of the refining and petrochemical industry. Companies such as ExxonMobil, Dow Chemical, and Shell are just a few of the companies that can be found in this region.

Due to its location on the interstate system and its high density of petrochemical manufacturing factories, it would be logical to assume that air quality would be a serious issue. The potential for low air quality is substantial. Federal and state regulatory agencies have significant amounts of guidance in the law and sciences capable of mitigating this risk to some extent, but what exactly are those risks? The potential health implications are either unknown or hotly debated, but research has continued to increase. During the summer of 2004, a $30 million grant was awarded to the University of Washington to conduct a national study on the effects of particulate air pollution on health. This was the largest research grant ever awarded by the Environmental Protection Agency to study the effects of fine particulate matter and other ambient pollutants on health.

Many studies have been conducted to determine effects for acute and chronic air pollution exposures, but most of these studies were conducted in tightly controlled environments where ambient concentrations could be controlled and therefore could be highly correlated to actual body burden or exposure. Epidemiological studies are an attempt to use

realistic measurements of ambient air quality to determine if there is a statistically significant correlation. These studies have limitations, but the intent is to utilize "real world" data to correlate mortality and morbidity to those controlled studies that better represented actual exposures.

As the literature review will show, there are a multitude of diseases and illnesses that are associated with inhalation exposures to air pollutants. In addition, there are epidemiological studies that correlate ambient levels of certain federally-regulated pollutants to specific illnesses, especially those that affect the cardiovascular and respiratory systems. Given all of the criteria pollutants and their associated toxicological properties, and the fact that the Baton Rouge Air Quality Control region is classified as severe for its non-attainment of federal ozone standards, a valuable link may be established between levels of these pollutants and mortality cases in the 5-parish area. The intent of this thesis is to compare air quality data for ozone and particulate matter to mortality cases for respiratory and cardiopulmonary disease for between 2000 and 2003, and to determine whether or not levels of these criteria pollutants have had a significant effect in this region.

2. LITERATURE REVIEW

2-1: Air Quality Regulations

The history and evolution of current air quality regulations begins in 1967. While there was some air pollution legislation on the books prior to this point, it was in this year that the Air Quality Act was passed (Ferrey, 2001). One of the primary changes that this new legislation imposed was that Air Quality Control Regions (AQCRs) were to be established. The AQCRs are essentially "bubbles" over certain geographic areas of the country where ambient air quality standards are to be monitored and enforced. These areas are typically set up over higher-density population centers. For this thesis, the AQCR of concern consists of the area over Baton Rouge and the surrounding parishes.

It was not until 1970 when national standards for levels of pollutants and the technology used to implement them were written into the laws (Ferrey, 2001). The Clean Air Act (CAA) Amendment was passed to create uniformity of which pollutants were to be monitored and what concentrations were authorized. The CAA also solidified the responsibility of the states in this process. State Implementation Plans (SIPs) were required by the EPA from every state, and it was the SIPs which laid down the foundation for how Louisiana would police itself in the monitoring of air quality. The Louisiana Department of Environmental Quality (DEQ) is the state agency responsible for this task.

The EPA established a list of airborne pollutants that it deemed deserving of specific monitoring. These pollutants are referred to as "criteria pollutants", and their associated regulated levels are the National Ambient Air Quality Standards (NAAQS). Over the years, through various additional amendments and changes, the criteria pollutants and their NAAQS

have been adjusted. The current list of criteria pollutants and their associated maximum values are listed in Table 1.

Two separate standards exist for the criteria pollutants -- the primary and secondary standards. According to 40 C.F.R., primary standards are established to protect public health, while secondary standards are set up to protect the public welfare (Revesz, 2002). Essentially, the primary levels are meant to establish a certain level of safety to protect the health of humans, particularly those segments of the population who are "at risk" for subsequent illnesses due to exposure (Leikauf, et al., 1995). The secondary levels are meant to protect the environment, such as wildlife and natural resources.

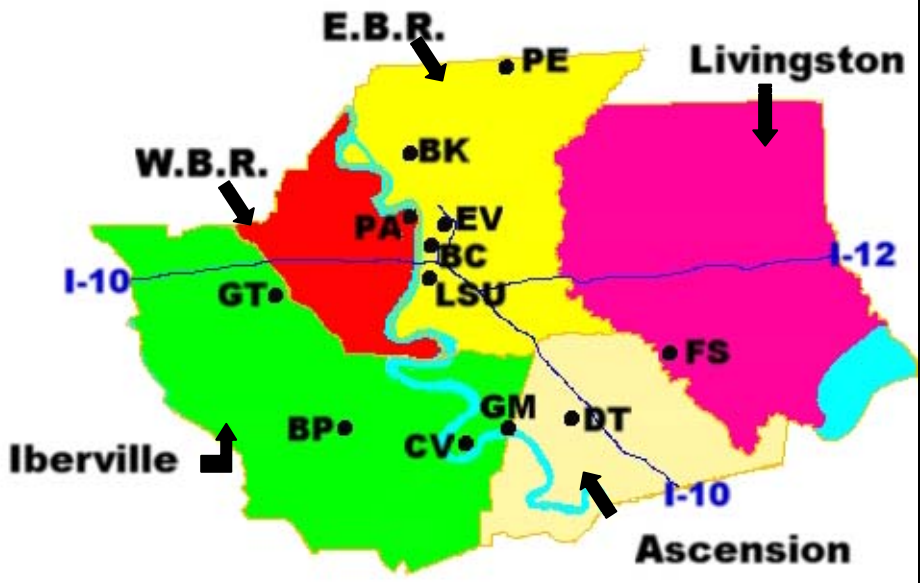
Table 1: Criteria Pollutants and Associated NAAQS Standards
(http://www.epa.gov/air/criteria.html)

Criteria Pollutant	Interval	Primary Standard	Secondary Standard
Carbon monoxide	8-hour average	9 ppm	None
	1-hour maximum	35 ppm	None
Lead	3-month average	1.5 $\mu g/m^3$	1.5 $\mu g/m^3$
Nitrogen dioxide	Annual average	0.053 ppm	0.053 ppm
Sulfur dioxide	Annual average	0.03 ppm	
	24-hour average	0.14 ppm	
	3-hour average		0.5 ppm
PM_{10}	Annual average	50 $\mu g/m^3$	50 $\mu g/m^3$
	24-hour average	150 $\mu g/m^3$	150 $\mu g/m^3$
$PM_{2.5}$	Annual average	15 $\mu g/m^3$	15 $\mu g/m^3$
	24-hour average	65 ug/m^3	65 ug/m^3
Ozone	1-hour maximum	0.12 ppm	0.12 ppm
	8-hour average	0.08 ppm	0.08 ppm

2-2: Baton Rouge NAAQS Monitoring

The Baton Rouge Air Quality Control Region has multiple sensor arrays set up throughout the five parishes (figure 1). These monitoring stations resemble small shacks with transmission antennae, which allow much of the NAAQS data to be relayed real-time to the

DEQ (figure 2). Each monitoring station contains a variety of sensors capable of measuring one or all of the criteria pollutants, as well as some co-located meteorological data.



Arrows and colored regions indicated individual parishes.
Black circles indicate monitoring sites.

Fig. 1: Locations of Air Monitoring Sites within the Baton Rouge 5-Parish Area

Fig. 2: Louisiana State University NAAQS Monitoring Station

Since the BR-ACQR is not in compliance with ozone standards, the DEQ has taken more steps to protect the public. Each site now has redundant ozone monitoring units, and a mobile sensor station has been purchased to better locate the source of emissions. In addition, whenever an ozone sensor records a spike in the ambient concentration of >25 ppb, a signal is sent to the DEQ offices to alert the appropriate personnel.

Particulate matter readings are different from ozone since some of the filters must be sent out of state for weight and analysis by a contracted commercial company. Other particulate sensors are capable of real-time measurements. The variations of the particulate matter sensors will be described in greater detail in the Methods section.

Baton Rouge has gained the attention of the Environmental Protection Agency and several environmental action groups due to what has been labeled as poor air quality. Over the last decade, the Baton Rouge AQCR has been in compliance with all but one of the NAAQS standards. This five parish area is classified as an area of non-attainment for the 1-hour ozone standard of 0.12 ppm[*]. Effective June 23, 2003, the EPA reclassified the Baton Rouge AQCR to a "severe" rating under the CAA, a one-step increase from its previously lower "serious" classification (www.deq.state.la.us). The only higher category in the CAA air quality rating schemes is "extreme".

Comparing these numbers to the past two decades, the number of non-attainment days has decreased. However, even with SIP revisions, vehicle emissions inspections, and public information on controlling ozone precursors, Baton Rouge has a challenge ahead before compliance in ozone concentrations is reached and the state can be reclassified to a new air quality category.

[*] Since the NAAQS stand for 1-hour ozone is 0.12 ppm, only measurements in excess of 124 ppb are recorded as exceedances due to rounding up/down of values.

When an ozone sensor detects an exceedance, this failure date goes against the three-year three-failure per sensor count for non-attainment. An AQCR has to maintain a net number of exceedances within a three year period of a maximum three days of non-attainment. This means that the same sensor location must record more than three exceedances. If three different individual sensors each record an exceedance over the three year period, that is not classified as non-attainment. In the event the number of failures in a three year period drops below three, the area can be reclassified as an attainment area. Table 2 lists the number of ozone exceedances by location for 2000-2003 as well as the official number of days of 1-hour ozone non-attainment. There is a difference in the quantity of exceedances by station and days of non-attainment because some of the exceedance days occurred on the same day for different locations. An important point to note is that any sensor with three exceedances classifies the entire Baton Rouge area as non-attainment. Table 2 shows that the parishes of Ascension and Livingston have zero exceedance days. However, since these parishes fall under the bubble of the BR-AQCR, they are also in non-attainment by default, and will have to comply with any additional restrictions, such as fuel additives or stricter automobile emission inspections.

Specific AQCRs are responsible for any non-attainment dates, whether the pollution levels were generated within that region or were transported by surface or upper level winds. In Baton Rouge, LA DEQ continues to argue that while there are sources of ozone and ozone precursors in this area, the problem is being exacerbated by the Houston AQCR. DEQ argues that winds are carrying ozone and precursors from Houston to Baton Rouge and is making ambient conditions worse.

A study which took advantage of a major power outage in the northeast United States may help Louisiana's claim. In 2003, a blackout of power plants in the northeast U.S. and Canada occurred. Power plant emissions were essentially stopped, which allowed a unique opportunity to examine how the shutdown of these plants could affect ambient conditions. After 24 hours, ozone, particulates, and sulfur dioxide levels decreased 50%, 70%, and 90% respectively at significant distances downwind from the sources (Marufu, et al., 2004).

Table 2: BR-AQCR Ozone Monitor Exceedances by Location

Sensor	Parish	Number of Exceedances				Attainment
		2000	2001	2002	2003	
Grosse Tete	Iberville	2	0	0	0	Yes
Port Allen	West Baton Rouge	1	0	1	2	Yes
Capitol	East Baton Rouge	2	0	2	2	No
LSU	East Baton Rouge	3	0	1	3	No
Baker	East Baton Rouge	3	0	0	1	Yes
Pride	East Baton Rouge	1	0	0	0	Yes
Bayou Plaquemine	Iberville	1	1	1	1	Yes
Carville	Iberville	1	0	2	0	Yes
Dutchtown	Ascension	2	0	0	0	Yes
French Settlement	Livingston	3	0	0	0	Yes
Total Exceedances by station		19	1	7	9	
Total AQCR days of non-attainment		11	1	2	6	

With respect to other criteria pollutants, the BR-ACQR has reached attainment. This does not mean that there is not a health risk associated with these other pollutants. For instance, particulate matter has been found to show detrimental physiological effects at levels lower that the NAAQS standards (Costa, 2000; Dreher, 2000). Oxides of nitrogen (NOx) are important because not only do they possess toxic properties, but they also affect other criteria pollutant concentrations. In the presence of volatile organic compounds (VOCs), NOx serves as a critical constituent in the production of ozone (Manahan, 2000). Higher levels of NOx

can contribute to higher levels of ozone, and thereby increase the human health risk without acting as the hazardous agent. Next, the detrimental effects of criteria pollutants on health is explained in greater detail.

2-3: Criteria Pollutant Toxicity

The previous sections were intended to give a brief overview of legislation of air quality standards, procedures for monitoring, and some background on the Baton Rouge AQCR. This section examines the criteria pollutants individually and delineates their harmful health effects. While each of the criteria pollutants is important for determining the potential adverse health effects due to inhalation exposure, this thesis focuses on ozone and particulate matter. Ozone is reviewed because of the non-attainment status of the Baton Rouge AQCR and particulate matter because of the recent literature that asserts that the adverse effects of fine particles can be found at levels much lower that the EPA's current NAAQS levels.

Each criteria pollutant can induce deleterious health effects at certain levels. The question of concern is what happens at doses that are considered "low" and below the NAAQS primary standards – even doses where the effects may be below the threshold of measurement. Many of these pollutant levels, by nature of their interactions with various biological processes, may not be the primary causal factor for a mortality or morbidity case but rather that they are risk factors. Increases in the levels of these criteria pollutants increase the risk of illnesses by exploiting predisposed weaknesses in metabolism, physiology, or other medical conditions (Koren 1995).

Asthma is a prime example of a chronic respiratory disease which can be exacerbated by elevated levels of airborne pollutants. This disease is characterized by lymphocyte-induced inflammation, changes in the levels of mucus and cytokine secretion by epithelial

cells, and obstruction of airflow which can be triggered by a variety of airborne pollutants (Leikauf, et al., 1995). Non-specific hyper-reactivity asthma would be classified as asthma in response to compounds that are not the traditional allergens, such as pet dander or dust. Allergic asthma mostly Immunoglobin-E antibody mediated, which could be useful as a biomarker in studies where the researchers want to differentiate between allergic asthma and non-specific hyper-reactivity asthma cases.

The next sections describe some of the detrimental effects of each of the criteria pollutants, with the focus being primarily on ultrafine particulate matter and ozone. In addition, some samples of air quality data from the BR-AQCR are included to illustrate the relative ambient levels of these pollutants.

2-3.1: Carbon Monoxide

Carbon monoxide is an odorless, tasteless gas that is a byproduct of incomplete combustion (Manahan, 2000). The mention of CO as a toxicant typically brings to mind the asphyxiation of suicide victims from being in a closed environment with a running automobile. The basis for this toxicity is that CO has a higher affinity than oxygen for the active binding sites on hemoglobin (Klaassen, 2001). While the airborne concentration in the environment is much less than in a closed garage, the health affects are still similar, but on a smaller scale. Automobile exhaust and factory smokestacks have the potential to increase carbon monoxide levels where they may be hazardous to health. Certain groups, such as the elderly or very young, have increased risk factors for the effects of carbon monoxide. In addition, other groups may be at higher risk due to personal lifestyle, such as smoking, which results in an initially higher level of carboxyhemoglobin than non-smokers.

As with all of the criteria pollutants, mounting scientific evidence is revealing that adverse health effects occurring at levels below the NAAQS standards, and below what scientists and health professionals had felt were "safe". Singh et al., (1995) examined the effects of carbon monoxide on the blood cell constituents in pregnant and non-pregnant mice. Results of this study found an increase in erythrocyte concentrations and a decrease in hemoglobin percentages. This study also showed a simultaneous decrease in placental hemoglobin. Therefore, it is reasonable to conclude that fetal development could be impaired from a decrease in oxygen crossing the placental barrier. While the CO levels that could be fatal could be potentially calculated, it is the subtle effects of a minor elevation in environmental carbon monoxide concentrations that could have an effect on the development of a fetus. Changes in cell quantity and oxygen-carrying capacity could affect the heart, brain, and any other high-oxygen-demand tissues.

CO also exhibits a vasodilation effect. Inspired carbon monoxide directly affects the potassium channels in pulmonary arteries at a level of 50 ppm in rats (Dubuis et al., 2003). This study is interesting in that CO, while it is harmful in terms of oxygen transport by hemoglobin, may actually have a therapeutic value as well. The risk-to-benefit ratio, however, has not been identified.

Baton Rouge CO data:

Number of sensors: 1 (Baton Rouge Capitol monitor)

Figure 3 is a plot of daily carbon monoxide levels for 2003.

2003 1-Hr Max = 4.5 ppb (35 ppb is 1-hr NAAQS limit)

2003 Daily Average = 0.7 ppb

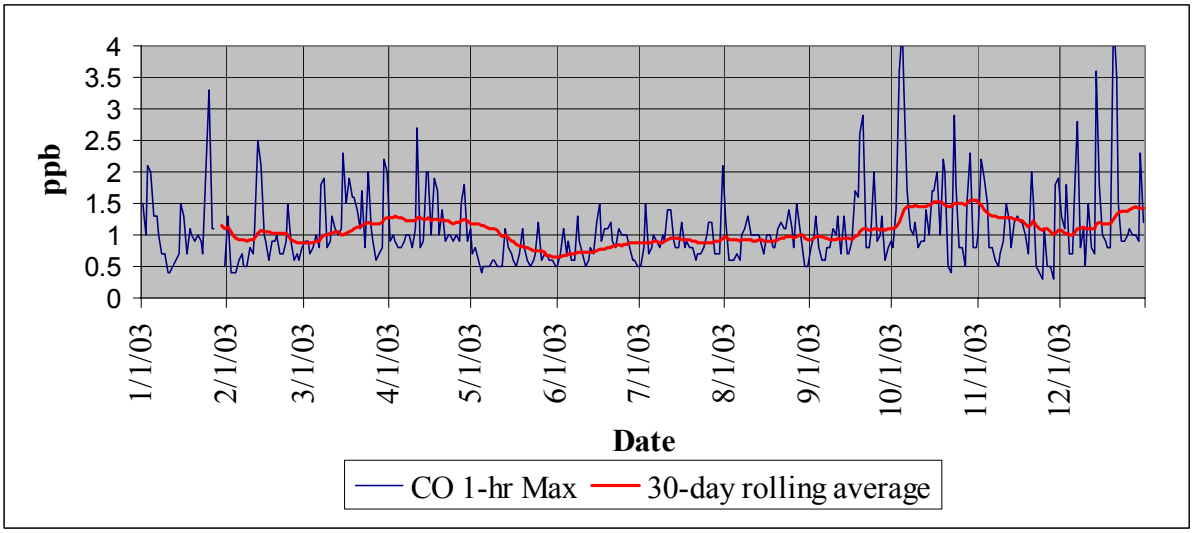


Fig. 3: 2003 Carbon Monoxide 1-Hour Maximum Values for Baton Rouge Capitol Site

2-3.2: Lead

Lead is a heavy metal which has been used for a wide variety of applications for centuries, and its toxicity has been known for almost as long. Lead came to the forefront of air pollution regulations when toxicity studies discovered the detrimental effects lead has on the nervous system. Lead affects the central nervous system of young children due to the fact in can cross the fledgling blood-brain barrier, and it affects the peripheral nervous system in adults (Klasssen, 2001). In the CNS, lead can damage neurons, leading to a decrease in IQ and other mental defects. In adults, lead cannot pass through the fully developed blood-brain barrier, but instead affects peripheral neurons, resulting in disorders such as wrist and foot drop.

The major source of airborne lead pollution originally was from automobile fuels. Lead was used as an anti-knocking agent in fuel up until 1970 (Ferrey, 2001). Since the EPA banned the use of lead in gasoline, airborne concentrations have decreased significantly. Now, lead comes from various industrial processes, especially smelting and the burning of coal.

Much of the toxicological data on lead poisoning comes from both ingestion and inhalation. Ingestion studies came about from the realization that lead paint chips eaten by children were a primary source for CNS neurotoxicity. There has been a significant amount of research in inhalation toxicity derived from the use of lead in gasoline. In a study of Navajo teenagers who were hospitalized for lead poisoning from sniffing gasoline, 65% of them exhibited encephalopathy, as well as other traditional symptoms of ataxia and tremors (Coulehan et al., 1983).

Unlike the other criteria pollutants, lead is unique because the risk of toxicity is tied to its retention in the tissues and bone. Over time, chronic exposure contributes to the overall body burden (Garcia Vargas et al., 2001). Therefore, even if lead aerosol is found in low concentrations, it is slowly but surely accumulating with every breath.

2-3.3: Nitrogen Dioxide

Nitrogen dioxide is another product of the combustion process. NO_2 has been shown to have a small number of known adverse human health effects, but it is seen more as an environmental pollutant, damaging crops and buildings. Probably even more important is the contribution of NO_2 to the creation of ozone. Keeping the level of NO_2 as low as possible results in a two-fold benefit: a decrease in health problems directly from this pollutant, and a decrease in the creation of ozone. This is critical for the BR-AQCR non-attainment status since ozone is considered more deleterious, and it is one of the reasons why the 5-parish area has the stigma of being a highly polluted area. It is important to mention LDEQ airshed models, in which computer modeling of ozone formation shows that decreasing VOCs has a greater effect on reducing ozone than nitrogen dioxide (www.deq.la.state.gov). Regardless, decreasing NO2 emissions can alleviate air pollution concerns both directly and indirectly.

Louisiana monitors nitrogen dioxide and nitric oxide under the heading of nitric oxides. The literature often refers to NOx when talking about this category of pollutant. The EPA delineates only a NAAQS limit on dioxides of nitrogen, and not NOx as a whole category. The literature may take NOx into consideration because NO is a precursor to NO_2.

Hatton et al. (1977) examined the accidental exposure of astronauts to nitrogen dioxide during the Apollo-Soyuz mission. Nitrogen dioxide used as an oxidant used in rocket fuel had leaked into the capsule during this mission's re-entry. Post-landing examination of the astronauts revealed that inhalation of NO_2 was painful and marked by an increase in the excretion of hydroxylysine, a breakdown product of collagen damage and a marker for lung injury.

A pertinent study into the toxic effects of NO_2 examined inhalation exposures of 1-2 ppm on human test subjects. At these doses, erythrocyte acetylcholinesterase activity decreased, and there was a higher level of lipid peroxidation of RBCs (Posin et al., 1978). The two studies mentioned in the previous paragraphs illustrated that nitrogen dioxide poses pulmonary and hematological risks.

Baton Rouge NO_2 data:

Number of sensors: 9

2003 BR-AQCR 1-hr Max = 107 ppb (LSU)

2003 Baton Rouge Capitol Site Annual Average = 15.5 ppb

Figure 4 is a plot of daily nitrogen dioxide levels at the Baton Rouge Capitol for 2003. This graph shows that NO_2 levels for the Capitol monitor are 70% less than the NAAQS annual average limitation. It is interesting that nitrogen dioxide levels seem to reach their lowest

values during the summer months. This may be due to higher surface level reactions with VOCs, resulting in a decrease in NO_2 with an increase in ozone production.

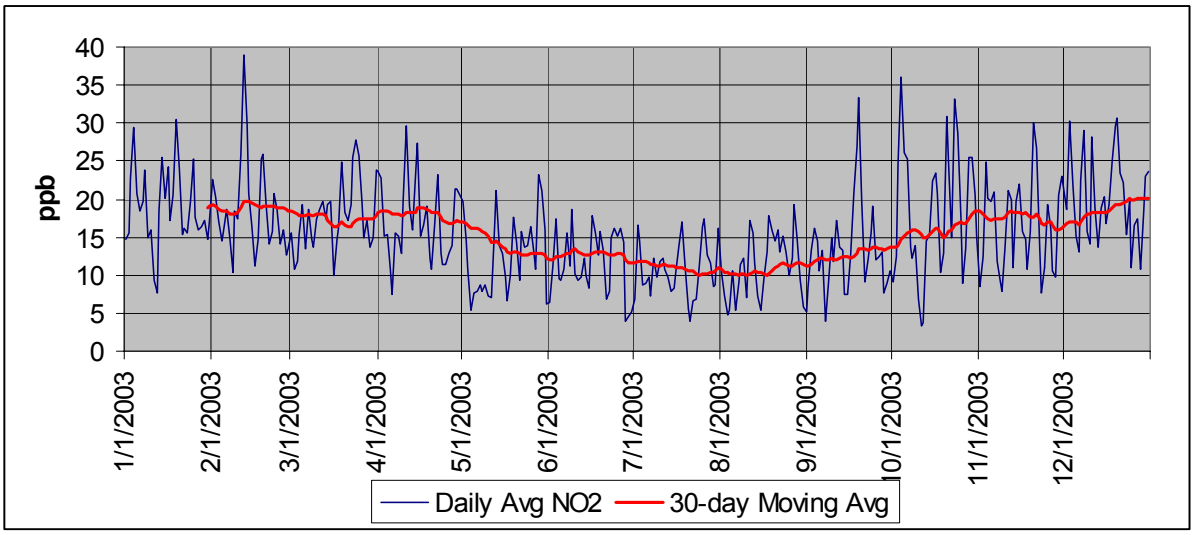


Fig. 4: 2003 Daily Average NO_2 for Capitol Monitor Site

2-3.4: Sulfur Dioxide

Sulfur dioxide, the precursor to acid rain, is a product of the combustion of fossil fuels, with coal-powered electrical plants being the largest offender. While this pollutant can result in physical damage to buildings, automobiles, crops, and bodies of water, there is evidence that SO_2 has direct adverse effects on humans as a respiratory toxicant.

In addition, sulfur dioxides can transform into particulate sulfates, thereby having a multi-faceted indirect effect on health by contributing to particulate pollution as well. Nucleation, or the formation of new particulates, has been found to increase in the presence of sulfuric acid or high humidity (Zhang, R., et al., 2004). This data further increases the difficulty of projecting the effects of other pollutants on the fine particulate matter problem. Traditional fine particulate matter is typically categorized as the product of incomplete combustion. However, as SOx and subsequent H_2SO_4 interacts with VOCs, one can project

that these reactions can occur further downwind, thereby increasing fine particulate levels much further from the locations of "traditional" sources.

SO_2 can have serious effects at high doses, since this compound will convert to an acid when in the presence of water, as in the lungs. At lower doses, sulfur dioxide will act as a pulmonary irritant due to acidity and will affect blood composition such as hematocrit and oxygen transport in hemoglobin (Baskurt, 1988). This irritation could be an explanation of why SO_2 can trigger asthma. Sulfur dioxide and ozone in the ambient environment, however, cannot be always individually identified as the causative agent (Koren, 1995). This makes epidemiological analysis without biomarkers difficult.

In vivo studies of mouse bone-marrow cells have shown the potential for mutagenicity by SO_2. Ziqiang, M., et al. (2002) found that inhalation of sulfur dioxide in a murine model resulted in an increase in micronuclei formation in polychromatic erythrocytes. This adds another dimension to the air quality problem. No longer is SOx linked solely to respiratory distress or particulate nucleation. Instead, it appears that sulfur dioxide might be a systemic toxicant and pervasive down to the cellular level, resulting in genotoxicity.

Baton Rouge SO_2 data:

Number of sensors: 2

2003 max at Capitol site = 13.8 ppb (140 ppb is NAAQS 24-hr max)

2003 annual average at Capitol site = 3.4 ppb (30 ppb NAAQS annual limit)

Figure 5 is a plot of daily Baton Rouge sulfur dioxide levels for 2003. This monitor shows that SO_2 is only at levels around 10% of the NAAQS limits.

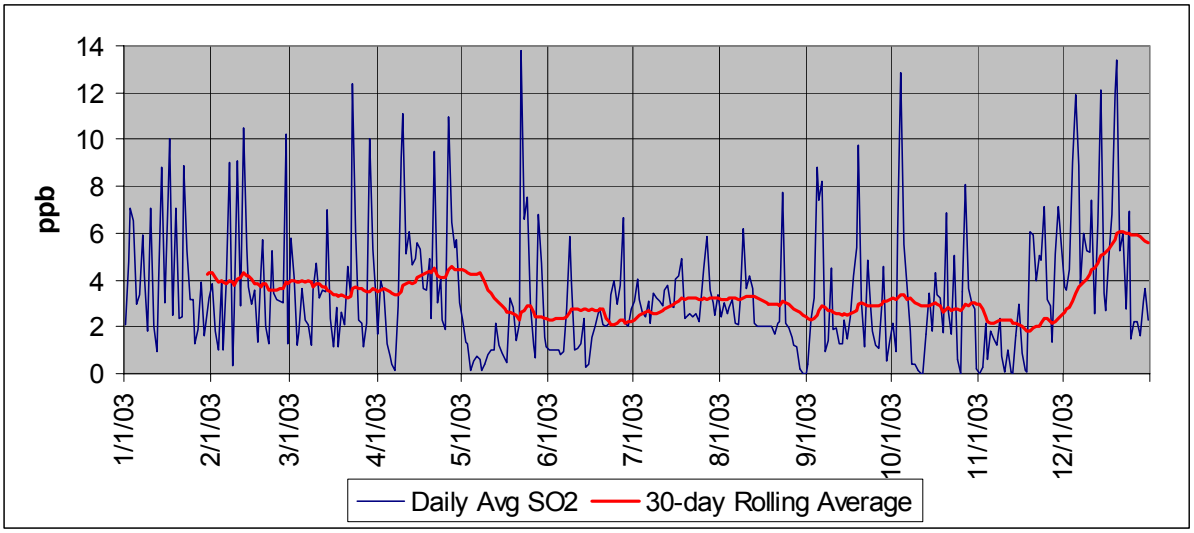


Fig. 5: 2003 Daily Average SO_2 for Capitol Monitor Site

2-3.5: Ozone

Ozone is a reactive oxygen species that can be formed by the interaction of VOCs and nitrogen dioxide and, to a much lower degree, from electrical discharges from lightning and electronics. Substantial evidence exists in the literature on the inhalation toxicology of ozone, especially for acute exposures. Primarily, ozone affects the lungs and can manifest itself into a variety of clinical diagnoses. High ozone levels are often linked to incidences of cardiopulmonary complications (Klaassen 2001) and bronchial hyper-reactivity and asthma induction (Boushey, et al., 1995)

Ozone, while it is a relatively short-lived chemical species poses serious health consequences due to its reactive nature. The ozone MSDS lists free radical oxygen as the major hazardous decomposition product. Ozone Solutions, Inc., a supplier of commercial ozone and related products, provides data on the temperature-dependent half-life of O_3 in both the gaseous and dissolved (H_20) phases. At 20°C, the half-life in the gaseous phase is 3 days and only 20 minutes in water (www.ozoneapplications.com). These numbers are significant

in terms of inhalation toxicity. When ozone enters the lungs it will encounter a moist environment in which it can dissolve. This allows ozone to decompose faster, making the reactivity much quicker.

Li, et al. (1991) examined the effect of a 0.3 ppm ozone concentration on spleen T lymphocytes *in vivo* for 1-3 weeks. The resulting decrease in T cells suggests that an exposure that is almost three times higher than the NAAQS concentration limit will impair the immune system. While this dose is much higher than an average individual in the BR-AQCR might encounter, the study was useful because it looked at a dose applied over weeks, illustrating effects between the typical acute and chronic exposure models.

In the United States, 6.3 million children have asthma according to a 2004 *Journal of Environmental Health* article. In this summary article, reference is made to a Yale School of Medicine study which found that even when ozone limits are at 60 ppb, a significant increase in asthma resulted. Airways are extremely sensitive to ozone, and respond in very dynamic ways, including changes in expiration volume and immune sensitivity. The onset of asthma can be characterized as a cascade-style reaction. Helper T-cells, after exposure to a causative agent, over-produces interleukin-3, -4, -5, and -10 and certain cytokines (Boushey, et al., 1995). Production of these messengers results in stimulation of granulocytes, mast cells, and eosinophils. Additionally, IL-4 stimulates production of IgE in CD4+ lymphocytes, which also amplifies the mast cell and eosinophil activation. This hyperreaction to ozone or other irritant results in an extremely oversensitive immune response within the lungs, causing constriction of bronchial tubes and symptoms that are characteristic of an asthmatic response.

Airway resistance can increase after exposure to ozone, but these exposures can be at levels five times higher than the 0.12 ppm NAAQS standard (Koren 1995). It becomes

difficult to draw direct comparisons at these extremely acute levels, since the general population would not be exposed to these levels on a daily basis Anyone living in the Baton Rouge AQCR will be exposed for a longer duration, but at lower doses. Studies that examine dosing regimens bordering on the sub-acute/sub-chronic will help to elucidate some toxicological effects that may not have been detectible in the past.

Changes in the ratio of collagen types I and III are found to result in fibrosis in tissues due to improper cross-linking after ozone exposures (Klinge et al., 2000). In rats exposed to the NAAQS ozone 1-hour limit of 0.12 ppm for 6 hours a day, five days a week, for almost two years, changes in the collagen ratio were observed (Last, et al., 1998). Even though the model mammal utilized was the rat, this study showed a direct result of repeated ozone exposure that could be a realistic exposure scenario in an area with high ozone pollution.

In a novel study, the neurological effects of ozone on bronchial innervations were examined. Human nasal tissue was exposed to 0.1 ppm ozone, and that exposure was followed by an observed increase in neurokinin A, a neuropeptide that is released when stimulated by irritants (Schierhorn, K., et al. 2002). This is valuable data because a specific method for ozone triggering a nerve response has been observed. This could lead to more effective treatment of ozone-induced asthma therapy by targeting specific components of the nervous system along with traditional methods to decrease the immune response.

Illustrating the large and relatively unknown dynamics between criteria pollutants, ozone has been found to increase the response to SO_2 in teenage asthmatics (Koenig, et al., 1990). A study such as this shows that there could be significant synergistic and antagonistic effects between different variables. A model which takes into consideration all exposures

may provide more realistic data when compared to mortality but extends beyond the confines of this study of solely ozone and particulate matter exposures.

In 1997, a change was made to the NAAQS ozone standards. The limits are now based on an 8-hr rolling average as opposed to a 1-hour standard. This change, however, applies only if the AQCR is in attainment with the 1-hour standard. Since the 5-parish area is not in compliance, the BR-AQCR will not be allowed to use the more flexible 8-hr average method.

Baton Rouge O_3 data:

Number of sensors: 10

2003 BR-AQCR 1-hr max = 174 ppb (124 ppb NAAQS 1-hr limit)

Figure 6 is a plot of the 2003 1-hour ozone daily maximum measurements for the Louisiana State University Station. The three labeled peaks indicate three days of non-attainment. On 18 July 2003, two of the three sensors were within five miles of each other, but the third was located approximately 20 miles from the other two. This could serve to validate an epidemiological study of hospitalizations and ozone levels because the "blanket" of low-level ozone can be stretching far beyond an individual sensor. It is also interesting to note that on different exceedance days, the other sensors that did not have a sample over the 0.12 ppm limit were relatively close to exceeding the NAAQS value.

2-3.6: Particulate Matter

The latest trend in evaluation of air quality is particulate matter (PM). Particulate matter can consist of a variety of organic and inorganic components. When NAAQS standards were first enacted, particulate matter less than 10 microns (PM_{10}), also referred to as

"coarse", was considered to be the high risk particle size. However, recent research has pointed out that

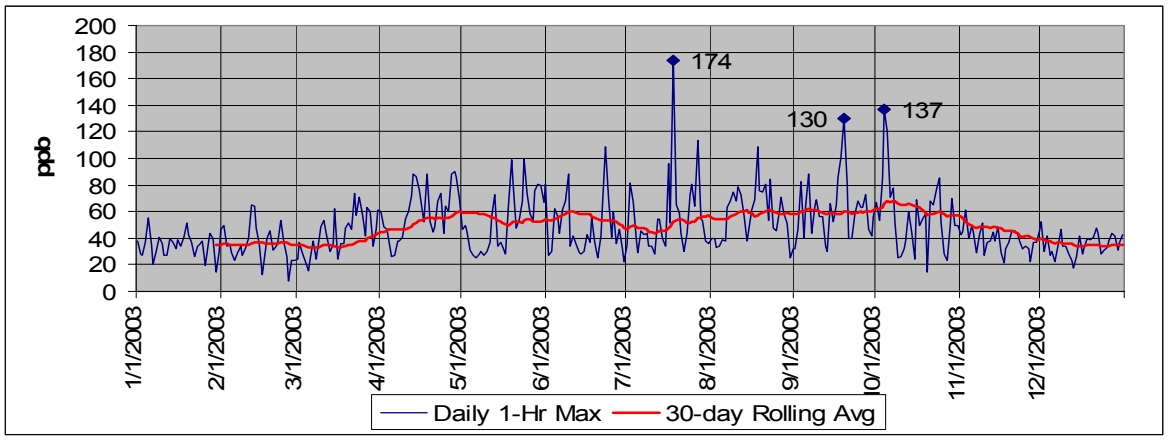


Fig. 6: 2003 1-hour Maximum Ozone at LSU Monitor Site

it is in fact particulates smaller than 2.5 microns ($PM_{2.5}$) which pose the greatest threat (Lippmann, et al., 2003). Thus, the CAA was amended in 1997 to create a separate category for monitoring purposes. Categorical classifications of fine particulates vary within the literature. Generally, $PM_{2.5}$ having a mean aerodynamic diameter <0.1 um may be classified as ultrafine (<0.1 um) and result from combustion, and fine (0.1 um-1 um) from combustion and inorganic materials (Schlesinger & Cassee, 2003).

The reasoning for the 1997 change related to two studies that were completed in the mid-'90s. The Harvard Six-City Study (Dockery, et al., 1993) and the American Cancer Society Particulate Matter Study (Pope, et al., 1995) served as the scientific basis for the EPA's current stance on particulate air pollution. These studies, through some innovative statistical analysis, showed a correlation between "fine" particulate matter, $PM_{2.5}$, and mortality from cardiovascular and respiratory illness. An independent review was conducted in 2000 (Krewski, et al.), and experiments by other research groups (Daniels, et al., 2000)

have validated the original study methodologies and results with negligible discrepancies that did not substantially affect the statistical significance of the conclusions.

Besides diameter, the composition and origin of $PM_{2.5}$ and PM_{10} vary significantly. Ultrafine PM is made up of approximately 45% organic carbon, while PM_{10} is made up of crust particles and metals (Li, et al., 2002). Given the nature of compounds such as PAHs, it would be expected that more of these toxic compounds would be found bound to and associated with organic compounds from combustion processes, and therefore in higher concentrations with $PM_{2.5}$ as opposed to PM_{10}.

Deposition of particulate matter in the lungs is dependent upon size. One model shows that humidity and hygroscopic particle size are intimately related to lung deposition. The smaller the particle, the less time it takes to reach a size within the lungs which would result in deposition as opposed to expiration. Particles <0.1 micron can reach this deposition size in a single breath, while particles greater than 1um take at least 10 seconds to reach a equilibrium and deposit (Broday, et al., 2001). If the model is correct, then it again illustrates that very fine particulates have the greater chance of interacting with lung tissue, and therefore are more hazardous.

Particles that are deposited in the lung tissue can cause a wide variety of medical problems. These include, but are not limited to: oxidative stress (Li, et al., 2002), lung hyperinflation (Calderon Garciaduenas, et al., 2000), and instigation of inflammatory response of lung macrophages (Zhang and Kusaka, 2000). Smith et al. (2003) found that neutrophil counts increased in rats after recurrent exposure to ultrafine PM for repeated exposures over a 4-6 week period. However, some animal models may not be appropriate for

drawing conclusions on human exposures due to differences in respiration rates and lung morphology (Hofmann and Bergmann, 1998).

Part of the rationale behind this thesis is that larger particles become trapped in the upper respiratory pathways, and it is the fine particles that can actually make it to the smaller bronchioles and alveoli (Klaassen, 2001). The idea is that mortality and morbidity rates increase as these smaller particles penetrate the deeper parts of the lungs. The particles themselves may serve as the causal factor, or it may be the compounds that have adhered to the particulates, such as organic compounds or even ultra-fine metals, that induce the toxicity (Zhang, et al., 2000; Broday and Georgeopoulos, 2001). Additional PM properties that may affect health are acidity/alkalinity, surface area, raw quantity, affinity for ingestion by macrophages, and adsorbed co-pollutants (Dreher 2000).While the research has shown many associations between particulate matter and certain pathology, the mechanisms are still under investigation.

Prolonged exposure to particulates from certain mining operations suggest that overloading the clearance capacity for the lungs can result in fibrosis from localized inflammation (Borm, et al., 2004). This chronic exposure to particulates is suspected to be a causal factor in the initiation of lung cancer. While this study is concerned with diseases excluding cancer, one cannot rule out the fact that, along with certain lifestyle risk factors, very long-term exposures to individuals with compromised lung particle clearance capacity may be more at risk for lung cancer. The ambient concentration of particulate matter may not directly cause the damage but may instead be the factor which saturates the clearance capacity, allowing cigarette smoke or other carcinogens more residence time in the lungs to inflict genotoxic effects.

Bacterial lipopolysaccharide endotoxin has been found to have synergistic effects between $PM_{2.5}$ and other pollutants (Lippmann, et al., 2003). The mechanisms by which these amplifying effects occur are unknown, but it could be hypothesized that either clearance capacity, hyperreactivity reactions, or particle-particle interactions may be the underlying factors.

Particulates have been shown to cause physiological problems beyond the site of initial exposure. Cardiovascular implications can be found in the literature. Residual oily fly ash exposure resulted in EKG changes resembling ischemia or hypoxemia in rats (Costa 2000). While this study shows that particulates can impact other major organ systems other than the lungs, the mechanisms are not known and therefore only very generalized statistical inferences can be made (Dreher 2000).

In yet another example that air pollution effects extend beyond the lungs or circulatory system, mice were exposed to ambient air at varying distances from a steel mill. There was a 1.5 – 2X increase in germline mutations at one kilometer from the mill as compared to 30 kilometers downwind (Somers, et al., 2002). This was a well-controlled study, and the mice were exposed only to ambient air. Food and water were administered by study personnel and animals were contained in cages and not in direct contact with soil. What is unclear in this study, however, is which specific agents caused the mutations.

Moyer, et al. (2002) conducted a study whereby mice were exposed to a variety of particulate matter types over a two-year period. The results indicated a significant increase in the incidence of inflammation and vascular damage occurred with long-term exposures. In this same study, a 90-day evaluation was completed, and the results did not show the same pathology, indicating that the exposure time varies significantly to induce this type of

systemic toxicity. Some rodent models that are attempting to elucidate criteria pollutant toxicity may not be good measures for human exposures. For instance, elastase-induced rat emphysema as an endpoint may have similar structural problems as humans but not nearly as much inflammation (Costa 2000). Accurate and relevant endpoints for toxicity are critical for generating conclusions on what diseases and illnesses are the result of airborne toxicant exposures.

One of the constituents of ultrafine PM is small quantities of water-soluble metal particles that are fine enough to be inhaled and absorbed. These metals can cause heart damage or directly affect lung biochemistry, which in turn also affects heart function. A rat line with genetic predisposition to hypertension was exposed to $PM_{2.5}$ from a Boston power plant at levels that were not high enough to directly damage lung tissue (Kodavanti et al., 2003). The study found two important results. First, different dosing regimens were used which showed that if the exposure is acute, but followed by a longer recovery time, clearance was significant in reducing effects of the initial dose. Second, since the rat strain utilized was predisposed to hypertension compared to a "normal" strain, cardiac lesions and myocardial degeneration were frequent in the compromised rats. This lends credence to the idea that ambient pollutants exacerbate pre-existing conditions, and is therefore a risk factor, not a causal one. This does make epidemiological studies more difficult, because an entire medical history, personal profile, and biochemical workup would have to been done to account for any and all potential pre-existing conditions.

The use of smoking as a surrogate for chronic PM exposure can provide valuable data in estimating the detrimental effects of ambient air quality. It was found that placental CYP1A1 and polycyclic aromatic hydrocarbon adducts were found to increase in smoking

mothers. Whyatt et al. (1995) hypothesized that a developing fetus does not have a strong detoxification capacity, potentially making them more susceptible to PAH-laden particulates. If one draws a parallel between cigarette smoke and combustion products resulting in fine PM, it would appear that ambient concentrations may have effects that extend far beyond the respiratory system of an exposed individual.

The study of biomarkers which can indicate exposure to $PM_{2.5}$ can help fine tune future statistical models of ambient exposures. One study in particular showed a strong contrast in oxidative stress markers after exposure to either $PM_{2.5}$ or PM_{10}. Heme oxygenase is an enzyme that is responsible for eliminating reactive oxygen species (ROS). After exposure to different sized particulates, heme oxygenase levels were found in significantly higher levels after exposure to $PM_{2.5}$ as compared to $PM_{2.5}$ (Li, et al., 2002).

This same study also examined the cytotoxic effects of these two sizes of particulates. Glutathione GSH/GSGG ratios after exposure to $PM_{2.5}$ decreased, and thereby exhausted a major component of the cellular ability to deal with ROS. PM_{10} particles did not affect the GSH/GSGG ratio at a statistically significant level. This data lends more evidence to the idea that it is the ultrafine particulates that present the greater health hazard, especially at the cellular level.

It is important to note that while $PM_{2.5}$ may appear to elicit more adverse health effects, especially those that are systemic or at the cellular level, PM_{10} is not without certain hazards in its own right. Some classifications of larger particulates have a higher level of reactivity and detrimental effects. Coarse particulate matter can be more reactive than $PM_{2.5}$, and it is dependent on the composition of the PM_{10} and the target. In the case of cat allergens and asthma, these particles affect the larger bronchial spaces. Nebulized cat allergens of

different sizes were administered to asthmatics. The most effective particle size for the induction of an immediate asthma response was 10.3 microns, not 1.4 or 4.8 microns (Lieutier-Colas, et al., 2003). A dose of 20X the amount of large particles was required to elicit a similar degradation on forced expiratory volume. Since it is the larger particles that impinge and interact with these surfaces, they would have a greater effect because they are able interact with those surfaces while the smaller particles would reach the alveolar spaces.

Studies have found differences in the relative effectiveness of inducing cellular stress between PM_{10} and $PM_{2.5}$. This is vital information to the study of air quality because regulations must take into consideration the potential severity when determining pollution limitations. Controversy arose due to the CAA revisions in 1997 which added $PM_{2.5}$ as a criteria pollutant. Researchers, policy makers, and industry have different views on the scientific basis for ultrafine particulate matter regulations. Fortunately, more and more data has been produced to strengthen the epidemiological results of the Harvard Six City Study and the American Cancer Society's Cohort Study on ultrafine particulates.

Baton Rouge $PM_{2.5}$ data:

Number of sensors: 8

2003 24-hr max at Capitol site =47.5 ug/m^3 (NAAQS 24-hour max is 65ug/m^3)

Figure 7 illustrates trends in the daily average $PM_{2.5}$ concentrations at the Baton Rouge Capitol site. The spike in particulate matter around 4 December appears to be an anomaly compared to the relatively low daily averages for the year. It could be useful if the cause of the peak could be determined and then to specifically look at the mortality or hospitalizations that occurred in close vicinity to this monitor site.

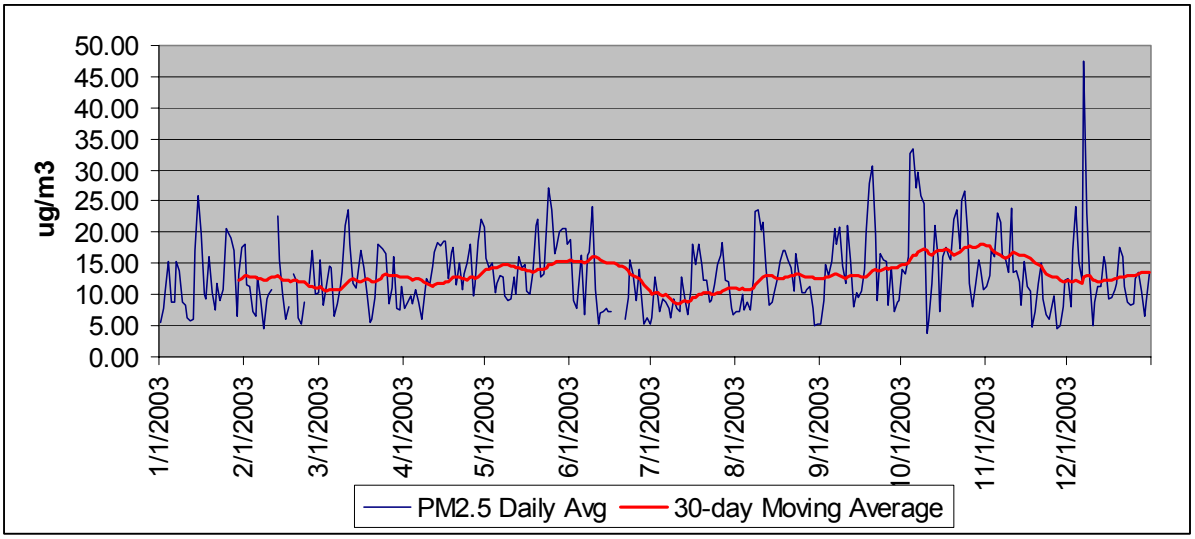


Fig. 7: 2003 Daily Average $PM_{2.5}$ at Capitol Monitor Site

2-4: Contemporary Epidemiological Studies

The following case studies list three specific research projects which examined the relationship between air quality and mortality or asthma cases. Each study utilizes different techniques and procedures for examining data and are presented here as a means of comparison to the approaches taken in this thesis.

PM_{10} and Mortality - In a study examining PM_{10} and mortality which spanned seven years and encompassed 20 major U.S. cities, Daniels et al. (2000) found a linear relationship with no threshold for cardio-respiratory and all-cause death cases. For all other death causes, no increase in risk was found until PM_{10} concentrations reached 50 ug/m3. Mortality and air quality data were obtained at the county level. Individual PM_{10} sensors within a designated area were averaged, and values in the extreme 10% were removed from the analysis to eliminate outliers. Age was considered in the analysis by dividing the population into three groups: <65, 65-74, and >75 years of age. Current-day and one-day lag were examined, and

the curves did not change significantly. Examples of ICD-9 codes considered were cardiac disease, chronic obstructive pulmonary disease, influenza, and pneumonia. The authors examined linear and spline models. In the linear model, a 0.69% increase in respiratory and cardiovascular mortality was found for every 10 ug/m3 increase in PM10, and a 0.54% increase was found for all-cause mortality. Using a spline model, total and cardio-respiratory mortality were linear with no threshold, and for other cause mortality, a 50 ug/m3 threshold was observed.

Ozone and Asthma Emergency Room Visits - A study was conducted in New Brunswick, Canada, to determine if there was an association between ambient ozone levels and asthma emergency room visits (Stieb, et al., 1996). This study did not look at mortality or inpatient admissions, but rather ER visits. Asthma incidence was the only disease-related endpoint that was examined. This study did take into consideration weather and particulates as a co-pollutant, but these factors were not found to be significant. It is important to note that the ambient PM measurements were taken only every six days, which makes it difficult to draw a direct comparison to a daily ozone measurement or hospital admission count.

The study looked at a zero- to three-day lag. Some studies suggest that there are exposure thresholds (Lippmann, et al., 2003), which is a logical assumption since the literature points to the saturation of enzymes that neutralize ROS or physically clear particles from the lung spaces. Ozone was examined both as a daily average and the 24-hour maximum value, which is how this thesis is designed. The area of interest only consisted of a population of 125,000, or 1/6 of the Baton Rouge AQCR. The statistical analysis utilized was a Poisson regression, and a non-linear correlation was found with a lag of two days from

ozone measurement to ER visits with a $p<0.05$ when ozone was >75 ppb. Researchers also found that a quadratic model fit better than a linear model, which they suggest is because that there is a low threshold of ozone concentration to asthma.

Ozone and Asthma-related Hospital Admissions - Lu et al. (2003) took a spatial relationship approach to their analysis of ozone and asthma admissions in the California South Coast Air Basin. The study looked at Air Quality Index (AQI) levels with a 1-day lag and asthma admissions at hospitals in the area and compared those values to over 1.1 million hospital visits for asthma over a three year period. The AQI is a formula used to convert air quality measurements into a numerical scale of risk that is often broadcast to the public in a color-coded risk index. Each 50 "points" is usually color coded as green, yellow, red, etc to relate a relative risk.

This area contained 30 ozone monitors, which is a much larger array than can be found in the BR-AQCR. Since the sensors are spread out over a large geographic area, GIS was used to determine concentration estimates between sensors. These spatially interpreted values were then compared to the 100 meter by 100 meter census tracts and the incidents of asthma within each one. The study found no significant relationship between ozone and asthma on these variables alone. One would expect the summer months to produce higher levels of ozone due to higher temperatures, and by that logic, one would also expect to see higher incidences of asthma. However, the opposite was the case, and it was in fact the winter months that had the greater incidents of asthma. For the month of August, the lowest asthma rate was found when modeled with ozone levels with a $p<0.001$.

A multivariate analysis was then conducted with time factors (seasons) included in the model. Using a Poisson regression, the researchers found that when the AQI increased by 50, asthma increased by 11% in children less than 5 years of age. The study's key point is that ozone, by itself, may not be a significant risk factor in ambient concentration levels, and that seasonal variability may be important as well.

Toxic effects are not directly caused from ambient concentration, but rather from interaction and deposition of PM on tissue surfaces and subsequent cellular interaction (Broday et al., 2001). The purpose of studies such as these is to draw some conclusions on measurements that are practical and that can be made, and to draw some general conclusions based on the data available.

3. MATERIALS

Air quality data for ozone and particulate matter was obtained from the Louisiana Department of Environmental Quality. These data sets cover the period of time from 1 January 2000 to 31 December 2003 for monitoring sites in East Baton Rouge, West Baton Rouge, Ascension, Livingston, and Iberville Parishes. Request for data was made by phone to the Air Quality Evaluation Division, and data was obtained in three working days. For perspective, there were approximately 350,000 individual data points for ozone and 72,000 for particulate matter between 2000 and 2003.

Since this study consists of potentially sensitive medical data and history, a Louisiana State University Institutional Review Board (IRB) application was submitted on 23 June 2004 and approved on 25 June 2004. IRB approval was mandatory prior to making the requests for mortality statistics.

Mortality statistics were obtained through the Louisiana Department of Health and Hospitals (LA DHH). An application was submitted for mortality statistics on 5 July 2004 to the Vital Records Review Panel, and data was obtained in electronic format in September 2004. The process for release of this data involved the review of the application by four members of the LA DHH staff, and the subsequent approval by at least three of the four members.

The mortality data request consisted of a query of all cases of mortality under the "I" and "J" alphanumeric codes in the International Classification of Diseases, Tenth Revision (ICD-10) for 2000-2003. Diseases with an "I" classification refer to cardiovascular conditions, while "J" codes refer to respiratory illness. Specifically, the query requested each mortality case, by date of death, for all cardiovascular and respiratory illness sub-codes.

Parish of residence, zip code of residence, gender, and age at death were also obtained. The parish of residence consisted only of the five parishes of the BR-AQCR – the target area of this study. In addition, for comparison purposes, mortality cases for all causes were obtained with the same data parameters. This resulted in 20,008 total deaths during the four year study period, 7,200 of which were I-codes and 1,569 were J-codes.

Statistical analysis was completed on a Dell Inspiron 8500 notebook computer running SAS 9.0. The software and license for SAS 9.0 were obtained from the Experimental Statistics Office at Louisiana State University.

4. METHODOLOGY

The literature review illustrated there is evidence that the criteria pollutants exert detrimental effects that impair or damage the proper functionality of the cardiovascular and respiratory systems. This is due to direct interactions with lung tissues due to route of exposure, as well as reactions due to subsequent transport in the blood and throughout the circulatory system. This does not infer that theses pollutants exert no effects on other systems. Since ambient concentrations can interact directly with the lungs followed by entry into circulation, and also that these two systems are also intimately related (functionality of one can affect functionality of the other), the relation between ambient pollution and effects is highly relevant.

4-1: Site Selection

The BR-AQCR was selected for the study for several reasons. First, Baton Rouge has received its share of critical press for having exceeded the EPA's NAAQS standard every year since at least 1980 according to tabular data on the LA DEQ's ozone evaluation website. (http://www.deq.state.la.us/evaluation/ozone). Currently, this Air Quality Control Region is not in compliance for the 1-hour ozone standard of 0.12 ppm. This violation, in the legal and possibly the public health sense, affects the parishes of East and West Baton Rouge, Ascension, Iberville, and Livingston.

Second, the high level of industrial activity and volume of transportation situated relatively close to a large population center could provide a more substantial and defendable link between ozone, fine particulate matter, and illness. Third, the opportunity to examine a region that is not in compliance with NAAQS standards for ozone with a simultaneous

analysis of particulate matter concentrations may provide insight into potential synergistic or antagonistic effects.

Finally, this location is convenient in terms of access to state agencies – the Louisiana Department of Environmental Quality is located in Baton Rouge and the Department of Health and Hospitals is in New Orleans. This made communications and assistance fairly simple.

4-2: Data Validation

4-2.1: Ozone

There are 10 ozone sensors within the BR-AQCR (table 3). Figure 1 in the literature review section illustrated the geographic dispersion of the sensors within the study region. The majority of the sensors are located near the more dense population areas, which is beneficial for this study in the sense that the measurements would be more representative of the ambient concentration to which the majority of individuals could be exposed. However, there are significant areas with no sensor coverage, and data could potentially be erroneous due to a lack of substantial sensor coverage, especially in Livingston Parish (only one sensor for ozone in the second most highly populated parish).

Table 3: Population Counts and Ozone and $PM_{2.5}$ Sensor Density by Parish

Parish	Population	% BR-AQCR	# of Ozone Sensors	# $PM_{2.5}$ Sensors
East Baton Rouge	412,852	64.9%	4	4
Livingston	91,814	14.4%	1	1
Ascension	76,627	12.1%	1	0
Iberville	33,320	5.2%	3	2
West Baton Rouge	21,601	3.4%	1	1
Totals	636,214	100%	10	8

Since ozone is primarily formed as a sunlight-driven reaction, it is imperative to have relatively complete hourly data sets that incorporate the times of peak illumination. During times of maintenance, power failures, or equipment malfunctions, there are dates for which a full compliment of 24 hourly measurements is not available. Daily maximum values are found between noon and sunset, and a station which has detected only 50% of the daily readings may only have taken samples during the nighttime or other less-than-optimal time frames. This may have the effect of skewing the data from the true daily average, or may obscure or omit the peak daily value. 40 CFR Part 50 delineates the procedures and protocols for determining the primary and secondary NAAQS measurements, and Appendix H of this CFR specifies that for an ozone daily maximum value to be valid, at least 75% of the daily measurements must have been obtained. For the BR-AQCR, the percentage of the 1-hour measurements that were recorded by individual station ranged from 89.6% to 98.3% per year, with an average of 96.3% over the four-year period.

This is critical in the context of ozone. 75% covers a reasonable span of time and provides a rational threshold for using a daily value. To account for this in the analysis, data sets were examined for missing values, and any dates which were missing more than 25% of their measurements were discarded. By removing individual data sets based upon this preliminary screen, the statistical analysis should better reflect actual conditions with a slightly smaller data set as opposed to performing a statistical analysis on inaccurate daily measurements.

Ozone hourly values for each station, after applying the 75% threshold, were examined for two specific values. First, the highest hourly value obtained during a 24-hour period was determined and was labeled as the Daily Peak Value (DPV), which is directly tied

to the EPA's regulations; it is this value that determines if a location has had an exceedance. The second value, Daily Average Value (DAV), was calculated to be the mean value of the hourly measurements in a 24-hour period. Figure 8 is a plot of BR-AQCR daily peak and daily average ozone concentrations over the four-year study period. This figure shows a definite seasonal trend, with higher ozone values in the summer and fall, and lower concentrations in the winter.

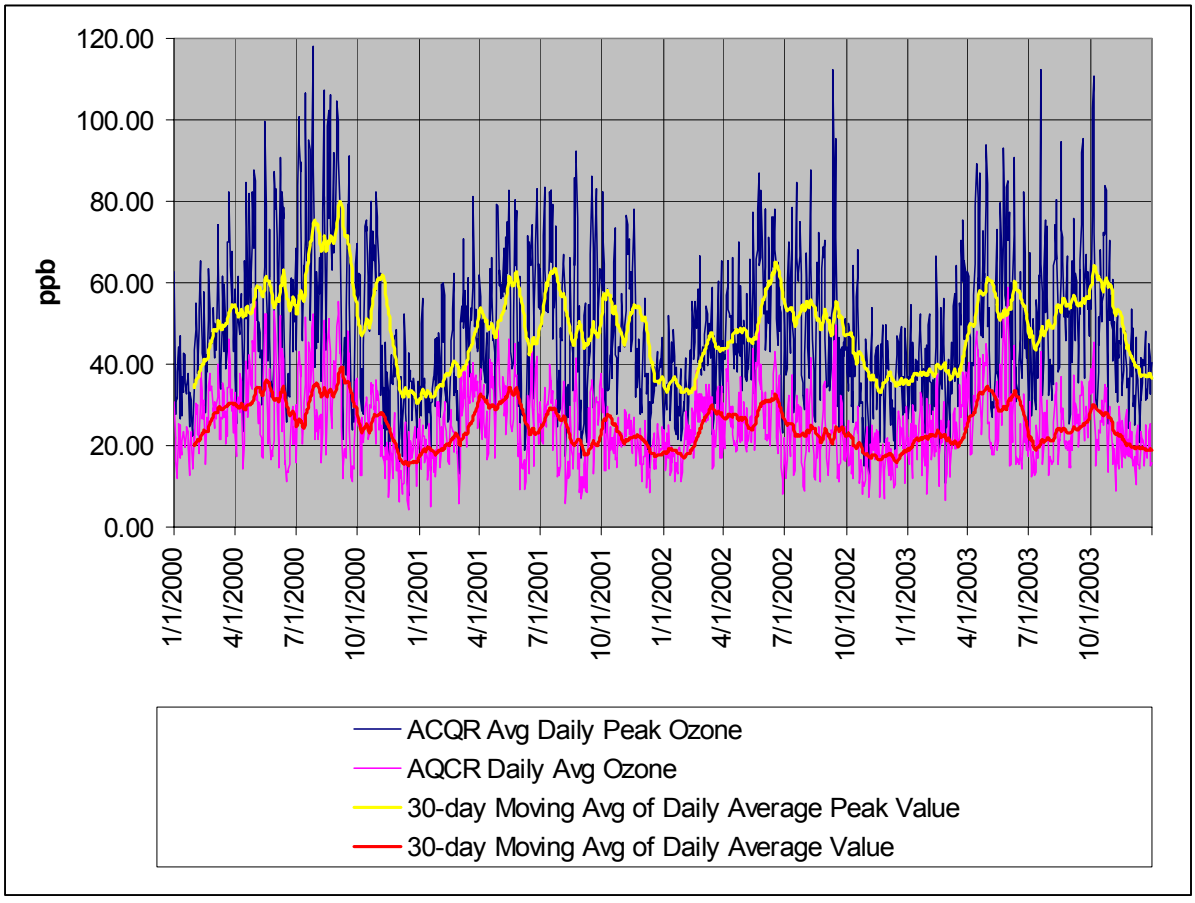


Fig. 8: BR-AQCR Ozone Trends

After application of the 75% threshold, 251 data points were removed from the analysis. This resulted in 98.3% of the ten daily validate ozone sensor locations available for producing a value for DPV and DAV calculations. Once DPV and DAV were determined for each of the ten sensor locations, an average was calculated for the entire region, resulting in

the AQCR Average Peak Value (APV) and the AQCR Average Daily Value (ADV). These are the values that represent the ambient exposure concentration for an individual residing within the BR-AQCR. An example of the results are provided in Table 4. The twenty dates listed are the days of non-attainment for the 24-hour ozone standard in the BR-AQCR between 2000 and 2003.

4-2.2: Particulate Matter

There are 10 particulate matter sensors (eight $PM_{2.5}$ and two PM_{10}) located within the BR-AQCR. Unlike the ozone sensors, the PM sensors consist of several different types. There are six $PM_{2.5}$ sensors which utilize the Federal Reference Method (FRM), which is the method that is mandated by the EPA for NAAQS monitoring purposes. Within this group of sensors, $PM_{2.5}$ is measured at three different intervals as listed in table 5. Three-day samples are all taken on the same date, which allows for calculation of averages on a time scale that is more useful than every six days, but not nearly as detailed as daily concentrations. This temporal difference in monitoring times may pose problems in the statistical analysis since the intent is to look at illness on a daily basis, not an every three- or six-day time frame.

The Partisol-Plus Model 2025 Sequential Air Sampler is manufactured by Rupprecht & Patashnick Co., Inc. This device is designated as the $PM_{2.5}$ reference method for EPA monitoring. It is a combination of particle separator and sequential filter system in which PM2.5 is separated from larger particles and then deposited on a filter disk over a 24-hour period. Air is drawn into the device at 16.7 l/min ($1m^3$/hr), which establishes the baseline for the units of measurement. When the sampling period is complete, the device pneumatically slides the used filter to a holding chamber and moves a clean filter into place for the next

Table 4: Daily Peak and Daily Average 1-Hour Ozone Values for All BR-AQCR Monitor Sites

	Grosse Tete		Port Allen		B.R. Capitol		LSU		Baker		Pride		Bayou Plaquemine		Carville		Dutchtown		French Settlement		AQCR	
Date	**DPV**	**DAV**	**DPV**	**DAV**	**DPV**	**DAV**	**DPV**	**DAV**	**DPV**	**DAV**	**DPV**	**DAV**	**DPV**	**DAV**	**DPV**	**DAV**	**DPV**	**DAV**	**DPV**	**DAV**	**APV**	**ADV**
05/16/00	79	51.25	96	49.33	84	42.13	97	55.25	**136**	56.38	**129**	53.83	85	51.88	84	57.38	88	45.42	117	58.38	99.50	52.12
07/05/00	91	38.38	107	47.29	107	48.83	109	47.57	108	43.75	76	37.33	87	37.91	93	40.79	97	36.58	**131**	41.83	100.60	42.03
07/06/00	96	38.70	74	36.61	86	43.65	106	46.83	78	38.57	70	38.32	**126**	47.14	112	53.18	100	43.91			94.22	42.99
07/15/00	95	44.83	110	49.08	102	57.08	117	54.13	98	47.46	101	43.04	80	43.54	114	58.75	120	55.67	**128**	51.96	106.50	50.55
07/26/00	**128**	51.96	118	53.54	**130**	56.21	**139**	65.00	110	53.25	111	45.83	113	46.48	110	52.92	114	49.50	107	47.46	118.00	52.21
08/11/00	74	39.50	**150**	58.88	**144**	58.74	**157**	63.88	**128**	60.38	73	43.88	63	39.08	110	52.96	104	48.22	70	42.11	107.30	50.76
08/17/00	81	38.48	109	46.52	107	50.18	114	52.57	104	48.22	69	31.59	76	37.45	108	51.55	**128**	55.41	87	39.14	98.30	45.11
08/18/00	89	47.26	97	48.29	97	50.50	103	55.17	103	51.50	109	42.04	86	42.71	83	51.88	**131**	53.71	**127**	54.04	102.50	49.71
08/21/00	82	34.57	122	45.95	109	39.45	**135**	57.00	99	46.91	81	30.25	121	46.46	**127**	54.38	109	42.30	77	34.86	106.20	43.21
08/25/00	**136**	43.92	89	41.63	89	43.75	90	44.04	94	43.71	68	30.38	98	36.17	94	41.67	72	33.13	69	27.71	89.90	38.61
08/31/00	93	53.83	124	53.26	118	47.67	115	50.91	**151**	62.09	105	46.82	84	48.73	84	52.05	92	38.74	79	38.05	104.50	49.21
07/15/01	96	35.00	70	40.75	82	44.9	92	43.96	66	37.17	61	30.00	**125**	46.63	97	59.62	67	29.92	67	31.79	82.30	39.97
08/08/02	83	29.52	**128**	44.52	**131**	43.35	102	39.96	84	45.35	64	37.48	66	28.04	67	29.30	95	25.83	58	30.83	87.80	35.42
09/11/02	102	29.00	102	40.79	**164**	50.17	**154**	48.33	81	39.46	79	31.13	**139**	37.29	116	37.23	98	29.88	89	33.29	112.40	37.66
04/27/03	78	38.13	81	44.71	78	38.25	88	42.21	67	37.42	51	30.92	78	44.92	**129**	55.67	79	33.58	62	28.58	79.10	39.44
07/18/03			**129**	46.46	**147**	50.54	**174**	56.04	106	46.92	69	35.79	78	30.58	**129**	46.17	105	42.04	73	34.00	112.22	43.17
08/18/03	81	34.43	**144**	45.90	**139**	43.43	109	45.35	92	39.39	66	31.39	89	31.17	74	35.43	94	34.09	57	30.00	94.50	37.06
09/19/03	89	28.50	95	36.54	105	39.08	**130**	42.54	105	42.83	40	16.50	**127**	35.75	98	42.71	91	30.33	73	28.50	95.30	34.33
10/04/03	101	37.00	98	36.38	114	40.08	**137**	42.38	106	47.92	90	40.38	112	36.50	104	48.08	89	32.38	77	32.25	102.80	39.33
10/05/03	116	37.25	119	54.21	108	45.63	120	49.92	**138**	56.92	104	44.79	94	33.50	115	53.71	102	38.13	92	37.92	110.80	45.20

1) Values in ppb
2) Underlined values are 1-hour exceedance values resulting in BR-AQCR non-attainment status
3) The empty cells on 7/6/00 and 7/18/03 are due to the respective sensors not attaining the established 75% threshold

sampling cycle. When all filters have been used, the collection tube (containing up to 16 filter disks) is removed and sent to a contracted company for mass and composition analysis.

The FRM sensors take $PM_{2.5}$ samples by being "open" to the environment for a 24-hour period from midnight to midnight. Fine particulate matter is separated from the coarse particles, filtered, and collected during that time frame. The mass is then divided by the volume of air that was sampled, giving the NAAQS 24-hour average concentration. FRM sensors were not held to any 75% validation standard as there was in ozone because the values were discrete daily averages and not composed of 24 separate values that had to be averaged.

Table 5: Summary of $PM_{2.5}$ Monitoring Site Types in the BR-AQCR

Sensor Location	**Code**	**Parish**	**$PM_{2.5}$**	**Periodicity**
Baton Rouge Capitol	BC	East Baton Rouge	X	Daily
Pride	PE	East Baton Rouge	X (TEOM)	Hourly
Baker	BK	East Baton Rouge	X	Every 6 days
Baton Rouge (Evangeline)	BE	East Baton Rouge	X	Every 3 days
Port Allen (WLUX)	PA	West Baton Rouge	X	Daily
French Settlement	FS	Livingston	X (TEOM)	Hourly
Geismar	GM	Iberville	X	Every 3 days
Bayou Plaquemine	BP	Iberville	X	Every 3 days

1) Two sensors for PM_{10} are located within this region, but are not included in this study.

In the search for better sensor technology and a more effective sampling timeframe, LA DEQ has put into place two tapered-element oscillating microbalance (TEOM) sensors. The TEOM Series 1400a Ambient Particulate Monitor is also manufactured by Rupprecht & Patashnick Co., Inc. This sensor is capable of real-time measurements of $PM_{2.5}$. It employs an inertial mass measurement technique. As particulates accumulate on the filter at the tip of

the tapered element, the oscillations begin to slow down. The rate of decrease in motion is then transformed into a mass measurement that is representative of the ambient particulate concentration. This particular sensor has been approved by the EPA for correlation with FRM sensors but cannot be used as the sole monitor for reporting purposes.

TEOM $PM_{2.5}$ sensors operate by being "open" to the environment for an unspecified amount of time. The manifold allows fine particulate matter to enter a chamber and the sensor essentially subtracts the mass from the previous measurement from the current measurement – resulting in a real-time measurement of concentrations. The limiting factor in the measurements is that the filter has a critical level at which the filter must be replaced and the time/weight must be zeroed out.

While these are not authorized by the EPA for NAAQS reporting requirements, they can provide valuable data for this study for two reasons. They increase the number of sensors in the BR-AQCR, and they provide $PM_{2.5}$ concentrations on an hourly basis instead of over a 24-hour period. This results in time-series data which is similar to the ozone sensors, and provides a more accurate comparison to hourly ozone measurements for statistical analysis.

Validation of the TEOM data is identical to the ozone data. $PM_{2.5}$ hourly values for each station were subjected to the 75% threshold, and dates below the threshold were discarded. The Daily Average Value (DAV) was calculated for $PM_{2.5}$ to be the mean value of the hourly measurements in a 24-hour period. This conversion served the purpose of placing the TEOM measurements on the same 24-hour average scale as the other sensors, which allowed for the calculation of a more representative area average of $PM_{2.5}$ concentration. The DAV for all eight $PM_{2.5}$ sensors was then averaged to give the BR-AQCR average concentration (figure 9). Again, it is important to reiterate that while the TEOM monitors

sample the airborne concentrations much more frequently, it is in fact the FRM sensors that the EPA uses for determining compliance with NAAQS standards.

Two of the sensors measure coarse particulate matter, or PM_{10}. These sensors obtain a 24-hour sample every six days, and the samples are taken concurrently on the same date. While it would have been valuable to determine if there was an effect of PM_{10} on health, the six-day gap in data between the days of sampling would make it difficult to make an accurate comparison. In addition, if one of the two sensors did not provide a value for a particular date, then there is no way to calculate any semblance of an average concentration for the five parishes. Therefore, PM_{10} was not analyzed in this study.

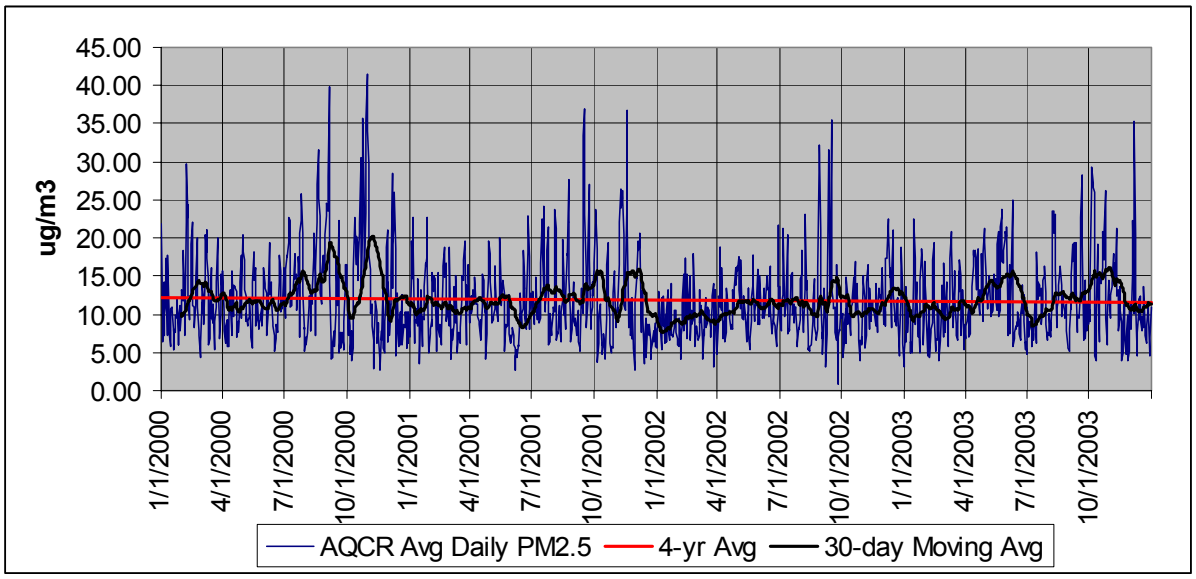


Fig. 9: 2000-2003 BR-AQCR $PM_{2.5}$ Daily Average Concentration

4-3: Mortality Data

Mortality data was obtained through the Louisiana Department of Health and Hospitals. LA DHH acted as a clearing house for all health related data for this study. For mortality statistics, whenever an individual dies, the personal information and the cause of

death, as determined by a coroner under the ICD-10 coding scheme, is sent to the DHH for record-keeping.

To list all of the specific ICD-10 codes and sub-codes, even those that fall only under the cardiovascular and respiratory illness categories, would take up a significant amount of space. The purpose of table 6 is to list the general disease categories to give the reader an idea of some of the causes of death that were targeted in this study. These specific codes were selected based upon the literature review and the evidence that both ozone and fine particulate matter can have adverse effects on the lungs, heart, and vasculature.

This is not an all-inclusive list.

Table 6: ICD-10 Codes Used in Mortality Statistics

ICD-10	**Major mortality category**	**Sub-categories**
I00-I01	Acute rheumatic fever	
I05-I09	Chronic rheumatic heart diseases	
I10-I15	Hypertensive diseases	Hypertension, hypertensive heart/renal disease
I20-I25	Ischemic heart disease	Angina, AMI, coronary insufficiency, atherosclerotic heart disease
I26-I28	Pulmonary heart disease	Diseases of pulmonary circ.
I30-I51	Other forms of heart disease	Valve disorders, cardiomyopathy, cardiac arrest, heart failure (I50)
I60-I69	Cerebrovascular disease	Cerebral aneurysm and stroke
I70-I79	Disease of arteries/capillaries	Atherosclerosis
I80-82, I87-I89	Diseases of veins	Phlebitis and embolisms
I95, I97, I99	Other circulatory diseases	hypotension
J00-J06	Acute upper respiratory infections	
J10-J18	Influenza/pneumonia	
J20-J22	Other acute lower resp. infections	Acute bronchitis
J30-J32	Rhinitis/sinusitis	
J40-J47	Lower respiratory disease	Emphysema, COPD, asthma
J80-J84	Other resp. diseases affecting interstitium	Edema
J90-94	Diseases of pleura	
J95-J99	Other respiratory diseases	Post-op disorders, respiratory failure

5. STATISTICAL ANALYSIS

5-1: Sensor Measurement Correlations

It is clear that the air quality measurements will differ to some degree from location to location within this 5-parish area. Since air quality measurements will vary, it is logical to believe that exposures would differ depending on where the individual spends most of their time. Gaps in sensor coverage already make it impossible to pinpoint concentrations for a single household, much less a zip code. Even geocoding and spatial statistics will only be an approximation based upon 8-10 individual monitoring stations. Concern exists on the validity of a statistical model which does not account for the potential error. This thesis intends to determine how the sensors correlate.

It is assumed for the purposes of this study that the air in the BR-AQCR is a homogeneous mixture and that ozone and particulate matter concentrations are equal at any location within this area. To make this assumption with some qualitative measure of certainty, correlation matrices were constructed using SAS to determine how differently each sensor type varied by geographic position. The ten ozone sensors are all composed of the same equipment and taken on equal time intervals. This makes correlation simpler than for the variety found in the $PM_{2.5}$ monitors.

DPV and DAV for each ozone sensor were examined for the degree of linear correlation for each daily measurement, and the scatterplots for each site show a high degree of correlation between sensors (figures 10 and 11).

After further examination of the correlation matrix, the correlation coefficients for individual sites confirm the strong linear relationships for both DPV and DAV, significant to $p<0.0001$ (tables 7 and 8). However, they also illustrate the problems referred to previously.

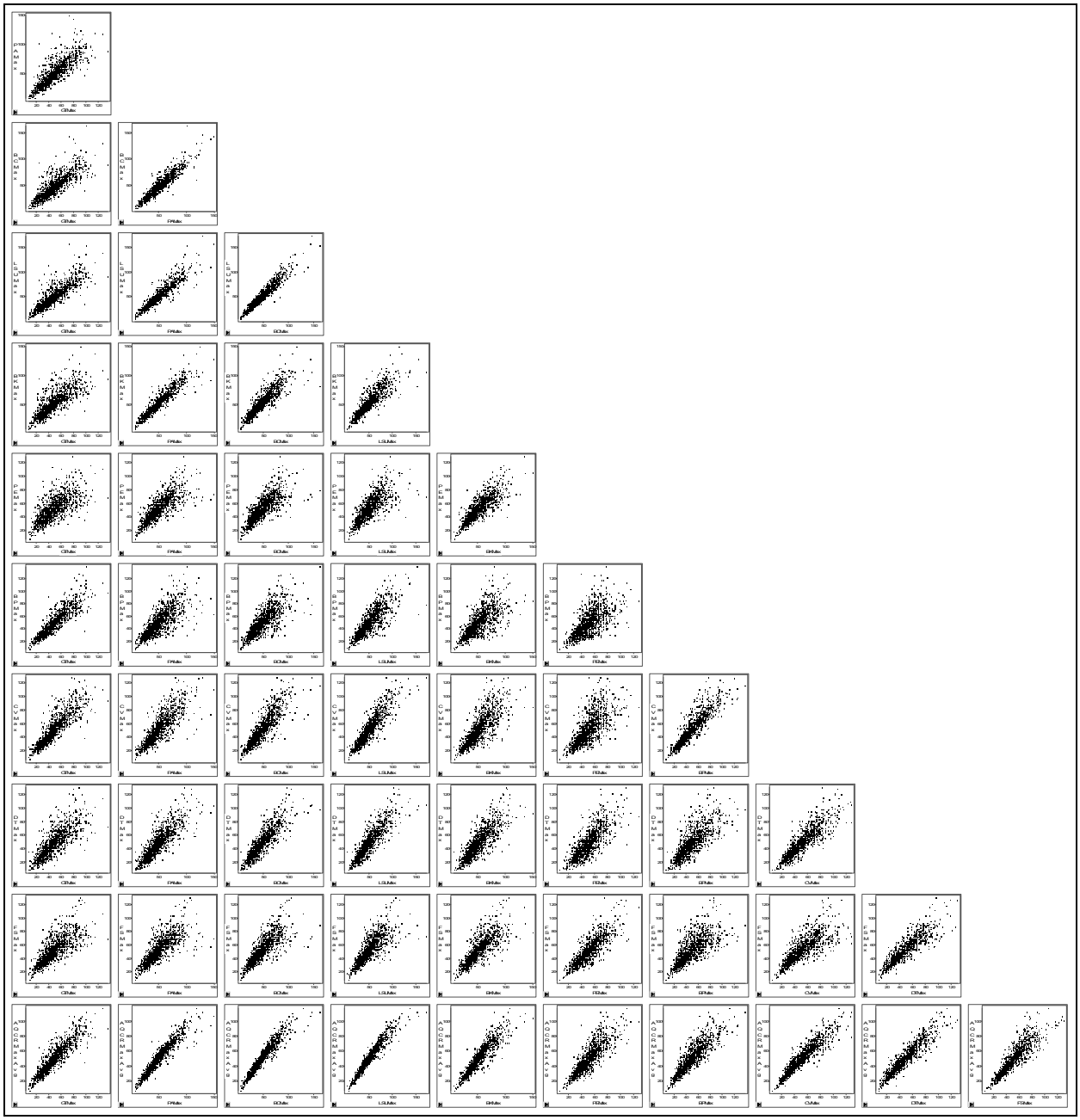


Fig. 10: Correlation Scatterplots of Ozone Daily Peak Values (DPV)

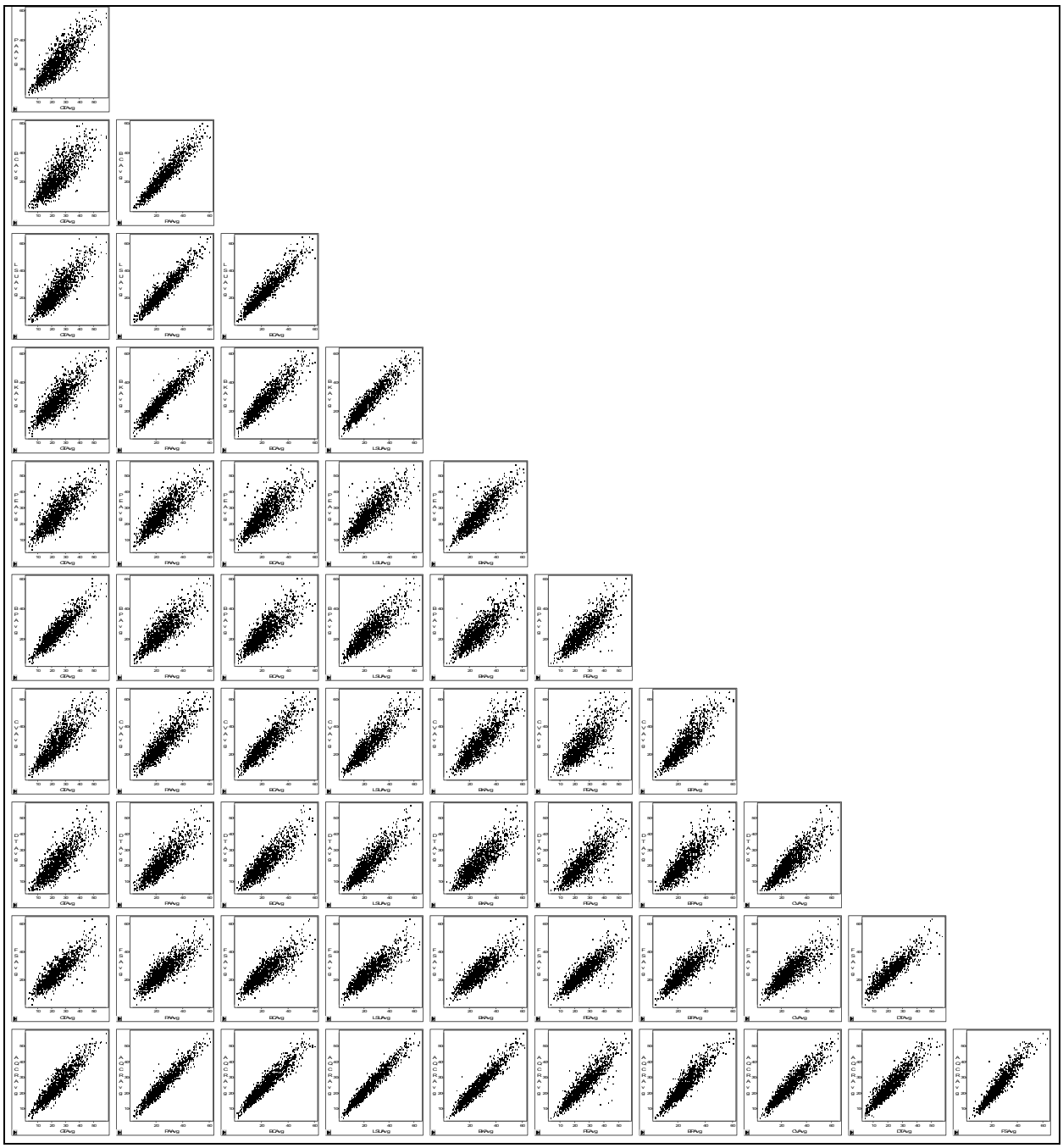


Fig. 11: Correlation Scatterplots of Ozone Daily Average Values (DAV)

Table 7: Correlation Matrix for Ozone DPV

Location	Grosse Tete	Port Allen	Capitol	LSU	Baker	Pride	Bayou Plaq.	Carville	Dutchtown	French Sett.
Grosse Tete		0.8486	0.8373	0.8613	0.8032	0.7163	0.9080	0.8833	0.8101	0.7840
Port Allen	0.8486		0.9432	0.9352	0.9409	0.8137	0.7990	0.8549	0.8620	0.8140
Capitol	0.8373	0.9432		0.9587	0.8974	0.7979	0.8125	0.8792	0.8909	0.8272
LSU	0.8613	0.9352	0.9587		0.8897	0.7915	0.8480	0.9144	0.8938	0.8273
Baker	0.8032	0.9409	0.8974	0.8897		0.8565	0.7367	0.8077	0.8380	0.8278
Pride	0.7163	0.8137	0.7979	0.7915	0.8565		0.6748	0.7259	0.8133	0.8697
Bayou Plaq.	0.9080	0.7790	0.8125	0.8480	0.7367	0.6478		0.9114	0.7906	0.7422
Carville	0.8833	0.8549	0.8792	0.9144	0.8077	0.7259	0.9114		0.8859	0.7922
Dutchtown	0.8101	0.8620	0.8909	0.8938	0.8380	0.8133	0.7906	0.8859		0.9005
French Sett.	0.7840	0.8140	0.8272	0.8273	0.8278	0.8697	0.7422	0.7992	0.9005	
Avg. Corr.	0.8280	0.8657	0.8716	0.8800	0.8442	0.7814	0.8026	0.8513	0.8539	0.8205
BR ACQR Avg Correlation = 0.8399 (n=1216)										

Table 8: Correlation Matrix for Ozone DAV

Location	Grosse Tete	Port Allen	Capitol	LSU	Baker	Pride	Bayou Plaq.	Carville	Dutchtown	French Sett.
Grosse Tete		0.8409	0.7952	0.8517	0.8402	0.8218	0.9173	0.8368	0.8464	0.8579
Port Allen	0.8409		0.9464	0.9476	0.9482	0.8242	0.8482	0.8921	0.8566	0.8544
Capitol	0.7952	0.9464		0.9393	0.9149	0.8039	0.8188	0.9116	0.8712	0.8556
LSU	0.8517	0.9476	0.9393		0.9314	0.8253	0.8567	0.9048	0.8789	0.8664
Baker	0.8402	0.9482	0.9149	0.9314		0.8714	0.8374	0.8620	0.8454	0.8694
Pride	0.8218	0.8242	0.8039	0.8253	0.8714	0.8374	0.8620	0.8454	0.8694	0.9539
Bayou Plaq.	0.9173	0.8482	0.8188	0.8567	0.8374	0.8288		0.8688	0.8376	0.8641
Carville	0.8368	0.8921	0.9116	0.9048	0.8620	0.7797	0.8688		0.8699	0.8408
Dutchtown	0.8464	0.8566	0.8712	0.8789	0.8454	0.8095	0.8376	0.8699		0.9040
French Sett.	0.8579	0.8544	0.8556	0.8664	0.8694	0.8757	0.8641	0.8408	0.9040	
Avg. Corr.	0.8454	0.8843	0.8730	0.8891	0.8800	0.8278	0.8568	0.8702	0.8644	0.8741
BR ACQR Avg Correlation = 0.8665 (n=1216)										

There is definitely a difference in daily ozone readings based on geographic location within the BR-AQCR. The values for LSU, Capitol, and Port Allen exhibit the highest correlation coefficients with each other (a value of 1.00 equals complete linear correlation). This is expected since they are geographically the closest to one another. Pride and Bayou Plaquemine exhibit the least correlation, which is also expected because these two monitors are at the northern and southwestern extremes.

Additionally, the average correlation for all sensors across the BR-AQCR was calculated for both daily peak (0.8399) and average ozone measurements (0.8665). This calculation is very useful since it shows that sensors throughout the area tend to measure similar values over a 24-hour sampling period, as opposed to measuring correlating daily peak values which may be extremely transient. The importance of this is twofold.

First, in the context of this study, DAVs should be better measures of homogeneity for the ambient conditions throughout the BR-AQCR. Second, from a regulatory standpoint, this data shows that peak values are not as consistent through a geographic area. If the purpose of NAAQS limits is to protect public health, then an average should be a better indication of potentially hazardous exposure levels. A high, transient peak value may be detrimental, but if it occurs at a location that is far from the more dense population areas, it will not translate into a good estimate of exposure and risk. This supports the EPA's decision to transition to an 8-hour moving average limitation for ozone for areas that are already in attainment for the 1-hour limit.

Given the strong correlation between the sensors, especially those which are situated near some of the most dense population areas, this data supports using an non-weighted average of all ten ozone sensors for the mortality regression analysis. BR-AQCR average

peak values (APVs) and average daily values (ADVs) will be examined independently to determine if there is a difference in their affects on mortality.

Again, for $PM_{2.5}$, it is assumed that the air in the BR-AQCR is a homogeneous mixture. However, the particulate matter data consists of two different types of technology and is comprised of four different time scales. Correlation matrices were constructed using SAS to determine how differently each sensor type varied temporally, geographically, and by sensor type. DAVs for each ozone sensor were examined for the degree of linear correlation for each daily measurement, and the scatterplots for all sites, 3- and 6-day measurement sites, and daily measurement sites are provided in figures 12, 13, and 14 respectively. At a glance, it is apparent that the different $PM_{2.5}$ sensor types do not correlate as well with each other as compared to the ozone sensors. After further examination of the correlation matrices, the correlation coefficients for individual sites confirm relationships between sensors, significant to $p<0.0001$, but less so than for ozone (tables 9, 10 and 11). The Capitol monitor exhibits the highest average correlation when compared to all sensor types, and when compared only with sensors scheduled for daily sampling. This makes sense because it is the most centrally located monitoring site. The French Settlement monitor sits at the opposite of the geographic and correlation spectra with lowest correlation coefficient averages when compared to all sensors and within the daily sampling group.

Technology and sampling frequency plays a significant part in the correlation between sensors. The two TEOM sensors exhibited some of the lowest correlation values. This may be because they are in very rural locations or possibly because of the technology employed in the sensors. Sensors that operate on 3- and 6-day sampling cycles exhibited the highest average correlation. These results further complicate the process of determining which

monitors should be used to calculate a daily average concentration for use in the mortality regression. It would be a much more valid study if all sensors were the same type and took readings during the same time frames. Since there are no co-located monitors, this analysis cannot determine whether the FRM or TEOM sensors are more accurate.

If all sensors are averaged together, regardless of technology or frequency, Day-1 will incorporate seven to eight $PM_{2.5}$ values. Day-2 and -3 will not include four of the sensors, which may present an average that is skewed geographically. It is important to include as many of the sensors as possible without skewing the daily averages towards the 1-day monitoring site values. Since the average correlation coefficients are at least 0.2 from perfect linearity, all daily values were averaged together for regression analysis. To provide a comparison between daily average sensors and 3- and 6-day sensors, mortality will also be examined for a three-day sampling cycle.

Table 9: Correlation Matrix for all $PM_{2.5}$ Daily Average Values

Location	Capitol	Evangeline	Baker	Bayou Plaq.	Gesimar	Port Allen	Pride (TEOM)	French Sett. (TEOM)
Capitol		0.9079	0.9505	0.8416	0.8982	0.9403	0.7836	0.7871
Evangeline	0.9079		0.8689	0.7398	0.8039	0.8452	0.8731	0.7097
Baker	0.9505	0.8689		0.8070	0.8535	0.9186	0.8428	0.7706
Bayou Plaq.	0.8416	0.7398	0.8070		0.8304	0.7975	0.6513	0.6491
French Sett.	0.7871	0.7097	0.7706	0.6491	0.7387	0.7098	0.7647	
Geismar	0.8982	0.8039	0.8535	0.8304		0.8512	0.6953	0.7387
Port Allen	0.9403	0.8452	0.9186	0.7975	0.8512		0.7489	0.7098
Pride	0.7836	0.7305	0.8428	0.6513	0.6953	0.7489		0.7647
Avg. Corr.	0.8727	0.8008	0.8588	0.7595	0.8102	0.8302	0.7657	0.7328
BR ACQR Avg Correlation = 0.8039 (n = 146)								

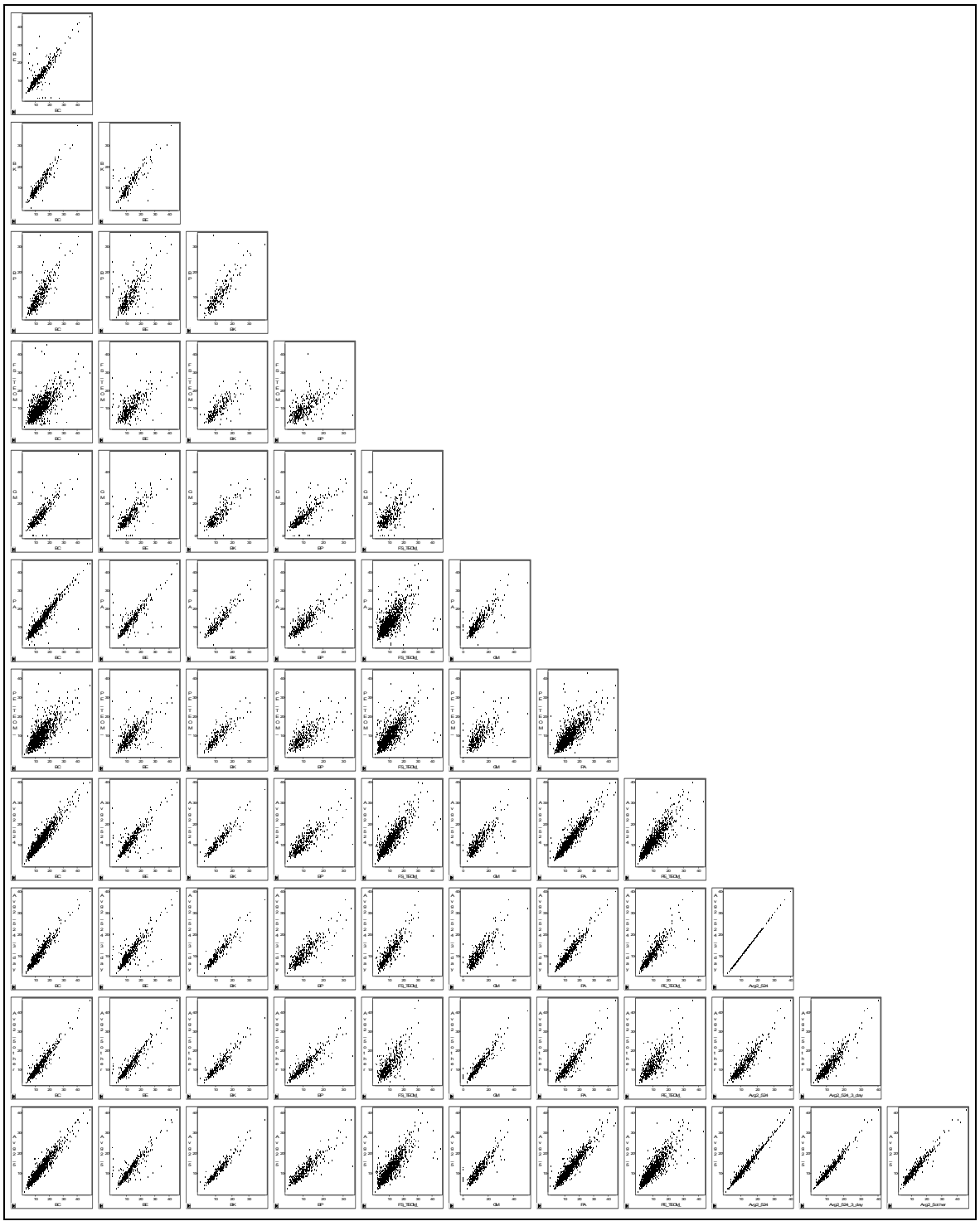


Fig. 12: Correlation Scatterplots of all $PM_{2.5}$ Daily Average Values

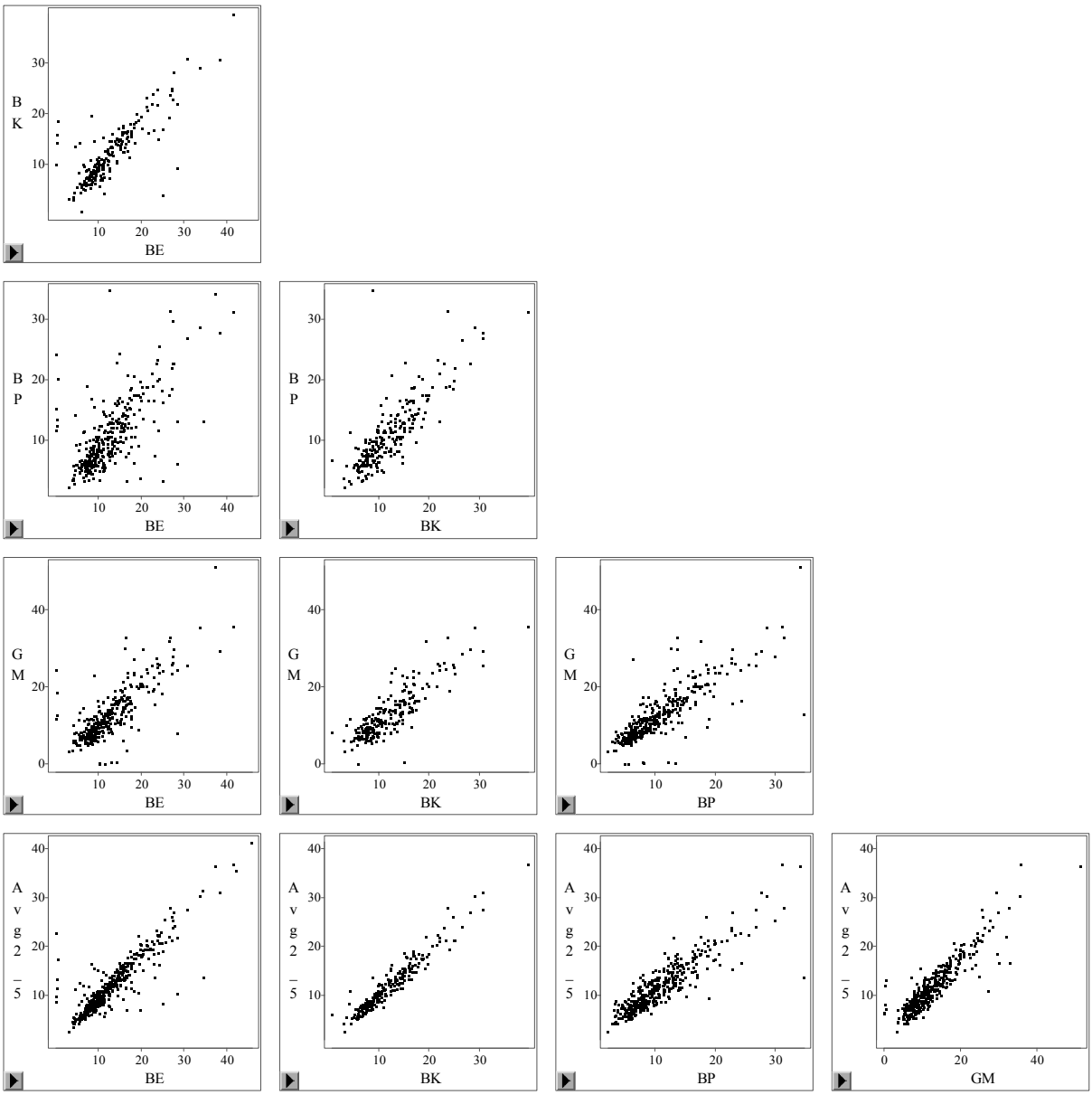


Figure 13: Correlation Scatterplots of 3-day and 6-day $PM_{2.5}$ Daily Averages

Table 10: Correlation Matrix for 3-day and 6-day $PM_{2.5}$ Monitors

Location	Evangeline	Baker	Bayou Plaq.	Geismar
Evangeline (3-day)		0.8768	0.7735	0.8039
Baker (6-day)	0.8768		0.8251	0.8584
Bayou Plaq. (3-day)	0.7735	0.8251		0.8563
Geismar (3-day)	0.8200	0.8584	0.8563	
Avg. Corr.	0.8234	0.8534	0.8183	0.8395
BR ACQR Avg Correlation = 0.8337 (n = 183)				

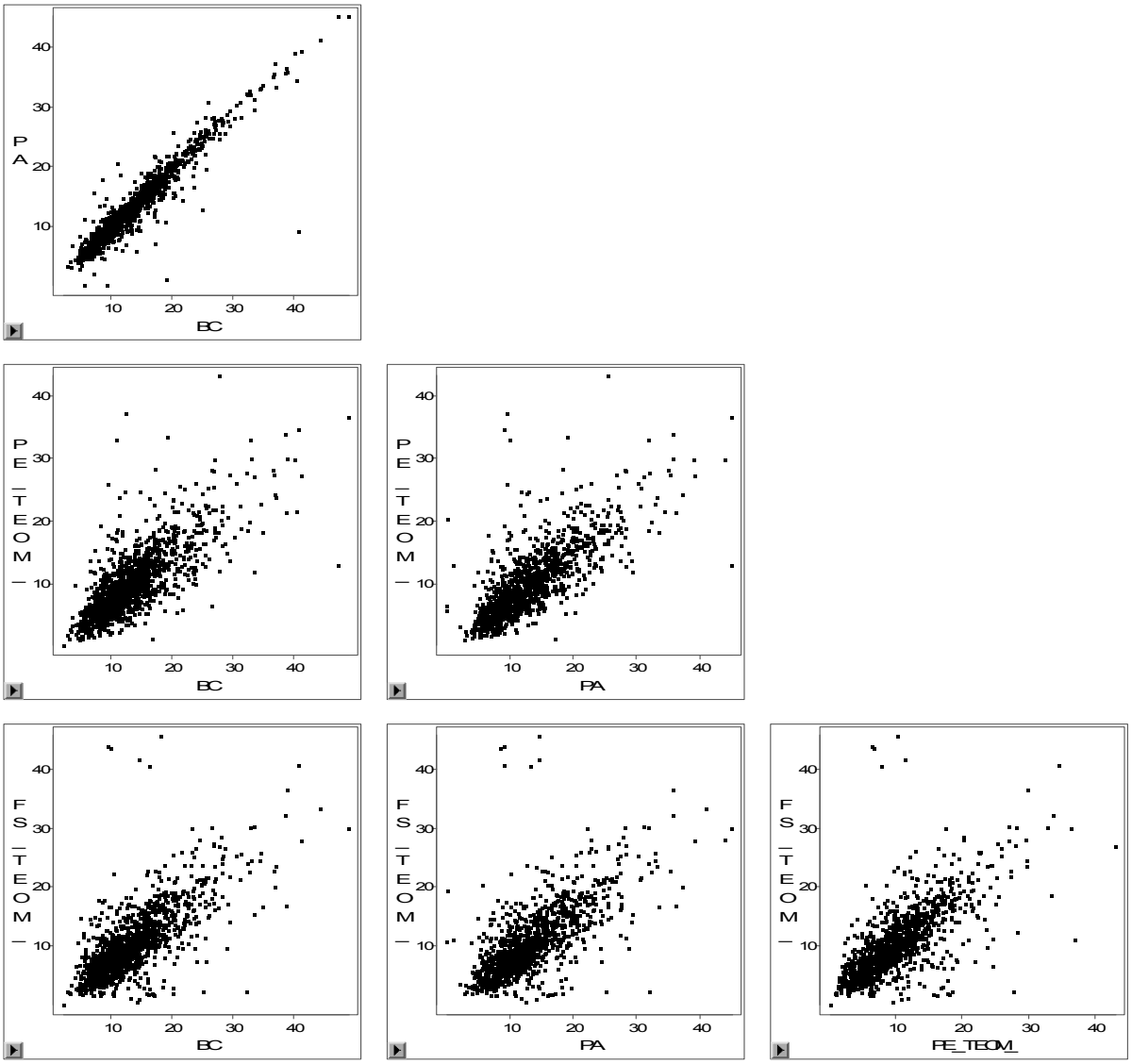


Figure 14: Correlation Scatterplots of Daily $PM_{2.5}$ Monitor Averages

Table 11: Correlation Matrix for Daily $PM_{2.5}$ Monitors

Location	Pride (TEOM)	French Sett. (TEOM)	Port Allen	Capitol
Pride (TEOM)		0.6998	0.7712	0.7873
French Sett. (TEOM)	0.6998		0.6788	0.7412
Port Allen	0.7712	0.6788		0.9476
Capitol	0.7873	0.7142	0.9476	
Avg. Corr.	0.7528	0.6976	0.7992	0.8254
BR ACQR Avg Correlation = 0.7687 (n = 1146)				

5-2: General Mortality Statistics for the Baton Rouge Area

The purpose of the following analyses is to determine the general trends of age, parish, and cause of death in the BR-AQCR (figures 15 through 19). There were 20,009 deaths by all causes and 8,769 cases of death due to cardiovascular (7,200) and respiratory (1,569) causes.

Figure 15 graphically shows that there is a seasonal trend in the number of mortality cases per day. For the period, the average number of deaths by I and J mortality codes is six. The 3-day moving average shows that the mortality rate increases during the winter months. This is an important observation because ambient ozone levels are lowest during this time of year, and therefore may not be a significant risk factor for mortality.

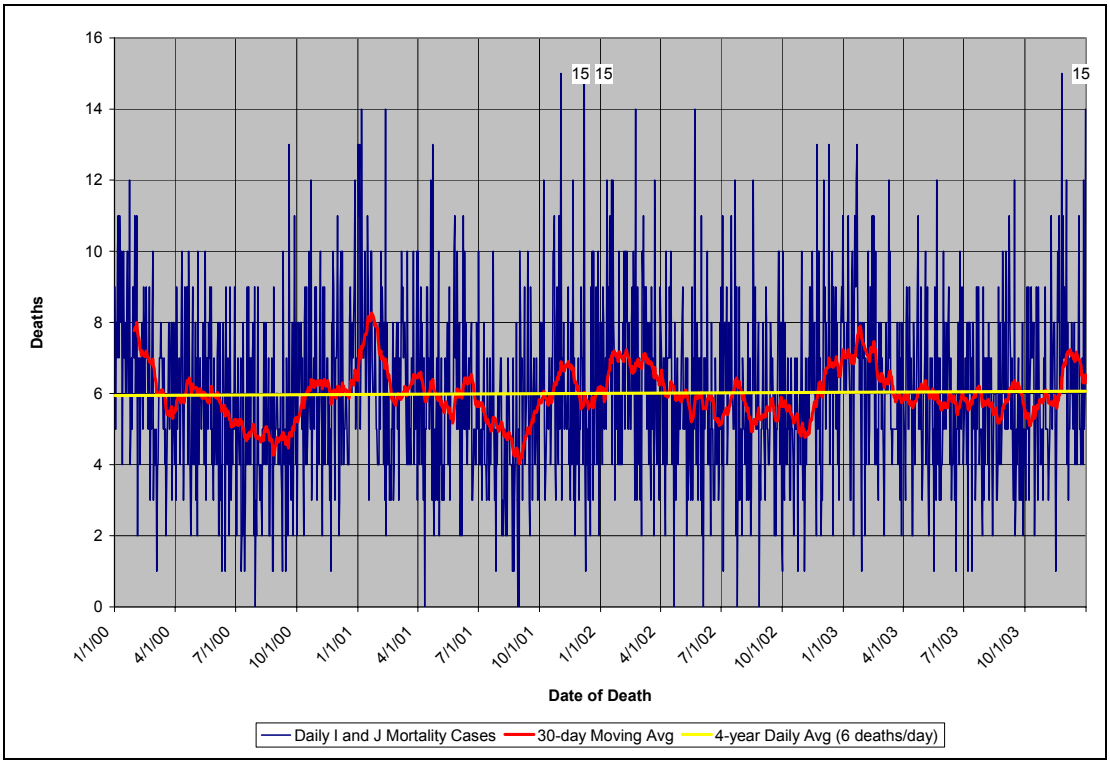


Fig. 15: 4-Year Cardiovascular and Respiratory Mortality Cases

Figure 16 is a histogram of the frequency of I and J mortality cases between 2000 and 2003. With a mean of 6.002 deaths per day and a variance of 6.213, this data very closely

follows a Poisson distribution, which will be used as the method to perform the regression analysis.

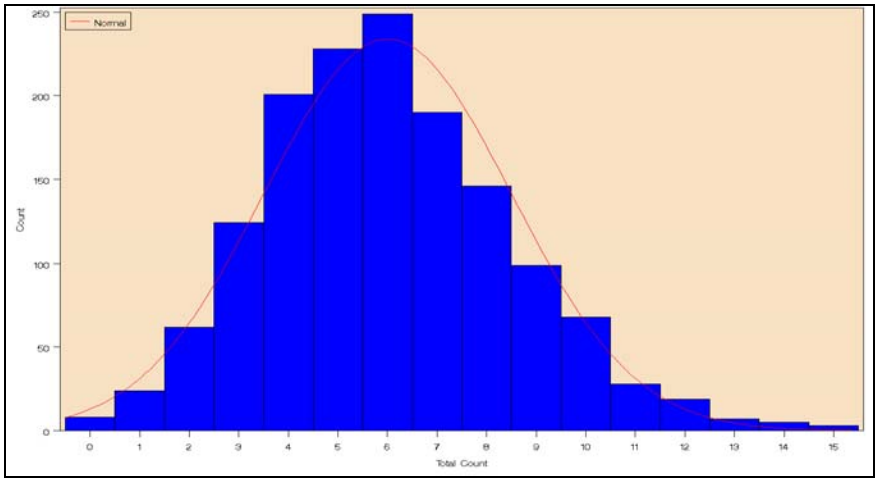


Fig. 16: Histogram of Cardiovascular and Respiratory Mortality Frequency

Figure 17 is composed of a series of box plots, by parish, indicating the average age at death due to cardiovascular and respiratory illnesses. The mean age at death for the five parishes falls within a range of 2.67 years. The mean age at death for the BR-AQCR is 74.3 years. Individual parish mean ages of death do not vary substantially from the overall mean.

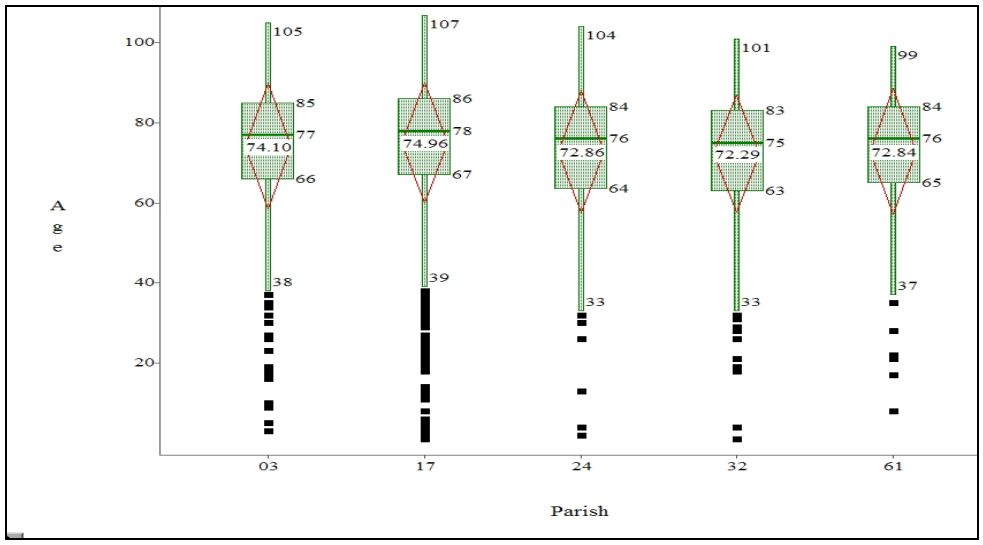


Fig. 17: Distribution of Age at Death Due to I and J Mortality by Parish of Residence
(03 - Ascension, 17 - E. Baton Rouge, 24 - Iberville, 32 - Livingston, 61 - W. Baton Rouge)

Figure 18 illustrates the numbers of the major subcategories of cardiovascular and respiratory causes of death. For the Baton Rouge area, acute myocardial infarctions are the leading cause of death in the cardiovascular and respiratory disease categories.

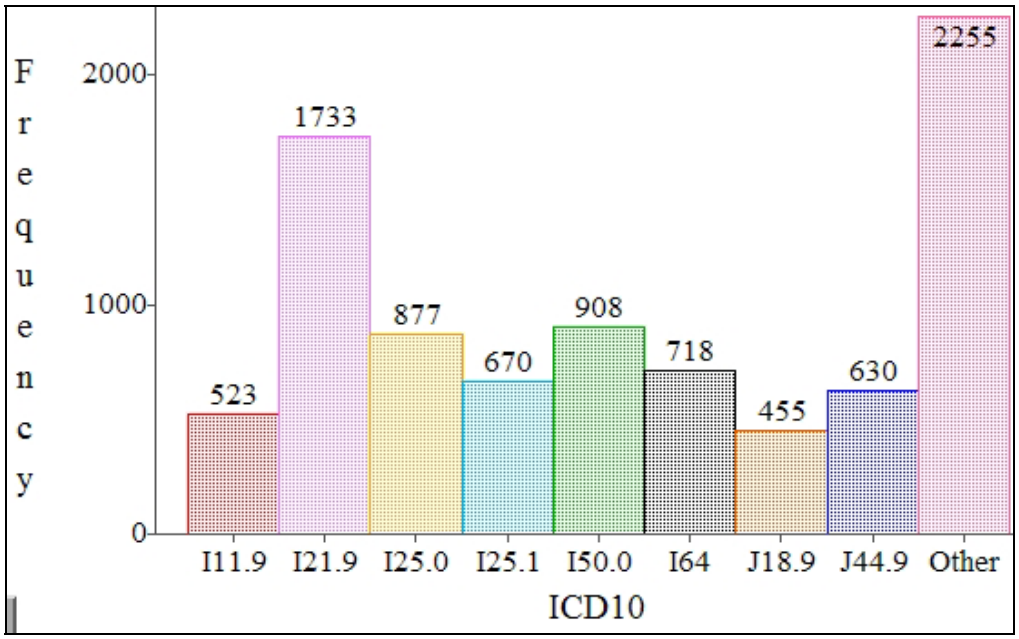


Fig. 18: Frequencies of Cardiovascular and Respiratory Mortality Cases by ICD-10 Code

I21.9 = Acute Myocardial Infarction
I50 = Heart Failure
I25.0 = Atherosclerotic Cardiovascular Disease
I11.9 = Hypertensive Heart Failure
I64 = Stroke
I25.1 = Chronic Ischemic Heart Disease
J44.9 = Chronic Obstructive Pulmonary Disease

The most frequent all-cause mortality cases by ICD-10 code in the 5-parish area are displayed in figure 19. The number one cause of death is by acute myocardial infarction (I21.9), followed by lung cancer (C34.9). Heart failure (I50.0) and atherosclerotic disease (I25.0) are the top third and fourth highest causes of death, respectively. These statistics are significant in the study of air quality because out of any possible cause of death, the top four can be linked potentially to air quality. In addition, the fact that lung cancer is the number

two cause of death may indicate that long-term, chronic exposures to the air quality found in Baton Rouge may have significant effects on tumor formation. However, this statement is not statistically proven in this thesis and would take a substantial epidemiological study to determine.

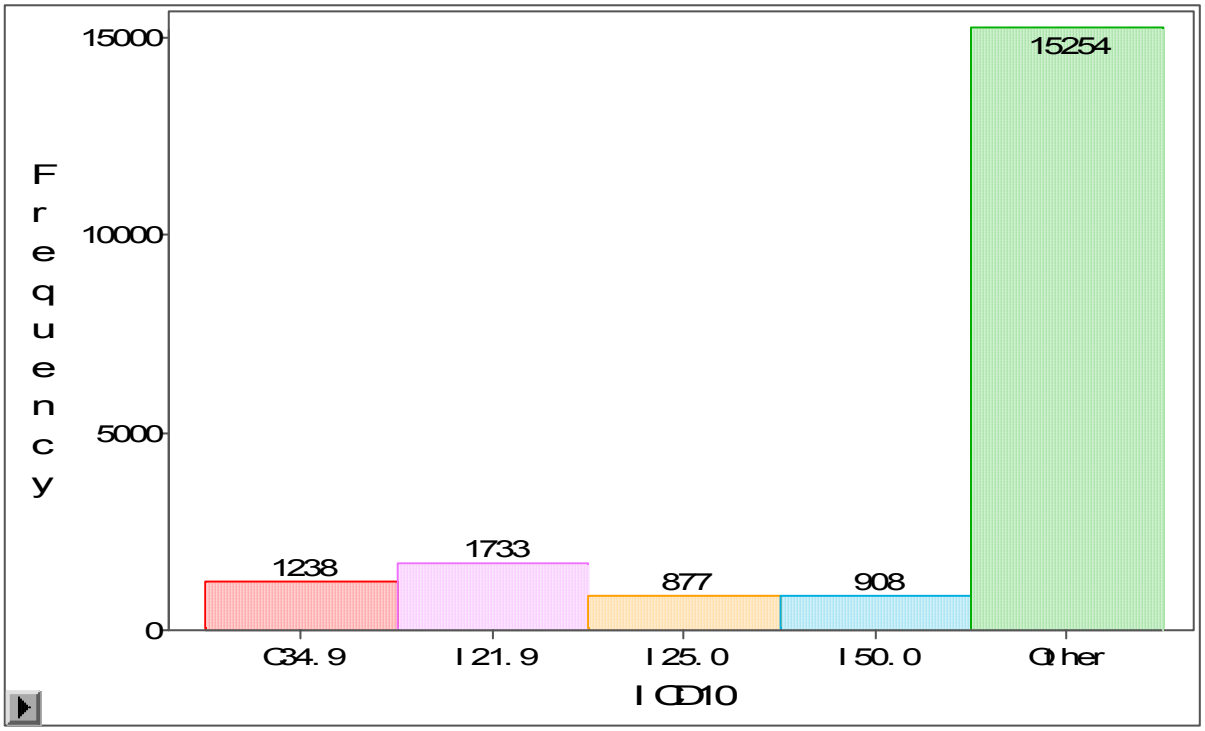


Fig. 19: Frequencies of All-Cause Mortality Cases by Specific Mortality Code.
I21.9 = Acute Myocardial Infarction
C34.9 = Lung Cancer
I50.0 = Heart Failure
I25.0 = Atherosclerotic Disease

5-3: Regression Analysis

Based upon the Poisson distribution of mortality cases, a log-linear regression was performed to determine what effects ozone and fine particulates have on mortality in the Baton Rouge 5-parish area. Mortality frequency was the dependent variable which was log-transformed in the analysis, and air quality averages were the explanatory variables. Ozone and $PM_{2.5}$ values were entered into regression models in SAS in a variety of different

permutations. Each of the following tables will illustrate the different parameters utilized, such as the lag factor that was applied (0-3 days), which air quality value(s) are being used, the number of observations in the regression, etc. Lag is the potential effect that an air pollution concentration has on mortality on a day that is in the future – that is, the number of days between an air quality measurement and the associated number of deaths. In a regression equation, the β_1 (and β_2) value is the factor that, when multiplied by the air quality measurement, serves to give the slope of the regression line. β_1 is then added to β_0, the Y-intercept, which then gives the total regression equation for the line.

5-3.1: Ozone Effects on Mortality

Table 12 contains the regression equations and p-values for Daily Average Values and Average Peak Values of ozone and both I and J and other-cause mortality. All log-linear regressions of ozone for both concentrations show a statistically significant negative relationship between ozone and mortality when using a threshold of $p<0.05$. This data indicates that as ozone levels increase, mortality rates decrease.

For the other-cause mortality regressions, all of the regression slopes were also negative, but only the Average Peak Value at three days of lag was statistically significant. The cardiovascular and respiratory deaths show a slightly steeper negative relationship than the other-cause regressions. As for differences in the lag factors for I and J mortality, β_1 increases approximately 20-30% between day 1 and 2 with an associated decrease in p-values. For other-cause mortality, β_1 remains lower and constant across lag, which when coupled with the previous observations, makes sense because ozone should not have any significant effect on mortality cases not involving an I or J cause of death.

Table 12: Regression Results of Ozone Daily Averages and Peak Averages to Mortality

X	Y	Lag (days)	Equation Log(Y) = $\beta_0 + \beta_1 X$	p-value	n
Ozone ADV	Other Mortality	0	Log(Y) = 2.0609 + (-0.0009)X	0.4005	1457
Ozone ADV	I and J Mortality	0	Log(Y) = 1.8510 + (-0.0024)X	0.0399	1457
Ozone ADV	Other Mortality	1	Log(Y) = 2.0634 + (-0.0010)X	0.3458	1457
Ozone ADV	I and J Mortality	1	Log(Y) = 1.8490 + (-0.0024)X	0.0445	1457
Ozone ADV	Other Mortality	2	Log(Y) = 2.0629 + (-0.0009)X	0.3631	1457
Ozone ADV	I and J Mortality	2	Log(Y) = 1.8788 + (-0.0036)X	0.0024	1457
Ozone ADV	Other Mortality	3	Log(Y) = 2.0760 + (-0.0014)X	0.1628	1457
Ozone ADV	I and J Mortality	3	Log(Y) = 1.8718 + (-0.0033)X	0.0054	1457
Ozone APV	Other Mortality	0	Log(Y) = 2.0678 + (-0.0006)X	0.2772	1457
Ozone APV	I and J Mortality	0	Log(Y) = 1.9007 + (-0.0023)X	0.0002	1457
Ozone APV	Other Mortality	1	Log(Y) = 2.0824 + (-0.0009)X	0.0970	1457
Ozone APV	I and J Mortality	1	Log(Y) = 1.8910 + (-0.0021)X	0.0007	1457
Ozone APV	Other Mortality	2	Log(Y) = 2.0767 + (-0.0008)X	0.1547	1457
Ozone APV	I and J Mortality	2	Log(Y) = 1.9283 + (-0.0028)X	<0.0001	1457
Ozone APV	Other Mortality	3	Log(Y) = 2.1017 + (-0.0013)X	0.0184	1457
Ozone APV	I and J Mortality	3	Log(Y) = 1.9207 + (-0.0027)X	<0.0001	1457

5-3.2: $PM_{2.5}$ Effects on Mortality

Poisson regressions were performed on $PM_{2.5}$ data and the results are listed in table 13. Analysis was completed for three averages: daily averages of all eight monitors, daily averages of sensors that ran on a daily cycle, and averages composed of only the sensors that obtained air quality measurements every 3 and 6 days. For other-cause mortality, none of the regressions produced a statistically significant log-linear association with fine particulate matter.

Only one regression model produced a statistically significant relationship ($p = 0.0487$) between cardiovascular and respiratory mortality cases and $PM_{2.5}$ utilizing a lag of three days. This relationship was negative, as were most of the others, indicating that as particulate matter concentrations increased, mortality from I and J causes decreased. As with

ozone, regression slopes for all monitor types and sampling frequencies did tend to increase between day 1 and 2, however no p-values were equal to or less than $p<0.05$.

Table 13: Regression Results of $PM_{2.5}$ Averages by Sampling Period to Mortality

X1	Y	Lag (days)	Equation $Log(Y) = \beta_0 + \beta_1 X$	p-value	n
Avg2.5	Other Mortality	0	Log(Y) = 2.0315 + (0.0007)X	0.6964	1457
Avg2.5	I and J Mortality	0	Log(Y) = 1.8100 + (-0.0015)X	0.4336	1457
Avg2.5	Other Mortality	1	Log(Y) = 2.0180 + (0.0018)X	0.3011	1457
Avg2.5	I and J Mortality	1	Log(Y) = 1.8030 + (-0.0010)X	0.6083	1457
Avg2.5	Other Mortality	2	Log(Y) = 2.0372 + (0.0002)X	0.9032	1457
Avg2.5	I and J Mortality	2	Log(Y) = 1.8293 + (-0.0033)X	0.1025	1457
Avg2.5	Other Mortality	3	Log(Y) = 2.0660 + (-0.0022)X	0.2159	1457
Avg2.5	I and J Mortality	3	Log(Y) = 1.8378 + (-0.0039)X	**0.0487**	1457
Avg2.5 (24-hr)	Other Mortality	0	Log(Y) = 2.0335 + (0.0005)X	0.7686	1457
Avg2.5 (24-hr)	I and J Mortality	0	Log(Y) = 1.8125 + (-0.0018)X	0.3730	1457
Avg2.5 (24-hr)	Other Mortality	1	Log(Y) = 2.0166 + (0.0019)X	0.2691	1457
Avg2.5 (24-hr)	I and J Mortality	1	Log(Y) = 1.8043 + (-0.0011)X	0.5687	1457
Avg2.5 (24-hr)	Other Mortality	2	Log(Y) = 2.0365 + (0.0003)X	0.8751	1457
Avg2.5 (24-hr)	I and J Mortality	2	Log(Y) = 1.8276 + (-0.0031)X	0.1179	1457
Avg2.5 (24-hr)	Other Mortality	3	Log(Y) = 2.0698 + (-0.0025)X	0.1561	1457
Avg2.5 (24-hr)	I and J Mortality	3	Log(Y) = 1.8374 + (-0.0039)X	0.0507	1457
Avg2.5 (3-day)	Other Mortality	0	Log(Y) = 2.0679 + (-0.0007)X	0.7936	480
Avg2.5 (3-day)	I and J Mortality	0	Log(Y) = 1.8130 + (0.0030)X	0.9301	480
Avg2.5 (3-day)	Other Mortality	1	Log(Y) = 2.0347 + (0.0050)X	0.8461	480
Avg2.5 (3-day)	I and J Mortality	1	Log(Y) = 1.7609 + (0.0004)X	0.8938	480
Avg2.5 (3-day)	Other Mortality	2	Log(Y) = 1.9949 + (0.0017)X	0.5479	480
Avg2.5 (3-day)	I and J Mortality	2	Log(Y) = 1.8479 + (-0.0052)X	0.1026	480
Avg2.5 (3-day)	Other Mortality	3	Log(Y) = 2.0658 + (-0.0005)X	0.8468	480
Avg2.5 (3-day)	I and J Mortality	3	Log(Y) = 1.8778 + (-0.0045)X	0.1543	480

5-3.3: Ozone and $PM_{2.5}$ effects on mortality

The literature review discussed several examples of how some airborne pollutants can have synergistic and antagonistic health effects. The study attempted to determine by multiple regression methods whether or not ozone and $PM_{2.5}$ had any such interactions (table 14). The regressions were performed using both daily average ozone and average peak ozone values with the BR-AQCR average $PM_{2.5}$ concentrations for all sensors.

All regression coefficients for ozone, regardless of the type of average, indicated a negative relationship, just as the analysis performed on ozone alone. The largest increase in the relationship again occurred at a lag of two days. All but one of the cardiovascular and respiratory mortality relationships were significant, and that p-value for zero lag for daily average ozone concentration was still close at $p = 0.0571$. Regression coefficients for both daily average and peak average concentrations of ozone were similar, illustrating that there is not much difference in changes in the frequency of mortality where a daily spike or a 24-hour average is concerned. There was a substantial difference between daily average and peak average p-values for I and J cases; peak values were significant to less than or equal to $p = 0.0001$ and daily average values were several orders of magnitude higher.

Fine particulate matter had only one statistically significant effect when average $PM_{2.5}$ is analyzed with daily ozone peak values. This indicated a positive correlation between mortality and particulates with a negative relationship to ozone. However, this is for other-cause mortality, which is not expected nor explainable since particulates should not have any effect on these non-cardiorespiratory conditions. The data for multiple regressions do not indicate that there is a statistically significant synergistic or antagonistic relationship between these two NAAQS pollutants.

Table 14: Multiple Regression Results of Ozone and $PM_{2.5}$ Concentrations

X_1	X_2	Y	Lag (days)	Equation $Log(Y) = \beta_0 + \beta_1X_1 + \beta_2X_2$	p-value β_1	p-value β_1	n
O_3 ADV	Avg2.5	Other Mortality	0	Log(Y) = 2.0519 + (-0.0012)X1 + (0.0015)X2	0.2731	0.4201	1457
O_3 ADV	Avg2.5	I and J Mortality	0	Log(Y) = 1.8502 + (-0.0024)X1 + (0.0001)X2	0.0571	0.9510	1457
O_3 ADV	Avg2.5	Other Mortality	1	Log(Y) = 2.0460 + (-0.0017)X1 + (0.0029)X2	0.1348	0.1191	1457
O_3 ADV	Avg2.5	I and J Mortality	1	Log(Y) = 1.8447 + (-0.0025)X1 + (0.0007)X2	0.0483	0.7344	1457
O_3 ADV	Avg2.5	Other Mortality	2	Log(Y) = 2.0568 + (-0.0012)X1 + (0.0010)X2	0.2929	0.5874	1457
O_3 ADV	Avg2.5	I and J Mortality	2	Log(Y) = 1.8844 + (-0.0034)X1 + (-0.0009)X2	0.0092	0.6641	1457
O_3 ADV	Avg2.5	Other Mortality	3	Log(Y) = 2.0843 + (-0.0011)X1 + (-0.0014)X2	0.3298	0.4637	1457
O_3 ADV	Avg2.5	I and J Mortality	3	Log(Y) = 1.8838 + (-0.0028)X1 + (-0.0020)X2	0.0299	0.3576	1457
O_3 APV	Avg2.5	Other Mortality	0	Log(Y) = 2.0591 + (-0.0011)X1 + (0.0027)X2	0.1063	0.2079	1457
O_3 APV	Avg2.5	I and J Mortality	0	Log(Y) = 1.8874 + (-0.0030)X1 + (0.0040)X2	<0.0001	0.0934	1457
O_3 APV	Avg2.5	Other Mortality	1	Log(Y) = 2.0655 + (-0.0018)X1 + (0.0052)X2	0.0055	0.0137	1457
O_3 APV	Avg2.5	I and J Mortality	1	Log(Y) = 1.8768 + (-0.0028)X1 + (0.0043)X2	0.0001	0.0728	1457
O_3 APV	Avg2.5	Other Mortality	2	Log(Y) = 2.0686 + (-0.0022)X1 + (0.0025)X2	0.0662	0.2422	1457
O_3 APV	Avg2.5	I and J Mortality	2	Log(Y) = 1.9178 + (-0.0034)X1 + (0.0031)X2	<0.0001	0.1927	1457
O_3 APV	Avg2.5	Other Mortality	3	Log(Y) = 2.1005 + (-0.0013)X1 + (0.0003)X2	0.0436	0.8716	1457
O_3 APV	Avg2.5	I and J Mortality	3	Log(Y) = 1.9151 + (-0.0030)X1 + (0.0017)X2	<0.0001	0.4892	1457

6. CONCLUSIONS

6-1: Results of Regressions

In this thesis, the hypothesis was that as ambient ozone or particulate matter concentrations increase or decrease, there will be a corresponding increase or decrease in deaths due to cardiovascular and respiratory illness. The regression analysis of the air quality data to mortality cases does not support this. For ozone, the analysis shows quite the opposite. All regression coefficients for the effects of ozone on mortality show a negative relationship – that is, as ozone concentrations increase, mortality decreases in a log-linear fashion. This conclusion mirrors the results of the study completed by Lu et al. (2003) in the Los Angeles Air Basin, which found a negative relationship when strictly examining ozone and asthma incidences. For $PM_{2.5}$, there was only one statistically significant value for I and J mortality, and that too indicated a negative relationship just as ozone.

Another goal of this study was to determine if there was a certain day after exposure which resulted in the larger effect. The data supports that the largest effect occurred at Day 2, or two days after exposure to a specific level. As explained in the literature review section, many studies found a lag of between one to three days from exposure to effect. This study does confirm that aspect, even though the relationship was opposite of what the hypothesis proposed.

Multiple regression analysis confirmed that ozone concentrations were having a negative effect, but showed that fine particulates were not having any statistically significant effect regardless of lag factor. However, the p-values were several orders of magnitude smaller for ozone in conjunction with $PM_{2.5}$ as opposed to ozone alone.

The regression of fine particulate matter by separation of sensor types did not produce a substantial number of significant values. Since only one of the monitors provided a statistically significant value, none of the results support the idea that one sensor type is a better measurement device for examining mortality risk. However, since all of these monitor measurements agree in terms of the significance of the regression, this does validate the idea of using an average of all monitoring sites in future analyses.

6-2: Potential Confounders and Sources of Error

Since this study utilized air quality data from an outside source, analysis is based on information that did not have author-derived quality control. Selection of monitoring sites and data collection were at the mercy of the LA DEQ and the sensor array that was already in place. Spatial differences in air quality measurements could have had a two-fold effect on the analysis. First, some of the sensors are in locations with a relatively low population density. The average value of ozone or $PM_{2.5}$ used in the regression may be skewed to values of air quality that are derived from the outskirts of the BR-AQCR.

Second, because daily air quality readings are dependent variables, that is, concentrations at one location may eventually affect the measurements at another site, the layout of the sensor array does not give a good indication or prediction of how plumes and "clouds" are traversing the area. The prevailing winds at one location will eventually push some proportion of ozone or particulates over to another sensor. That measurement will be a ratio of pollution derived locally and pollution derived from distant sources. This may cause fluctuations in the areas between monitoring stations, and those values are not currently measured with equipment. They can only be estimated with an all-encompassing average value or with spatial interpolation using GIS or complex statistical models.

As discussed, $PM_{2.5}$ sensors come in multiple varieties with four different sampling time schedules. Temporal discrepancies almost certainly exist due to 1-hour, daily, 3-day, and 6-day sampling schedules. Statistically, a correlation coefficient can be determined between the different sensors, but none of the monitoring site values completely match up. As such, there will always be a source of error within any statistical method that utilizes an average ambient concentration to estimate an exposure concentration.

The problem with attempting to relate ambient concentration to actual exposure is one of great contention between environmental enforcement bodies and industry. In studies such as this, where ambient concentrations are analyzed along with mortality cases, a potential source of error comes from the assumption that a measurement of air quality directly translates into exposure or body burden. Ambient conditions can only be used as an estimate, and actual exposures may be above or below this value. For instance, the elderly may stay indoors on hot, high ozone days or an individual may smoke, resulting in particulate exposures that are higher than ambient. Leikauf, et al., (1995) noted that ten cigarettes raise particle levels 20-50 ug/m3 above ambient in a closed environment. Therefore, the amount of time spent indoors in a smoky environment versus time outdoors in true ambient conditions would be very difficult to measure and incorporate into an epidemiological study.

After the attacks on the World Trade Centers on September 11, 2001, fire and rescue personnel at ground zero were exposed to elevated levels of particulates from smoke. Fumes and metallic compounds from burning buildings and equipment exposed these crews to higher than normal levels of co-pollutants adhered to and embedded in the soot. Stokstad (2004) describes the biomonitoring process of the firefighters in New York City and how it is a valuable tool to measure exposure more accurately than by measuring just the airborne

concentration. Use of nicotine metabolites and possibly PAH concentrations bound to serum albumin could be used to establish a baseline in some cohort and other types of epidemiological studies. While biomonitoring may be relatively easy to detect compounds such as cotinine, the metabolite of nicotine in cigarette smoke, it is much more difficult to measure a reactive oxygen species such as ozone. The potential for biomonitoring to produce tests for detection of either the detrimental effects of criteria pollutants or the criteria pollutants themselves could provide additional data for analysis with air quality measurements.

The composition of particulates could definitely be a confounding factor since particulate composition varies from region to region and source to source. PM originating in the eastern U.S. is typically more acidic with more sulfates, while the western U.S. will typically see more nitrates in the PM composition (Schlesinger & Cassee 2003). Further studies could examine samples of particulates collected at each monitoring site, separate the various organic and inorganic constituents, and determine the concentrations of individual metals and compounds.

7. SUMMARY

For ozone, it appears that there is an effect in the Baton Rouge AQCR, and that effect was the opposite of what is expected. $PM_{2.5}$ did not show an effect positively or negatively. Ozone levels can be high in this region, which at least made for a good statistical comparison between ranges of ozone concentrations. It is important to note that particulate levels are not at levels as close to the NAAQS limit as ozone is, which may show that the current EPA standards are sufficient to protect human health, and that the BR-AQCR is more "healthy" than expected for the effects of this pollutant.

Mortality as an endpoint for fluctuations in ambient exposure to pollutants has its limitations. The concept that ozone and $PM_{2.5}$ is a risk factor and could exacerbate a pre-existing cardiac or pulmonary condition to the point of death is substantiated in the literature review. There are many effects that these two criteria pollutants possess that could exploit a physiological or anatomical flaw in the human body. However, this study does not take into account many considerations such as lifestyle, socio-economic factors, or personal history. The intent of the study was to look strictly at an air pollution variable with known adverse health effects and to determine if it exhibited a relationship to mortality.

Fine particulate matter may not have been attributable to mortality in this study for several reasons. One substantial consideration for the results obtained from the regressions may be that the BR-AQCR simply has too low a level of $PM_{2.5}$. It is plausible that there is an exposure threshold where, clearance capacity and neutralization of harmful co-pollutants are not saturated and can handle the ambient particulates. Baton Rouge may be below that threshold and would not show a significant effect on cardiovascular and respiratory mortality.

Subsequent studies could compare the relative air quality between AQCRs of equivalent size to determine if that is the case.

It is important to look at inhalation toxicity as a combined and possibly synergistic effect. Very few studies have examined the toxicological effects of multiple airborne pollutants, and even these studies only look at 2-3 different pollutants. Breathing is a vital function and as such, exposes individuals to a plethora of compounds in different concentrations. One may say that carbon monoxide problems in the Baton Rouge AQCR are far below the NAAQS limits. However, the data is not there to say if these low levels are safe, and there is even less evidence to show that these low levels of CO, when combined with a certain level of particulate matter on an ozone exceedance day, will not have a significant effect on individuals with asthma or high blood pressure. That is why it is critical to look at air pollution problems from a total pollution constituent aspect, rather than breaking down the air we breathe into individual components and examining the adverse health effects piecemeal.

It would be difficult to take all potential confounding factors into consideration. Socioeconomic status, seasonal variability, lifestyle risk factors, relative health, and genetics could all potentially play a part in whether ambient conditions can trigger or increase the risk of certain predisposed medical conditions. Relative levels of individual "health" can be an objective or subjective measurement, and can be a potential confounder. Healthy subjects exposed to PM2.5 at a level 2.5-4X higher than mandated by EPA's NAAQS 24-hour standards for 2-hours while at rest resulted in no problems with pulmonary function, blood oxygen levels, or EKGs both during exposure and 22 hours after (Gong, et al., 2000). The results of this study could result in two opinions, either that acute ultrafine particulate

exposure has no effect on health or that healthy individuals without compromising physiological factors are more resistant to the health effects. Ultrafine particulate matter at ambient concentrations should be considered as a risk factor for cardiovascular and pulmonary illness, not as a primary causal agent.

Other avenues for data analysis could be found by examining smaller subsets of the population, such as examining school absences for hospital admission correlations or geocoding mortality cases and using GIS to determine which air monitor will best represent the exposure. These absences may or may not result in a documented hospital admission, depending on the severity, which further complicates the issue. Also, retrieving data on prescriptions for nebulized asthma inhalers may show a seasonal trend that could be taken into account in the statistical models.

Effects of ozone and fine particulates have been documented, and there is no doubt that they exert adverse effects at some level, but this thesis does not support that conclusion. The limitations of this study do not support the idea that these criteria pollutants do not have a deleterious effect on health by increasing the death rate. More variables should be incorporated into further analyses to determine if other mitigating factors are having an effect. In addition, the examination of hospital admissions for cardiovascular and respiratory illnesses as an endpoint could prove to represent better the health effects of air quality from acute exposures than would mortality.

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