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Estimating The Impact of a Select Criteria Pollutant (PM2.5) on Childhood Asthma in Florida

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Estimating The Impact of a Select Criteria Pollutant ($PM_{2.5}$) on Childhood Asthma in Florida

by

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DEDICATION

I dedicate my dissertation work to my loving family and many friends. A special feeling gratitude to my loving parents, Pradeep and Prabha Khanna, who were my first teachers. Their encouragement is the reason I could reach the finish line. My husband, Karan, who has been there for good and bad. My loving children, Saloni and Nalin, their never-ending stimulation, "*Mom you can do it*", kept me going through this journey. Both of you have been my cheerleaders. My sister Swati has never left my side, my life support and is too dear to express in words. My two very special friends Dr. Shalini Kesar and Dr. Niketa Patel who have been there with me in this journey listening and encouraging every time I wanted to give up.

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ABSTRACT

Asthma has been reported in children as a leading chronic illness in the US and around the world. It is also the third leading cause of hospitalization among children under the age of 15, and is also one of the most common causes of school absenteeism. Children are at higher risk of asthma attacks and they pose a higher burden on health care system. Nearly 20.6% of middle and high school children in Florida have been told they have asthma, this prevalence has grown over 3% from 2006 to 2012. Changes in air pollutant levels are often related to health outcomes, e.g. prevalence of chronic asthma. Exposure to ambient air pollutants have been reported to exacerbate asthma attacks especially in children. Often agencies and governing bodies utilize national level health impact assessments (HIAs) to estimate local levels of health impacts. The US EPA (Environmental Protection Agency) developed the Benefit Mapping and Analysis Program (BenMAP) to estimate impacts on health due to changes in air pollution. Recent studies have shown that assessment of regional exposure is important to understand health impacts of pollutants at the local level. To use BenMAP effectively for HIA in Florida, one may have to update the prevalence rates and concentration response (CR) functions in BenMAP with Florida data.

The main purpose of the research was to develop a method which can estimate impact of change in criteria pollutants on childhood asthma outcomes in Florida. The rates present in BenMAP are based on national estimates, which are higher than the rates for Florida. If these

rates are used for the HIA method then the change in asthma emergency department visits estimated by BenMAP may be an overestimate with higher uncertainties. There are no baseline rates for asthma exacerbation ED visits in BenMAP, an asthma exacerbation is a more severe and poorly managed case of asthma. Asthma ED visit prevalence rates will tend to overestimate the asthma exacerbation rates by 64%, if used. Detailed review of US-EPA's BenMAP software and peer reviewed literature was performed to identify the gaps in BenMAP for asthma assessments. The CR functions were developed using local pollutant and outcomes data. CR functions were added to BenMAP to bridge the gaps. The baseline prevalence and exacerbation rates at county level by age group, gender and race ethnicity were developed.

This study highlights that an increase of 10 $\mu g/m^3$ of $PM_{2.5}$ contributes about 2% to asthma ED visit rate, in children 5-12 and is lower, for 13-18 olds (0.6%). The baseline prevalence and exacerbation rates at county level for asthma in children differed by race/ethnicity. This study publishes the ED rates by county and by gender, race and ethnicity from 2010 to 2014, which are recent rates and have not been published to such granularity by the State or by any other researcher. Current pollutant data in BenMAP is only available through 2008, and EPA has recommended it should be updated for analysis purposes. This study has updated the monitor data in BenMAP for Florida counties for 2010-2014.

There are three major contributions of this study. Firstly, the study contributes to publishing childhood emergency department prevalence rates for asthma and exacerbation in the State of Florida by age group, race/ethnicity and gender. Secondly, development of concentration response functions specific to Florida using the time series analysis to show the impact of $PM_{2.5}$

on asthma exacerbation emergency department visits, incorporating both temporal and spatial variability of $PM_{2.5}$ during the study period. Finally, the study demonstrates the utility of using local (county-level) baseline asthma prevalence rates and local pollutant data for State HIA in Florida. The local $PM_{2.5}$ data in BenMAP can be used for other health outcome assessments, researchers will only have to update the prevalence rates for the health outcome used in their study. Estimation using local data will be less prone to uncertainties using National level data, the use of local data has been emphasized by several researchers.

The study recommends future work in refining spatial grid resolution in BenMAP to zip code level to facilitate studies at neighborhood level. Another recommendation is to further design research to study SES in context to dietary changes and better understand social injustices in areas with diverse population. A population-based study in conjunction with Florida Asthma Coalition (FAC) asthma cases from doctors' offices is recommended which will be able to control for misclassifications, and include weather and allergens in analysis while studying individual pattern of exposure and diet.

CHAPTER ONE: INTRODUCTION AND AIM

1.1 Introduction

Asthma has been reported as a leading chronic illness among children in the US and around the world. Asthma affects nearly 334 million people globally and 14% of children experience asthma symptoms. It often causes reduced quality of life among patients and their families and imposes physical, psychological and social effects (GAN, 2014). The estimates of its economic burden due to loss in productivity, disability, lost school days, absence from work and premature death, measured as disability adjusted life years (DALYs) are significantly high, placing asthma as the 14th most important disorder in the world. The burden of asthma is highest among children aged 10-14 and the elderly aged 75-79 (GAN, 2014). Low and middle-income countries have higher prevalence rates of acute asthma, and the rate of prevalence seems to be increasing at an alarming rate compared to high-income countries (GAN, 2014).

Asthma is a major chronic disease in the United States, nearly 6.8 million children are affected by asthma (Bloom *et. al.*, 2013). The prevalence of current asthma has increased from 2001 to 2010. However, between 2005 to 2008 it seemed to have reached a plateau, but continued to grow since 2008 (Moorman *et. al.*, 2012). The number of person with an asthma attack in the previous 12 months has increased 2.6% per year from year 2003 to 2010, 11.0 million to 13.9 million (Moorman *et. al*, 2012). With increasing asthma prevalence, the ambulatory care for asthma use has continued to grow since 2000 (Akinbami, 2007). Asthma is also the third leading

cause of hospitalization among children under the age of 15; and has been known to be one of the most common causes of school absenteeism (Akinbami, 2007; Akinbami *et. al.*, 2012).

The prevalence of asthma for children in Florida was estimated to be 8.3% as compared to 9.0% nationally in 2007 (CDC, 2008). It was estimated 32,007 children in Florida had asthma using the Behavior Risk Factor Surveillance System (BRFSS) 2007. The gender difference in Florida is higher than nationally, 10.5% of boys in Florida have current asthma compared to 10.1% nationwide, while only 5.9% of girls in Florida have current asthma compared to 7.9% nationally (CDC, 2008).

Changes in air pollutant levels are often related to health outcomes, e.g. prevalence of chronic asthma. High levels of air pollution, indoors and outdoors, can contribute to and possibly cause various health problems ranging from respiratory illness, heart disease, and cancer to death (Akinbami, 2006; Akinbami *et. al.*, 2012). In particular, exposure to ambient air pollutants may exacerbate asthma attacks especially in children (Akinbami, *et. al.*, 2012).

Air pollutants and health outcome data are available publicly or from agencies for research. Even though these data are available, the biggest challenge is linking air pollutant data with health outcomes. Several studies have combined health outcomes with US EPA exposure data but are aggregated to specific counties or zip codes, due to the unavailability of data for smaller levels of geographic detail (e.g. residence). A study by CDC elaborates the linkage methods for US National Health Interview Survey (NHIS) with US Environmental Protection Agency's (EPA) air monitoring data. (Charleston *et.al.,*2008; Talbot *et.al.,*2009)

The US EPA developed the Benefit Mapping and Analysis Program (BenMAP) to estimate impacts on health due to changes in air pollution. BenMAP is a powerful tool, and its flexibility allows the users to input their own site-specific data on pollutant type, ambient concentrations, demographics, and health outcomes, to create visual maps of air pollution and incidence rate changes of health outcomes. It helps in economic evaluations and cost benefit analysis of air pollution control. EPA regularly performs national-level health impact assessments (HIA) for regulatory purposes. Often agencies and governing bodies utilize national level HIAs to estimate local levels of health impacts. Recent studies have shown that assessment of regional exposure is important to understand health impacts of pollutants at the local level (Fann *et. al.*, 2008; Hubbel *et. al.*, 2009). National level HIAs are unlikely to describe true impacts of pollutants at a sub-national scale or local-scale as they often assume distribution of exposure and outcome in all the sub regions similar to the national level and hence mask the true spatial variations (Fann *et. al.*, 2008). Recent research has indicated that there is a great need for developing local or regional level health impact assessments (Hubbel *et. al.*, 2009; Fuentes 2009).

1.2 Public Health Significance

Asthma is an important public health problem. Change in diagnosis or possible alterations in the population gene pool cannot explain the increase in asthma incidence or prevalence. Protection against environmental factors becomes a practical approach to reducing and controlling asthma. Thus, better understanding and quantifying the impact of environmental pollutants on pediatric asthma is critical. Children are at higher risk of asthma attacks and they pose a higher burden on health care system. Nearly 20.6% of middle and high school children in Florida have

been told they have asthma, this prevalence has grown over 3% from 2006 to 2012. The increase in asthma prevalence imposes higher emergency department and hospital visits. In Florida, the number of asthma emergency visits, for children under age of 18, have increased from 34,855 in 2005 to 48,674 in 2014. This poses a great financial burden on state and payer as the cost of care has had a sharp increase in the past decade. Current published studies focusing on asthma morbidity have shown health care disparities but few have national or state estimates of current asthma incidence or prevalence rates, the published rates are usually 3 or more years older.

There are three major contributions of this study. First, the study will contribute to publishing childhood asthma emergency department prevalence and exacerbation rates in State of Florida. These rates will be age group, gender and race/ethnicity specific.

Second, the study will contribute to the development of CR functions are local to Florida; and will show the impact of $PM_{2.5}$ on asthma exacerbation emergency department visits while incorporating both temporal and spatial variability of $PM_{2.5}$.

Finally, the study demonstrates the utility of using local (county-level) asthma exacerbation rates, local pollutant data and localized CR functions for State Health Impact Assessments. Current published studies have not validated this for Florida.

1.3 Specific Aims

There were five specific aims of this research study:

Aim One: To assess the efficacy/utility of Concentration Response (CR) functions and Asthma prevalence rates present in EPA's BenMAP for estimating childhood asthma exacerbation rates due to changes in $PM_{2.5}$.

The goal for this aim was to identify the appropriateness of the CR function incorporated in EPA's assessment software BenMAP, while paying close attention to age and gender specificity. The CR functions are used to estimate asthma outcomes like exacerbation, due to changes in concentration of ambient air pollutants. BenMAP has built-in CR functions for estimating asthma exacerbation, these functions may not be age or gender specific. Since asthma rates vary with age and gender, using CR functions which are not age and gender specific will incorrectly estimate the asthma health impact due to air pollutants. This makes it necessary to develop age and gender appropriate CR functions. A detailed review of published peer reviewed literature, EPA's published documentation and technical manuals were used to identify the gaps in the CR functions currently present in BenMAP.

Aim Two: To determine the baseline emergency department asthma prevalence and exacerbation rates for childhood asthma at local county level in Florida.

The goal of this aim was to develop baseline asthma outcome rates using local health data obtained from Florida Agency for Health Care Administration (FL-AHCA). The local region defined in this study was Florida county level. The asthma cases or asthma exacerbation cases identified using ICD-9 were used to estimate the quarterly rate of prevalence and exacerbation of

asthma for children seen in emergency departments in Florida counties by age and gender. Nationally representative health data are collected by The National Center for Health Statistics, Centers of Disease Control and Prevention (NCHS/CDC), while national/state representative data are collected by the Agency for Health Care Administration (AHCA).

Even though the data are available at the national level, there are confidentiality restrictions and the geographic details on health outcome data are not available most of the time. As a result, baseline asthma prevalence/ incidence or exacerbation rates at local or state levels are not available. Developing accurate baseline rates are critical for developing appropriate methods for health impact analyses (HIA) at the local county or State level.

Aim Three: To evaluate the temporal and spatial patterns of $PM_{2.5}$ in Florida and incorporate for Florida into BenMAP for HIA estimations.

The goal of this specific aim was to evaluate temporal and spatial patterns of $PM_{2.5}$ across Florida and to incorporate air pollution monitoring data for Florida counties into BenMAP. This objective was accomplished by using Florida EPA's monitoring data from 2010-2014, 24-hour average $PM_{2.5}$, were averaged across all monitoring sites in a county to determine daily county level concentration of pollutant. The data was analyzed to see the temporal and spatial patterns of $PM_{2.5}$ at county level across Florida. The data was also loaded into BenMAP to match the county level spatial grids in BenMAP.

Aim Four: To develop county-level CR functions to be used in HIAs performed by local or State agencies.

The goal of this aim was to develop CR functions which can be used in HIA methods, for evaluating impact of ambient air pollutants $PM_{2.5}$ on childhood asthma exacerbation in Florida. Often, local regulatory or State agencies perform local health impact assessments of air pollutants HIA methods with national level data. The CR functions used in these methods are derived from studies conducted nationally or at another location in the US and are not really local to a state. Local HIA require appropriate regional or robust CR function for regional HIA to be reliable (Hubbell *et.al.*, 2009).

This aim was achieved by linking local daily asthma exacerbation outcomes to local daily $PM_{2.5}$ concentrations at county level and using Poisson model in Generalized Linear Estimating Equations (GEE), to estimate CR function. The CR function estimated from this study was pooled with CR functions from peer reviewed studies to increase the reliability of the HIA method (Fuentes *et. al.*, 2009).

Aim Five: To estimate age and gender specific asthma exacerbation rates in children due to change in $PM_{2.5}$ concentrations at the county level.

My final objective was to estimate asthma exacerbation rates in Florida at county level due to changes in local air pollutants, while controlling for age and gender using the HIA method in BenMAP. This objective was accomplished by incorporating results from specific aims two, three and four in BenMAP software and estimating the asthma exacerbation rates at county level due to the change in $PM_{2.5}$.

1.4 Hypotheses

Hypothesis One:

It is hypothesized that rates for prevalence and exacerbation of asthma at the National level cannot be generalized to county levels.

Local county level rates will be compared to State of Florida and National rates available for age and gender. These comparisons will highlight differences in county-specific and national-level rates by age and gender.

Hypothesis Two:

It is hypothesized that using aggregated National levels of pollutant concentration for local level pollutant concentrations will mask true estimates of asthma exacerbation rates in children due to changes in level of criteria pollutants at the county level.

Developing the required spatial grids at the county level and inserting monitor data of $PM_{2.5}$ in BenMAP will reveal the importance of air quality management at the local area to control asthma, and the costs associated with changes in air pollution.

Hypothesis Three:

It is hypothesized that if CR functions, corresponding to asthma exacerbation and levels of air pollutants, are derived from national or global research studies then the reliability of local HIAs will be low.

Local HIA methods use CR functions developed from national or international research studies. However, estimates of CR functions from just one study may be accompanied with low reliability of HIA. To improve reliability of air quality HIA, it is necessary to select CR functions which have been derived from peer-reviewed studies (Hubbell *et. al*, 2009). CR functions using local and peer reviewed studies can be pooled to further increase the reliability (Fuentes, 2009).

Hypothesis Four:

It is hypothesized that using local CR functions and asthma prevalence rates will result in reliable estimates of the impact of $PM_{2.5}$ on ED visits for childhood asthma/asthma exacerbation in Florida.

HIA methods using local response functions, and asthma prevalence rates

1. will have lower uncertainties:

   a) lower variability; b) smaller confidence intervals c) greater precision, and
2. will be able to better estimate the asthma exacerbation rate due to change in $PM_{2.5}$ in Florida.

1.5 Definition of terms

Please see Appendix 1 for definitions of terms used throughout this dissertation.

CHAPTER TWO: REVIEW OF THE LITERATURE

2.1 Background

Asthma is a complex pulmonary disease known to have many triggers, including allergens, infections, exercise, abrupt changes in the weather, and exposure to air pollutants (O_3, NO_2, Particulate Matter (PM)) and smoke (ALA,2012; Akinbami 2007). It is characterized by recurrent episodes of chest tightness, coughing and wheezing. Many environmental and genetic factors play an important role in the induction and exacerbation of asthma. Exacerbation or recurrent asthma episodes range in severity from inconvenient to life threatening situations, and involve shortness of breath, coughing, wheezing, chest pain or tightness, or a possible combination of these symptoms (ALA, 2007).

Current asthma prevalence during 2006-2010 was estimated to be 8.0% among the US population. Asthma prevalence varied by demographics, it was higher among females (9.0%) than males (6.9%), and children had higher prevalence than adults (9.4% versus 7.6%) (Moorman *et. al.* 2013) . Children are also more susceptible to asthma incidence and exacerbation than adults. In 2005, 9 million children younger than 18 (12.7% of US children) had a life time diagnosis of asthma; among them 6.5 million had *current* asthma, and 3.8 million had asthma attacks in the past 12-months (Akinbami 2006; Akinbami 2007). Nearly 60% of children had experienced greater than one asthma attack in past year (Akinbami 2007). Asthma led to about 3.5 million visits to physicians, 0.5 million visits to hospital outpatient departments, 593,000 emergency department

(ED) visits, and 155,000 hospitalizations in children under the age of 18 (Akinbami 2007; Akinbami *et.al.* 2009).

2.2 Definition Of Asthma

Asthma is defined by WHO as "*a heterogeneous disease, usually characterized by chronic airway inflammation. It is defined by the history of respiratory symptoms such as wheeze, shortness of breath, chest tightness and cough that vary over time and in intensity, together with variable expiratory airflow limitation.*" (WHO, 1999). The natural history of this disease includes exacerbations or acute episodes in addition to chronic persistent inflammation and reduced lung function. (Reddel *et. al.,*2009). The ICD-9 definition and codes of asthma are given in Appendix 2. These codes help in clinical diagnosis of asthma and help in differentiating between intrinsic asthma, extrinsic asthma, chronic obstructive asthma, other forms of asthma and asthma unspecified.

Asthma exacerbations are defined as "*episodes characterized by a progressive increase in symptoms of shortness of breath, cough, wheezing or chest tightness and progressive decrease in lung function, i.e. they represent a change from the patient's usual status that is sufficient to require a change in treatment.*" (GINA, 2016). The ICD9 definition and codes of asthma exacerbation are given in Appendix 2. The codes help in diagnosis and differentiating intrinsic asthma with acute exacerbation, extrinsic asthma with acute exacerbation, Chronic obstructive asthma with acute exacerbation, other forms of asthma exacerbations and asthma unspecified with acute exacerbations.

2.3 Asthma Etiology

Development of asthma has been associated with family history, which is not sufficient explanation for development of asthma (Burke *et. al.*, 2003). Phenotypes characterizing asthma differ in etiology and pathophysiology. Geographical variation in prevalence, incidence and magnitude of increases in asthma over the past two decades have been suggestive of that environmental changes play an important role in asthma prevalence and incidence. Gene by environment interactions have been suggestive of probable cause of varying prevalence across the world. During a person's lifetime the environmental triggers may affect differently at different time points and risk factors may change over a time. (Subbarao *et. al.*, 2009). Short term and long term studies on risk factors of asthma have been suggestive of contrary results. Long term studies have shown asthma to be associated with risk factors like air pollutants with higher magnitude on the other hand short term studies have been suggestive of lower likelihood of asthma or even protective sometimes. Allergic sensitization, environmental tobacco smoke, exposure to animals, breastfeeding, decreased lung function in infancy, family size and structure, socio-economic status, antibiotics and infections, and gender have been suggestive factors influencing childhood asthma (Subbarao *et. al.*, 2009).

2.4 Trends in Childhood Asthma

<u>Asthma Prevalence, Incidence and Exacerbation</u>

Asthma is the leading serious chronic illness of children in the United States. Since the early 1980's, the number of children (<14 years) diagnosed with asthma has increased rapidly, making it the most common chronic disease among children (Akinbami, 2006). In 2006, the American Lung Association (ALA) estimated 6.8 million children under the age of 18 currently

had asthma, 4.1 million of which had an asthma attack, and many others have "hidden" or undiagnosed asthma (ALA, 2007). In 2011, ALA estimated 8.7 million children ages 5-17 had been diagnosed with asthma in their lifetime, 7.1 million children had current asthma. (ALA, 2012).

The prevalence of current asthma has increased from 2001 to 2010, and was highest in 2010 (Akinbami *et. al.*, 2012). Between 2005 to 2008 it seemed to have reached a plateau but continued to grow since 2008 (Moorman *et. al.*, 2012). Despite the plateau in asthma prevalence between 2005 to 2008, ambulatory care for asthma during this period had continued to grow (ALA, 2007).

Based on data from the National Health Interview Survey (NHIS), incidence of asthma increased from 1980 to 1996 from 3.65% to 6.2%, but had reached a plateau between 1996 and 2004 to 5.4% (Akinbami *et. al.*, 2006).

The number of person with an asthma attack among those with current asthma in the previous 12 months has increased 2.6% per year from year 2003 to 2010, 11.0 million to 13.9 million (Moorman *et. al*, 2012). With increasing asthma prevalence, the ambulatory care for asthma use has continued to grow since 2000 (Akinbami, 2007).

Asthma Morbidity

Asthma is also the third leading cause of hospitalization among children under the age of 15. Hospitalization rates for asthma have a similar trend to prevalence rates.(ALA, 2007; ALA, 2012). Asthma is one of the most common causes of school absenteeism. (Akinbami, 2007; Meng *et,al*;2012). In 2003, children aged 5 to 17 years who reported at least one asthma attack in the

previous year, missed 12.8 million school days (Akinbami, 2007). Asthma inpatient admission rates have strong seasonal variation, for all the subgroups stratified by race, age and ethnicity (Johnston *et al, 2006a;2006b).* Asthma hospital admissions decrease in summer months (May-Sep) in comparison to the rest of year. (Johnston *et al, 2006a;2006b* ; Winquist *et al, 2016)* Another study indicated that lower admission rates were seen during rainy season (Valença *et.al,* 2006) Patients with any pulmonary infection showed a different seasonal pattern, having significant variation from January through March (USEPA, 2008).

Asthma Mortality

Asthma mortality rates illustrated a sharp rise from 1980 to 1990's but have declined recently; however, they are still higher than the rate in 1980.((Akinbami, 2006) Asthma mortality, deaths per 1,000 persons has declined from 2001to 2009 (Akinbami *et. al.*, 2012). The average annual death rate for 2007-2009 was 0.15 per 1,000 persons with asthma. For children under the age of 18 it was lower than 0.05, however, annual death rate was higher for females and blacks for all ages (Akinbami *et. al.*, 2012).

2.5 Race, Gender and Age Differences in Asthma

Racial disparity in Asthma

Racial disparity for asthma has been well documented in many studies (CDC, 2008; FAP, 2014). Higher asthma prevalence is reported among non-Hispanic black children as compared with non-Hispanic white children (Blixen 1999; CDC, 2008; ALA, 2007; and FAP, 2014). African Americans are more susceptible to asthma attacks with change of weather than non-African Americans (Blaisdell,*et.al*, 2002). A recent study showed that national estimates of current asthma

prevalence among the children in the selected minority subgroups ranged from 4.4% in Asian Indian children to 13.0% in American Indian/Alaska Native children (Brim *et.al.,* 2008; CDC, 2008). Adverse outcomes, like emergency department visits, hospitalizations and mortality, were also higher for non-Hispanic black children (Blixen *et.al.,* 1999) Recently, the disparity has increased significantly between white and black children for asthma mortality (Akinbami, 2007; Akinbami *et. al.,* 2016)

Gender Disparities and Asthma

Asthma occurrence, prevalence and mortality vary with gender. Among younger children, boys have a higher risk of asthma development than girls and this difference diminishes with increasing age (Bloom *et.al.,* 2007). The influence of gender on asthma has a different trend among teenagers and adults. The prevalence of asthma is predominant in boys in early ages of life and appears higher in girls after puberty (Horwood *et.al.,* 1985). Under the age of 15 years, more boys than girls have been admitted to hospitals for asthma (Bloom *et.al.,* 2007). The differences in asthma risk by gender disappear during teenage years, as girls surpass boys with more asthma attacks (Akinbami 2007; ALA 2007; Akinbami *et.al.,* 2016). How hormones or sex related hormones contribute to asthma incidence or asthma prevalence is not well understood yet. Overall boys have a higher asthma prevalence, hospitalizations and death rate as compared to girls (Blaisdell 2002; Akinbami 2007; ALA 2007; Akinbami 2016)

Age and Asthma

In 2006, the rate in those under 18 years of age (92.8 per 1,000) was much greater than those over 18 (72.4 per 1,000), and the highest rate of 106.3 per 1,000 populations was seen in those 5-

17 years of age (Akimbani *et al,* 2016) In 2005, it was reported that approximately 32.6 percent of hospitalizations due to asthma were in children under the age of 15 years (Akimbani *et al,* 2016; Akimbani ,2006). The current prevalence rates and mortality seemed to increase with age for those under the age of 18 (Akimbani, 2007; Akimbani *et al,* 2016). While, hospitalizations and emergency visits decreased with age (ALA, 2007; Akimbani *et al,* 2016).

2.6 Asthma and Genetics

Many genes have been linked to asthma and asthma exacerbation but the role of genetics in asthma remains unclear (Ober 2005, Holgate 1999). Genetics plays a major role in regulation of inflammatory mediators, like cytokines and growth factors, and IgE production. The clinical diagnosis, etiology and pathophysiology have been of onset of clinical asthma, the complexity of IgE and genetic environment involvement in clinical asthma is noted by linkages to certain phenotypic characteristics, but not necessarily the pathophysiologic disease process or clinical picture itself. The relevance of polymorphisms in the beta-adrenergic and corticosteroid receptors in determining responsiveness to therapies is of increasing interest, but the widespread application of these genetic factors remains to be fully established.

2.7 Air Pollutants and Asthma

Outdoor and indoor air pollutants have been associated with asthma. Outdoor pollutants affect indoor air concentration and are also used to define the Air Quality Index (AQI). Regulated outdoor air pollutants in the US can be categorized as criteria pollutants and hazardous air pollutants.

Criteria pollutants

Criteria pollutants are a group of very common air pollutants regulated by EPA on the basis of health and/or environmental effects. They are particle pollution often referred to as particulate matter (PM), ground-level ozone (O_3), carbon monoxide (CO), sulfur oxides (SO_x), nitrogen oxides (NO_x) and lead (Pb). These pollutants are considered to be the most widespread nationally and the most immediately dangerous to human health (Rodriguez, 2007). According to the Clean Air Act ("Act"), the USEPA is responsible for setting the National Ambient Air Quality Standards (NAAQS), and must use health-based criteria and cannot consider estimates of compliance cost. Two sections of the Clean Air Act ("the Act") govern the establishment and revision of NAAQS. Section 108 (42 U.S.C. 7408) directs the Administrator to identify pollutants that "may reasonably be anticipated to endanger public health or welfare" and to issue air quality criteria for them. These air quality criteria are intended to "accurately reflect the latest scientific knowledge useful in indicating the kind and extent of all identifiable effects on public health or welfare which may be expected from the presence of [a] pollutant in the ambient air." PM is one of six pollutants for which the EPA has developed air quality criteria. Section 109 (42 U.S.C. 7409) directs the Administrator to propose and promulgate "primary" and "secondary" NAAQS for pollutants identified under section 108. Section 109(b)(1) defines a primary standard as "the attainment and maintenance of which in the judgment of the Administrator, based on [the] criteria and allowing an adequate margin of safety, [are] requisite to protect the public health." A secondary standard, as defined in section 109(b)(2), must "specify a level of air quality the attainment and maintenance of which in the judgment of the Administrator, based on [the] criteria, [are] requisite to protect the public welfare from any known or anticipated adverse effects associated with the presence of [the] pollutant in the ambient air." Welfare effects as defined in section 302(h) [42 U.S.C. 7602(h)] include but are not

limited to "effects on soils, water, crops, vegetation, manmade materials, animals, wildlife, weather, visibility and climate, damage to and deterioration of property, and hazards to transportation, as well as effects on economic values and on personal comfort and well-being." 1-2 Section 109(d) of the Act directs the Administrator to review existing criteria and standards at 5-year intervals. When warranted by such review, the Administrator is to retain or revise the NAAQS. After promulgation or revision of the NAAQS, the standards are implemented by the states.

EPA calculates the AQI for five major air pollutants regulated by the Clean Air Act: ground-level O_3, PM, CO, sulfur dioxide (SO_2), and nitrogen dioxide (NO_2). Four of these pollutants (CO, Pb, NO_2, and SO_2) are emitted directly from a variety of sources. Ozone is not directly emitted, but is formed when oxides of nitrogen (NO_x) and volatile organic compounds (VOCs) react in the presence of sunlight. PM can be directly emitted, or it can be formed when emissions of NO_x, SO_x, ammonia (NH_4), organic compounds, and other gases react in the atmosphere. Ground-level ozone and airborne particles are the two pollutants that pose the greatest threat to human health in the US (USEPA, 2008). Criteria air pollutants are responsible for many adverse effects on human health, causing thousands of cases of premature mortality and tens of thousands of emergency room visits annually (USEPA,2008). Since 1980, emissions of the six criteria pollutants have declined significantly, with the greatest drop in lead (EPA,2008). NO_x emissions have dropped by one third, whereas, SO_x and CO emissions have been cut roughly by one half. Since 1980, ambient concentrations of CO, Pb, SO_2, and NO_2 have decreased steadily, and currently are below the National Ambient Air Quality Standards (NAAQS) in most parts of U.S (USEPA, 2012a). Concentrations of ozone (O_3) and Particulate Matter less than 2.5µm ($PM_{2.5}$) have exceeded the NAAAQS at several places within the United States (USEPA, 2008). In 2005, concentrations of O_3 and/or $PM_{2.5}$ did not meet the standards in 68 Metropolitan Statistical Areas (MSA) where

approximately 128 million people live. The majority of the days in which AQI exceeded 100 were mostly due to O_3 or $PM_{2.5}$ (USEPA, 2008).

Particulate matter (PM)

PM is a complex mixture of solid or liquid particles that are airborne and dispersed. These particles include dust, dirt, soot, smoke, acids (e.g. nitrates and sulfates), organic chemicals, metals, etc. PM originates from a variety of anthropogenic sources, including diesel trucks, power plants, wood stoves, industrial processes, windblown dust, construction sites, and burning waste (Jacobson, 2002). Particles are also formed in the atmosphere by condensation or the transformation of emitted gases such as sulfur dioxide (SO2), nitrogen oxides (NOx), and volatile organic compounds (VOCs) (Jacobson, 2002). PM also have a seasonal pattern, increased operations of power plants in the third quarter of the year leads to a higher concentration of PM in the eastern half of the U.S. (USEPA, 2008). Use of wooden stoves and fireplaces during winter months leads to increased PM concentration in the western US during the fourth quarter (USEPA, 2008). The size of particles is directly linked to their potential for causing health problems (Godish, 2002). PM_{10} known as "inhalable coarse particles" (particulate matter with an aerodynamic diameter less than 10 μm and greater than 2.5 μm), provides a better correlation of particle concentration with human health. A significant portion of $PM_{2.5}$ known as "fine particles" (particulate matter with an aerodynamic diameter less than 2.5 μm) are secondary in nature, and are especially detrimental to human health as they can perforate deep into the lungs (USEPA, 2008). Exposure to PM is unfavorable for vulnerable populations, especially those with chronic heart and lung disease, young children, elderly and pregnant women. Long-term exposure may cause reduced lung function, development of chronic bronchitis and even premature death (USEPA, 2008). Short-term exposures to particles

(hours or days) can aggravate lung disease causing asthma attacks and acute bronchitis and may also increase susceptibility to respiratory infections (USEPA, 2008).

In 2006, the EPA's final PM rule established NAAQS 35 $\mu g/m^3$ as 24-hour standard and retained the annual standard of 15 $\mu g/m^3$. The EPA revised the secondary standards for fine particles by making them identical in all respects to the primary standards. In 2009, several agencies and state governing bodies signed a petition for review of 2006 standards, as stated below. This resulted in investigation and clarification by US EPA in a form of written document known as Regulatory Impact Assessment (RIA), outlines detail analysis and explanation of standards for public welfare (USEPA, 2012b).

> "*Following promulgation of the final rule in 2006, several parties filed petitions for its review. On February 24, 2009, the U.S. Court of Appeals for the District of Columbia Circuit remanded the primary annual PM2.5 NAAQS to the EPA citing that the EPA failed to adequately explain why the standard provided the requisite protection from both short- and long-term exposures to fine particles, including protection for at-risk populations. The court remanded the secondary standards to the EPA citing that the Agency failed to adequately explain why setting the secondary PM standards identical to the primary standards provided the required protection for public welfare, including protection from visibility impairment."*

Source : https://www3.epa.gov/ttn/ecas/docs/ria/naaqs-pm_ria_proposed_2012-06 (USEPA 2012b)

Mixtures of Pollutants

A number of pollutants may be present in the atmosphere. These tend to form complex mixtures, such as smog from fine particulate matter and smoke. The nature and activity of pollutants in the environment pose a challenge to investigators to study the health effects of air pollutants. Complex mixtures of pollutants can be classified into three groups that 1) originate from single sources (e.g., environmental tobacco smoke from active smoking); 2) result from physical

mixing of primary emissions from multiple sources (e.g., a range of volatile organic compounds [VOCs] emitted from building furnishings); or 3) result from physical mixing of emissions from multiple primary sources with agents created by chemical transformations of those emissions (e.g., precursors of smog [like nitrogen oxides, hydrocarbons, and sulfur oxides] reacting to form ozone and acid particles mixed with other oxidants and metals). Complex mixtures have been associated with increased respiratory symptoms and hospital admissions for asthma. Several investigators have postulated that measurements of criteria pollutants may serve as exposure surrogates for a complex mixture of criteria pollutants mixed with regional hazardous air pollutants (HAPs) (Fujita *et.al.* 2014; Fujita *et.al.* 2016; Scheffe *et.al.* 2016; Santamaría *et.al.* 2017). Some studies have investigated the association of asthma surrogates, i.e. hospital admission, to levels of multiple air pollutants (Thurston 2007; Burnett 1990), but little on the cumulative effects of pollutant mixtures was found in the published literature.

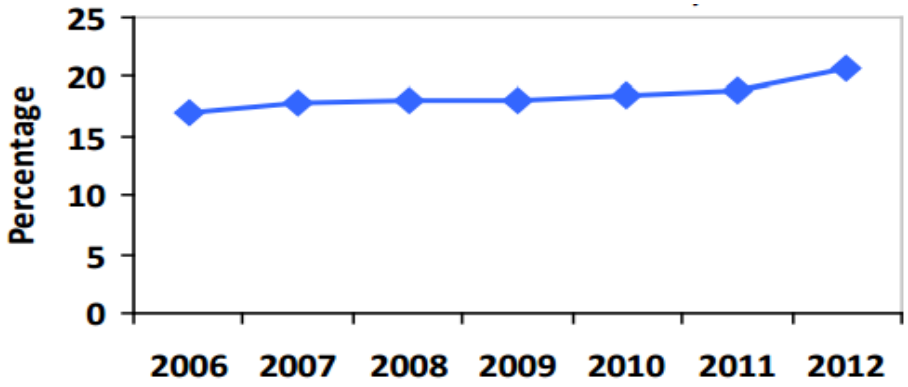
2.8 Seasonality, Air Pollution and Asthma

Air pollution, during photo-chemically active periods, has been associated with respiratory morbidity (Tresende *et. al.,* 2005). Seasonal variations in asthma are widely recognized (Khot, *et.al.*1984; McCormick *et.al.,* 1995; Marks *et.al,*1997; Sears 1997; Fleming *et. al.,* 2000; Buckley et al.2012) Seasonal trends in hospitalization for asthma peaked in January through March, followed by a sharp decline beginning in April through June among adults is well documented in peer reviewed literature (Fleming *et. al.*, 2000; Chen *et.al.*, 2006). The seasonal trend for asthma was significantly correlated with peaks in air pollution and climate. (Grech *et. al.,* 2002; Chen *et.al.*, 2006; Johnston *et.al.*, 2006) A study reported that childhood asthma admissions increased from

May–Oct by 2.7% with an increase of 12 µg/m^3 in PM_{10} with a 3 day lag (Blixen *et.al.,* 1999). Asthmatic children exhibit much higher variations in asthma exacerbations and larger sensitivity to air pollution over time (Johnston *et.al.*, 2006).

2.9 Asthma in Florida

The prevalence of asthma has been shown to be increasing among all ages nationally as well as in Florida. The prevalence of lifetime asthma has increased from 17% in 2006 to 20.5% in 2012 among Florida public middle and high school going children (FDOH, 2014, FAP 2014; Appendix 3:Table1). Figure 1 shows by 2012, lifetime asthma in school going children increased to nearly 21% . Nearly 17% of middle and high school students reported an asthma attack in the past twelve months. (FAP 2014)



Source: FAP 2014, *Asthma in school aged children, Florida 2013.*

Figure 1 Lifetime Asthma Prevalence, FYTS 2006-2012

The lifetime asthma prevalence for middle and high school students increased for all the race/ ethnicity categories from 2006 to 2012, however the highest increase was in non-Hispanic Black and Hispanic categories, which increased from 17.7 to 21.8 in year 2012. Approximately

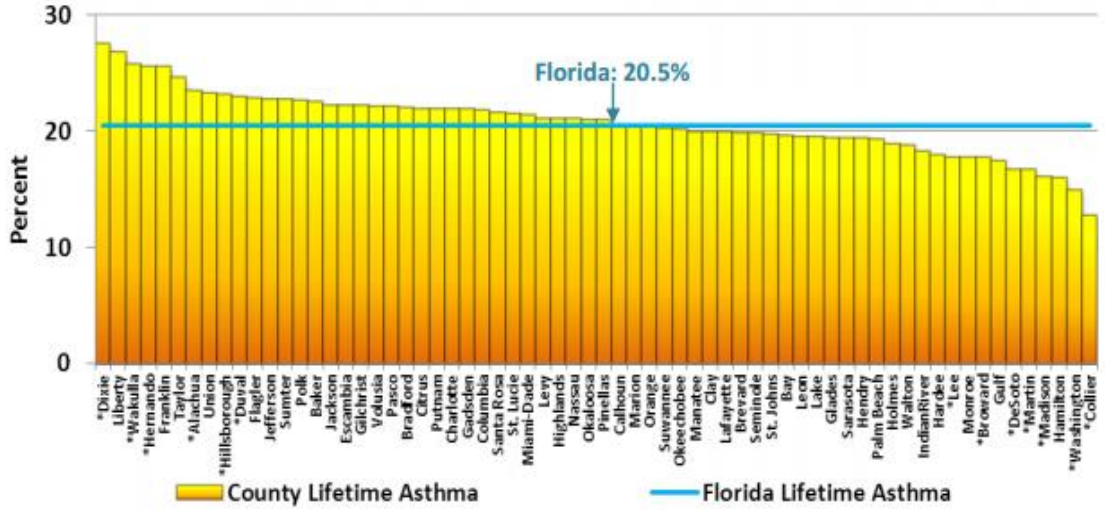
21.7% public high school students (165,000) reported lifetime asthma in Florida for 2011, this was lower than national prevalence which is 25% (FAP 2014).

Females in Florida had a significantly lower prevalence of lifetime asthma than their national counterparts. Nationally, non-Hispanic Black public high school students had the highest prevalence of lifetime asthma. However, Florida Hispanics have the highest lifetime asthma prevalence. This prevalence is higher than US counterparts. (FAP 2014)

The gender difference in Florida is higher than the gender difference nationally. In Florida, a higher percentage of boys (10.5%), have current asthma prevalence than nationwide (10.1%), while only 5.9% of girls in Florida have current asthma as compared to 7.9% nationally (CDC 2008). The analysis of Youth Risk Behavior Surveillance System (YRBSS) by FLDOH shows that ethnic difference in Florida are different than that of US. In US, Hispanic have lower current asthma prevalence than White and Black. While in Florida White non-Hispanics have the least current asthma prevalence rate, and Hispanics have the highest prevalence rate in Florida. (FLDOH, 2014).

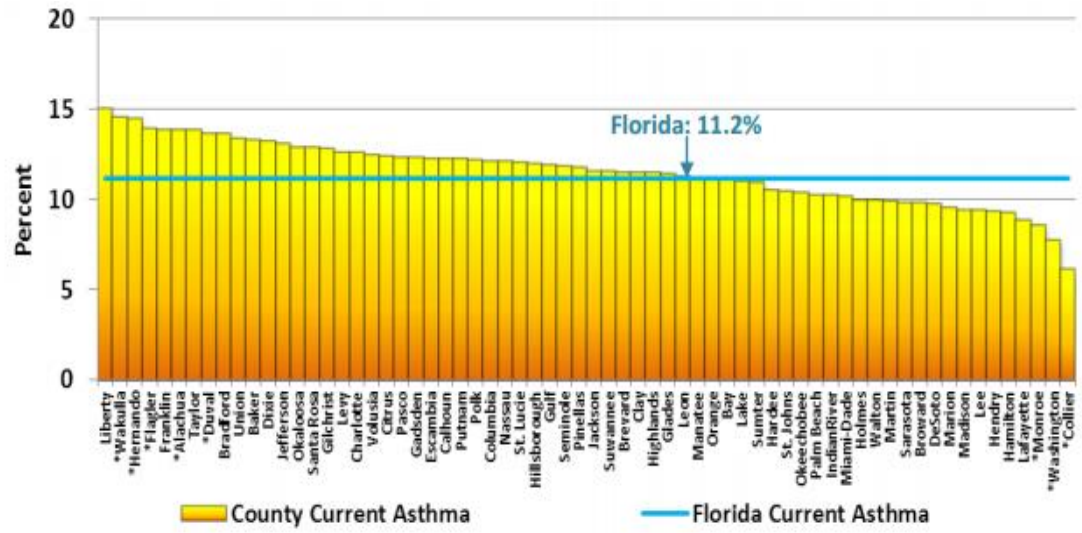
ED visits for asthma in age group 5-17 was 29,150 for 2012, the highest among all age groups in Florida. For the age group 5-17 females (8.01 per 1, 000) had lower ED visit rates than males (11.7 per 1,000). The asthma ED visit rates increased from 6.82 per 1,000 in 2008 to 9.89 per 1,000 in 2011 (FAP 2014). The asthma ED rate for non- Hispanic Blacks was three time that of non Hispanics Whites and twice that of Hispanics.

The Florida Youth Tobacco Survey (FYTS) 2012 shows that middle and high school lifetime asthma prevalence and current asthma prevalence by county differs between Florida counties. Alachua, Duval, Flagler, Hernando, and Wakulla had prevalence greater than Florida for lifetime and current asthma (Figure 2). The current adolescent asthma prevalence for Florida was 11.2% in 2012 (FAC 2013, Appendix 3).



Source: FAP 2014: *Asthma burden 2013.*

Figure 2 Lifetime Adolescent Asthma Prevalence by County, FYTS 2012



Source: FAP 2014: *Asthma burden 2013*

Figure 3 Current Adolescent Asthma Prevalence by County, FYTS 2012.

2.10 Health Impact Assessment and BenMAP

There is enough evidence that factors outside the health care delivery system affect human health. Our economic, social and physical environments have been shown to affect public health. (WHO, 2007). Several environmental factors including criteria air pollutants have been linked to risk of disease and an impact on human health. As this evidence, has accumulated over time, quantification of impact of air pollution on the public health has also increased (WHO, 2000). Quantitative estimates of these health impacts have become critical input components in policy decisions, establishment of environmental regulations and research planning around the world. (WHO 2002; Martuzzi *et. al.* 2003; Harris *et. al.* 2012; Hebert *et. al.* , 2012; Fakhri *et. al.* 2015). Often global, national and local agencies and governing bodies assess the risk or impact of changes in air pollution on health using quantitative methods like Health Risk Assessments (HRA) or Health Impact Assessments (HIA). As per the Clean Air Act ("ACT") for setting NAAQS the USEPA must use health-based criteria. For this purpose, USEPA uses Regulatory Impact Analysis (RIA) to estimate the human health and welfare costs and benefits of attaining NAAQS for criteria pollutants nationwide.

HRA is an analytical process which is used to quantify and estimate the risk of adverse human health effects associated with exposure to contaminants or pollutants. HRA and HIA are interchangeably used by agencies and researchers in the process to quantity the effects of air pollution on public health. HRA looks at the risk associated with air pollutants at the population level, whereas HIA is the impact on public health outcomes due to changes in an air pollutant level, due to policy changes or higher air quality control regulations. Results from HRA can be used within an HIA to predict human health effects of specific exposures.

HIA can be understood as a tool that is used by decision makers to improve public health through community design. It estimates the health impact on an entire population, or selected subpopulation as a result of exposure and by decisions and activities that created them. The WHO defines HIA as "*A combination of procedures, methods and tools by which a policy, programme or project may be judged as to its potential effects on the health of a population, and the distribution of those effects within the population"* (WHO, 1999). HIA methods identify the actions or policies to manage the health effects on the population (Quigley *et. al.*, 2006). Thus, HIA methods use available results from epidemiological studies with data from environmental and health outcomes to assess impact on health, and to help policy makers plan and implement measures to protect public health.

There is no single assessment method of HIA, as assessments may vary across projects depending on particular questions under consideration. The HIA could be integrating other assessments like Environmental Impact Assessment (EIA) or Sustainability assessment (SA) or the scope of assessments vary from international to local projects. In general, HIAs have five stages namely screening, scoping, risk assessment, reporting to decision makers, and monitoring and evaluation of the consequences of implementation.

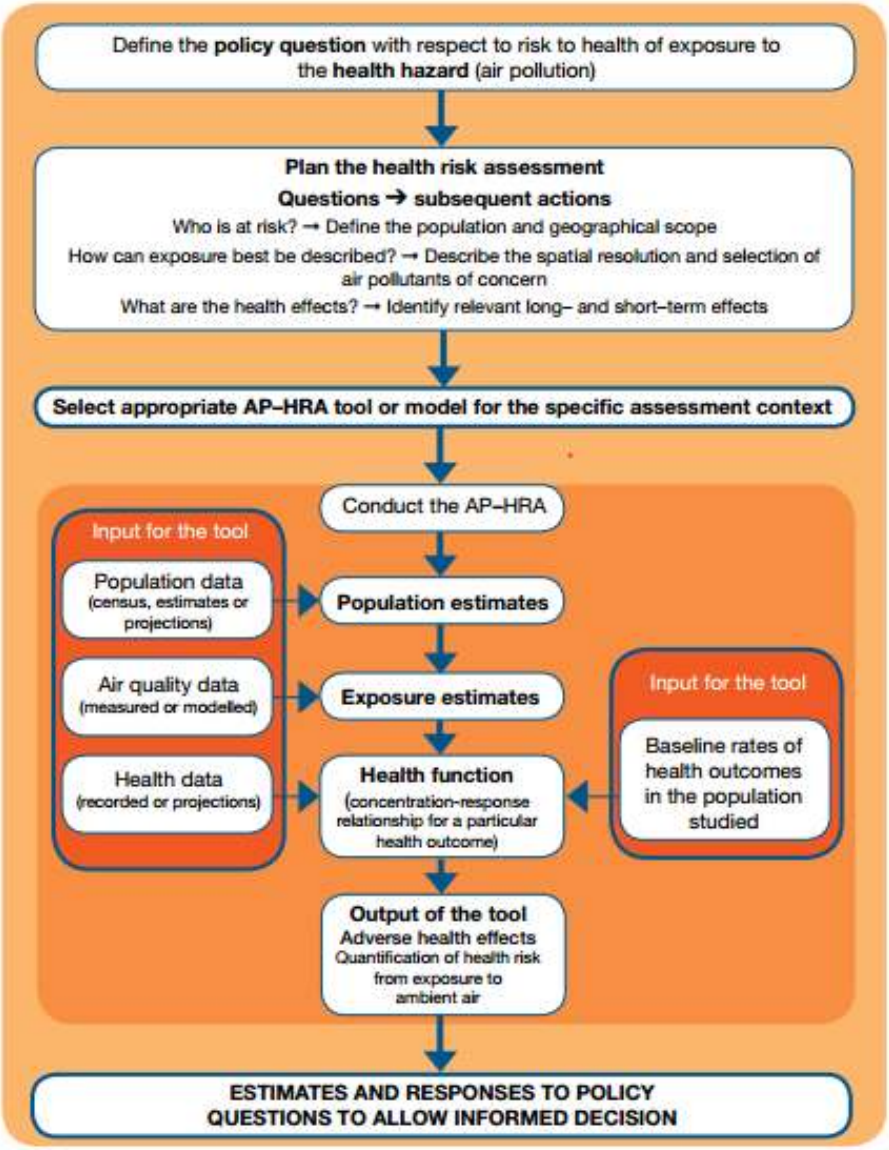
HRA Methodology

The main steps involved in an air pollution HRA (AP-HRA) as recommended by WHO guidelines on Assessment and Using Epidemiological Evidence for Environmental Health Risk Assessment (WHO 2000a, 2000b) are:

- Specify exposure which is hazardous to health
- Define health outcomes
- Specify the exposure response relationship
- Derive population baseline frequency measures for health outcomes
- Calculate the number of cases or lives that can be saved or attributed to the exposure.

The overview of AP-HRA is given in Figure 4, which starts with defining the policy question, then planning the HRA, next selecting the appropriate tool for the HRA, then conducting a HRA using the selected tool and finally responding to policy question and making informed decisions using the results of the HIA.

To conduct an HRA appropriate data, resources and the selection of appropriate tools/methods are required. Several computer based tools are available for AP-HRA, most of these tools are similar in approaches and rely on CR functions derived from epidemiological studies (Anenberg *et. al.*, 2016). Simple tools like Epi-Info or ESRI's Arc-GIS can be used to visualize the results from HIA. A WHO expert meeting on methods and tools for assessing the health risks of air pollution at local, national and international level has described twelve tools across the world to assess the health risks of air pollution (WHO, 2014). USEPA's Benefits Mapping and Analysis Program (BenMAP) is one of the twelve tools in the WHO expert meeting. BenMAP is used in Regulatory Impact Analysis to quantify health impacts associated with changes in exposure, and these estimates may be expressed as Attributable Risk (AR) estimates (USEPA, 2014b; USEPA, 2015).



Source: WHO (2016) "Health-risk-assessment-air-pollution-General-principles"

Figure 4 An Overview of AP-HRA

Role of Risk Impact Assessment (RIA) in the Process of Setting the NAAQS

Legislative Roles

In setting primary standards for NAAQS, USEPA has responsibility under the law of the United States of America to establish criteria that protect public health, regardless of

implementation cost. The Act requires USEPA, "*for each criteria pollutant , to set a standard that protects public health with an adequate margin of safety."* (USEPA 2012b). The Act requires that these standards are based on health consideration only. The USEPA believes that " consideration of cost and benefits is essential to making efficient, cost-effective decisions" for implementing NAAQS. The RIA's are intended to inform the public about potential cost and benefits of implementing new standards (USEPA 2012b).

The RIAs also are used to create public documentation of cost benefit analysis, performed to satisfy statutory and executive orders before implementing NAAQS. RIA illustrate cost benefit analysis different scenarios of ambient pollutant concentration, it gives selected regulatory scenario and alternative combinations of primary standards that could be implemented by State Implementation Plans (SIP) or Federal regulations.

Illustrative Nature of RIA

The RIA for setting or revising NAAQS is basically an illustrative analysis that provides insights of hypothetical control scenarios if implemented federally or by individual states. RIA analysis mainly attempts to estimate the costs and human welfare benefits of new proposed implementation strategies, which a governing body may use as cost-effective control. (USEPA 2012b).

Overview and Design of USEPA's RIA

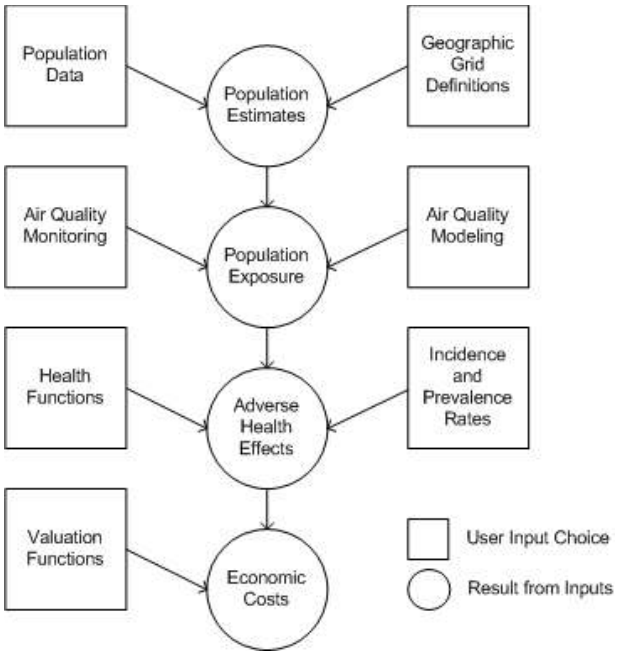
A typical RIA evaluates the costs and benefits of hypothetical national strategies to attain several alternative standards for criteria pollutant in question. It follows the following steps:

1. Models the levels of criteria pollutant (i.e. $PM_{2.5}$) in the future analysis year.

2. For a criteria pollutant it reviews existing air quality standards and compares it to alternative proposed standards.
3. Benefits analysis approach – under this approach USEPA estimates human health outcomes under alternative pollutant concentrations as compared to current standards. USEPA considers an array of health impacts attributable to changes in air pollutant for this type of analysis.
4. Cost analysis approach– under this approach USEPA estimates total cost under partial and full attainment of several alternative standards.
5. Comparison of benefit and cost- estimates the total benefit and cost and summary of net benefit of several alternative standards.
6. Economic impacts- a qualitative discussion of economic impact, i.e. employment, of air quality regulations.

Benefit analysis approach and HIA

Under a benefit analysis approach, USEPA quantifies the health related benefits of pollutant related air quality improvements, to attain an alternative proposed NAAQS level in the future year, e.g. 2020. For an RIA, health impact analyses are normally limited to health effects, that are directly linked to ambient levels of air pollution, specifically linked to the pollutant in question (USEPA, 2012b). HIA quantifies the changes in the incidence of adverse health impacts resulting from changes in human exposure to concentration of air pollutant. HIAs are a well-established approach for estimating the retrospective or prospective change in adverse health impacts expected to result from population-level changes in exposure to pollutants (Levy *et. al.*., 2009).



Source: USEPA 2015

Figure 5 Health Impact Assessment method used in US-EPA's BenMAP

Estimating health impacts typically utilizes data on an exposed population, changes in ambient air pollution levels, baseline incidence rate of the health endpoint, and, a health effect estimate. A typical HIA estimation has the following four key components (Figures 4 and 5):

1. Population estimates- through population data from census, local estimates or projections
2. Exposure estimates - estimate a change in air quality or pollutant concentration and combine air quality data with population information to determine changes in exposure.
3. Health function- Combine changes in population exposure to ambient air pollution with concentration response functions or impact functions.
4. Quantification of health risk from exposure to ambient pollutant through the use of summary statistics.

A typical health impact assessment equation using a log-linear form looks like:

$$\Delta y = (1 - e^{\beta * \Delta x}) \, y_0 * \text{Pop} \qquad \text{--- equation 1}$$

Where,

Δy: is the estimated change in health outcome (i.e. morbidity or mortality)

β: is the concentration response coefficient drawn from epidemiological studies, it is the slope of RR/OR in epidemiological studies.

y_0: is the baseline incidence/ prevalence rate of adverse health outcome being quantified

Pop: the potentially affected population by change in air quality or pollutant

Δx : is the estimated change in ambient pollutant concentrations.

A HIA equation depends on the functional form of CR function from which it is derived. The research literature and epidemiologic studies help to identify the relationship between the criteria pollutant and health outcome measure in question.

Concentration Response Functions

CR functions, also known as effect estimates or health impact functions, relate one unit change in air pollution (Δx) to one unit change in incidence or prevalence of health outcome (Δy). CR functions are obtained from published epidemiologic studies, where they are published as odds ratios (OR) or relative risk ratios (RR). The OR/ RR measures the odds or risk of the change in population health outcome with a specific change in pollutant concentration. Epidemiologic studies have several forms of CR functions, the most commonly published forms in the literature for $PM_{2.5}$ are linear regression, Poisson regression, logistic regression and conditional logistic regression.

Linear Regression Model

A linear regression model defines the relationship between the adverse health outcome rate (prevalence rate) and pollutant concentration, i.e. $PM_{2.5}$, to be linear:

$$y=\alpha+\beta*PM$$

The change in adverse health outcome rate (Δy) from baseline y_0 to y_i can be defined as

$$\Delta y = y_0 - y_i = \beta*(PM_0-PM_i)$$

$$\Delta y = \beta*\Delta PM$$

The linear relationship is reported in literature for asthma and PM2.5 exposure in several time series models (Ostro *et. al.*, 1991; Loftus *et. al.*, 2015 ; Freitas *et. al.*, 2016).

Logistic Regression Model

A logistic regression model is used to estimate the probability of occurrence of adverse health event in some epidemiological studies. The logistic regression gives the probability of occurrence as:

$$y= \text{prob (event} \mid x*\beta) = \frac{e^{x*\beta}}{1+e^{x*\beta}} = \frac{1}{1+e^{-x*\beta}}$$

Where, β is the vector of coefficients of explanatory variables x. In epidemiology the results are often represented as odds ratio (OR) or relative risks (RR). The odds of adverse event can be given by

$$\text{Odds} = \frac{y}{1-y} = \frac{1}{e^{-x*\beta}} = e^{x*\beta}$$

The odds ratio for control or alternate scenario versus baseline ($odds_0$) can be written as

$$\text{Odds Ratio (OR)} = \frac{odds_i}{odds_0} = \frac{e^{x*\beta}}{e^{x_0*\beta}}$$

The change in probability of adverse health event from baseline to i^{th} scenario , Δy , can be derived from odds ratio and can be expressed by

$$\text{Odds Ratio (OR)} = e^{\beta * \Delta x} = e^{\beta * \Delta PM}$$

Log-Linear

The prevalence rate (y) is exponentially dependent on the outcome in a log linear relationship, and is defined as :

$$y = a * e^{\beta x};$$

where x is the matrix of outcomes and the equation can be written as

$$y = \mathbf{A} * e^{\beta * PM};$$

or $\ln(y) = \alpha + \beta * PM$

where A is the prevalence rate of y when the concentration of PM is zero, β is coefficient of PM and $\alpha = \ln(A)$;

A is matrix of other covariates affecting prevalence, that are evaluated at their means

$$A = A_0 * e^{\beta_1 x_1 + \beta_2 x_2 + \ldots + \beta_n x_n}$$

A_0 Is the prevalence of y when all the covariates in the model are zero.

The change in PM can rewritten as

$$\Delta y = y_o - y_c = \mathbf{A} * (e^{\beta * PM_0} - e^{\beta * PM_c})$$

$$= \mathbf{A} * e^{\beta * PM_0} (1 - e^{\beta * (PM_c - PM_0)})$$

$$= y_0 (1 - e^{\beta * \Delta PM})$$

Where y_0 is the baseline prevalence rate.

The relative risk (RR) is simply a ratio of two risks and can be written as

$$RR = \frac{y_o}{y_c} = e^{\Delta PM * \beta}$$

Taking natural log of both the sides, the coefficient of PM or coefficient of CR functions can be derived as

$$\beta = \frac{\ln(RR)}{\Delta PM};$$

Poisson Regression Model

Poisson regression is commonly used when the dependent variable is the count of a rare event (e.g. number of cases of asthma or number of deaths), or studying the hazard using Cox proportional hazards model and is believed to be Poisson distributed. The CR function for Cox's model is believed to be log-linear in form, which is the natural logarithm of health response and is a linear function of pollutant concentration. The most commonly used form of CR function for $PM_{2.5}$ to estimate the association between health outcome is the log-linear form of the model.

The proportional hazard model is based on a hazard function, and is defined as the probability of adverse event at time t, conditional on having survived up to time t (Collet 1994). The proportional hazard model takes the form:

$$h(X, t) = h_0(t)\, e^{X*\beta}$$

Where $h_0(t)$ is the baseline hazard, it is the hazard or risk when the all the covariates (X) are set to zero, and β is the vector of coefficients. Relative risk (RR) can be calculated using ratio of hazard functions for baseline and i^{th} scenario.

$$RR = \frac{y_i}{y_0} = \frac{h(X_i, t)}{h(X_0, t)} = \frac{h_0(t)\, e^{Xi*\beta}}{h_0(t)\, e^{Xo*\beta}} = e^{(Xi-Xo)*\beta-}$$

$$= e^{\Delta PM*\beta}$$

For this functional form, it is assumed that the only difference between baseline and the i^{th} scenario is the level of pollutant, ΔPM and everything else (h_0) is constant. For the above equation β can be estimated by taking the natural log on both sides

$$\beta = \frac{\ln(RR)}{\Delta PM};$$

The above equation shows that the natural logarithm of Δy, change in health outcome, is linearly related to ΔPM or change in pollutant concentration

$$\ln(y_i - y_0) = \ln(\Delta y) = \beta * \Delta PM;$$

Choosing Functional Form Of CR Function

A functional form of the CR function is often chosen by a researcher and the parameters of this function are estimated from the literature using data on pollutant and health outcome. In the epidemiologic literature RR or OR are normally presented as measures of association rather coefficient β. Hence, it becomes necessary to estimate β before developing the functional relationship between pollutant change (ΔPM) and change in health outcome, Δy for CR functions. The underlying relationship assumed in epidemiological studies affects the estimation of CR functions.

Role of the Shape of CR Functions

The magnitude, shape, and degree of certainty in the association between population exposure to an ambient pollutant ($PM_{2.5}$) and the risk of health outcome is one of the most intensely studied issues in environmental health (Fann *et. al.* 2016; Pope *et. al.* 2006; Burnett *et. al.* 2014).

The shape of CR functions may influence the overall estimates of risk or benefits from HIA analysis. The shape of these functions is particularly important at lower concentrations of ambient pollutant as a larger number of population may be exposed at lower concentrations, and hence the overall impact may be much higher (NRC 2002). There is sufficient evidence in literature that the shape of CR functions is curvilinear for adverse outcomes of mortality and long term exposure to high concentrations of $PM_{2.5}$ (Krewski *et. al.* 2000, Burnett *et. al.* 2014; Nasari *et. al.* 2016). However, short term exposure studies, and acute health outcomes studies typically use time series models. For time series studies which analyze a large number of repeated measurements, linearity of CR functions has been reported with reasonable power (NRC 2002; Dominici *et. al.* 2003b; Dominici 2004). For PM_{10} the slope of CR functions increases with concentration of pollutant, and appears to be fairly linear for acute health outcomes (Samet *et. al.* 2000a,b,c; Pope *et. al* 2006; Pope *et. al.*2015). For $PM_{2.5,}$ there is no evidence of departure from linearity for CR functions over a range of short term exposures and acute outcomes (NRC2002). Current epidemiologic studies do not support non-linearity of CR functions at low concentrations of pollutants in the world's cleanest places like the US. Instead these CR functions are prone to higher uncertainties at low and very high concentrations of pollutants (Pope *et. al.*2015). The uncertainties in the CR function may impact conclusions when conducting HIA (Fuentes, 2009)

CR Functions in BenMAP

BenMAP has the capability to aggregate CR functions from multiple studies using *Pooling.* It is a type of meta-analysis that combines beta estimates from different studies while accounting for heterogeneity across studies. BenMAP offers several options of pooling i.e., addition, subtraction, user-assigned weights, random-effect model, fixed effect model. (USEPA, 2014b;

USEPA, 2015). The individual studies in the literature report individual risk estimates from that distribution. Random effects pooling accounts for heterogeneity in the individual risk estimates to generate a single mean risk estimate

BenMAP software has many preloaded CR functions for $PM_{2.5}$ and ozone and many common health outcomes, two of which are asthma and asthma exacerbation. The preloaded CR functions can be selected by the user or may add new functions. CR functions can be identified by short and long term epidemiologic studies, and can be added to the existing functions. However, current CR functions in BenMAP are only for O_3 and $PM_{2.5,}$ and are based on older literature and need updating as recommended in the provisional assessment for ozone and $PM_{2.5}$ (Researcher was selected for National Network for Environmental Management Studies (NNEMS) 2009 Fellowship at USEPA, North Carolina). CR functions for chronic asthma are not present in BenMAP, hence intense literature review needs to be performed and the functions need to be developed using the odds ratio and relative risk estimates in the literature. The current CR functions present in BenMAP were developed by the EPA for national assessments from published literature and may need modification for local or regional assessments.

Uncertainties due to CR functions

CR functions extracted from epidemiology studies or estimated through local models may be associated with uncertainties inherent in the epidemiologic model used. These may include uncertainties present in the choice of statistical model, arising from lag structures, meteorological variables included in the model and or adjustments of seasonality. The uncertainties

in epidemiology studies can be quantified by using confidence intervals, standard errors and distribution analysis. The errors quantified by experts using distribution analysis has been shown to vary largely due to factors which may have influenced uncertainties while developing the association (Roman *et.al.*, 2008).

Population Estimates

The population estimates used in HIA are baseline incidence or prevalence rates and the exposed population.

The *Exposed population* is the number of people, living with in a geographic area, who are affected by changes in air quality. The exposed population information is used to determine changes in population exposure, see in Figure 5. The government census office is a good source for this information. The census could be used to obtain populations at the local level, like county, census tract or zip-code level. Projections incorporating population growth are used for future estimations. The e*xposed population data available in BenMAP are the census 2010 data at county level*. When conducting an HIA at the National or State level, county data are aggregated to National or State level with the software. For resolution below county level, users have a choice of importing the data in BenMAP. However, this step requires extensive cleaning and aggregation to local resolution level like zip-code or block level prior to importing into BenMAP, and requires creating spatial grid cells to match the other input data, i.e. air quality and case data.

Baseline rates can be obtained from published literature or should be determined by the researcher using available health outcomes data. These rates are generally available at the national level, and in the US the rates are published by CDC. Local baseline rates for health outcomes may be available from local governing body, and should be used instead of national baseline rates when estimating health impacts at local levels. Using national level baseline incidence/prevalence rates could bias the results of local HIA. Recent research shows that using national incidence rates could underestimated the asthma hospital- admissions from changes in $PM_{2.5}$, and could not identify the spatial and demographic differences in the health outcome. The prevalence rates have been shown to differ by race/ethnicity, gender and age (ALA2007; Akinbami 2007; Akinbami 2016). These could be effect modifiers when estimating health impacts. One should utilize age, gender and race/ethnicity specific baseline incidence/ prevalence rate when available, for estimating population exposure to ambient air pollution.

In BenMAP baseline rates are present at the national level and match the year of census information in it. Local baseline rates for health outcomes may be available through local agencies and can be easily incorporated in BenMAP for local analysis.

Uncertainties due to population estimates

The exposed population estimates normally come from Census or surveys conducted in the areas for housing or other area development projects. The estimates obtained from the census or other surveys are innate with uncertainties, some of which could be due to design or sampling. The sampling errors and variations can be estimated standard errors. The standard errors represent uncertainty due to several sources, mainly due to sampling variation between counties or lowest unit of analysis. In addition to sampling, uncertainties may be present due to non-sampling

variations, i.e. errors made in data collection, administrative errors, high non-response, failure to represent all units in the sample. The non-sampling errors can be random or non-random, for non-sampling error the agency conducting the survey makes imputations at the lowest available unit. (Census Bureau 1995). These imputations add to the uncertainties if the analysis unit is not similar to that in the data, meaning if the data is in census tract then getting data to zipcode level without using weights will add uncertainties which cannot be controlled in analysis.

The health outcomes baseline rates are estimates based on either survey data (i.e NHIS or NSCH) or from hospital admission/discharge records. These estimates from surveys are also bound to have uncertainties due to sampling and non-sampling errors. The sampling errors can be estimated using standard errors while non-sampling errors are best estimated by sensitivity analysis.

Exposure Assessment Estimates

Estimating changes in population exposures to air pollutants is an essential component of health impact analyses. It provides the link between anticipated air pollution changes and resulting changes in health outcomes. The goal is to provide the necessary input for a health impact equation. Change in population exposure can be assessed or calculated by multiplying the observed changes in concentration of air pollutant to exposed population. Changes in air pollutant can be obtained through the use of simulated exposure models, or the use of actual monitoring data or a combination of modeling and monitoring data.

Simulated exposure modeling is a complex process that depends on many assumptions about the future, including pollution emissions reductions resulting from proposed regulation,

changes in emissions due to factors other than the proposed regulation, meteorological conditions, the physical and chemical processes in the atmosphere affecting pollution dispersion, transformations, and deposition, and the nature and degree of pollutant contact with future human populations. (USEPA, 2014b)

Estimating Change in Ambient Pollutant Concentration in BenMAP

The change in ambient pollutant concentration can be calculated as the difference between two air pollution levels at different points in time. The starting air pollution levels are also called the baseline level, and the second air pollution levels are normally calculated after some change, such as that caused by a regulation.

The exposure to air pollution at two different points can be estimated several ways, using air quality monitoring data or air quality modeling data or a combination of modeling and monitoring data. The four broad categories BenMAP uses to estimate exposure for HIA are *model direct*, *monitor direct, model and monitor relative, and model rollback.* (US EPA, 2015)

Model Direct uses modeling data that project air quality spatially and temporally when studying the effect of chronic health outcomes. BenMAP converts the input air modelled data into a spatial grid file that matches the grid structure of the population input data and baseline health outcome data. The model data can be easily entered in BenMAP come from Community Multi-Scale Air Quality (CMAQ), Comprehensive Air Quality Model with Extensions (CAMx), Regional Modeling System for Aerosols and Deposition (REMSAD), or air quality models generated by researcher. The spatial grid resolution available in BenMAP are 12x12km or 36x36km grids. The

appropriate methodology, scale, and resolution of air quality assessment is dependent on available pollutant data and research context.

Monitor Direct method uses ambient air quality data from EPA's local monitoring stations. Data can be interpolated from point based monitor values to grid cell based exposure estimates using Closest Monitor and Voronoi Neighbor Averaging (VNA). The VNA method was used by the Florida DEP to estimate the pollution removal by trees and its effect on human health (Nowak et. al., 2013). The closest monitor approach is a location specific approach, in which monitor data are used to estimate concentration level of exposure are recorded at the nearest (i.e. shortest distance) monitor location within the study area. This approach is used to estimate county level population exposure in epidemiological studies (Brauer et al., 2008; Jerrett et al., 2005; Ritz et al., 2002). For closest monitor approach BenMAP assigns the closet monitor to the population grid-cell, the annual and daily air pollution metrics are calculated using monitoring data and then used in the health effects estimations (USEPA, 2015).

Voronoi Neighbor Average (VNA) algorithm uses monitor data directly. It interpolates air quality of every population grid cell by first identifying the set of monitors in adjacent polygon cells that surround the center of population grid cells, the values from neighboring monitors are used to interpolate air pollutant value for the population grid cells. Using the VNA method, BenMAP draws "Voronoi" cells or polygons around the center of each population grid cell, and identifies the neighboring monitors. The "Voronoi" cells have a special property that the boundaries of the polygon are the same distance from the two closest points. BenMAP then chooses those monitors that share the boundaries to the grid cell, and uses these neighboring monitors to estimate

the air pollution level for the grid cell. To estimate the air pollution exposure in each grid cell, BenMAP calculates the inverse distance weighted average of the neighboring monitors. The inverse distance weighting interpolation is based on an assumption that the degree of influence of nearby monitors should be greater than the effect of distant points. The interpolant is weighted average of the sample point values and the weights *wi* are expressed as an inverse function of distance and is expressed as :

$$wi = \frac{1/d_i}{\sum_1^N 1/d_i}$$

Where N is the number of sample points, d is the distance of target point from each of the monitors.

The VNA method is highly advantageous, it uses monitor data and provides clear and consistent definition of spatial relationships between unconnected points. The method can be applied to estimate temporal and spatial interpolations when using scaling. Also the shape and size of "Voronoi" cells are adaptive to spatial distribution of population and monitors(USEPA, 2015. Du *et. al.*, 2002). Also the distance to the monitors can be defined by the user, so the user has the flexibility to use monitors which are within a certain distance. The VNA method is highly reliable, and replicable; and is currently being used by FL- DEP in several evaluations and studies (Tolbert et al., 2007Sarnat *et. al* 2008; Sarnat *et. al* 2010; Nowak *et. al* 2013).

Model and Monitor relative: Uses ambient air quality data from EPA's local monitoring stations in combination with modeling to get local pollutant levels. This is the method used by the EPA to quantify the burden of O_3 and $PM_{2.5}$ on health in the United States (Fann, *et. al.*, 2011). The modeling data can be combined with monitoring data using VNA method, to give better predictions. This method provides predictions in areas or time periods when monitoring data are

not available. The method is highly recommended by EPA, air monitoring and risk analysis unit. There are several published studies promoting the model and monitor combination method (Kheirbek et. al 2013; Kheirbek et. al 2016; Fann et. al. 2008).

Monitor Rollback method is used to reduce or increment the available monitor data in BenMAP by a certain value, this method is referred as rollback. The monitor data can be rolled back using three methods: percentage rollback, incremental rollback, and rollback to a standard. Percentage rollback reduces all monitor observations by the same percentage. Incremental rollback increases all observations by the same increment. This method is useful in studying the effect of change in pollutant concentration on health outcomes. (USEPA, 2014b; USEPA2015)

All four methods mentioned here group individuals spatially into grid cells in BenMAP. The grid cells help to estimate the average exposure to ambient air pollution of persons living in some specified area. These spatial estimating grid cells are referred to as "*air quality*" grids in BenMAP. The "*air quality*" grids can be regular shape, such as 12 x 12 km grid cell, or domain, such as delineated by models like CMAQ, CAMX, REMSAD, as well as irregular shapes like counties or zipcode. It is assumed that all persons in a spatial grid cell are exposed to the same pollution levels.

Uncertainties due to Exposure Assessment

As in all other stages of the benefits analysis, the assumptions and methods used in the exposure assessment should be well-justified and clearly described, with careful attention paid to assessing and communicating key sources of uncertainty. Measurement error is inherent in

estimates of exposure based on ambient pollutant monitors. Data for exposure to pollutants is typically based from monitors at central sites and do not adequately represent personal exposure (Haran *et. al.* 2002). Uncertainties arise using central sites because of spatial variations in ambient concentrations across cities, wide ranges of personal activity patterns, and differences in ambient air penetration indoors. On the contrary, single city studies have shown that in time series studies central-site fine-particle measurement correlate well with average population exposure over time (Samat *et. al.* 2000; Dominici *et. al.*, 2003b). The measured ambient concentrations at a central site may differ across cities, and little is known about reliability of population exposure averages measured from central-site across cities. The penetration of ambient PM to indoor environments has been shown to vary with weather conditions (Samat *et.al.*, 2000). Including weather components will help decrease these uncertainties but can remain inherent and limitations should be clearly stated in the analysis.

The use of statistical models or air quality numerical models used to help in exposure assessment of PM can add more sources of uncertainty to health risk assessments as these models have their own uncertainties. Evaluation of air quality models help to quantify the different sources of error in the models. Using monitoring data in conjunction with CMAQ model data helps to amount uncertainty (Fuentes, 2009). In most cases probabilistic models can help characterize the uncertainties. In Bayesian approach models the joint distribution of exposure rather than just means of the distribution and can help characterize uncertainties better (Fuentes, 2009). Sensitivity analysis can be conducted to understand the impact of uncertainty in the exposure on HIA (Fuentes, 2009).

Health Benefits Analysis using BenMAP

Health benefits are calculated by linking the impact function and the modeled changes in air pollution (Charleston *et.al.,*2008; Talbot *et.al.,*2009). The commonly used health benefits are changes in mortality, chronic illness, hospitalizations, emergency department visits, acute illnesses not requiring hospitalization, exacerbations and repeat episodes, work/school loss days, and minor restricted activity days (MRADs). In BenMAP these health endpoints have unit values available to calculate the cost associated or to monetize the benefits (Fann et. al. 2008; USEPA 2015).

Uncertainties and Sources of Uncertainties

Confidence intervals generated using standard errors associated with effect estimates have been used traditionally to characterize the overall uncertainties in the HIA. This describes a narrow range of the total uncertainty. There is no comparable information available for the baseline incidence/prevalence rates, exposure estimates and air quality changes. The local HIA imposes additional uncertainties due to lack of comparable information—which are difficult to characterize quantitatively. Sensitivity analyses, which vary key input parameters such as effect estimates and baseline incidence rates, may be useful substitutes to traditional confidence intervals. Uncertainties may be alleviated by careful selection of effect estimates, baseline incidence rates and other input data.

2.11 Developing HIA functions for Local Scale

National HIA functions do not describe impacts of pollutants at the local or on the sub-national scale accurately. Development of national HIA functions use pooling techniques. Under

this approach, studies are weighted by the inverse of their variance, and can generate more robust national effect estimates. These pooled functions developed for national level HIA are less useful for generating local effect estimates as they induce heterogeneity in populations and exposures across all local areas. Local-scale HIAs require more geographically resolved air quality data, CR functions, and baseline incidence rates than are often used. However, comprehensive local data may not be available or may be incomplete for developing CR functions, since small-scale local epidemiologic studies will often be underpowered.

Recent epidemiologic studies on $PM_{2.5}$ and adverse health outcomes (Levy *et. al.* 2000; Fann *et. al.* 2008; Hubbell *et. al.* 2009) indicate that national mean estimates may need to be adjusted to account for local factors that are related to the effect estimate. Epidemiologic studies conducted in Detroit have generated city specific estimates, but suffer from poor statistical power, and do not cover the different health outcomes typically assessed in a benefits analysis. The process for selecting appropriate effect estimates for HIA requires development of profile characteristics of the study locations (e.g. demographics, disparity, weather , population density, etc), and finding the closest match along a range of attributes that can impact effect estimates. However, in cases where local estimates lack statistical power, it may be best to apply national effect estimates (Fann *et. al.* 2008; Hubbell *et. al.* 2009).

BenMAP is a powerful computer based tool for HIAs at the national level, and is recognized to be a reasonable approach for cost benefits analysis with some inherent uncertainties by the National Research Council (NRC, 2002, 2008). However, for local or state level HIA's, it needs modification to minimize the uncertainties of the assessments. Local baseline incidence and

prevalence rates by age, gender and race/ethnicity are not available for Florida presently, and need to be developed for local Florida assessments.

CHAPTER THREE: METHODOLOGY

3.1 Study Design

A population-based ecological study was conducted to study the impact of ambient $PM_{2.5}$ concentrations on childhood asthma exacerbation emergency department rates.

3.2 Data Sources

Florida Agency of Health Care Administration Emergency Department Claims Data

Emergency Department (ED) discharge data extract files for state of Florida for 2010, 2011, 2012, 2013 and 2014 were obtained from Florida AHCA. The data obtained contained variables such as zip code of patient residence, patient county, age of patient and dates of service, however other identifiers like the residence address, patient name and SSN were not provided by Florida AHCA. The data obtained from AHCA was at patient record level with a record of each visit of a patient.

Study Subjects

Based on Florida Emergency department data the study populations were created as children between the age of 5-18 who were seen for asthma in emergency departments in Florida counties and who resided in Florida counties. The records with patients residing in counties out of Florida (patient county=99) were excluded in the analysis stage.

An asthma case was defined as a case with emergency department record having a primary diagnosis of asthma (ICD-CM, 9th revision =493, 493.0, 493.00, 493.01, 493.02, 493.1, 493.10, 493.11, 493.12, 493.22, 493.8, 493.81, 493.82, 493.9, 493.90, 493.91, 493.92). An asthma exacerbation was defined as having primary diagnosis of exacerbation (ICD-CM, 9th revision =493.02, 493.12, 493.22, 493.92). Individual subjects could have multiple emergency department visits during the study period. However, since the subject unique identifier were not shared by AHCA due to HIPAA regulations, a repeat visit of the patient could not be identified in this study. There is not direct method to account the repeat measure of patient, the only way to account is to use percentages of repeat measurements from the published literature. FAP had estimated that in 2011, nearly 17.2 % have repeated asthma visits to ED or inpatient hospitalizations. These accounted for 36.0% of all asthma ED visits or hospitalizations and 36.1% of the total charges for all asthma ED visits or hospitalizations in general Florida population. (FAP, 2014). These estimates can be used to calculate repeated measures. However, these estimates are for overall population ages 0-99 and not specific to 5-18 years. It is estimated that for 5-18 the repeated visits will be higher since asthma visit are higher for 5-12 age group.

Population Estimates

The demographic estimates for population in each county for 2010, 2011, 2012, 2013 and 2014 were obtained from Florida Health Charts via a web query accessed on June 05, 2015, through the link : *http://www.flhealthcharts.com/FLQUERY/Population/PopulationRpt.aspx*. Population estimates for Florida Health Charts are provided by the Department of Health, Office of Health Statistics and Assessment in consultation with the Florida Legislature's Office of Economic and Demographic Research (EDR). The population data for 2010 for Florida Health Charts, along with

rates affected by the population data were updated in 2012 by EDR based on information from the 2010 census. Age-group data estimates available from Florida Health Charts were in 1-year age intervals. The population estimates for each county were obtained for each age year and age groups were then calculated by summing across the categories within gender and race/ethnicity. Two age group categories 5-12 and 13-18 were calculated, 5-12 are elementary and middle school children and 13-18 are middle school and high school children. The race /ethnicity categories where combined to White not Hispanic, Black not Hispanic, Hispanic (any race) and Other.

U.S. Environmental Protection Agency – Ambient Air Monitoring Data

$PM_{2.5}$ measurements for 2010, 2011, 2012, 2013 and 2014 were obtained from publicly available U.S. EPA Air Quality System (AQS) DataMart. Data were available from 24 $PM_{2.5}$ monitors in 17 counties across Florida. Daily summary data for 24-hour mean for $PM_{2.5}$ retrieved from DataMart, using AQS Application Programming Interface (API). It is noticed that sampling data collection varied from daily to every three days. Nearly, 80% of locations were every 3 days. Temperature and Precipitation data were also obtained from AQS Data mart, these data are grouped under meteorological section.

The data downloaded for $PM_{2.5}$, were measured by USEPA under federal reference methods or federal equivalent method (FRM/FEM, 88101). The FRM/FEM are monitoring data used for checking atmospheric air quality, and for purposes of determining compliance with the US. National Ambient Air Quality Standards (NAAQSs) – specified in 40 CFR Part 50. While the data retrieved for temperature were reference number 62101 (AQS,2015). The relative humidity is the

ratio (percent) of actual pressure of water vapor in air to the saturation vapor pressure at the same temperature, this is also summarized as average daily values by USEPA (AQS,2015). .

The data for temperature, precipitation and $PM_{2.5}$ are listed by the year and data tables were linked to each other by county. The files downloaded were comma separated text with a header. These files were daily summary and were downloaded from AQS API, using the following link https://aqsdr1.epa.gov/aqsweb/aqstmp/airdata/download_files.html (AQS,2015). Similar to $PM_{2.5}$ data the temperature data is submitted by tribal, state and local agencies and goes through several quality control steps and is certified annually by the submitting agency. The data from AQS were already aggregated to daily averages (approximately foe every third day) by USEPA.

3.3 Procedures

The procedures to the specific aims were

Aim One: To assess the efficacy/utility of Concentration Response (CR) functions and Asthma prevalence rates present in EPA's BenMAP for estimating childhood asthma exacerbation rates due to changes in $PM_{2.5}$.

This was evaluated by performing a detailed review of documentation of user's manual and appendices for BenMAP-community edition (BenMAP-CE). Publicly available documents were downloaded from the EPA's website (USEPA, 2015); and more detailed documentations which included details on studies in BenMAP were obtained in Summer 2009 during my NNEMS Fellowship at EPA, RTP, and from personal communication with EPA staff , Neal Fann at US EPA-RTP. BenMAP-CE training sessions hosted by the US-EPA Air Pollution Training Institute were also attended to help understand calculation of health impacts using CR (USEPA, 2014b). These

documentations gave details on CR functions that are already present in BenMAP for health outcomes and $PM_{2.5}$ (USEPA, 2014b; USEPA, 2015). CR functions for only O_3 and $PM_{2.5}$ are currently present in BenMAP-CE and are accessible in the publicly available version (USEPA, 2014b).

To assess the efficacy of CR functions in BenMAP a detailed review of peer reviewed published literature was performed. The published literature CR functions were compared to those already present in BenMAP. The CR functions for asthma exacerbation and $PM_{2.5}$ which are not present in BenMAP, or are based on older studies will be the focus of objectives 2 -5.

Aim Two: To estimate annual baseline emergency department asthma and asthma exacerbation prevalence rates for children at the local county level in Florida from 2010-2012.

This objective was addressed by analyzing emergency department data received from Florida AHCA, and incorporating baseline emergency department asthma and asthma exacerbation prevalence rates into BenMAP. These data were analyzed and descriptive statistics for each year were calculated using Statistical Analysis Software (SAS®, Version 9.4). Annual emergency department visits for Florida and baseline ED visit prevalence rates for Florida were calculated. In addition, rates specific to age groups (5-12, 13-18), gender, race/ethnicity, season and county were calculated. The stratified rates by race/ethnicity, age and gender for asthma and asthma exacerbation were also calculated. These stratified rates were compared to overall rates by age group (5-12, 13-18), gender, race/ethnicity.

The annual emergency department visits for all causes, asthma and asthma exacerbation for school age children (i.e. ages 5-18) were calculated. This was done by aggregating the visits in each category for each year, i.e. 2010, 2011, 2012, 2013 and 2014.

Annual ED_a Visits for Florida in 'y^{th}' year = $\sum_{i=1}^{67} Annual\ ED_a\ Visit_{iy}$

Where, i = 1- 67 co*unties in Florida*

$y \in \{2010, 2011, 2012, 2013, 2014\}$

a $\in$ *{All visits, Asthma visits, Asthma Exacerbation visits}*

Average visits per county was calculated using sum of all the visits across Florida in a year and dividing it by number of counties in Florida, i.e. 67. These stratified rates by county give the variance of average annual visits in Florida.

Average Annual Visit per County= Annual ED_b Visits for Florida in 'y^{th}' year /67

Where, $y \in \{2010, 2011, 2012, 2013, 2014\}$

b $\in$ *{Asthma visit, Asthma Exacerbation}*

The annual trends over time 2010-2014 for Florida were tabulated and were presented graphically for comparison.

A prevalence rate is defined by the CDC as "*the proportion of persons in a population who have a particular disease or attribute at a specified point in time or over a specified period of time*" (CDC, online). Hence, the annual prevalence rate for asthma ED visits is defined as the proportion of children (5-18) residing in Florida who had ED visits for asthma within a year. The

annual prevalence rate for asthma exacerbation ED visits is defined as the proportion of children (5-18) residing in Florida who had ED visits for asthma exacerbation within a year.

The overall prevalence rates for Florida were calculated for each year using the following formula:

$$\textbf{Annual Prevalence for Asthma Visits } \textit{per 1,000} = \frac{\text{All asthma ED visits in a given year}}{\text{Population of children in the same year}} \times 1000$$

$$\textbf{Annual Prevalence for Asthma Exacerbation } \textit{per 1,000} = \frac{\text{All asthma exacerbation in a given year}}{\text{Population of children in the same year}} \times 1000$$

Baseline annual averages were calculated using 2010-2012 data, by averaging the annual visits over the three years. The data was averaged over three years to smooth the variance due to between the years.

$$\textbf{Baseline ED}_{a}\textbf{ Visit for Florida} = \frac{(\text{Annual ED}_{a}\text{ Visit}_{2010} + \text{Annual ED}_{a}\text{ Visit}_{2011} + \text{Annual ED}_{a}\text{ Visit}_{2012})}{3}$$

Where, $a \in$ *{All visits, Asthma visit, Asthma Exacerbation}*

Baseline prevalence rate for asthma ED visits was calculated using the baseline asthma ED visits as numerator and population of children in the 2010 Census year as denominator. Baseline prevalence rate for asthma exacerbation ED visits was calculated using the baseline asthma ED visits as numerator and population of children in the 2010 Census year as denominator.

Baseline Florida Specific Rates

$$\text{Baseline Prevalence } \boldsymbol{ED_b} \text{ Asthma Rate}_{jkl} = \frac{\text{Baseline } \boldsymbol{ED_b} \text{ Asthma Visit} \times 1000}{2010\ Census\ Population\ of\ Florida}$$

Where, b $\in$ *{ Asthma visit, Asthma Exacerbation}*

j = age group, i.e. 5-12, 13-18

k = gender, i.e. Male, Female

l = race/ ethnicity, i.e. White, Black, Hispanic, Other

Baseline Prevalence rates were calculated for gender, age group (5-12, 13-18), race/ethnicity (White non-Hispanic, Black non-Hispanic, Hispanic, all other).

For each county, total annual visits were obtained by adding all visits within gender, race/ethnicity and age group specific for the county in a specific year (ie. 2010, 2011, 2012).

County Specific Rates

$$\textbf{Annual } \boldsymbol{ED_b} \textbf{ Asthma Visit}_{\boldsymbol{i}} = \sum_{jkl} ED_b \text{ Asthma Visit}_{ijkl}$$

Where, b $\in$ *{ Asthma visit, Asthma Exacerbation}*

i = 1- 67 co*unties in Florida*

j = age group, i.e. 5-12, 13-18

k = gender, i.e. Male, Female

l = race/ ethnicity, i.e. White, Black, Hispanic, Other

Baseline **ED_b Annual Visit for County$_i$**

$$= \frac{(\text{Annual } ED_b \text{ Visit}_{i,2010} + \text{Annual } ED_b \text{ Visit}_{i,2011+} \text{Annual } ED_a \text{ Visit}_{i,2012})}{3}$$

Where, b ϵ *{Asthma visit, Asthma Exacerbation}*

Baseline ED_b Prevalence i^{th} County

$$\text{Baseline Prevalence } \boldsymbol{ED_b} \text{ Asthma Rate}_i = \frac{\text{Baseline Annual ED}_b \text{ Visit} \times 1000}{2010\ Census\ Population\ of\ County_i}$$

The baseline annual rates for emergency department visits which were specific for gender, race/ethnicity and age group for state of Florida were calculated by dividing the baseline annual ED visits in each group with the population estimates in each group level obtained from Florida Health Charts(*http://www.flhealthcharts.com*). The baseline annual ED visits in each group was the average of annual ED rates for years 2010-2012, similar to the county level shown above. These annual county level rates were compared to rates at the State and National level rates. These comparisons highlight the differences between county and national level rates, specific to age, race/ethnicity and gender.

Aim Three: To evaluate the temporal and spatial patterns of $PM_{2.5}$ in Florida and incorporate $PM_{2.5}$ data monitor for Florida into BenMAP for HIA estimations.

The goal of this specific aim was to define temporal and spatial patterns of $PM_{2.5}$ across Florida, and to incorporate air pollution monitoring data for Florida counties into BenMAP. Temporal and spatial patterns of $PM_{2.5}$, were defined using data from Florida EPA's monitoring data from 2010-2012. This objective was accomplished by using Florida EPA's 24-hour average $PM_{2.5}$ daily summary data for all monitoring sites operating during 2010-2012.

The data downloaded from Florida EPA, for 24 monitoring locations in 17 counties were used for this analysis. The monthly trend of daily $PM_{2.5}$ concentration ($\mu g/m^3$) in Florida were obtained by averaging daily concentrations across all operational monitoring sites in Florida during the month in these years. These daily $PM_{2.5}$ concentrations help in understanding monthly temporal trends and any seasonal variations. The descriptive analysis of these trend included in averaging of daily $PM_{2.5}$, minimum, maximum and standard deviation in the month across all monitoring stations, and are given in the results section.

To study the spatial and temporal patterns of daily $PM_{2.5}$ concentrations monthly averages of daily $PM_{2.5}$ concentrations for each county were calculated. The monthly $PM_{2.5}$ concentrations were calculated by averaging daily concentrations across all monitoring sites in that county during that month. Comparing the county level data of monthly averages contributed to the spatial trends at county level.

Time series analysis were performed to study the temporal and seasonal trends of $PM_{2.5}$ in Florida. Peer reviewed literature over the past two decades has reported time series analysis as one of the major methods used to study long-term variation in the mean concentration (trend) and periodic components (season) (Slini, *et. al.* 2002; Peng *et.al.,* 2004, 2006; Andria *et. al.,* 2008). PROC TIMESERIES procedure in SAS© was used to study the trend analysis of $PM_{2.5}$ while testing for differences in monthly averages for the three years of data, and any seasonal pattern. This analysis was also run for each county to see if there were any visual differences between counties.

The temporal and spatial trends at county level were further studied by Panel Design for time series (Hsiao, 2014). The panel data analysis is used when time series and cross sectional data are combined. Panel data have been extensively used in linear econometric models when analyses included observations in time on households, countries, trade and air pollution (Hsiao, 2014). A Panel is similar to stratifying the data by location group. In this study, panel was synonymous with county of diagnosis or ED visits. The PROC SGPANEL procedure was used in SAS© to perform panel analysis. The time series regression procedure in SGPANEL was used to study the temporal trend for each county. For Florida the county estimates are pooled by the size of the county. The weights used in the pooling of the county was the area in square miles. The area size versus population size was used in this study. The main reason to use area was to see the spread of pollutants in the counties versus the number of people affected. The monitoring locations are mandated by federal regulations and are based on population density of an area. Using area weights adds to the knowledge of distribution of $PM_{2.5}$ concentrations in counties with lower population density. The weight matrix, $W=(w_{ij})$ is NxN positive matrix in which the rows and columns correspond to the cross-sectional county units, and $w_{ii}=0$. The weights were standardized so that the sum of each row, $\sum_{j=1}^{N} w_{ij} = 1$.

Aim Four: To develop county-level CR functions to be used in HIAs performed by local or State agencies.

The goal for this objective was to develop county level CR functions using local ambient pollutant concentrations and asthma emergency department visits during 2010, 2011 and 2012. Counts of rare events are discrete numbers and are not normally distributed (Schwartz and Morris, 1995) and have been analyzed using Time-Series models to study the effect of environmental

exposures on rare events (Samet *et. al.* 2000a,b; Dominici *et. al.*, 2004; Bell *et. al.*, 2005; Fuentes, 2009).

Generalized Poisson linear regression models which can account for over-dispersion of health outcome data was used (Fuentes, 2009). Over-dispersion generally happens when the variance in the population is more than expected (McCullagh *et. al.*, 1989). Over dispersion can be due to unaccounted covariates in the model. For this study the other allergens and multi-pollutants were not included, these could introduce over-dispersion. When an over-dispersion is present it tends to distorts the standard error and test statistics (McCullagh *et. al.*, 1989). Generalized Estimating Equations (GEE) using generalized Poisson distribution, were used to investigate the association between different timescales of PM2.5 and childhood asthma /asthma exacerbation ED visits.

GEE is an iterative process which uses iterative estimating equations that use estimates of regression parameters and their variance under time dependence assumptions. (Liang and Zeger, 1986). The parameter estimates from GEE are consistent and insensitive to covariance structures. The estimators are known to be robust as they rely on the first two moments (i.e mean, variance). GEE tend to focus on estimating the average response or "population -averaged effects" rather than effect on individual of changing covariates (Zeger *et. al.*,1985)

$$\boldsymbol{Y_N(t)} = \sum_{\boldsymbol{N}} \boldsymbol{Y_j(t)}$$

Where N are number of estimating equations, ranging from 1 to n.

If $Y_j(t)$ is the number of asthma/ asthma exacerbations ED visits of county *j* for day *t* and j=1,…,J and t=1,…,T. It is was assumed that $Y_j(t)$ follows a generalized Poisson distribution

(GPoi) with dispersion parameter ϕ, mean parameter $\mu_j(t)$

$$Y_j(t) \sim GPoi(\phi, \mu_j(t)),$$

$$\log(\mu_j(t)) = \beta_j PM_{(t-l)} + S(temp_j(t), \lambda_1) + S(precp_j(t), \lambda_2) + \gamma_1 * k + \gamma_2 * weekend + \gamma_3 * month + \tau_{jn} Z_{jn}(t)$$

and variance for $Y_j(t)$ is given by

$$\boldsymbol{Var}\left[\boldsymbol{Y_J(t)}\right] = \boldsymbol{\mu_j(t)}\emptyset$$

Where :

j refers to county (1-67 for Florida Counties);

k refers to the Season,

k=1 is cold season (November – April) and

k=2 is warm season (May-October)

weekend =Yes/No

month= January to December

t refers to time, is day 1-365 for each year

l refers to lag of $PM_{2.5}$ which varies from 0-2 days

$\gamma_1, \gamma_2, \gamma_3$– refer to the coefficients of season , weekend and month

Z refers to matrix of independent variables, confounders and effect-modifiers

τ_{jn} refers to the coefficients of confounders.

The effect of each orthogonal decompositions of $PM_{2.5}$ time-series is allowed to vary by county (j) and by season(k). The S (*w* , λ) are smooth functions of the weather covariates and

w=(temperature, and precipitation); λ is the smoothing parameter and represents the degrees of freedom (df). For temperature and precipitation the splines with df=2 were used. These weather variables are important covariates in explaining air pollution.

The confounders and effect modifiers considered in this study were:

gender (male, female);

race/ethnicity (White, Black, Hispanic, Other);

age groups 5-12 and 13-17

counties 1-67 Florida counties

The gender and ethnicity were controlled in the GEE models, while to control for age group, separate models were used to get the beta estimates. The PROC GENMOD procedure was used in SAS© to perform GEE. For each age group the models were separately run for comparing them with those obtained from peer-reviewed studies and those present in BenMAP. The CR functions from this aim are local to Florida.

In addition, a detailed literature review of epidemiological and environmental studies was performed. The CR functions or the odds ratios from the peer-reviewed studies, which analyzed the change in childhood asthma associated with change in pollutant concentration, were tabulated. This analysis evaluated studies using selection criteria including its location, design, study population and if the study was peer-reviewed. The priority was given to the US based studies that have focused on $PM_{2.5}$ pollutant and childhood asthma as health effect. The CR functions from peer-reviewed studies were compared to CR functions local to Florida and effort

was made to pool the results using BenMAP (USEPA, 2015; Fuentes 2007).

Aim Five: To estimate age and gender specific asthma exacerbation rates in children due to change in $PM_{2.5}$ concentrations at county level.

In BenMAP one can estimate the pollution concentration in a specific year using the Monitor Rollback method (USEPA, 2015). This method uses available monitor data in BenMAP incremented or reduced by a certain value. This value can be the percentage increase or decrease as expected for a future period. The rollback method increases/decreases all observations by the same percentage. This method is useful in studying the effect of change in pollutant concentration in future years on health outcomes.

Currently, data for $PM_{2.5}$ present in BenMAP are for 2000-2008. USEPA has acknowledged that the pollution data in BenMAP are not current and should be updated by users periodically for analysis. The data were uploaded into BenMAPfor Florida counties using monitor data from FLEPA for 2010 to 2014. The monitor data was cleaned and entered in BenMAPusing the instructions from the BenMAP manual. (USEPA, 2015)

The 2008 data for Florida counties in BenMAP were used to estimate average daily $PM_{2.5}$ concentration (in $\mu g/m^3$) at the county-level for 2011, by using percentage change for each county. The percentage change from 2008 to 2011 for county was obtained using the data from EPHT (FLDEP, 2016). The average annual data for $PM_{2.5}$ by Florida counties that were extracted from EPHT are given in Appendix 4. The percentage changes were calculated for each county, and the results were tabulated. These values of percentage change were entered in BenMAP and

were used to estimate the $PM_{2.5}$ concentrations. The observed concentration values of $PM_{2.5}$ from BenMAP were compared to expected values using EPHT data. The error and % difference were calculated.

$$Error = \frac{|Expected - Observed|}{Expected}$$

The ED prevalence rates at the county level for asthma and exacerbation were calculated using log linear formulae built into BenMAP by the USEPA (USEPA, 2014a; USEPA, 2015). For this aim the ED prevalence rates at county level were estimated using:

a. National prevalence rates and CR functions already present in BenMAP, and are considered standard values by the EPA for regulatory HIAs
b. Florida count-level prevalence rates from Aim 2 and CR functions from Aim 4 to estimate asthma /asthma exacerbation rates in Florida at the county-level while controlling for gender and race/ethnicity and stratifying for ages groups 5-12 and 13-18.

Prevalence rates based on different combinations were examined for lower uncertainties utilizing error analysis. The county level data were aggregated to Florida by each method and the means were tested using T-test.

CHAPTER FOUR: RESULTS

4.1 Aim One: Functions and Prevalence Rates Present in EPA's BenMAP

To assess the efficacy/utility of Concentration Response (CR) functions and Asthma prevalence rates present in EPA's BenMAP

USEPA's BenMAP has CR functions pre-loaded in the software for estimation health outcome changes due to changes in the concentration of criteria pollutant $PM_{2.5}$. The derivation

Table 1. Asthma Related Health Effects Prevalence Rates in BenMAP

Endpoint	Age	Parameter	Annual Rate per 100	Source
Asthma Exacerbation, Shortness of Breath	8-13	Prevalence	7.40	Ostro *et. al.*. (2001)
Asthma Exacerbation, Wheeze	8-13	Prevalence	17.30	Ostro *et. al.*. (2001)
Asthma Exacerbation, Cough	8-13	Prevalence	14.50	Ostro *et. al.*. (2001)
Asthma Prevalence Rates, US population	All ages	Prevalence	7.8	American Lung Association (2008)
Asthma Prevalence Rates, US population	<5	Prevalence	6.14	American Lung Association (2008)
Asthma Prevalence Rates, US population	<18	Prevalence	9.41	American Lung Association (2008)
Asthma Prevalence Rates, US population	5-17	Prevalence	10.7	American Lung Association (2008)
Asthma Prevalence Rates, African American	<5	Prevalence	9.98	American Lung Association (2008)
Asthma Prevalence Rates, African American	5-17	Prevalence	17.76	American Lung Association (2008)
Asthma Emergency Department Visits	0-17		0.865	HCUP and NAMCS (2007)

Source BenMAP Community Edition Manual, USEPA, 2015 (pages:55, 58,59)

and estimation of these functions and rates are explained in detail in the Appendices D and E of BenMAP's Users manual (USEPA, 2015). The relevant prevalence and exacerbation rates present in manual are given in Table 1 and CR functions are outlined in Table 2.

Table 1 shows that asthma prevalence rates being used in BenMAP are much lower than the published rates for Florida by FLDEP (Appendix 3.1). Using CDC's BRFSS, the asthma prevalence for middle and high schoolers ages 10-20 is "defined as those who have been told that they have asthma" (Appendix 3.1). For 2006. The annual asthma prevalence rate was 17 per 100 cases in Florida, this rate has increased nearly 4% by year 2012 (Appendix 3.1). The results also shows that the rate varies by county. Counties with lower African American population seem to have lower prevalence rates (Appendix 3.1).

The crude rate for Asthma emergency visits in Florida for 2006 was 0.85 per 100, but this rate has shown a gradual increase since 2006 and has reached 1.18 per 100 in 2014 (Appendix 3.1). The 3% increase in emergency visit rates could be due to population growth in certain age groups or ethnicities with higher asthma rates, or simply a surge in severe asthma cases due to increase general awareness of asthma in schools, through asthma education programs implemented by the Florida Asthma Coalition (FAC), or due higher access to care during Obama Health Care. Hence, using the prevalence or asthma emergency rates present in BenMAP for HIA will underestimate the asthma rates in BenMAP, due to two main reasons. Firstly, these rates are derived using rates across the nation and do not represent true Florida population. Secondly, with an increase in asthma prevalence and emergency department visits in Florida using older

prevalence or asthma emergency rates will underestimate the rates. There are baseline rates for asthma exacerbation in BenMAP, asthma exacerbation is a more severe case and the prevalence for exacerbation ED visits is seen to be lower than asthma ED visits, similar to asthma prevalence is higher than asthma exacerbation prevalence in general population.

Table 2, highlights the already present CR functions in EPA's BenMAP. Different studies are used to estimate asthma hospital visit, asthma emergency rates and asthma exacerbation rates. Each of the studies are based in different locations, have different endpoints and have used different statistical models for analysis. The CR functions for asthma emergency room visits, which are based on studies from the west coast and are not representative of the east coast due to several factors. The main differences between the west coast populations and Florida population are the size of population, age, gender and race/ethnicity distribution, geographical location, weather, and pollutant concentration. The three studies are based in Seattle or nearby areas, which are at higher altitude than any area in Florida. The west coast climate patterns are more defined by four season while Florida is more tropical with just warm and cold seasons. Florida is more prone to hurricane and heavy rainfall during the summer season while Seattle is during winter and early spring season. Average humidity and due point is higher in Florida than in Washington state. The population density in Seattle and its neighboring areas is higher than in metropolitan cities of Florida, like Miami.

Norris *et. al.* (1999) studied the relationship between asthma hospital visits for ages <18 and air pollutants. They converted PM_{10} to $PM_{2.5}$ concentrations and saw significant association between $PM_{2.5}$ and emergency room visits. The study used Poisson regression model with

adjustments for day of week, time trend, temperature and dew point. Using a multiplier to convert PM_{10} to $PM_{2.5}$; overestimates the $PM_{2.5}$ concentrations, hence introducing uncertainty in the analysis and masking the true effect of $PM_{2.5}$ on asthma emergency room visits. The study population was 1995-1996 which is over 20 years ago, the populations and the risk has changed in 20 years.

Slaughter *et. al.* (2005) used log linear generalized liner model to study the effect of $PM_{2.5}$ and emergency room visit for asthma in Spokane, Washington, from 1995-2001 using log linear generalized regression models. The study population included all ages and was not specific to children. Mar *et. al.* (2010) studied the effect of $PM_{2.5}$ and asthma emergency room visits in Tacoma area, from 1998-2002 using Poisson regression models and GAM models. The population was all ages and was controlled for season trends, daily temperatures and relative humidity. Asthma prevalence rates are highest in children and elderly over ages 65, using all age estimation may underestimate the asthmas emergency rates.

Mar *et. al.* (2004) studied the effect of PM2.5 on asthma exacerbation cough and shortness of breath as a sub-study of a main study to determine the effect of pollen allergy on asthma in children and adults. This study was designed in a small area of Spokane and included nine children and sixteen adults from 1997 to 1999. Ostro *et. al.* (2001) studied the relationship of air pollution and asthma exacerbation in African American children ages 8-13 living in LA during August to November 1993.

The detail review of the studies in Tables 1 and 2, shows that all the studies used for prevalence and CR functions base their estimates on population studies conducted 15 or more years ago. The prevalence and incidence rates have changed for asthma and asthma exacerbation

Table 2 CR Functions for $PM_{2.5}$ and Asthma-Related Effects

Effect	Author	Study Year	Study Location	Age	β	Std. Err	Form
Asthma, Emergency Room Visit	Norris *et. al.*, 1999	1995-1996	Seattle, WA	0-17	0.0165	0.0041	Log-linear
Asthma, Emergency Room Visit	Slaughter *et. al.*, 2005	1995-2001	Spokane, WA	0-99	0.0029	0.0027	Log-linear
Asthma, Emergency Room Visit	Mar *et. al.*, 2010	1998-2002	Greater Tacoma, WA	0-99	0.0056	0.0021	Log-linear
Asthma Exacerbation Cough	Mar *et. al.*, 2004	1997-1999	Spokane, WA	6-18	0.0191	0.0098	Logistic
Asthma Exacerbation Shortness of Breath	Mar *et. al.*, 2004	1997-1999	Spokane, WA	6-18	0.0122	0.0138	Logistic
Asthma Exacerbation Cough	Ostro *et. al.*, 2001	1993	Los Angeles, CA	6-18	0.0010	0.0008	Logistic
Asthma Exacerbation Shortness of Breath	Ostro *et. al.*, 2001	1993	Los Angeles, CA	6-18	0.0026	0.0013	Logistic
Asthma Exacerbation Wheeze	Ostro *et. al.*, 2001	1993	Los Angeles, CA	6-18	0.0019	0.0008	Logistic
Asthma, Hospital Admissions	Babin *et. al.*, 2007	2001-2004	Washington, DC	0-17	0.0020	0.0043	Log-linear

Source BenMAP Community Edition Manual, USEPA 2015 (pages:101,107)

in the past ten years. In Florida, the rates are showing a general increase and do not match the rates in the data used for these studies. The location where the studies were conducted and timing of these studies makes it a valid argument that to use BenMAP effectively for HIA in Florida one should update the prevalence rates and CR functions in BenMAP with Florida data.

4.2 Aim Two: Childhood Asthma Prevalence and Exacerbation rates in Florida

To determine the baseline emergency department prevalence and exacerbation rates for childhood asthma in Florida from 2010-2012.

A total of 2,668,100 pediatric emergency department (ED) visits of school-aged children (ages 5-18) occurred in the State of Florida between January 01, 2010 and December 31, 2012, of which 76,576 (2.87 %) were asthma visits (ICD-9=493), and 44,503 (1.67%) were asthma exacerbation visits (ICD-9= 493.02, 493.22, 493.92). (Table 3).

Table 3 Emergency Department Visits in Florida for children ages 5-18

Year	ED Visits	ED Visits for Asthma	ED Visit for Asthma Exacerbation
2010	834,240	24,020	14,626
2011	871,551	24,139	14,068
2012	962,309	28,417	15,809
2013	977,896	29,045	16,706
2014	1,049,057	30,713	18,698
2010-2012	**2,668,100**	**76,576**	**44,503**
Average Annual *	**889,367**	**25,525**	**14,834**

*Uses only data for 2010-2012, for this study the average for 2010-2012 is considered baseline

Average annual ED visits for childhood asthma were 25,525 and for childhood asthma exacerbations were 14,834. The overall average prevalence for childhood asthma is 8 cases per 1,000 children aged 5-18 residing in Florida (Table 4). The annual trend for asthma and asthma exacerbation ED visits are shown in Figure 6 and Figure 7. The total asthma visits for children have increased from 2010 to 2014. The total visits in Florida (as shown by line graph) were steady from 2010 to 2011 and then there was a sharp increase from 2011 to 2012 (24,139 to 28,417), however, total visits gradually increased to 30,713 by 2014.

The annual asthma and asthma exacerbation rates increased from 2010 to 2014 by 27.8% (Table 4). The average baseline prevalence averaging over 2010 to 2012 for asthma exacerbation is 4.7 cases per 1,000 children aged 5-18 residing in Florida.

Table 4 Emergency Department Rate in Florida for children ages 5-18

Year	**Annual Asthma Visit Rate per 1000**	**Annual Asthma Exacerbation Rate per 1000**
2010	7.55	4.60
2011	7.59	4.42
2012	8.93	4.97
2013	9.13	5.25
2014	9.66	5.88
Average Annual *	**8.0**	**4.7**

*Uses only data for 2010-2012, for this study the average for 2010-2012 is considered baseline

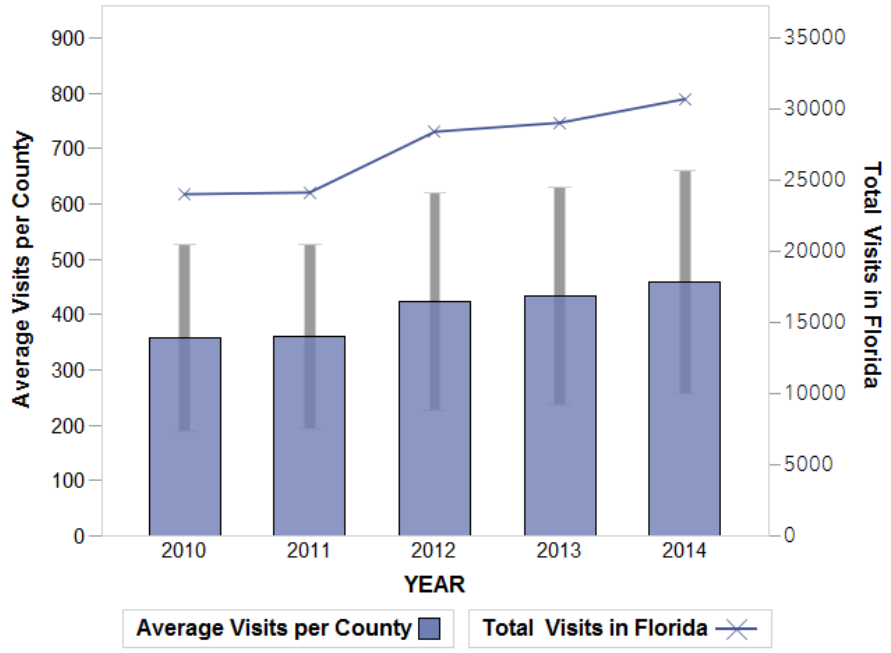


Figure 6 Trend of Asthma ED Visits in Florida for school age children

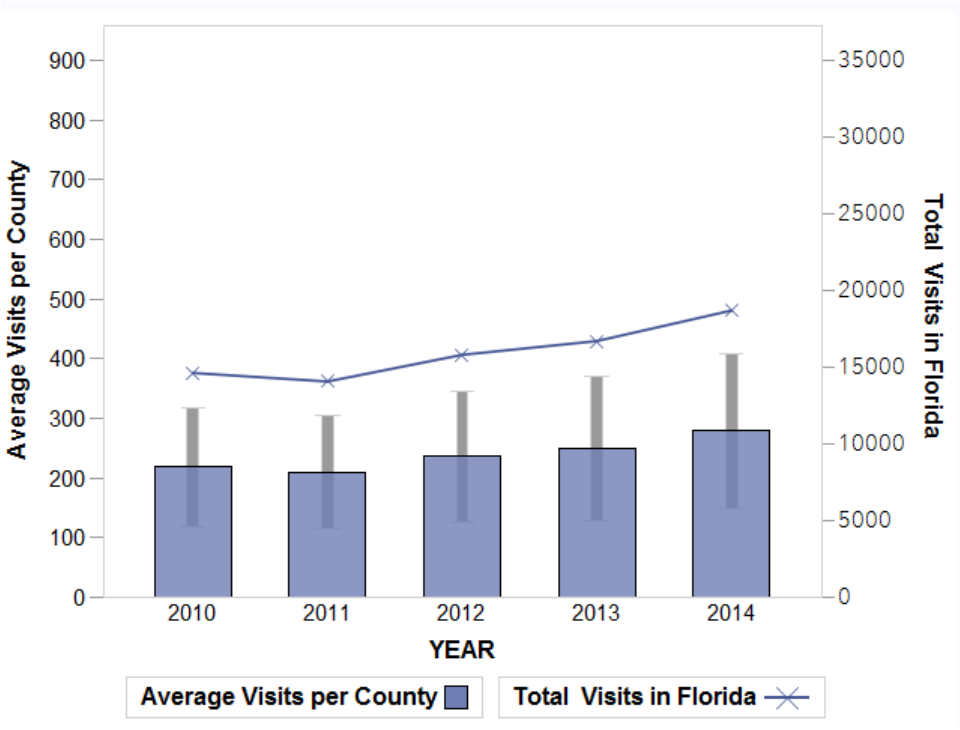


Figure 7 Trend of Asthma Exacerbation ED Visits in Florida for school age children

The prevalence of ED visit for childhood asthma and asthma exacerbation by demographic characteristics and by county of residence are given inTable 5 andTable 6 respectively. Table 5 shows that males have a higher prevalence rate than females for asthma exacerbation and all asthma ED visits. The difference between male and female asthma ED rates is 2.8 per 1,000 for childhood asthma and is 1.9 per 1,000 for childhood exacerbation. The difference between age groups 5-12 and 13-18 is 6.9 per 1,000 for ED visit rates. However, this disparity was lower for exacerbation where the difference between age group 5-12 and 13-18 was 2.8 per 1,000.

There are high disparities in prevalence of asthma and asthma exacerbations among different race/ethnicity groups. The Black not Hispanic group is 3.6 times more likely to have higher prevalence of asthma rates and four times more likely to have higher asthma exacerbations then the White non-Hispanic group. On the other hand, asthma ED visits and asthma exacerbation visits in the Hispanic group are nearly twice that of White non-Hispanics, and half that of Black non-Hispanics.

Asthma and exacerbation ED visit rates were higher for urban counties than rural counties. The ED visit rate for urban counties was 8.02 per 1,000 while for rural counties was 7.15, the difference for exacerbation rates between urban and rural counties was lower, 0.3 cases per 1,000 residents. The ED visit rates for asthma and asthma exacerbations were about two times higher for cold season versus for warm season. Cold season were categorized as months between November and April, while the warm seasons included months between May to October.

Table 5 ED Visit Rates for Childhood by Demographic Characteristics (2010-2012)

	Asthma Visit Rate per 1,000	Asthma Exacerbation Rate per 1,000
Florida	**7.57**	**4.51**
Gender		
Female	6.14	3.56
Male	8.93	5.42
Age Groups		
5-12	10.66	6.31
13-18	3.77	2.29
Race/Ethnicity		
White Not Hispanic	4.35	2.45
Black Not Hispanic	15.79	9.78
Hispanic	7.60	4.51
Other	4.47	259
Location		
Urban	8.02	4.72
Rural	7.15	4.48
Season		
Warm	4.89	2.94
Cold	11.13	6.45

Annual average asthma ED visit rates among ten rural counties, i.e. Madison, Gadsden, Franklin, Liberty, Suwannee, Jackson, Hardee, Calhoun, Hendry, Union, were higher than average ED visits for Florida. The urban counties with the higher annual ED asthma visits than Florida were Polk, Escambia, Marion, Volusia, Miami-Dade, Duval, Broward, Osceola, Orange, and Flagler.

Results in Table 6 show that the counties with higher asthma visits also had higher exacerbation rates. Annual average asthma exacerbation ED visits rates among ten rural counties higher than the average Florida counties were Madison, Gadsden, Hendry, Liberty, Taylor, Jackson, Suwannee, Holmes, Franklin, and Monroe. The urban counties higher than annual ED exacerbation visits were Escambia, Duval, Polk, Osceola, Volusia, Orange, Broward, Miami-Dade, Pinellas, and Marion.

Table 6 Prevalence of ED Visit for Childhood Asthma and Asthma Exacerbation by County of Residence (2010-2012)

County	Asthma Visit Rate per 1000	Asthma Exacerbation Rate per 1000	County	Asthma Visit Rate per 1000	Asthma Exacerbation Rate per 1000
Florida	**7.57**	**4.51**			
Alachua	5.44	3.77	Lee	7.21	4.18
Baker	5.55	2.46	Leon	5.80	4.48
Bay	5.90	2.84	Levy	3.85	2.32
Bradford	6.38	3.95	Liberty	9.89	5.65
Brevard	4.91	2.61	Madison	17.80	15.19
Broward	8.83	5.23	Manatee	5.13	2.58
Calhoun	8.15	2.72	Marion	10.04	4.54
Charlotte	5.21	3.40	Martin	4.42	2.22
Citrus	3.83	2.05	Miami Dade	9.64	5.18
Clay	3.98	2.67	Monroe	6.90	4.60
Collier	5.02	3.58	Nassau	5.91	2.43
Columbia	6.73	3.51	Okaloosa	6.40	4.28
DeSoto	5.73	3.03	Okeechobee	7.03	4.11
Dixie	1.21	0.81	Orange	8.58	5.58
Duval	8.88	6.43	Osceola	8.75	5.97
Escambia	10.30	6.87	Palm Beach	6.29	4.11
Flagler	7.87	2.48	Pasco	5.03	2.71
Franklin	14.50	4.72	Pinellas	7.47	4.70
Gadsden	15.77	11.61	Polk	11.31	6.14
Gilchrist	2.34	1.67	Putnam	6.91	3.76
Glades	5.47	4.10	St. Johns	5.30	3.96
Gulf	6.74	2.41	St. Lucie	4.80	2.90
Hamilton	5.75	4.05	Santa Rosa	5.25	3.37
Hardee	8.33	2.86	Sarasota	3.92	2.16
Hendry	7.99	6.44	Seminole	6.57	3.74
Hernando	4.98	2.86	Sumter	6.63	4.05
Highlands	6.40	3.27	Suwannee	9.12	5.00
Hillsborough	6.67	3.72	Taylor	7.02	5.56
Holmes	7.23	4.87	Union	7.83	4.45
Indian River	5.85	2.60	Volusia	9.99	5.69
Jackson	8.67	5.30	Wakulla	2.89	2.16
Jefferson	4.22	2.81	Walton	5.83	2.92
Lafayette	2.49	2.13	Washington	6.85	3.61
Lake	5.53	3.87			

The Tables 7 and 8 give the baseline prevalence rates for state of Florida stratified by race ethnicity for age groups and gender. Baseline rates for this study are the average rates over three years 2010-2012. The asthma ED visits prevalence rate is 10.66 per 1,000 for age group 5-12 , while it is 3.77 per 1,000 for age group 13-18. The asthma exacerbation ED prevalence rates which is for more chronic cases is lower than asthma ED visits and is 6.31 for age group 5-12 , while it is 2.29 per 1,000 for age group 13-18. The prevalence rates of asthma ED visits for age group 5-12 years was almost three times higher than age group 13-18. The prevalence rate for asthma exacerbation ED visits was also almost three time higher for the 5-12 age group than the 13-18 age group. The overall rates are adjusted over ethnicity and gender.

Table 7 Prevalence Rate per 1,000 for Childhood Asthma ED visit Stratified by Race/Ethnicity for Age and Gender (2010-2012)

	Race/Ethnicity				**All Races/ Ethnicity**
	White not Hispanic	**Black not Hispanic**	**Hispanic**	**Other**	
Age group	*Per 1,000*	*Per 1,000*	*Per 1,000*	*Per 1,000*	*Per 1,000*
5-12	5.82	22.76	10.92	6.02	10.66
13-18	2.61	7.38	3.34	2.25	3.77
Gender					
Female	3.72	12.46	6.11	3.31	6.14
Male	4.95	19.01	9.01	5.62	8.93
Overall	**4.35**	**15.79**	**7.60**	**4.47**	**7.57**

The asthma ED visits prevalence rates is 6.14 per 1,000 for females of all ages and race/ethnicity, while it is 8.93 per 1,000 for males. The asthma exacerbation ED prevalence rates which is for more chronic cases is lower than asthma ED visits and is 3.56 for females, while it is 5.42 per 1,000 for males. The prevalence rates of asthma ED visits for males was 1.4 higher

than females. The prevalence rate for asthma exacerbation ED visits for males was 1.5 times higher than females. The overall rates are adjusted over ethnicity and age groups.

The results in Table 7 show that for asthma ED visits the prevalence for the Black not Hispanic persons in the 5-12 age group was 22.76 per 1,000, while for Hispanic children it was 10.92 and for White not Hispanic was only 5.82 per 1,000 children in this age group. For age group 13-18 for Black not Hispanic the childhood prevalence was 7.38 per 1,000 , while for Hispanic children was the prevalence was 3.34 and for White not Hispanic the childhood prevalence was only 2.61 per 1,000 children in this age group. For both the age groups Black not Hispanics had the highest prevalence rates for ED visit for asthma of any race/ethnicity. In the younger age group this was almost 4 times higher than White not Hispanic and two times higher than Hispanic. Also, in this age group prevalence rated for asthma among Whites on Hispanic was not very different other from not Hispanic races. For the older age group this diversity was little less pronounced, Black not Hispanic have a prevalence rate only three times higher than White non-Hispanics. In this age group the prevalence rates among White non-Hispanic, Hispanic and other races were very similar.

Gender differences are observed across all race ethnicity groups, the prevalence rates were higher for males than females for asthma ED visits. Black not Hispanic males were 1.5 times more likely to visit the ED for asthma than females, the prevalence rates for males Black not Hispanic was 19.01 per 1,000. Other race ethnicity females had the lowest prevalence rate for asthma ED visits, 3.31 per 1,000 (Table 7).

Table 8 Prevalence Rate per 1,000 for Childhood Asthma Exacerbation ED visit Stratified by Race/Ethnicity for Age and Gender (2010-2012)

	Race/Ethnicity				All Races/ Ethnicity
	White not Hispanic	**Black not Hispanic**	**Hispanic**	**Other**	
Age group	*Per 1,000*	*Per 1,000*	*Per 1,000*	*Per 1,000*	*Per 1,000*
5-12	3.25	14.06	6.40	3.42	6.31
13-18	1.50	4.62	2.08	1.40	2.29
Gender					
Female	2.04	7.52	3.53	1.85	3.56
Male	2.84	11.97	5.43	3.32	5.42
Overall	**2.45**	**9.78**	**4.51**	**2.59**	**4.51**

The results in Table 8 show that for asthma exacerbation ED visits the prevalence for Black not Hispanic for the 5-12 age group was 14.06 per 1,000, while for Hispanic children prevalence was 6.40 and for White not Hispanic 3.25 per 1,000 children. For age group 13-18 Black not Hispanic prevalence was 4.62 per 1,000 , while for Hispanic children prevalence was 2.08 and for White not Hispanic prevalence was only 1.5 per 1,000. For both the age groups Black not Hispanics had the highest prevalence rates for ED visit for asthma than any other race ethnicity. In the younger age group this was almost 4 times higher than White not Hispanic and two times higher than Hispanic. These results are similar to asthma ED visits in Table 7. Also, in the younger age group prevalence rates for exacerbation among Whites not Hispanic was not very different other non-Hispanic races. For the older age group this diversity was a little less pronounced, Black not Hispanics have a prevalence rate only three times higher than White not Hispanics. In this age group the prevalence rates among White not Hispanic, Hispanic and other races were very similar. This ethnicity difference is similar to that seen for asthma ED visits.

Gender differences are seen across all race ethnicity groups, the prevalence rates were higher for males than females for ED visits. Black not Hispanic males were 1.5 times more likely to visit ED for asthma than females, the prevalence rates for males Black not Hispanic was 11.97 per 1,000. Other race ethnicity females had the least prevalence rate for asthma ED visits, 1.85 per 1,000.

Table 9 and Table 10 give the baseline prevalence rates for the state of Florida stratified by race ethnicity for age groups and gender. For childhood asthma and asthma exacerbation ED visits previous analyses have shown that the younger age group has higher prevalence rates. Within gender the age group 5-12 has 3.5 times higher ED visits for asthma and exacerbation than age group 13-18.

Table 9 Prevalence Rate per 1,000 for Childhood Asthma ED visit by Age and Gender (2010-2012)

	Gender		
	Male	**Female**	**All**
Age group	*Per 1,000*	*Per 1,000*	*Per 1,000*
5-12	13.24	7.97	10.66
13-18	3.67	3.87	3.77
All	8.93	6.14	7.57

Table 10 Prevalence Rate per 1,000 for Childhood Asthma Exacerbation ED Visits by Age and Gender (2010-2012)

	Gender		
	Male	**Female**	**All**
Age group	*Per 1,000*	*Per 1,000*	*Per 1,000*
5-12	7.99	4.56	6.31
13-18	2.27	2.31	2.29
All	5.42	3.56	4.51

The results in above Tables 5-10 lead to inference that the prevalence rates vary between Florida counties and for race ethnicity, gender and age group. The prevalence rates that are to be input into BenMAP should be adjusted for race ethnicity, gender and age group and Florida counties. One option would be to stratify by race ethnicity, gender, age group and Florida counties, the stratification at this minuscule level will add extreme burden on the analysis in BenMAP software. It will decrease efficiency of the software and increase analysis run time using the software. Another option would be to adjust for race ethnicity, gender and age group at county level, i.e. Table 6. These prevalence rates were uploaded into BenMAP for baseline prevalence. The results will be used to predict overall county level change in Asthma or Asthma exacerbation when there is a change in $PM_{2.5}$ after adjusting for race/ethnicity, gender and age group disparities. Using these rates will be helpful for Health Impact Assessments at county level to assess impact of $PM_{2.5}$ on asthma and asthma exacerbation.

Another option is to use the stratified rates for race/ethnicity, age group and gender for Florida adjusted for county. These prevalence rates are given in Table 11 and Table 12. These rates help to predict change in asthma or asthma exacerbation when there is a change in $PM_{2.5}$ after adjusting for county. It will be using adjusted Florida rates across all counties for an age group, gender or race ethnicity. Using these prevalence baseline rates will be helpful for HIAs when planning for a community or group of individuals based on race/ethnicity, age group and gender. These results in Table 11 and Table 12 were uploaded into BenMAP for baseline prevalence to be selected for studying differences based on race/ethnicity, gender and age group diversity.

Table 11 Prevalence Rate per 1,000 for Childhood Asthma ED visit stratified by Race/ethnicity, Age and Gender (2010-2012) for BenMAP input

Age group	Gender	Race/Ethnicity				All Races/ Ethnicity
		White not Hispanic	Black not Hispanic	Hispanic	Other	
		Per 1,000	*Per 1,000*	*Per 1,000*	*Per 1,000*	*Per 1,000*
5-12		5.82	22.76	10.92	6.02	10.66
	Female	4.48	16.89	8.12	3.93	7.97
	Male	7.09	28.47	13.60	8.13	13.24
13-18		2.61	7.38	3.34	2.25	3.77
	Female	2.81	7.11	3.50	2.41	3.87
	Male	2.42	7.64	3.19	2.08	3.67
All age groups						
	Female	3.72	12.46	6.11	3.31	6.14
	Male	4.95	19.01	9.01	5.62	8.93
Overall		**4.35**	**15.79**	**7.60**	**4.47**	**7.57**

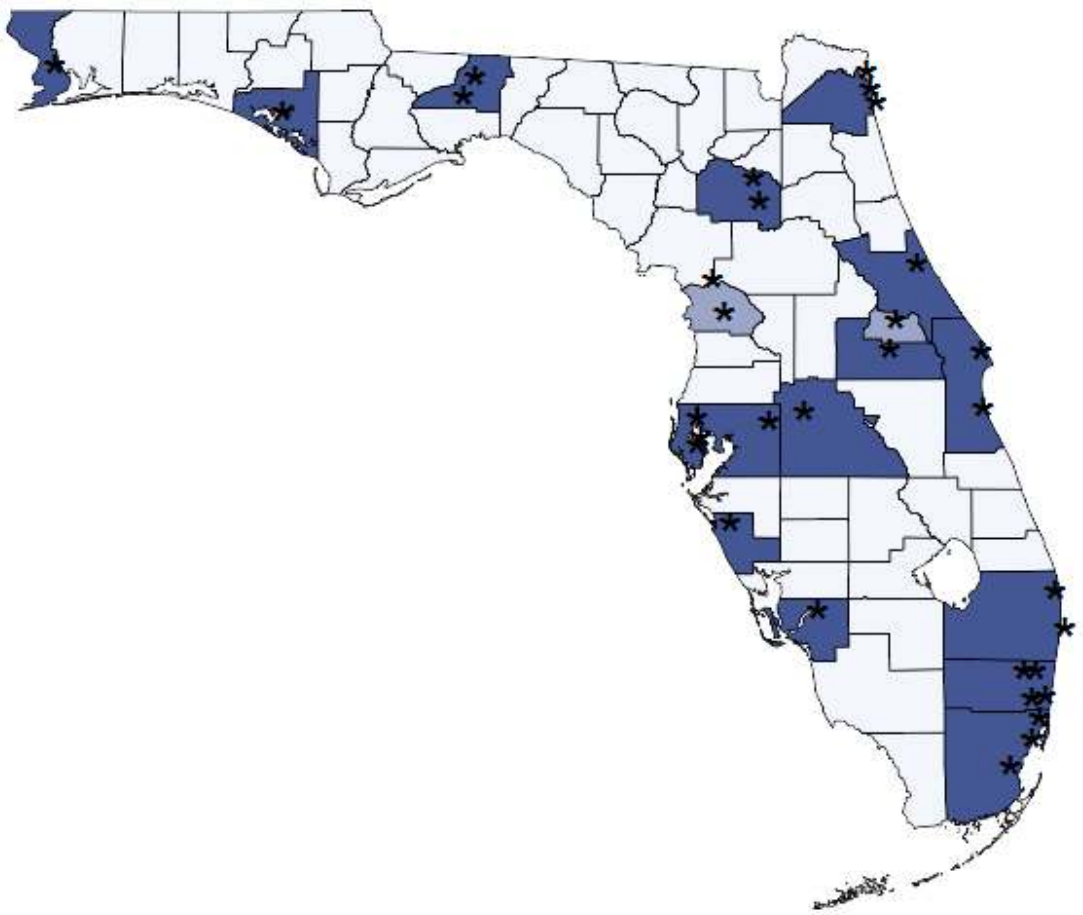
Table 12 Prevalence Rate per 1,000 for Childhood Asthma Exacerbation ED visit stratified by Race/ethnicity, Age and Gender (2010-2012) for BenMAP input

Age group	Gender	Race/Ethnicity				All Races/ Ethnicity
		White not Hispanic	Black not Hispanic	Hispanic	Other	
		Per 1,000	*Per 1,000*	*Per 1,000*	*Per 1,000*	*Per 1,000*
5-12		3.25	14.06	6.40	3.42	6.31
	Female	2.41	10.14	4.57	2.15	4.56
	Male	4.05	17.86	8.15	4.69	7.99
13-18		1.50	4.62	2.08	1.40	2.29
	Female	1.60	4.35	2.18	1.42	2.31
	Male	1.41	4.89	1.98	1.38	2.27
All age groups						
	Female	2.04	7.52	3.53	1.85	3.56
	Male	2.84	11.97	5.43	3.32	5.42
Overall		**2.45**	**9.78**	**4.51**	**2.59**	**4.51**

4.3 Aim Three: Temporal and spatial patterns of $PM_{2.5}$ in Florida

To evaluate the temporal and spatial patterns of $PM_{2.5}$ in Florida and incorporate for Florida into BenMAP for HIA estimations.

There are 67 counties in Florida but only 17 counties have $PM_{2.5}$ monitoring system set up by Florida Department of Environmental Protection (FDEP) using 24 monitor locations. The counties with $PM_{2.5}$ monitors are shown in Figure 8.



** Monitor location by latitude and longitude were present in data extracted from EPA (AQS, 2015)*

Figure 8 $PM_{2.5}$ Monitor Locations* in Florida during 2010-2014

The monthly trend of $PM_{2.5}$ averaged over 2010 to 2012 by county and for Florida is given in Figure 9. Table 13 gives the monthly concentration of daily $PM_{2.5}$ in Florida averaged across all the counties. The results show that $PM_{2.5}$ concentration varies during the year, the higher concentrations are observed in months of March to July.

Table 13 Monthly Concentration of $PM_{2.5}$ ($\mu g/m^3$) in Florida

	Month	N	MIN	MAX	MEAN	STDDEV
Florida	January	1504	1.6	53.3	8.6	0.85
Florida	February	1380	2.3	38.7	8.9	0.86
Florida	March	1511	2.1	43.0	9.0	1.01
Florida	April	1459	0.9	66.3	9.2	1.73
Florida	May	1494	0.9	35.9	9.6	0.49
Florida	June	1427	0.5	94.4	9.9	2.72
Florida	July	1472	0.1	34.2	9.5	0.97
Florida	August	1480	1.7	62.0	8.7	1.31
Florida	September	1409	0.4	43.4	7.6	1.21
Florida	October	1476	0.5	35.2	8.3	0.77
Florida	November	1427	0	20.2	7.7	0.44
Florida	December	1500	0.8	22.7	7.7	0.42

Figure 9 shows that some counties show a very clear pattern of seasonal trend. The counties which show a clear seasonal pattern are Alachua, Bay, Brevard, Broward, Duval, Escambia, Leon, Miami-Dade, Palm Beach. Escambia and Leon have a very high $PM_{2.5}$ concentration as compared to the other counties. Escambia has high $PM_{2.5}$ during May to September while Leon has high $PM_{2.5}$ during February to May. Brevard, Broward, Lee, Miami-Dade, Sarasota, Palm Beach, and Lee show a lowest concentration in the month of September.

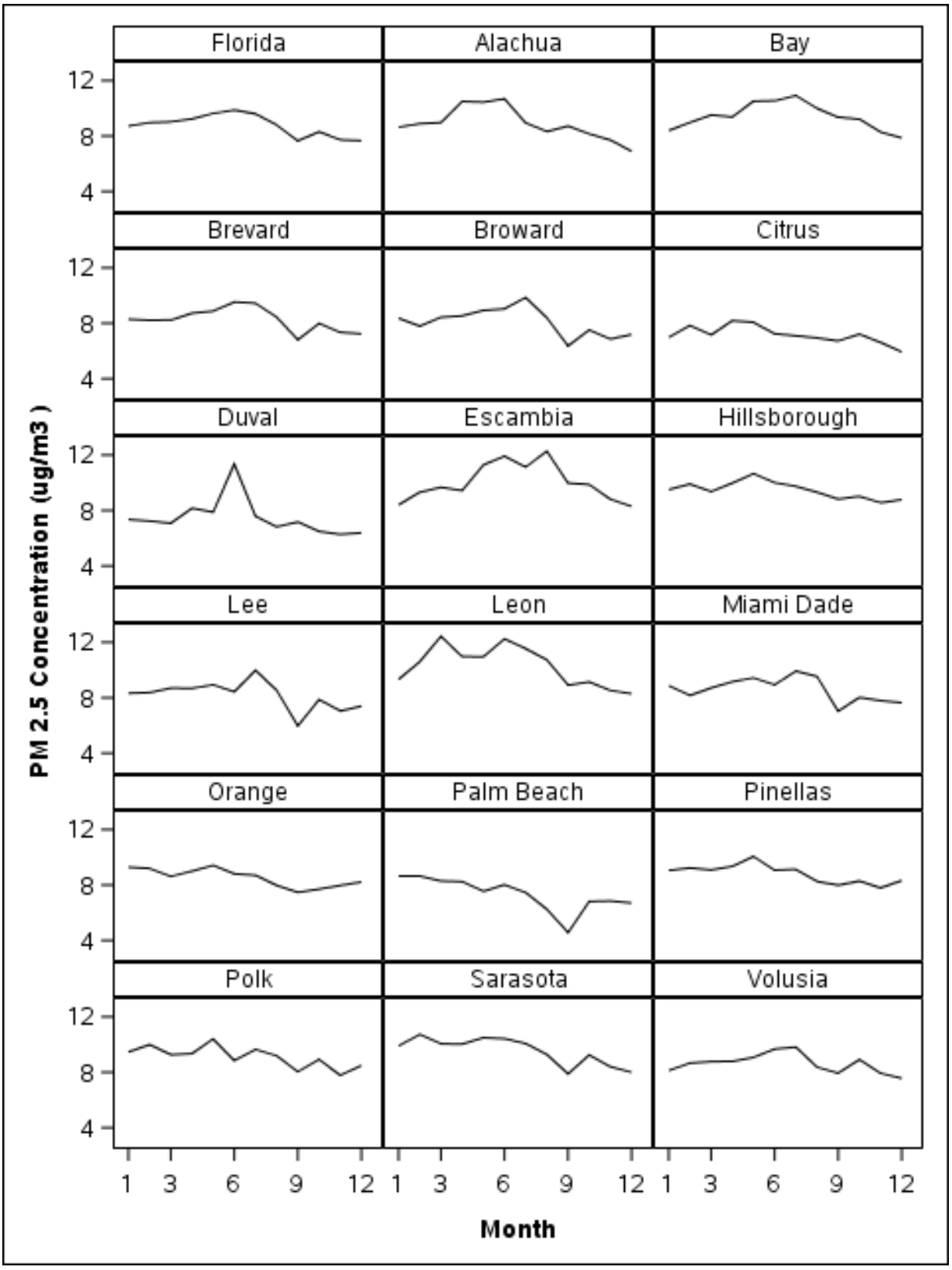


Figure 9 Monthly Average of $PM_{2.5}$ Concentration ($\mu g/m^3$) by County in Florida

Time series analysis by county or Panel Analysis was further conducted to understand the temporal and spatial trends of $PM_{2.5}$ in Florida. Figure 10 gives the results of time series analysis for all seventeen counties and for Florida for January 2010 to Dec 2012. The top panel gives the regression time series analysis for Florida across all counties. The analysis shows that on average the PM2.5 concentration decreased by 1.32 $\mu g/m^3$, the highest decrease was for Leon county by 3.53 $\mu g/m^3$ from January 01, 2010 to December 31, 2012. Decrease in PM2.5 was seen in all the Florida counties except for Miami- Dade which seems to increase by 0.078 $\mu g/m^3$, however this increase was not statistically significant. Other counties where the decrease in $PM_{2.5}$ was not statistically significant were Brevard, Palm Beach, Volusia.

The weather condition, i.e. temperature and precipitation was also studied at county level to see temporal patterns and any correlation with $PM_{2.5}$ concentrations in a county. The results are given in Figure 11 for Florida. The results showed a lot of variance in the three years studied in this analysis. The concentration of $PM_{2.5}$ was highest in June 2011 and has somewhat decreased from January 2010 to December 2012. There are several spikes in the concentration pattern but there is no definite pattern. The spikes are all through but there are higher spikes during April to July every year, the higher values are not that prominent during rest of the year. This could be due to variability in the data at county level and different counties experiencing higher concentration spikes at different times as a result Florida level.

Figure 11 also shows that temperature has a very clear pattern with highest temperatures in June, July and August. Mean daily precipitation also shows a pattern of higher rainfall from June to October. The mounds are not very high because the metric considered in the study is daily precipitation across Florida adjusted by county. If the metric of measurement was

total precipitation these mounds would be higher and more pronounced. Figure 11 shows that there is hardly any day during months of June to October which doesn't have some rain in Florida. Since higher temperatures and higher precipitations are observed during the same months of the year, these will be used to control for confounding when calculating association between $PM_{2.5}$ and asthma and asthma exacerbation ED rates.

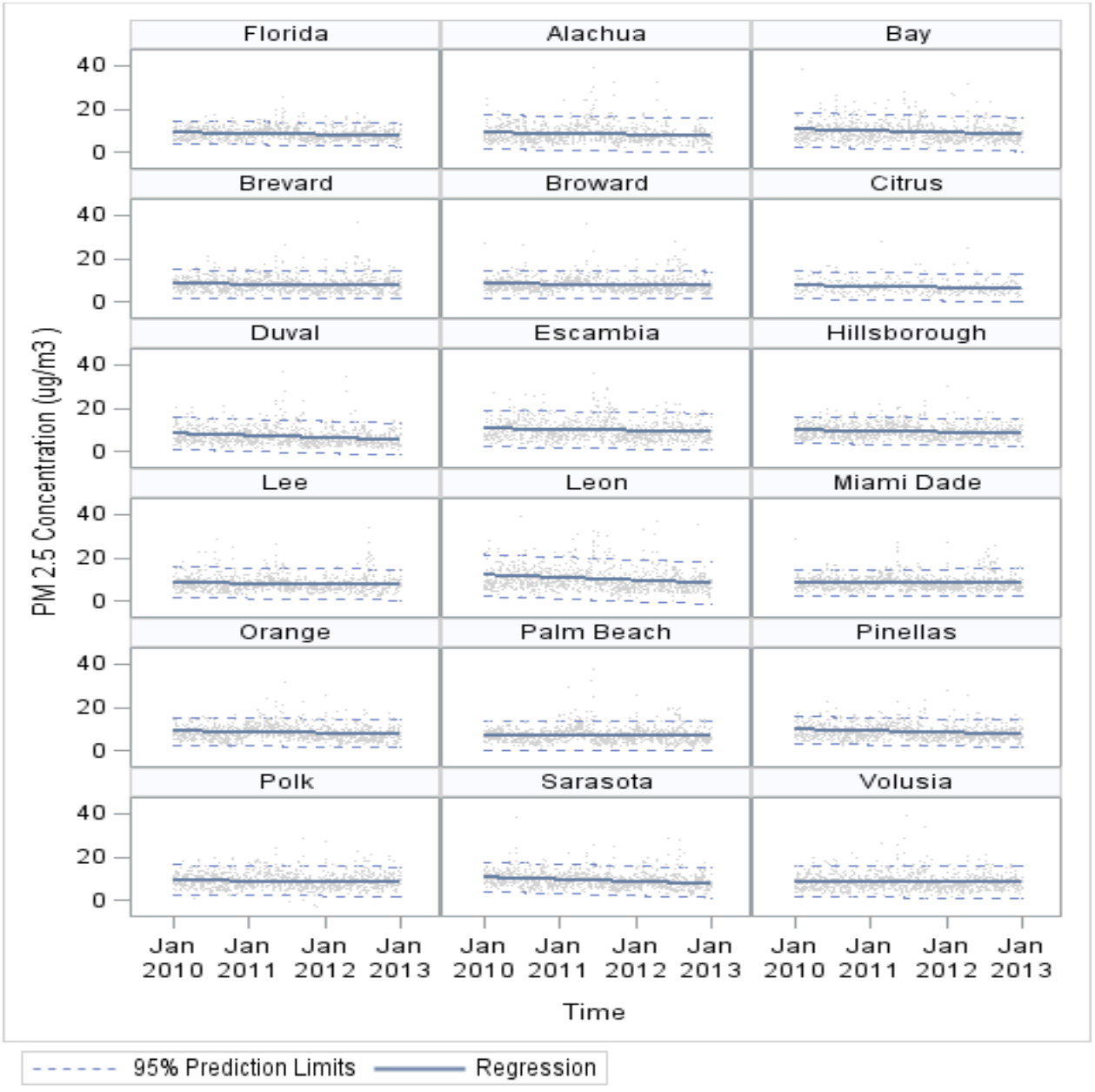


Figure 10 Time Series Analysis of $PM_{2.5}$ Concentration ($\mu g/m^3$) by County in Florida

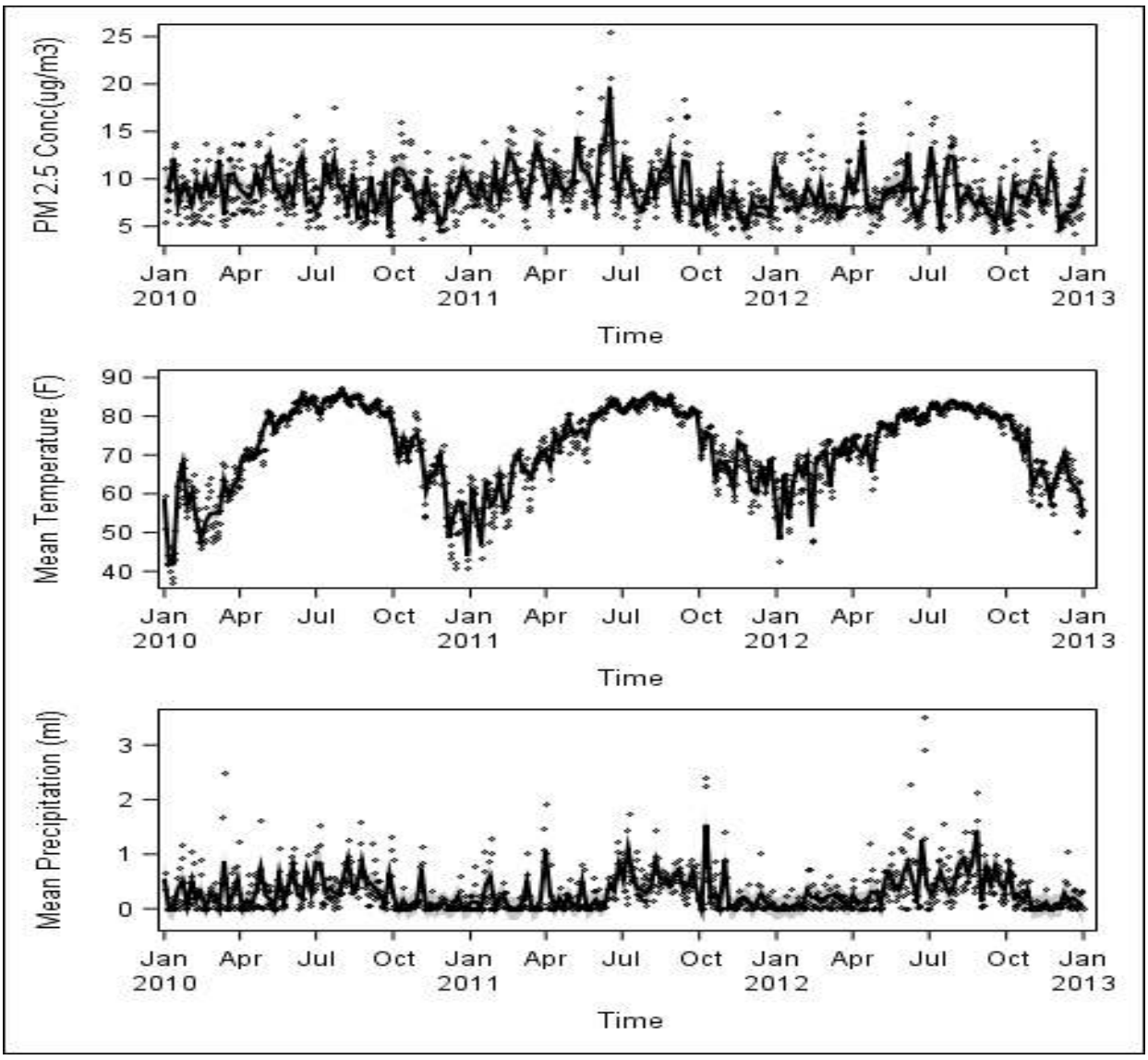


Figure 11 Time Series Analysis of $PM_{2.5}$ Concentration ($\mu g/m^3$) , Temperature (in ^{O} F) and Precipitation (in ml) in Florida

All the seventeen counties are urban counties and will be chosen for this analysis. The total population density and density of children under the age of 18 for the seventeen counties is given Table 14, all the seventeen counties ranked in the top 50th percentile of all the counties in Florida. The top five counties were Pinellas, Broward, Seminole, Orange and Hillsborough.

Table 14 Population Density By County For Children Under Of The Age Of 18 In Florida

	Area in square mile	Percentage of Population under Age<18	Population under Age<18	Population under Age<18 per square mile	Rank
Florida	53,625	21.3	4,200,091	72	
Alachua	875	17.9	44,285	51	25
Bay	758	22.0	37,076	49	26
Brevard	1,016	19.8	107,686	106	13
Broward	1,210	22.4	391,349	323	2
Citrus	582	15.9	22,394	38	31
Clay	604	26.3	50,170	83	17
Duval	762	23.5	203,514	267	7
Escambia	656	21.6	64,154	98	14
Hillsborough	1,020	23.9	294,208	288	5
Lee	785	19.5	120,869	154	8
Leon	667	19.6	53,973	81	18
Miami-Dade	1,898	21.9	545,728	288	6
Orange	903	23.6	270,147	299	4
Palm Beach	1,970	20.4	268,884	137	9
Pinellas	274	17.8	162,888	595	1
Polk	1,798	23.5	141,736	79	19
Sarasota	556	15.7	59,735	107	12
Seminole	309	23.0	97,181	314	3
Volusia	1,101	18.9	93,273	85	16

4.4 Aim Four: Concentration Response Functions for HIA

To develop county-level CR functions to be used in HIAs performed by local or State agencies.

Local ambient PM2.5 concentrations from 24 monitors in 17 counties was analyzed with count data of asthma ED visits and exacerbation ED visits using a time series Poisson regression to develop odds ratio, also known as CR functions. These CR functions will used in BenMAP for assessing the impact of PM2.5 on asthma and asthma exacerbation in Florida at the county level.

The sample size for each age group during each season used in time series models are given in Table 15. Sample size for time series model refers to total number of days in a season for which daily count data existed. The year was divided into season using inferences from analysis of temperature and precipitation in the previous aim (Figure 6). The warm season was considered to be the months of May to October when the temperature and precipitation was highest across Florida counties. The cold season was considered to be the months of November to April when temperature are lower and precipitation was below annual average for Florida. Table 5 in aim two shows that asthma and asthma exacerbation ED visits differ significantly by season, the ED visit prevalence rates were higher for the cold season than the warm season.

Asthma ED visits

Table 16 presents the ORs and 95% confidence interval for association between asthma Ed visits and $PM_{2.5}$ unadjusted and adjusted for lag, weather, season , gender and ethnicity, stratified for age groups 5-12 and 13-18. The unadjusted OR for age group 5-12 is 1.023 (1.008-

1.039) and the adjusted OR for age group 5-12 is 1.020 (1.09-1.034). The adjusted OR suggests that among 5-12 year olds there was 2% (0.9%-3.4%) average increase of asthma ED visits per 10 $\mu g/m^3$ increase of $PM_{2.5}$.

Table 15 Sample Size for time series models for each age group, in Florida (January 01, 2010-December 31, 2012)

Age Group Season	N
5-12 years	
Warm	552
Cold	544
13-18 years	
Warm	552
Cold	544
All Age Groups	
Warm	552
Cold	544

The unadjusted OR for age group 13-18 is 1.007 (1.001-1.013) and the adjusted OR for age group 13-18 is 1.006 (1.001-1.012). The results of the adjusted OR among 13-18 year olds suggest that there was 0.6% (0.1%-1.2%) average increase of asthma ED visits per 10 $\mu g/m^3$ increase of $PM_{2.5}$. The low association of PM2.5 with 13-18 year olds could be possible as these are ED visit rates which are indicative of severity and asthma management. These rates suggest that asthma in middle school and high school kids is more manageable and probable less exposure to outdoor air pollution and less cases in ED visits.

Table 16 Odds Ratio (OR) and 95% confidence intervals for Asthma ED visits and $PM_{2.5}$ concentrations

	Age group 5-12		Age group 13-18	
	OR	95%CI	OR	95%CI
Unadjusted	1.023	1.008-1.039	1.007	1.001-1.013
Adjusted for *Lag**				
Day One and Day Two	1.018	1.008-1.028	1.005	1.002-1.009
Lag* and Weather				
Day One, Day Two Temperature and Precipitation	1.015	1.007-1.023	1.005	1.001-1.009
Lag*, Weather and Season				
Day One, Day Two Temperature and Precipitation, Season, month, weekend	1.012	1.004-1.020	1.004	1.00-1.008
Lag*, Weather, Season and Gender				
Day One, Day Two Temperature and Precipitation, Season, month, weekend, Gender (Male/ Female)	1.012	1.004-1.020	1.004	1.00-1.008
Lag*, Weather, Season, Gender and Ethnicity				
Day One, Season, month, weekend, Gender (Male/ Female) and Ethnicity	1.020	1.009-1.034	1.006	1.001-1.012

* The lag PM2.5 concentration from previous day or two days before, day one and day two concentrations were added when both lag days were considered in the model

Asthma Exacerbation ED visits

Table 17 presents the ORs and 95% confidence interval for association between asthma exacerbation ED visits and $PM_{2.5}$ unadjusted and adjusted for lag, weather, season, gender and ethnicity, stratified for age groups 5-12 and 13-18. The unadjusted OR for age group 5-12 is 1.017 (1.005-1.028) and the adjusted OR for age group 5-12 is 1.014 (1.004-1.025). The adjusted OR suggests that among 5-12 year olds there was 1.4% (0.4%-2.5%) average increase of exacerbation ED visits per 10 $\mu g/m^3$ increase of $PM_{2.5}$.

The unadjusted OR for age group 13-18 is 1.005 (1.001-1.009) and the adjusted OR for age group 13-18 is 1.004 (1.001-1.008). The results suggest that the adjusted OR among 13-18 year old there was 0.4% (0.1%-0.8%) average increase of exacerbation ED visits per 10 $\mu g/m^3$ increase of $PM_{2.5}$. The low association with $PM_{2.5}$ could be due to the fact that majority of asthma exacerbations are reported to be mostly virus-induced (Skyes 2008). Literature has also shown that role of bacterial infections in exacerbations is also increasing (Sasaki 2015). Another reason for the low $PM_{2.5}$ associations with exacerbation could be due to the strong association of exacerbation to exposure of seasonal allergens (Murray 2004).

Table 17 Odds Ratio (OR) and 95% confidence intervals for Asthma exacerbation ED visits and $PM_{2.5}$ concentrations

	Age group 5-12		Age group 13-18	
	OR	95%CI	OR	95%CI
Unadjusted	1.017	1.005-1.028	1.005	1.001-1.009
Adjusted for *Lag**				
Day One, Day Two	1.014	1.006-1.021	1.004	1.001-1.007
Lag* and Weather				
Day One, Day Two, Temperature and Precipitation	1.010	1.004-1.015	1.004	1.001-1.007
Lag*, Weather and Season				
One Day and Two Day, Temperature and Precipitation, Season, month, weekend	1.007	1.002-1.013	1.004	1.001-1.007
Lag*, Weather, Season and Gender				
Day One, Day Two, Temperature and Precipitation, Season, month, weekend, Gender (Male/ Female)	1.007	1.002-1.013	1.004	1.001-1.007
Lag*, Weather, Season, Gender and Ethnicity				
Day One, Season, month, weekend, Gender (Male/ Female) and Ethnicity	1.014	1.004-1.025	1.004	1.001-1.008

* The lag PM2.5 concentration from previous day or two days before, day one and day two concentrations were added when both lag days were considered in the model

The CR functions or the odds ratios from the peer-reviewed studies, which analyzed the change in childhood asthma associated with change in pollutant concentration were reviewed and are tabulated in Table 18. There were 33 peer reviewed studies published between 2000 and 2015, the outcome for these studies was ED visits or ED and hospital admissions for childhood asthma Several of these studies were from locations outside of the United States, while eighteen studies were from US cities. The study design of nineteen published studies was time series, while ten were cross sectional, one was logistic regression and three were meta-analyses. Most of the studies looked at single pollutant model for PM2.5, while nine studies looked at two or multi-pollutant models. The age group in the studies varied from 0 to 18, while there were seven studies which looked at all ages. These studies did stratify by age groups, groups of 5-11 and <18 years.

Only thirteen out of 33 studies in Table 18 reported that $PM_{2.5}$ was positively associated with pediatric asthma. The ten studies are tabulated in Table 19 and the three meta-analyses are tabulated inTable20. The tables give the odds ratio as measure of association between $PM_{2.5}$ with pediatric asthma ED visits. In Table 19, one study was based in Canada and will not be included in BenMAP. Also, the studies showing association between asthma and $PM_{2.5}$ and published by 2010 have been already evaluated by EPA and will not be entered in BenMAP. In fact, Mar *et. al.*. 2010 study observed association is already present in BenMAP. The rest of the studies except one were part of three meta-analyses listed in Table 20. All the meta-analyses studies were selected to enter in BenMAP.

Table 18 Summary Of Studies Evaluating Association Of Ambient Pollutants On Pediatric Asthma, 2000-2015

Source	Location	Study Design	Sample Size	Age group	Definition of Exposure	Outcome Definition
Lin *et. al.* 2002	Toronto, Canada	Time series and case crossover	7319 patients	6-12	Daily predicted model using 6d period data	Hospital admissions for Asthma
Barnett *et. al.*, 2005	5 cities in Australia and 2 cities in New Zealand	Case crossover	Hospital Admission between 1998-2001	0, 1-4y, 5-14y	Daily PM2.5, PM10, NO2 and SO2	Hospital admissions for Respiratory condition and Asthma using ICD codes
Slaughter *et. al.* 2005*et. al.*	Spokane, Washington	Time Series	2373	All ages	24hr daily average for PM_{10}, $PM_{2.5}$ and PM_1	ED and Hospital Admission for Respiratory Conditions with Asthma defined by ICD-9 code of 493
Lee *et. al.* 2006	Seoul, Korea	Time Series, GAM	8.09 patients per pay	0-15y	Daily 24-h average PM10, SO2, NO2, 8h average CO and O3	Hospital admissions for Asthma, ICD-10 codes J45-J46
Akinbami *et. al.*, 2007	NHIS survey sampled in U.S. MSA's 2001-2004	Cross sectional	34,073 children	3-17y	Rolling 12 month average based on quarterly measures for $PM_{2.5}$ retrieved from EPA's AIR.	Current asthma: yes response to " Has doctor ever told you that your child has asthma?" and " Does your child still have asthma?" Asthma attack : Yes response to " During the past 12 months has your child has an episode of asthma or asthma attack?"

Table 18 Summary Of Studies Evaluating Association Of Ambient Pollutants On Pediatric Asthma, 2000-2015*(continued…)*

Source	Location	Study Design	Sample Size	Age group	Definition of Exposure	Outcome Definition
Ito *et. al.* 2007	New York City	Time series	5-11y-133,141 12-17y- 55,143	All ages	Daily 24-h average	Emergency department visits for asthma using ICD-9 code of 493
Vilineuve *et. al.*, 2007	Edmonton, Canada	Case crossover	2-4 y- 7247 5-14y- 13145	2-14y	24 hour average $PM_{2.5}$	ED visit ICD-9 code of 493
Andersen *et. al.* 2008	Copenhagen, Denmark	Time series	559 in single pollutant model, 318 in two pollutant model	5-18y	Daily 24-h average	Hospital Admissions ICD-9 code 493
Babin *et. al.* 2008	Washington DC Area Medicaid Beneficiaries, 1994-2005	Time series	11 year Medicaid data	5-12 y	Daily concentration PM2.5 and O3	Asthma exacerbations observed in acute care visits
Halonen *et. al.* 2008	Helsinki, Finland	Time series	4807 cases	0-15y	Fixed Monitoring site	Emergency department visits for asthma using ICD-9 code of 493
			1972 cases	0-15y	Daily 24-h average, during warm season	Emergency department visits for asthma using ICD-9 code of 493
Jalaludin *et. al.* 2008	Sydney, Australia	Time-Stratified case crossover	317,724 visits to Asthma ED visits	1-14	Daily 24-h average	Emergency department visits for asthma using ICD-9 code of 493
Stieb *et. al.* 2009	Seven Canadian Cities	Time series Analysis	400, 000 ED visits; 83563 Asthma cases	All ages	24hr daily average for $PM_{2.5}$, PM_{10}, , CO, NO_2, SO_2	ED visits for cardiac and respiratory, ICD-9 or 10 code of 493, J45 for asthma

Table 18 Summary Of Studies Evaluating Association Of Ambient Pollutants On Pediatric Asthma, 2000-2015*(continued…)*

Source	Location	Study Design	Sample Size	Age group	Definition of Exposure	Outcome Definition
Andersen *et. al.* 2010	105 ISAAC centers in 51 countries in arcoss world	Cross sectional	322529 with range of 1056–5521 in each center	13-14y	Annual concentration of PM10 at city level	Prevalence of self-reported asthma
Mar *et. al.* 2010	Tacoma, Washington	n/a		All ages	Daily 24-h average	ED visits Asthma defined by ICD-9 code of 493
Meng *et. al.* 2010	San Joaquin Valley, California	Logistic regression	1502 respondents of CHIS 2001	All ages	24-h daily average	Self -reported physician diagnosed asthma who resided in SJV, California
Silverman *et. al.* 2010	New York, USA	Time series	<6y -15,185 6-18 -10,322	<6, 6-18y	24-h daily average	Hospital admission for asthma ICD-9 code of 493
Strickland *et. al.* 2010	Atlanta, Georgia	Time series	91, 386 cases seen in ED departments for Asthma or Wheeze	5-17	24 hour average PM_{10}, $PM_{2.5}$ and $PM_{2.5}$ components	ED visit as reported in ** system With ICD-9 code of 493 for asthma or 786.07 for wheeze
Li *et. al.* 2011	Detroit, USA	Time series and case crossover	7063 ED and HA visits	2-18y	Daily 24-h average	Hospital admissions and ED visits based on ICD-9
Glad *et. al.* 2012	Pittsburg, Pennsylvania	Case crossover	978	0-17y	Daily 24h $PM_{2.5}$	Emergency department visits for asthma using ICD-9 code of 493
Winquist *et. al.* 2012	St. Louis	Time series Analysis	0-1yr 12,236 ED visits 1-18y 49.978 ED visits	01-8 y	Daily 24 hour average $PM_{2.5}$	ED and HA visit ICD-9 code of 493
Gleason *et. al.* 2014	New Jersey	Case crossover	21,854 cases	3-17y	12x12 grid multiscale air quality model	Emergency department visits for asthma using ICD-9 code of 493

Table 18 Summary Of Studies Evaluating Association Of Ambient Pollutants On Pediatric Asthma, 2000-2015*(continued…)*

Source	Location	Study Design	Sample Size	Age group	Definition of Exposure	Outcome Definition
Delfino *et. al.* 2014	California	Case crossover	11390 cases	0-18 y	Daily 24h $PM_{2.5}$	Emergency department and Hospital visits for asthma using ICD-9 code of 493
Strickland *et. al.* 2014	Atlanta, Georgia	Time series Analysis	109,758 children seen in ED	2-16	24 hour average PM10, PM2.5 and PM2.5 components	With ICD-9 code of 493 for asthma or 786.07 for wheeze
Wendt *et. al.* 2014	Boston, Massachusetts	Case crossover		0-17 yr	Daily 24 hour average $PM_{2.5}$	HA visit ICD-9 code of 493
Gleason *et. al.* 2015	Newark, New York	Time series, Case crossover	3,675 cases	3-17y	12x12 grid multiscale air quality model	Emergency department visits for asthma using ICD-9 code of 493
Ostro *et. al.* 2015	8 metropolitan cities California	Case crossover	43,094 Asthma visits in ED	All ages	24-h daily average	ED visits Asthma defined by ICD-9 code of 493
Zheng *et. al.* 2015	n/a	Meta-analysis	n/a	all	10µg of $PM_{2.5}$	Asthma emergency room and hospital admission
Alharti *et. al.*, 2016	Dallas, Texas	Time series	Mean daily counts 0-4 16.91 5-18y 25.75`	0-18	Daily 24-h average using monitor data	ED visit by ICD-9 code of 493 and exacerbation
Byers *et. al.* 2016	Indianapolis	Time series	33,981 cases	5-17y	Daily 24h $PM_{2.5\ h}$ with population weighting across monitors	Emergency department for asthma using ICD-9 code of 493

Table 18 Summary Of Studies Evaluating Association Of Ambient Pollutants On Pediatric Asthma, 2000-2015*(continued…)*

Source	Location	Study Design	Sample Size	Age group	Definition of Exposure	Outcome Definition
Ding *et. al.* 2016	China	Time stratified case crossover	2507 hospital visits in 2013	0-18y, stratified by 6-18	Daily 24h $PM_{2.5}$	HA visits
Fan *et. al.* 2016	n/a	Meta analysis	n/a	all	10µg of $PM_{2.5}$	Asthma emergency department visit
Lim *et. al.* 2016	n/a	Meta analysis	n/a	Pediatric	10 ug/m3 increase of $PM_{2.5}$	Hospital admissions and ED visits
Weichenthal *et. al.* 2016	Ontario, Canada	Case crossover	127,386	<9y	Daily 24 hour average $PM_{2.5}$	ED visit ICD-10 code of J45

Table 19 Measure of Association for $PM_{2.5}$ On Pediatric Asthma ED Visits, 2000-2015

Source	Study Year/ Location	Lag	Season	Age	Measure of Association	95% Confidence Interval
Babin *et. al.* 2008	1994-2005/ Washington DC	n/a	All	5-12	OR=1.05	1.01-1.09
Steib *et. al.* 2009	Early1990-2000/ Canadian Cities	n/a	Warm	All	OR=1.093	1.062-1.123
Mar *et. al.* 2010	1999-2002/ Tacoma, Washington	2	All	All	OR=1.058	1.014-1.102
	Tacoma, Washington	3	All	All	OR=1.043	1.000-1.087
Meng *et. al.* 2010	2001-2002/ San Joaquin Valley, California	n/a	All	1-17	OR=1.480	0.620-3.500
Li *et. al.* 2011	Jan01,2004-Dec31,2006/ Detroit, Michigan	3	All	2-18	OR=1.032	1.007-1.057
Glad *et. al.* 2012	/ Pittsburg, PA	1	All	All	OR=1.036	1.001-1.073
Winquist *et. al.* 2012	/ St. Lois	n/a	All	2-18	OR=1.054	1.023-1.086
Strickland *et. al.* 2014	2002-2010/ Atlanta, Georgia	n/a	All	5-17	OR=1.022	1.002-1.042
	2002-2010/ Atlanta, Georgia	n/a	Warm	5-17	OR=1.047	1.017-1.076
Gleason *et. al.* 2014	Jan01,2004- Dec 31, 2007/ New Jersey	0	Warm	3-17	OR=1.035	1.023-1.047
	Jan01,2004- Dec 31, 2007/ New Jersey	1	Warm	3-17	OR=1.012	1.000-1.024
Ostro *et. al.* 2016	2005-2009/ California	2	All	All	OR=1.2	0.0-2.4

Table 20 Summary of Meta Analysis Studies Published Between 2000-2015

Author	Number of Studies / Year of Studies	List Studies and Published year	Age	Measure of Association	95% Confidence Interval
Fan *et. al.* 2015	16 Published Between 2004-2015	Peel *et. al.*.2005; Slaughter *et. al.*. 2005; Babin *et. al.*. 2008; Halonen *et. al.*. 2008; Paulu and Smith 2008; Stieb *et. al.*. 2009; Mar and Koenig 2009; Mar *et. al.*.2010; Meng *et. al.*. 2010; Strickland *et. al.*. 2010; ; Li *et. al.*. 2011; Winquist *et. al.*. 2012; Chen *et. al.*. 2013; Delfino *et. al.*. 2014; Glad *et. al.*. 2012; Gleason *et. al.*. 2014;	All	OR=1.015	1.012-1.017
			<18	OR=1.036	1.017-1.044
Lim *et. al.* 2016	33 studies Published between 1999-2016	Norris *et. al.* 1999, Lin *et. al.* 2002, Lee *et. al.* 2006; Ko *et. al.* 2006; Villneneuve et. al. 2007; Andersen *et. al.*. 2003; Halonen *et. al.* 2008; Jalaludin 2008; Tacer *et. al.* 2008; Halonen *et. al.* 2010; Silverman *et. al.*. 2010 ; Strickland *et. al.* 2010; Li *et. al.* 2011; Glad *et. al.* 2012; Iskinder *et. al.* 2012; Winquist *et. al.*. 2012; Delfine *et. al.* 2014; Hua *et. al.* 2014; Strickland *et. al.* 2014; Wendt *et. al.* 2014	5-18	OR=1.027	1.011-1.043
			0-18	OR=1.048	1.028-1.067

Table 20 Summary of Meta Analysis Studies Published Between 2000-2015 ***(continued)***

Author	Number of Studies / Year of Studies	List Studies and Published year	Age	Measure of Association	95% Confidence Interval
Zeng *et. al.* 2016	32 studies Published between 1999-2015	Sheppard *et. al.* 1999; Lin *et. al.* 2002; Barnet *et. al.* 2005; Sluaghter *et. al.*. 2005; Lee *et. al.* 2006, Chardon *et. al.* 2007; Chimonas *et. al.* 2007; Ito *et. al.* 2007; Ko *et. al.* 2007; Andersen *et. al.* 2008; Babin *et. al.* 2008; Halonen *et. al.* 2008; Jalaludin *et. al.* 2008; Paulu *et. al.* 2008; Szyszkowicz *et. al.* 2008; Stieb *et. al.* 2009; Lavinge *et. al.* 2010; Lim 2010; Mar *et. al.* 2010; Strickland *et. al.* 2010; Li *et. al.* 2011; Iskandar *et. al.* 2012; Kim *et. al.* 2012; Santus *et. al.* 2012; Silverman *et. al.* 2012; Evans *et. al.* 2013; Malig *et. al.* 2013; Yamazaki *et. al.* 2013; Cheng *et. al.* 2014;Raun *et. al.* 2014;Gleason *et. al.* 2014;Hua *et. al.* 2014;	5-18	OR=1.023	1.015-1.031

CR functions for PM2.5 and Pediatric Asthma in BenMAP

The odds ratios and standard errors obtained for asthma (Table 16) and asthma exacerbation (Table 17) visits to ED due to visits per 10 $\mu g/m^3$ of PM2.5 after controlling for lag, weather, season, gender and ethnicity, were be loaded in BenMAP. The odds ratios from the three meta-analyses (Table 20) and Ostro (2016) were also entered in BenMAP for estimating health benefits. The odds ratio and standard deviations were converted to beta values to upload into BenMAP.

The beta values and standard error for the studies to be uploaded in BenMAP are given in Table 21. The different CR functions were combined in BenMAP by pooling method using random effects models. The random effects model assigns each study a weight based on two factors; the spread of estimates reported by that study (i.e. the variance), and how much that spread of estimates differs from the other studies. The random effects model helps to calculate the weighted average across the studies, using the weights calculated from the variance and overlap of the studies. These beta values were used in conjunction with preloaded beta values for asthma exacerbation in BenMAP to get a joint effect to be used in Aim 5 of this study. The goal of aim five is to **e**stimate age and gender specific asthma rates in children due to change in $PM_{2.5}$ concentrations at county level using BenMAP.

Table 21 CR-Functions for Input in BenMAP

Effect	Author	Study Year	Study Location	Age	Beta	Std. Err	Form
Asthma, Emergency Room Visit	Current Study	2010-2012	Florida all counties	5-12	0.002	0.0002	Log-linear
Asthma, Emergency Room Visit	Current Study	2010-2012	Florida all counties	13-18	0.0006	0.0023	Log-linear
Asthma Exacerbation, Emergency Room Visit	Current Study	2010-2012	Florida all counties	5-12	0.0014	0.0007	Log-linear
Asthma Exacerbation, Emergency Room Visit	Current Study	2010-2012	Florida all counties	13-18	0.0004	0.0098	Log-linear
Asthma, Emergency Room Visit	Zeng *et. al.* 2016	1999-2015	Meta-analysis	5-18	0.0023	0.0017	Log-linear
Asthma, Emergency Room Visit	Lim *et. al.* 2016	1999-2016	Meta-analysis	5-18	0.0027	0.0035	Log-linear
Asthma, Emergency Room Visit	Fan *et. al.* 2015	2004-2015	Meta-analysis	0-18	0.0035	0.0029	Log-linear
Asthma, Emergency Room Visit	Ostro *et. al.* 2016	2005-2009	California	0-99	0.0182	0.3521	Log-linear

4.5 Aim Five : Age And Gender Specific Asthma Rates In Children At County Level

To estimate age and gender specific asthma rates in children due to changes in $PM_{2.5}$ concentrations at county level.

Figure 12 displays the $PM_{2.5}$ county level data for 2008 in BenMAP. This is the most recent year for pollutant input present in BenMAP. Using 2008 data already present in BenMAP one can estimate the 2011 data by using Monitor rollback (percentage or incremental) by knowing the change in $PM_{2.5}$ concentrations at the county level as calculated using data from EPHT. Table 22 gives the comparison of $PM_{2.5}$ concentration data present in BenMAP, and the concentrations data extracted from EPHT for 2008. The results also give the difference between these two concentrations for each county. The results show that the $PM_{2.5}$ concentrations from the two sources for the same county were different. The concentrations for all counties, except for Sarasota, were lower in BenMAP than those extracted from EPHT. The data sources for both methods was FLEPA. The reason for the difference could be the methodology used to calculate the daily average for each county. Details of the methods were not available to further analyze the differences.

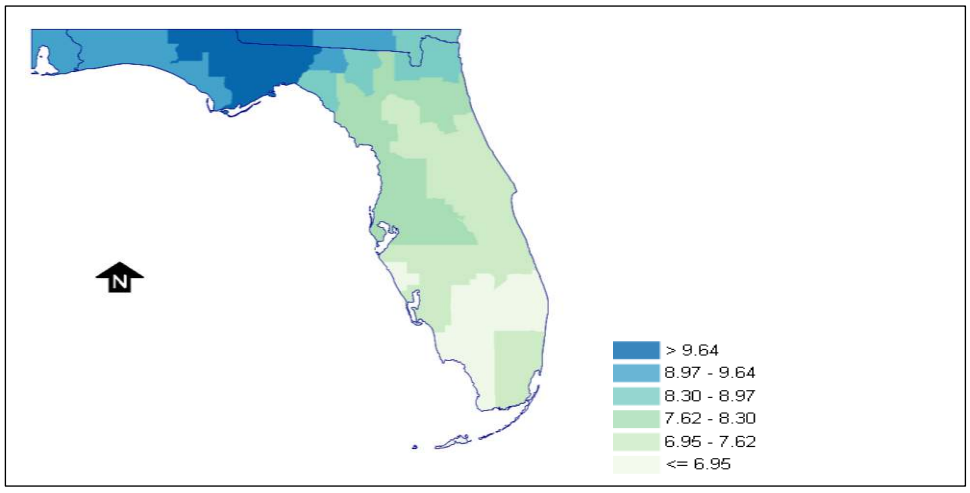


Figure 12 $PM_{2.5}$ Florida county level data for 2008 present in BenMAP

Table 22 Comparison of $PM_{2.5}$ Concentration in BenMAP to EPHT

FIPS County Code	County Name	$PM_{2.5}$ concentration for 2008 (in $\mu g/m^3$)		
		In BenMAP	In EPHT	Difference
1	Alachua County	7.49	7.93	-0.44
5	Bay County	9.67	9.9	-0.23
9	Brevard County	7.31	7.43	-0.12
11	Broward County	6.91	7.69	-0.78
17	Citrus County	7.77	7.81	-0.04
31	Duval County	8.69	9.24	-0.55
33	Escambia County	9.41	9.47	-0.06
57	Hillsborough County	8.09	8.51	-0.42
71	Lee County	7.02	7.07	-0.05
73	Leon County	10.12	10.64	-0.52
86	Miami-Dade County	7.18	8.1	-0.92
99	Palm Beach County	6.47	6.56	-0.09
103	Pinellas County	7.94	8.05	-0.11
105	Polk County	7.59	8.16	-0.57
115	Sarasota County	7.1	6.9	0.2
117	Seminole County	7.42	7.98	-0.56

The percentage change in $PM_{2.5}$ concentration for each county from 2008 to 2011 from EPHT data was calculated, and the results are given in Table 23. These results show that the change in $PM_{2.5}$ concentrations varied for counties, which ranged from -12.5% to 4.7%. All of the counties except Alachua county showed decrease in $PM_{2.5}$ concentrations. Alachua county had an increase by 4.7%. The reason for this increase in Alachua county was further investigated by checking news articles and published county health records for 2011. No public evidence was found to support this increase. It is also possible that the increase seen in Alachua county, while a decrease was seen in the other counties, could be an error or uncertainty of model data published by EPHT or due to the errors in reported data. This theory is supported by EPHT data in appendix 4, which shows that there were decreases in concentrations from 2008 to 2010.

Table 23 Change in $PM_{2.5}$ Concentration from 2008 to 2011 as per EPHT

FIPS County Code	County Name	Concentration of $PM_{2.5}$ (in $\mu g/m^3$)		Percentage Change
		2008	**2011**	
1	Alachua County	7.93	8.3	4.7%
5	Bay County	9.9	8.9	-10.1%
9	Brevard County	7.43	6.5	-12.5%
11	Broward County	7.69	6.8	-11.6%
17	Citrus County	7.81	7.6	-2.7%
31	Duval County	9.24	9	-2.6%
33	Escambia County	9.47	9.5	0.3%
57	Hillsborough County	8.51	7.8	-8.3%
71	Lee County	7.07	7.2	1.8%
73	Leon County	10.64	10.3	-3.2%
86	Miami-Dade County	8.1	7.4	-8.6%
99	Palm Beach County	6.56	6.4	-2.4%
103	Pinellas County	8.05	7.6	-5.6%
105	Polk County	8.16	7.5	-8.1%
115	Sarasota County	6.9	6.9	0.0%
117	Seminole County	7.98	7.6	-4.8%

The percentage changes for each county were added to BenMAP (Table 23), to estimate the $PM_{2.5}$ concentrations in 2011 for each county. The results are shown in Figure 13. The change in $PM_{2.5}$ concentrations from 2008 to 2011 in BenMAP are given in Figure 14 and Table 24. The results also give the error and percentage difference from the PM2.5 concentrations that were reported in EPHT (E), and that observed (O) in BenMAP. These differences range from 10.4% to 0.8% across the 17 counties. Orange County was not used in this analysis as data for 2011 were not available in EPHT. The range of differences and of errors, highlights that EPHT was prone to differences in the concentration models used in the two source for $PM_{2.5}$. Figures 12-14 show that

BenMAP is helpful in estimating the pollutant concentration and changes in counties where no monitors are present using the VNA method.

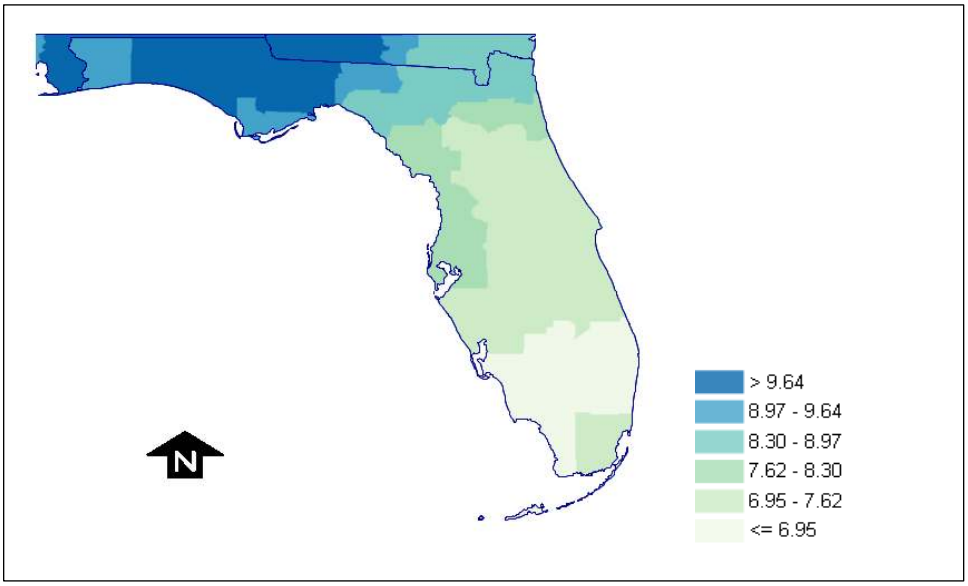


Figure 13 Estimated $PM_{2.5}$ concentration (in $\mu g/m^3$) at county level data for 2011 in BenMAP

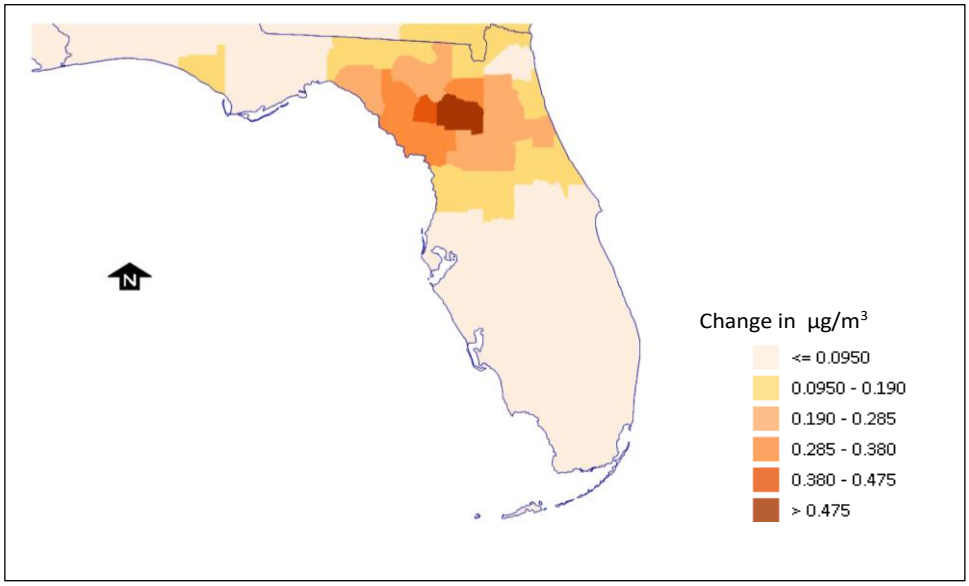


Figure 14 Change in $PM_{2.5}$ at Florida county level data from 2008 to 2011 in BenMAP

Table 24 Comparison of Estimated $PM_{2.5}$ Concentrations from BenMAP and EPHT

County Name	Concentration of $PM_{2.5}$ (in $\mu g/m^3$)		Error (| E-O|/E)	%Difference (Error/100)
	In EPHT (Expected)	In BenMAP (Observed)		
Alachua County	8.3	7.44	0.1036	10.4%
Bay County	8.9	8.59	0.0348	3.5%
Brevard County	6.5	6.55	0.0077	0.8%
Broward County	6.8	6.34	0.0676	6.8%
Citrus County	7.6	7.32	0.0368	3.7%
Duval County	9	8.21	0.0878	8.8%
Escambia County	9.5	9.19	0.0326	3.3%
Hillsborough County	7.8	7.47	0.0423	4.2%
Lee County	7.2	6.85	0.0486	4.9%
Leon County	10.3	9.79	0.0495	5.0%
Miami-Dade County	7.4	6.65	0.1014	10.1%
Palm Beach County	6.4	6.14	0.0406	4.1%
Pinellas County	7.6	7.49	0.0145	1.4%
Polk County	7.5	7.05	0.0600	6.0%
Sarasota County	6.9	6.83	0.0101	1.0%
Seminole County	7.6	6.9	0.0921	9.2%

The results in Table 25 show that the estimated $PM_{2.5}$ concentration values for all the counties using a simple rollback were under a 5% change except for Alachua County. Alachua County showed an increase instead of decrease as seen in other counties of Florida. BenMAP uses the VNA method for estimating $PM_{2.5}$ concentrations. The VNA method uses the inverse distance weighted average of the neighboring monitors. This can introduce uncertainties when estimating from neighboring counties, where change in $PM_{2.5}$ concentrations is in the opposite direction than that of the county of estimation, (ie, decrease in neighboring counties while an increase in Alachua county). Mixing increase and decrease concentrations in neighboring counties in BenMAP using the VNA method can lead to results which are less reliable. To overcome this problem, further analysis is needed to assess how limiting the distance in VNA can increase accuracy. Alachua County's change was set to missing to assist in the software with more reliable estimates. The new results are tabulated

in Table 26. The results show that the difference between the estimated and observed value decreased to nearly 5%. Also this change had minimal effect on the other county estimates, except for Citrus County, where the % difference decreased from 8.47% (Table 25) to 3.18% (Table 26).

Table 25 Comparison of Expected and Observed $PM_{2.5}$ Concentrations (in µg/m^3) using BenMAP

County Name	Concentration of $PM_{2.5}$ (in µg/m^3)				
	2008	%Change 2008	Expected 2011	Observed 2011	%Difference
Alachua County	7.49	4.70%	7.84	3.17	59.58%
Bay County	9.67	-10.10%	8.69	8.59	1.19%
Brevard County	7.31	-12.50%	6.40	6.55	2.40%
Broward County	6.91	-11.60%	6.11	6.34	3.79%
Citrus County	7.77	-2.70%	7.56	6.92	8.47%
Duval County	8.69	-2.60%	8.46	8.21	3.00%
Escambia County	9.41	0.30%	9.44	9.19	2.63%
Hillsborough County	8.09	-8.30%	7.42	7.47	0.69%
Lee County	7.02	1.80%	7.15	6.85	4.15%
Leon County	10.12	-3.20%	9.80	9.79	0.06%
Miami-Dade County	7.18	-8.60%	6.56	6.65	1.33%
Palm Beach County	6.47	-2.40%	6.31	6.14	2.77%
Pinellas County	7.94	-5.60%	7.50	7.49	0.07%
Polk County	7.59	-8.10%	6.98	7.05	1.07%
Sarasota County	7.10	0.00%	7.10	6.83	3.80%
Seminole County	7.42	-4.80%	7.06	6.90	2.32%

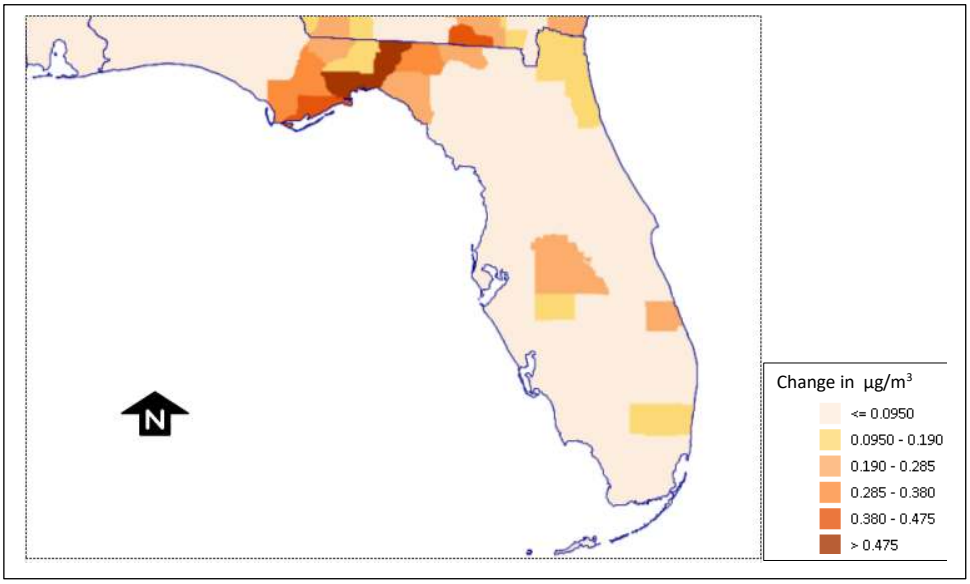


Figure 15 Change in $PM_{2.5}$ from 2008 to 2011 calculated using Rollback in BenMAP

Table 26 Estimated $PM_{2.5}$ Concentrations (in µg/m^3) after Correction

County Name	Concentration of $PM_{2.5}$ (in µg/m^3)				
	2008	%Change 2008	Expected 2011	Observed 2011	%Difference
Alachua County	7.49	-	7.84	7.44	5.13%
Bay County	9.67	-10.10%	8.69	8.59	1.19%
Brevard County	7.31	-12.50%	6.40	6.55	2.40%
Broward County	6.91	-11.60%	6.11	6.34	3.79%
Citrus County	7.77	-2.70%	7.56	7.32	3.18%
Duval County	8.69	-2.60%	8.46	8.21	3.00%
Escambia County	9.41	0.30%	9.44	9.19	2.63%
Hillsborough County	8.09	-8.30%	7.42	7.47	0.69%
Lee County	7.02	1.80%	7.15	6.85	4.15%
Leon County	10.12	-3.20%	9.80	9.79	0.06%
Miami-Dade County	7.18	-8.60%	6.56	6.65	1.33%
Palm Beach County	6.47	-2.40%	6.31	6.14	2.77%
Pinellas County	7.94	-5.60%	7.50	7.49	0.07%
Polk County	7.59	-8.10%	6.98	7.05	1.07%
Sarasota County	7.10	0.00%	7.10	6.83	3.80%
Seminole County	7.42	-4.80%	7.06	6.90	2.32%

The difference between estimated values and observed values was equal to or less than 5% for all counties. A graphical representation of the change in $PM_{2.5}$ data at Florida county level from 2008 to 2011 using BenMAP is given in Figure 15.

The results show that researchers can easily use the monitor rollback method for Florida to estimate $PM_{2.5}$ concentrations, by using change in pollutant concentration from year to year by county level. In this method one has to be careful to use only one direction of change, either increase or decrease as mentioned in the BenMAP manual. When using increase for some counties and decrease for other counties the results using VNA method with monitor rollback will give unreliable results.

USEPA has acknowledged that the pollution data in BenMAP are outdated and should be updated by users periodically for analysis. Data were updated for Florida counties using monitor data from FLEPA for 2010 to 2012, which is considered as baseline for this analysis. Monitor data for Florida counties was also updated for 2013 and 2014. The change in air pollution was calculated at the county level using VNA method in BenMAP. The results for change in $PM_{2.5}$ concentration for years 2013 and 2014 from 2010, which is considered baseline for this analysis, are given in Figures 16 and 17 respectively.

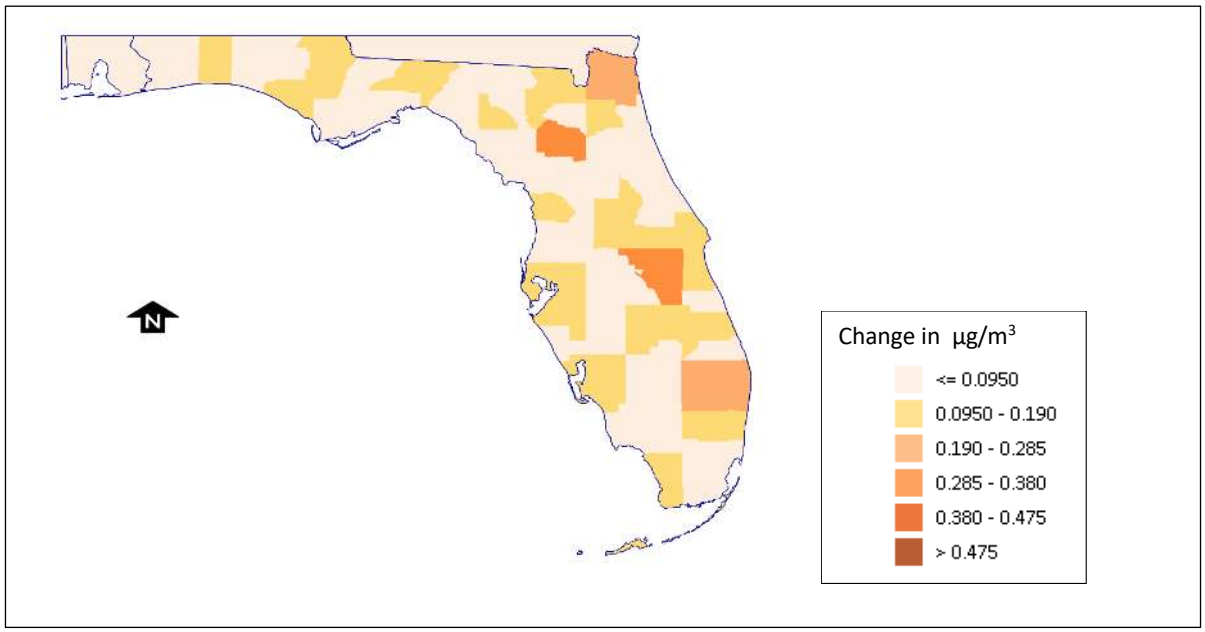


Figure 16 Change in $PM_{2.5}$ from Baseline(2010) to 2013

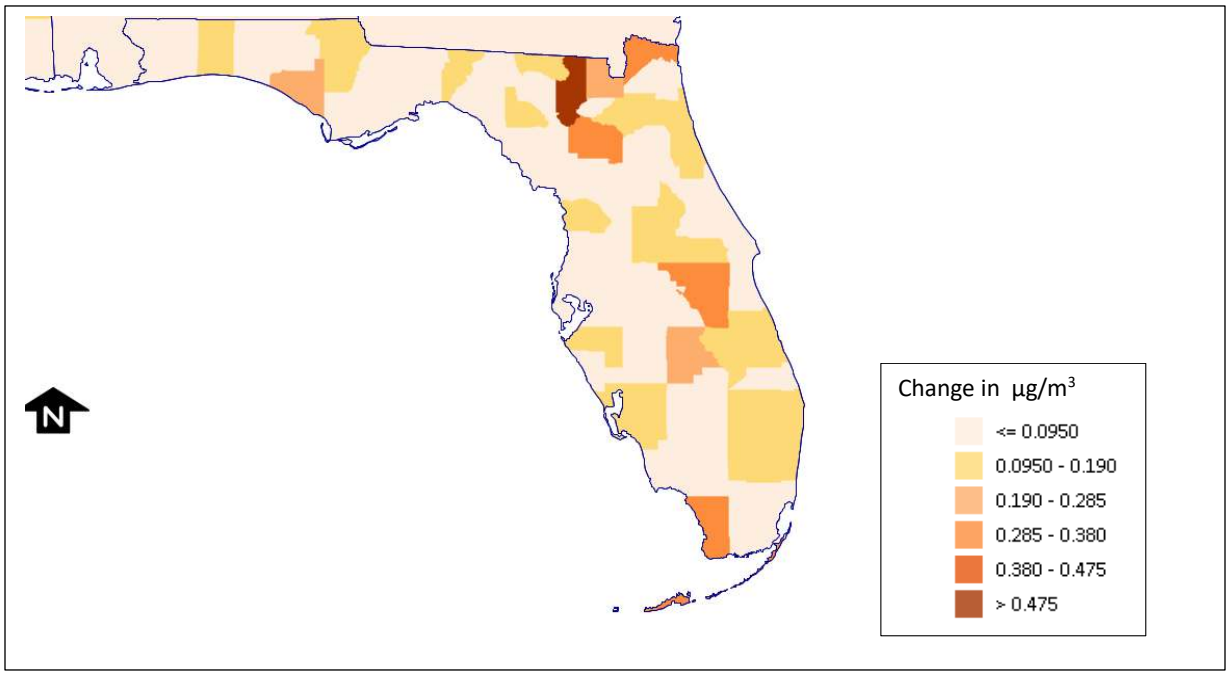


Figure 17 Change in $PM_{2.5}$ from Baseline (2010) to 2014

Table 27 gives the comparison of estimated asthma ED visits from BenMAP using beta (β) from this study-aim4 and pooled β-in BenMAP. The results show that the estimated change in annual

ED visits for asthma in Florida varied slightly by the two methods. The annual difference in the two methods was 6 asthma visits. The change in annual ED visits for asthma by the pooled method was 2% higher than the estimates using beta from this study. The pooled method using betas from studies already present in BenMAP, this study and three meta-analysis identified aim 4. Differences were also seen at each county level, these ranged from 0.3% - 6.7%, the statistics of difference is given in appendix 8.1.

Table 28 gives the comparison of estimated asthma exacerbation ED visits from BenMAP using beta (β) from this study-aim 4 and pooled β-in BenMAP. The results show that the estimated change in annual ED visits for asthma exacerbation varied slightly by the two methods. The annual difference in the two methods was asthma exacerbation visits. The change in annual ED visits for asthma exacerbation by the pooled method was 5% higher than the estimates using beta from this study. Differences were also seen at each county level, these ranged from 0% - 17%, the statistics of difference is given in appendix 8.2.

Table 29 gives the comparison of estimated asthma ED visits from BenMAP using prevalence of ED visits for the State of Florida from this study-aim3 and prevalence rate-in BenMAP. The prevalence rates from this study are controlled for age group, gender and race/ethnicity. The results show that the estimated change in annual ED visits for asthma in Florida varied a lot by the two methods.

Table 27 Estimated Change in Asthma ED visits from BenMAP using Pooled Beta

County	Change in Annual Asthma ED Visits		
	using Study β	using Pooled β	% Difference
Florida	-304	-310	2%
Alachua County	-5.8	-6.0	3.6%
Baker County	-0.5	-0.6	2.9%
Bay County	-1.7	-1.8	3.8%
Bradford County	-0.2	-0.3	4.1%
Brevard County	-5.6	-5.8	3.6%
Broward County	-32.4	-32.7	1.1%
Calhoun County	-0.3	-0.3	4.4%
Charlotte County	-1.1	-1.2	3.7%
Citrus County	-0.7	-0.7	3.7%
Clay County	-1.6	-1.6	3.0%
Collier County	-2.1	-2.2	2.6%
Columbia County	-1.1	-1.2	3.3%
DeSoto County	-0.2	-0.2	4.1%
Dixie County	0.0	0.0	3.4%
Duval County	-30.4	-30.5	0.4%
Escambia County	-0.7	-0.8	2.8%
Flagler County	-0.5	-0.6	3.0%
Franklin County	0.1	0.1	3.4%
Gadsden County	-0.4	-0.4	2.4%
Gilchrist County	0.0	0.0	3.4%
Glades County	0.0	0.0	2.3%
Gulf County	0.0	0.0	3.6%
Hamilton County	-0.1	-0.1	3.4%
Hardee County	0.5	0.5	3.5%
Hendry County	-0.1	-0.1	3.0%
Hernando County	-0.9	-1.0	4.4%
Highlands County	-1.0	-1.0	2.8%
Hillsborough County	-27.3	-27.2	0.3%
Holmes County	-0.2	-0.2	3.9%
Indian River County	-0.3	-0.4	3.6%
Jackson County	-0.8	-0.8	2.6%
Jefferson County	-0.1	-0.1	3.4%
Lafayette County	0.0	0.0	6.7%
Lake County	-3.6	-3.7	3.6%

Table 27 Asthma ED visits from BenMAP using Pooled Beta Beta *continued…*

County	Change in Annual Asthma ED Visits		
	using Study β	using Pooled β	% Difference
Florida	-304	-310	2%
Lee County	-7.2	-7.4	2.8%
Leon County	-4.6	-4.7	3.1%
Levy County	0.0	0.0	3.9%
Liberty County	0.1	0.1	4.0%
Madison County	1.0	1.1	3.4%
Manatee County	-2.9	-3.0	3.2%
Marion County	2.5	2.6	3.0%
Martin County	0.8	0.8	3.2%
Miami Dade County	-34.9	-35.8	2.5%
Monroe County	-1.1	-1.1	3.6%
Nassau County	-2.0	-2.1	3.6%
Okaloosa County	-2.3	-2.4	3.3%
Okeechobee County	-0.6	-0.7	3.4%
Orange County	-22.3	-22.4	0.3%
Osceola County	-6.0	-6.2	3.6%
Palm Beach County	-20.4	-21.0	3.1%
Pasco County	-5.7	-5.9	3.7%
Pinellas County	-28.0	-28.1	0.4%
Polk County	-38.1	-38.3	0.5%
Putnam County	-2.3	-2.4	3.3%
Santa Rosa County	-0.3	-0.3	4.1%
Sarasota County	-2.7	-2.8	3.1%
Seminole County	-3.0	-3.1	3.8%
St. Johns County	-4.2	-4.4	3.7%
St Lucie County	2.3	2.4	3.2%
Sumter County	-0.3	-0.3	4.7%
Suwannee County	0.4	0.4	4.0%
Taylor County	0.1	0.1	4.7%
Union County	0.1	0.1	4.6%
Volusia County	-1.9	-2.0	3.2%
Wakulla County	-0.4	-0.4	2.9%
Walton County	-0.9	-1.0	3.7%
Washington County	-0.3	-0.3	3.6%

Table 28 Estimated Asthma Exacerbation ED from BenMAP using Pooled Beta

County	Change in Annual Asthma Exacerbation ED Visits		
	using Study β	using Pooled β	% Difference
Total Change in Florida	-183	-192	5%
Alachua County	-4.0	-4.3	7%
Baker County	-0.3	-0.3	8%
Bay County	-0.9	-0.9	6%
Bradford County	-0.2	-0.2	2%
Brevard County	-2.9	-3.0	3%
Broward County	-19.0	-19.8	4%
Calhoun County	-0.1	-0.1	5%
Charlotte County	-0.7	-0.8	3%
Citrus County	-0.4	-0.4	7%
Clay County	-1.0	-1.0	4%
Collier County	-1.5	-1.6	3%
Columbia County	-0.6	-0.6	1%
DeSoto County	-0.1	-0.2	12%
Dixie County	0.0	0.0	13%
Duval County	-21.9	-23.8	8%
Escambia County	-0.5	-0.5	6%
Flagler County	-0.2	-0.2	15%
Franklin County	0.0	0.0	2%
Gadsden County	-0.3	-0.3	13%
Gilchrist County	0.0	0.0	13%
Glades County	0.0	0.0	5%
Gulf County	0.0	0.0	1%
Hamilton County	-0.1	-0.1	4%
Hardee County	0.2	0.2	11%
Hendry County	-0.1	-0.1	2%
Hernando County	-0.5	-0.5	3%
Highlands County	-0.5	-0.5	2%
Hillsborough County	-15.0	-15.5	3%
Holmes County	-0.1	-0.1	8%
Indian River County	-0.2	-0.2	6%
Jackson County	-0.5	-0.5	2%
Jefferson County	-0.1	-0.1	8%
Lafayette County	0.0	0.0	3%
Lake County	-2.5	-2.6	4%

Table 28 Estimated Asthma Exacerbation ED from BenMAP using Pooled Beta *continued…*

County	Change in Annual Asthma Exacerbation ED Visits		
	using Study β	using Pooled β	% Difference
Total Change in Florida	-183	-192	5%
Lee County	-4.2	-4.2	1%
Leon County	-3.5	-3.7	5%
Levy County	0.0	0.0	17%
Liberty County	0.1	0.1	16%
Madison County	0.9	0.9	0%
Manatee County	-1.5	-1.5	0%
Marion County	1.2	1.2	0%
Martin County	0.4	0.4	0%
Miami Dade County	-18.7	-19.9	6%
Monroe County	-0.7	-0.7	0%
Nassau County	-0.9	-0.9	2%
Okaloosa County	-1.6	-1.6	1%
Okeechobee County	-0.4	-0.4	0%
Orange County	-14.4	-15.6	8%
Osceola County	-4.0	-4.1	0%
Palm Beach County	-13.2	-13.4	1%
Pasco County	-3.0	-3.4	12%
Pinellas County	-17.4	-17.7	2%
Polk County	-20.2	-21.4	5%
Putnam County	-1.3	-1.4	8%
Santa Rosa County	-0.2	-0.3	15%
Sarasota County	-1.7	-1.7	3%
Seminole County	-1.9	-2.0	1%
St. Johns County	-2.3	-2.3	1%
St Lucie County	1.3	1.5	9%
Sumter County	-0.2	-0.2	9%
Suwannee County	0.2	0.2	12%
Taylor County	0.1	0.1	2%
Union County	0.1	0.1	3%
Volusia County	-1.1	-1.1	1%
Wakulla County	-0.3	-0.3	6%
Walton County	-0.5	-0.4	12%
Washington County	-0.2	-0.2	10%

The annual difference of change in asthma ED visits by the two methods was 134 asthma visits. The change in annual ED visits for asthma using study specific prevalence rates were 30% higher than the estimates using prevalence in BenMAP. Differences were also seen at each county level, these ranged from 21% - 69%, the statistics of difference is given in appendix 8.3. The observed differences were higher for counties were monitors per not present or which were smaller in area, where the asthma ED visits are less.

Table 30 gives the comparison of estimated asthma exacerbation ED visits from BenMAP using prevalence of ED visits for the State of Florida from this study-aim 3 and prevalence rate-in BenMAP. The results show that the estimated change in annual ED visits for asthma exacerbation in Florida varied a lot by the two methods. The annual difference of change in ED visits by two methods was 82 asthma exacerbation visits. The change in annual ED visits for asthma exacerbation using study specific prevalence rates were 31% higher than the estimates using prevalence in BenMAP. Differences were also seen at each county level, these ranged from 13% - 69%, the statistics of difference is given in appendix 8.4. The observed differences were similar to asthma visits and were higher for counties were monitors per not present or which were smaller in area, where the exacerbation ED visits are less.

The results show that estimating the health impact due to change in $PM_{2.5}$ is highly dependent on the prevalence of the health outcome and less dependent on the CR functions. The dependence on the CR function was probably less as the beta estimate for asthma and asthma exacerbation are very small, to the forth decimal place.

Table 29 Estimated Asthma ED visits using Prevalence in BenMAP and Prevalence by County

County	Change in Annual Asthma ED Visits		
	using Prevalence in BenMap	using Study Specific Prevalence	% Difference
Florida	-304	-436	30%
Alachua County	-5.8	-8.9	35%
Baker County	-0.5	-0.8	28%
Bay County	-1.7	-2.8	37%
Bradford County	-0.2	-0.4	41%
Brevard County	-5.6	-8.6	36%
Broward County	-32.4	-45.6	29%
Calhoun County	-0.3	-0.4	43%
Charlotte County	-1.1	-1.7	36%
Citrus County	-0.7	-1.1	37%
Clay County	-1.6	-2.2	29%
Collier County	-2.1	-2.8	24%
Columbia County	-1.1	-1.6	32%
DeSoto County	-0.2	-0.4	40%
Dixie County	0.0	0.0	33%
Duval County	-30.4	-40.6	25%
Escambia County	-0.7	-1.0	27%
Flagler County	-0.5	-0.8	29%
Franklin County	0.1	0.2	33%
Gadsden County	-0.4	-0.5	23%
Gilchrist County	0.0	0.0	33%
Glades County	0.0	0.0	21%
Gulf County	0.0	0.0	35%
Hamilton County	-0.1	-0.1	33%
Hardee County	0.5	0.8	35%
Hendry County	-0.1	-0.2	29%
Hernando County	-0.9	-1.6	45%
Highlands County	-1.0	-1.3	26%
Hillsborough County	-27.3	-39.7	31%
Holmes County	-0.2	-0.3	38%
Indian River County	-0.3	-0.5	36%
Jackson County	-0.8	-1.0	25%
Jefferson County	-0.1	-0.2	33%
Lafayette County	0.0	-0.1	69%
Lake County	-3.6	-5.5	35%

Table 29 Estimated Asthma ED visits using Prevalence in BenMAP and Prevalence by County *continued…*

County	Change in Annual Asthma ED Visits		
	using Prevalence in BenMAP	using Prevalence by County from Aim 3	% Difference
Florida	-304	-436	30%
Lee County	-7.2	-9.9	27%
Leon County	-4.6	-6.5	30%
Levy County	0.0	0.0	39%
Liberty County	0.1	0.2	40%
Madison County	1.0	1.5	33%
Manatee County	-2.9	-4.2	32%
Marion County	2.5	3.5	29%
Martin County	0.8	1.1	31%
Miami Dade County	-34.9	-45.8	24%
Monroe County	-1.1	-1.7	35%
Nassau County	-2.0	-3.2	36%
Okaloosa County	-2.3	-3.4	32%
Okeechobee County	-0.6	-1.0	33%
Orange County	-22.3	-31.4	29%
Osceola County	-6.0	-9.3	35%
Palm Beach County	-20.4	-29.2	30%
Pasco County	-5.7	-9.0	37%
Pinellas County	-28.0	-42.8	35%
Polk County	-38.1	-54.2	30%
Putnam County	-2.3	-3.3	32%
Santa Rosa County	-0.3	-0.5	41%
Sarasota County	-2.7	-3.9	30%
Seminole County	-3.0	-4.8	37%
St. Johns County	-4.2	-6.6	36%
St Lucie County	2.3	3.4	31%
Sumter County	-0.3	-0.6	48%
Suwannee County	0.4	0.6	40%
Taylor County	0.1	0.3	47%
Union County	0.1	0.2	46%
Volusia County	-1.9	-2.7	31%
Wakulla County	-0.4	-0.6	28%
Walton County	-0.9	-1.4	36%
Washington County	-0.3	-0.5	35%

Table 30 Estimated Asthma Exacerbation ED using Prevalence in BenMAP and Prevalence by County

County	Change in Annual Asthma Exacerbation ED Visits		
	using Prevalence in BenMAP County	using Prevalence by County from Aim3	% Difference
Total Change in Florida	-183	-265	31%
Alachua County	-4.0	-6.3	36%
Baker County	-0.3	-0.4	25%
Bay County	-0.9	-1.3	37%
Bradford County	-0.2	-0.3	44%
Brevard County	-2.9	-4.8	39%
Broward County	-19.0	-27.2	30%
Calhoun County	-0.1	-0.2	56%
Charlotte County	-0.7	-1.1	35%
Citrus County	-0.4	-0.7	45%
Clay County	-1.0	-1.5	31%
Collier County	-1.5	-2.0	25%
Columbia County	-0.6	-0.9	35%
DeSoto County	-0.1	-0.3	49%
Dixie County	0.0	0.0	13%
Duval County	-21.9	-28.9	24%
Escambia County	-0.5	-0.7	29%
Flagler County	-0.2	-0.3	29%
Franklin County	0.0	0.1	52%
Gadsden County	-0.3	-0.4	26%
Gilchrist County	0.0	0.0	42%
Glades County	0.0	0.0	26%
Gulf County	0.0	0.0	42%
Hamilton County	-0.1	-0.1	33%
Hardee County	0.2	0.3	27%
Hendry County	-0.1	-0.2	34%
Hernando County	-0.5	-1.0	48%
Highlands County	-0.5	-0.7	33%
Hillsborough County	-15.0	-22.5	33%
Holmes County	-0.1	-0.2	35%
Indian River County	-0.2	-0.2	35%
Jackson County	-0.5	-0.6	23%
Jefferson County	-0.1	-0.1	33%
Lafayette County	0.0	-0.1	69%
Lake County	-2.5	-4.0	37%

Table 30 Estimated Asthma Exacerbation ED from BenMAP using Prevalence in BenMAP and Prevalence by County *continued* …

County	Change in Annual Asthma Exacerbation ED Visits		
	using Prevalence in BenMAP County	using Prevalence by County from Aim3	% Difference
Total Change in Florida	-183	-265	31%
Lee County	-4.2	-5.8	28%
Leon County	-3.5	-5.0	29%
Levy County	0.0	0.0	34%
Liberty County	0.1	0.1	52%
Madison County	0.9	1.3	34%
Manatee County	-1.5	-2.2	32%
Marion County	1.2	1.6	26%
Martin County	0.4	0.7	37%
Miami Dade County	-18.7	-24.6	24%
Monroe County	-0.7	-1.1	38%
Nassau County	-0.9	-1.3	32%
Okaloosa County	-1.6	-2.3	33%
Okeechobee County	-0.4	-0.6	34%
Orange County	-14.4	-20.5	30%
Osceola County	-4.0	-6.4	37%
Palm Beach County	-13.2	-19.1	31%
Pasco County	-3.0	-4.9	38%
Pinellas County	-17.4	-26.6	35%
Polk County	-20.2	-29.9	32%
Putnam County	-1.3	-2.0	37%
Santa Rosa County	-0.2	-0.4	43%
Sarasota County	-1.7	-2.5	33%
Seminole County	-1.9	-3.0	36%
St. Johns County	-2.3	-3.7	39%
St. Lucie County	1.3	2.1	36%
Sumter County	-0.2	-0.4	50%
Suwannee County	0.2	0.4	38%
Taylor County	0.1	0.2	49%
Union County	0.1	0.1	49%
Volusia County	-1.1	-1.6	31%
Wakulla County	-0.3	-0.4	29%
Walton County	-0.5	-0.7	34%
Washington County	-0.2	-0.3	31%

CHAPTER FIVE: DISCUSSION

5.1 Summary of Findings

This study reviewed BenMAP manuals and published literature, and observed that the prevalence rates preloaded in BenMAP software varied from 7.4% to 17.3% for asthma exacerbation, 9.41%-17.76% for asthma prevalence rates and, asthma ED visit was 8.65 per 1,000. These prevalence rates for asthma differ from those published by FLDEP (20.59 %, Appendix 3.1). Asthma ED visit rates are 1.6 times higher than rates published by FLDEP (2016, Appendix1b). If rates present in BenMAP are used for the HIA method then the change in asthma ED visits estimated by BenMAP will be an overestimate. There are no baseline rates for asthma exacerbation ED visits in BenMAP, asthma exacerbation is a more severe case. The prevalence for exacerbation ED visits is noted to be lower than prevalence of asthma ED visits. For Florida, the exacerbation rates have been reported to be only 36% of all asthma ED visits (FAP 2014), using asthma ED visit prevalence rates will tend to overestimate the exacerbation by 64%.

CR functions in USEPA's BenMAP were developed using different studies to estimate asthma hospital visits, asthma emergency rates, and asthma exacerbation rates (USEPA, 2015). Each of the studies are based in different locations, have different endpoints and have used different statistical models for analysis. CR functions for asthma emergency room visits, which are based on beta values, utilized studies from the US West coast, and are not representative of the US East coast due to several factors. The main differences between West coast populations and the

Florida population are the size of populations, age, gender and race/ethnicity distribution, weather, and pollutant concentrations. All three West coast studies were based in Seattle or nearby areas, which are at higher altitude than any area in Florida. The West coast climate patterns are defined by four seasons while Florida is more tropical with warm/wet and cold/ drier seasons. Florida is more prone to hurricanes and heavy rainfall during the summer season, while in Seattle it rains during winter and early-spring season. Average humidity and dew points are higher in Florida than in Washington State. Population density in Seattle and its neighboring areas is higher than in metropolitan cities of Florida, like Miami. A detailed review of the BenMAP documentation and EPA's-IRA reveal that the studies used for prevalence and CR functions base their estimates on population studies conducted 15 years ago or more. Prevalence and incidence rates have changed for asthma and asthma exacerbation over the past ten years. In Florida, asthma rates are showing a general increase. The observed trend in Florida is opposite that of published rates in BenMAP, which show a general decrease in trend using National data. The location where the studies were conducted, and timing of the studies used in BenMAP, makes a valid argument that to use BenMAP effectively for HIA in Florida, one may have to update the prevalence rates and CR functions in BenMAP with Florida data.

The prevalence rate for asthma ED visits for school-aged children ages 5-18 for Florida was 7.57 per 1,000, and for exacerbation was 4.51 per 1,000. The rates varied by race/ethnicity where Black not Hispanic had higher prevalence rates for asthma and asthma exacerbation. This disparity is also seen in the report published by FAP (2014). Rates published by FAP are higher than in this analysis (21.7), since the FAC 2014 report considered primary and secondary diagnosis for asthma in the prevalence rates. The present analysis considers only primary diagnosis of

asthma. To overcome the variation in the prevalence rates, for this analysis the prevalence rates were averaged over three years 2010-2012. Prevalence rates have increased over time in Florida which is contrary to changing trends in asthma prevalence published by Akimbami *et. a*l (2016). Akinbami *et. al.* (2016) have published that asthma prevalence rates will decrease in the North East region of USA. This further strengthens the reasoning why local level prevalence rates should be used when available to decrease uncertainties and improve estimation of change in health outcome like asthma ED visits or asthma exacerbation ED visits.

The prevalence of asthma and asthma exacerbation is higher for the younger age group (5-12) than the older age group (13-18). This is similar to the results published in FLDOH 2014. According to the report, elementary school children had higher rates of asthma attacks, and significantly higher lifetime prevalence than middle and high school children. These findings are similar to Gan et. al (2014), where the author described that the burden of asthma is highest for ages 10-14. The asthma and exacerbation ED prevalence rates were higher for males than for females. Similar gender difference has been published in several studies (CDC 2008; ALA 2007; and FAC 2013). The gender disparity seems to diminish in older age groups similar to the published literature, which has documented that under the age of 15 years, boys have higher morbidity, hospital admission and emergency visits than girls (Bloom 2007, CDC 2008, FAP 2014).

Racial disparity for asthma has been well documented in several studies which is similar to the results found in this study (Akinbami 2002, ALA 2007, Akinbami 2016, FLDOH 2013). Non-Hispanic Blacks have been documented to have higher asthma prevalence than non-Hispanic

White and Hispanics. The results of this analysis show that the prevalence rates used for calculating the impact of $PM_{2.5}$ should be at the county level, and stratified by race/ethnicity, age group and gender for Florida. These rates help to predict change in asthma or asthma exacerbation when there is a change in $PM_{2.5}$ after controlling for county. Using race ethnicity, age group and gender specific prevalence baseline rates will be helpful for Health Impact Assessments when planning for a community or group of individuals based on race ethnicity, age group and gender. Adjusting or controlling for county will help in decreasing uncertainties and variance across counties in Florida.

Asthma exacerbation is a consequence of poor management of severe cases resulting in multiple visits to ED or hospitals. The analysis of FLDOH and FAC (2013) shows that 36% of all asthma visits are ED visits or hospitalization. The results of the present study show that almost 60% of all asthma ED visits are coded as exacerbation visits. This study highlights that in the younger age group asthma management may be more crucial and important to control asthma and prevent exacerbations and repeat patient visits.

Monitor data were used in the present study instead of model data, since the monitoring data were available for the same time frame as the asthma ED visit data from FLDEP. Model data in BenMAP is over 7-8 years old and needs to be updated. Using any other model data than CMAQ would require reconfiguring of the grid definitions in BenMAP, which compromise the accuracy of the grid analysis in BenMAP.

In this study, using time-series analysis to study temporal and spatial variation, monthly concentration of $PM_{2.5}$ varies over the year. The highest concentrations are found from March to July. The concentration of $PM_{2.5}$ also varies across the counties, and has decreased over time. The decrease over time is due to more stringent air quality controls by EPA, and the result of ongoing emission reductions from industrial facilities and motor vehicles. The downward trend has been reported by FL DEP since a decade.

Time series Poisson models were used to develop odds ratios to estimate the association between asthma ED visits and $PM_{2.5}$ concentrations. Since monitors are located in only 17 of the 67 counties, and all of those are urban counties, the time series model only used asthma ED visit and exacerbation ED visits for these 17 counties. The location of these monitors is mandated by FL DEP, and is based on population orientation and multi-pollutant sites. Using the time series analysis of $PM_{2.5}$ localized CR functions were obtained. These CR functions have a low association between $PM_{2.5}$ (10µg /m^3), asthma exacerbation odds ratio for age group 13-18 was 1.004, (CI=1.001-1.008), while for 5-12 age group was 1.014, (CI=1.0004-1.025). The lower association of $PM_{2.5}$ in the older age group could be due to the fact that the majority of asthma exacerbations are reported to be virus-induced or stronger association of exacerbation to exposure of seasonal allergens (Murray 2004). This study did not evaluate exacerbation due to viral infections or seasonal allergens. However, it should be noted that asthma exacerbations are conditionally dependent on asthma prevalence. This study highlights that an increase of 10 µg/m^3 of $PM_{2.5}$ contributes about 2% to asthma ED visit rate in children 5-12, and lower for 13-18 olds (0.6%). One could estimate the change in asthma prevalence in Florida by age, gender and race/ethnicity, and use these to estimate asthma exacerbation in Florida counties, paying particular

attention to areas which have higher Black non-Hispanic populations. The literature highlights that if asthma is not managed properly, then it can result in decrease of lung function and leading to respiratory failure, imposing asocial and medical burden (Kurai, 2013).

5.2 Consistent with Literature

The prevalence rate for asthma ED visit for school-aged children ages 5-18 for Florida was 7.57 per 1,000 and for exacerbation was 4.51 per 1,000; these rates are similar to those published by FAP (2014), where ED visit for 0-17 was 8.2 per 1,000 and exacerbation was 5.1 per 1,000. The rates published by FAC (2014) are higher since they include children below age of 5, who have been reported to been seen at a higher rate (Akinbami, 2007).

National ED rates for asthma are 8.65 per 1,000; the CR function in BenMAP for asthma ED visits ranges from 0.0029 -0.0191 while in this study beta for asthma Ed visits was found to be 0.0020 for 5-11year old and 0.0006 for 12-18year old. The present study highlights that local Florida CR functions and local prevalence rates differ from national CR functions and local prevalence rates. These results are consistent with results published for a Detroit study by Hubbell *et. al.* (2006), which states that there is a need for regional CR functions for local HIA to be reliable and have lower uncertainties.

Literature published by Murray (2004), Sykes (2008) and Kurai(2013) have indicated that viral and bacterial infections are major contributors to asthma exacerbations while air pollution is a minor contributor. The results of this study confirm that for exacerbation the association with $PM_{2.5}$ is very weak, it may have stronger association with other lung irritants like NO_2. However,

the literature has clearly documented that there are clear differences between gender and race ethnicity this is also highlighted in the results of the present study.

5.3 Study Contributions

There are three major contributions of this study. Firstly, the study contributes to publishing childhood asthma emergency department prevalence and exacerbation rates in the State of Florida by age group, race/ethnicity and gender.

The second contribution is, development of concentration response functions specific to Florida using the time series analysis to show the impact of $PM_{2.5}$ on asthma exacerbation emergency department visits, incorporating both temporal and spatial variability of $PM_{2.5}$ during the study period.

Finally, the study demonstrates the utility of using local (county-level) asthma rates and local pollutant data for State Health Impact Assessments in Florida. The local $PM_{2.5}$ data in BenMAP can be used for other health outcome assessments, researchers will only have to update the prevalence rates for the health outcome used in their study.

This study supports recommendations to avoid high levels of $PM_{2.5}$ concentration to avoid the financial burden of asthma on state and payer, as the cost of care has had a sharp increase in the past decade. The results of this study inform the asthma management programs of the areas in Florida which have higher pollutant concentrations. This information is useful to asthma management programs to formulate recommendations for potentially susceptible groups such as

asthmatic school age children in communities with high rates of asthma ED visits and high air pollutant concentrations. Even though this study is based on a single pollutant model, it brings an important contribution to the medical society that knowing the pollutant measurements and surges of pollutant levels, physician offices can facilitate better planning for asthma management, and provide recommendations to established patients for management of asthma through medications or avoiding areas with higher pollutant surges to avoid ED or hospital visits. Alerts to physician's offices can be generated via an application from FL-EPHT which monitors the air pollution surges issued by air pollution advisories of FL-EPA.

5.4 Study Strengths

A major strength of this study is that it included a larger number of pediatric asthma and asthma exacerbation ED visits in comparison with other published studies. This study publishes the ED rates by county and by gender, race and ethnicity from 2010 to 2014, which are recent rates and have not been published to such granularity by the State or by any other researcher. The reported prevalence rates of childhood asthma and asthma exacerbation rates are also published for Florida counties.

Another strength of this study includes the use of air pollution measurements from the monitors in Florida to quantity exposure at county level. The study uses USEPA's BenMAP to quantify the $PM_{2.5}$ exposure at the county level for counties which do not have monitors using Voronoi neighbor averaging (VNA) spatial scaling method (Wang *et al*, 2013; EPA 2014). The study also contributes to input of $PM_{2.5}$ monitor data for Florida during 2010-2014 in BenMAP. Current pollutant data in BenMAP is only available through 2008, and EPA has recommended it

should be updated for analysis purposes. These data can be used for other health outcomes measures estimations using $PM_{2.5}$ as the exposure. The study uses local air pollutant data and local asthma prevalence rates to estimate the effect of change in pollutant on asthma exacerbation. Estimation using local data will be less prone to uncertainties using National level data, the use of local data has been emphasized by Levy (2005), Fann *et. al.* (2008) and Hubbel et. al. (2009).

Further, the study uses an established time series and GEE method to quantify the association between $PM_{2.5}$ and asthma rates. The CR from the present study were pooled with CR functions in BenMAP, and those identified in the peer reviewed literature, using a random effects method. The random effects model assists in combining overlapping estimates of health outcome from various models to generate a single mean effect while accounting for heterogeneity across studies. The random effects model helps to calculate the weighted average across the studies, using the weights calculated from the variance and overlap of the studies.

5.5 Study Limitations

Several limitations should be considered when interpreting this study. The limitations observed in this study are common to environmental and observational studies that have assessed the environmental predictors associated with childhood asthma or outcomes associated with criteria air pollutants including the uncertainties imposed by CR-functions, pollutant concentration, baseline incidence rates and estimates of the exposed population.

This study relies mainly on AHCA ED visit data for asthma outcome data, and EPA's air monitoring data for $PM_{2.5}$. None of these data sources were collected for research purposes or with the intent of addressing research aims of this study. Hence, the data collection process, reporting

protocols, overall quality and timing of onset of outcome may have resulted in uncertainties and misclassification in analysis.

Misclassification of Asthma and Asthma Exacerbation

These analyses use ED visits as reported to Florida AHCA by the hospital, and the cases of asthma and asthma exacerbations were identified using the ICD-9-CM diagnosis codes available in ED discharge records. There could be misclassification of asthma cases due to inaccurate diagnostics coding on medical records, distinguishing other respiratory illnesses, i.e. bronchitis, from asthma especially in the pediatric population (Brauer *et. al.*. 2007). ED claims records also reflect healthcare utilization patterns, which differ by income level, race or age (Boudreaux et. al. 2003; Shields *et. al.*. 2004), and may have introduced selection bias in this study.

Misclassification of Onset Asthma or Asthma Exacerbation Attack

Onset of asthma and exacerbation attack is an important determination of exposure to pollutant in this study, its accuracy measurement is fairly important. Since the data were collected previously for administrative purposes, the exact date on onset of asthma or asthma exacerbation cannot be determined, and solely relies on when the patient is seen in the ED setting, the lag calculated in the study is delayed from true onset to actual recording of a case in ED.

Misclassification of Exposure

The study did not collect personal pollutant exposure, it rather relied on values averaged across monitor data in the county or to the nearest monitor to county near patient county. This method introduces potential uncertainties and exposure misclassification. This method assumes

same risk estimates across all neighborhoods within the county, and, in addition, the personal effects are not taken into account (i.e., the subject in same county are assigned the same $PM_{2.5}$ level exposure). As within-county or city exposure variations are not considered in this study the misclassification to personal exposure, which are inherent, cannot be quantified in this study.

Deviation from Temporal Overlapping Assumption

The association models using GEE to calculate CR functions are based on the *"temporal ordering assumption"* (Diggle *et. al.*, 2005). This assumption specifies that exposure must occur before the onset of outcome. It is assumed that asthma and exacerbation cases resided in the same county as they were admitted in and the onset of asthma or exacerbation was due to ambient air pollutant concentration before onset of outcome, inferring causality between outcome and exposure. Cox (2012) has questioned this assumption, whether any relationship should be seen as causal at $PM_{2.5}$ level in environmental epidemiological studies, where evidence lacks the rigor to infer causality.

Uncertainty of Risk Estimates due to CR functions

The magnitude of pollutant attributable estimates is limited by uncertainty of risk estimates using CR functions derived from the epidemiological literature (Hubbell *et. al.*, 2009). These risk estimates rely on multi-city or county level estimates, and introduce uncertainty in the risk estimates and cannot quantify how neighborhood-level CR functions vary across urban cities. This study minimized this uncertainty by using local Florida level CR function for counties. However, due to data limitations this study could not examine effect modification due to city-level

characteristics. It does, provide evidence that race/ethnicity, age group and gender may modify short-term risks attributable to asthma ED visits and exacerbation ED visits.

5.6 Future Direction

The present research study highlights the need to further refine "*air quality*" or spatial grids in BenMAP to the zip code level in order to facilitate a study of childhood asthma at the neighborhood level. Subjects who grow up in poorer areas with lower SES may be more susceptible to air pollution-related asthma and asthma exacerbation. There is enough evidence from published literature that ethnicity, family income and education effect dietary intake among adolescents (Xie *et. al,*2003). The food intake will effect the vitamin and mineral intake and absorption into the body. Food habits and quality of food has been linked to body weight, cognitive skills and performance in children and adolescents (Rampersaud *et.al.*, 2005). Observed changes in intake dietary trends among children and adolescents from 2000 to 2010 has lead to a change in intake of macronutrients and sources of energy in children (Ervin *et. al.,*2013). With changes in dietary habits which have been published in literature the becomes even more important to study, how diet influences individual level reaction to change in pollutant levels. Vitamin B and its components have been known to help individual cells in body to perform many different jobs including release of energy and fight during infection. A recent published study states that vitamin-B supplementation prevents DNA methylation changes due to $PM_{2.5}$ (Zhong *et.al,,* 2017). More research is needed to understand the interaction between pollutant exposures, diet intake and individual-level response to exposure leading to short-term asthma exacerbations. Studies designed to study diet and individual level response to air pollutnats may help us understand social injustices in areas with diverse populations.

A population-based study in conjunction with Florida Asthma Collation (FAC) using asthma cases from doctor's practices in different cities in Florida is needed to understand asthma case management at the community level. Working with doctor's offices directly, researchers could quantify personal exposures, and decrease their misclassification. This method will also help in reducing misclassification of asthma outcome and deviations from the "*temporal ordering assumption*". Working with doctor's offices will further minimize the misclassification of asthma cases due to inaccurate diagnostics coding on medical records, and help to distinguish other respiratory illnesses from asthma. This future work can include urban and rural area analyses to document how asthma differs with location, development and social factors.

Future work should include weather-related variables (e.g. wind speed, barometric pressure and relative humidity) and other allergen variables in the analyses. Wind speed, barometric pressure and relative humidity can affect exposure to the pollutant concentration and allergens. Resultant analyses should provide a better measure of the true association of the pollutants with asthma or asthma exacerbations. Effort should also be made to study multi-pollutant models to understand collinearity between pollutants and other markers of $PM_{2.5}$. Experimental and observational studies can continue to provide indications of key exposure parameters, and PM sources associated adverse effects of asthma in a managed care setting.

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APPENDICES

Appendix 1: List of Acronyms

AHCA- Agency of Health Care Administration

BenMAP - Benefit Mapping and Analysis Program

BRFSS - Behavior Risk Factor Surveillance System

CDC - The Center of Disease Control

CR function–Concentration Response Function

ED - Emergency Department

EPA - Environmental Protection Agency

EPHT- Environmental Protection Health Tracking

FYTS – Florida Youth Tobacco Survey

HIA - Health Impact Analysis

RTP- Research Triangle Park, North Carolina

NAMCS - National Ambulatory Medical Care Survey

NHAMCS - National Hospital Ambulatory Medical Care Survey

NHIS - National Health Information Survey

NNEMS- National Network for Environmental Management Studies Fellowship

NSCH - National Survey of Children's Health

OR - Odds ratio

VOCs - Volatile Organic Compounds.

VNA- Voronoi neighbor averaging

YRBSS- Youth Risk Behavior Surveillance System

Appendix 2: ICD9 Codes for Asthma

Asthma 493- >

- A chronic disease in which the bronchial airways in the lungs become narrowed and swollen, making it difficult to breathe. Symptoms include wheezing, coughing, tightness in the chest, shortness of breath, and rapid breathing. An attack may be brought on by pet hair, dust, smoke, pollen, mold, exercise, cold air, or stress.
- A chronic respiratory disease manifested as difficulty breathing due to the narrowing of bronchial passageways.
- A form of bronchial disorder with three distinct components: airway hyper-responsiveness (respiratory hypersensitivity), airway inflammation, and intermittent airway obstruction. It is characterized by spasmodic contraction of airway smooth muscle, wheezing, and dyspnea (dyspnea, paroxysmal).
- Asthma is a chronic disease that affects your airways. Your airways are tubes that carry air in and out of your lungs. If you have asthma, the inside walls of your airways become sore and swollen. That makes them very sensitive, and they may react strongly to things that you are allergic to or find irritating. When your airways react, they get narrower and your lungs get less air symptoms of asthma include
    - wheezing
    - coughing, especially early in the morning or at night
    - chest tightness
    - shortness of breath

  not all people who have asthma have these symptoms. Having these symptoms doesn't always mean that you have asthma. Your doctor will diagnose asthma based on lung function tests, your medical history, and a physical exam. You may also have allergy tests when your asthma symptoms become worse than usual, it's called an asthma attack. Severe asthma attacks may require emergency care, and they can be fatal asthma is treated with two kinds of medicines: quick-relief medicines to stop asthma symptoms and long-term control medicines to prevent symptoms.
- Form of bronchial disorder associated with airway obstruction, marked by recurrent attacks of paroxysmal dyspnea, with wheezing due to spasmodic contraction of the bronchi.

- 493 Asthma
  - 493.0 Extrinsic asthma
    - 493.00 Extrinsic asthma, unspecified convert 493.00 to ICD-10-CM
    - 493.01 Extrinsic asthma with status asthmaticus convert 493.01 to ICD-10-CM
    - 493.02 Extrinsic asthma with (acute) exacerbation convert 493.02 to ICD-10-CM
  - 493.1 Intrinsic asthma
    - 493.10 Intrinsic asthma, unspecified convert 493.10 to ICD-10-CM
    - 493.11 Intrinsic asthma with status asthmaticus convert 493.11 to ICD-10-CM
    - 493.12 Intrinsic asthma with (acute) exacerbation convert 493.12 to ICD-10-CM
  - 493.2 Chronic obstructive asthma
    - 493.20 Chronic obstructive asthma, unspecified convert 493.20 to ICD-10-CM
    - 493.21 Chronic obstructive asthma with status asthmaticus convert 493.21 to ICD-10-CM
    - 493.22 Chronic obstructive asthma with (acute) exacerbation convert 493.22 to ICD-10-CM
  - 493.8 Other forms of asthma
    - 493.81 Exercise induced bronchospasm convert 493.81 to ICD-10-CM
    - 493.82 Cough variant asthma convert 493.82 to ICD-10-CM
  - 493.9 Asthma unspecified
    - 493.90 Asthma,unspecified type, unspecified convert 493.90 to ICD-10-CM
    - 493.91 Asthma, unspecified type, with status asthmaticus convert 493.91 to ICD-10-CM
    - 493.92 Asthma, unspecified type, with (acute) exacerbation convert 493.92 to ICD-10-CM

Source : ICD9data.Com by Alkaline Software
http://www.icd9data.com/2015/Volume1/460-519/490-496/493/default.htm#493

Appendix 3: Prevalence Of Childhood Asthma , 2006-2012

Appendix 3.1 Prevalence of Lifetime Asthma in Middle and High School student in Florida

Percent of middle and high school students who have ever been told they have asthma Percent by County, All Genders, All Races, All Ethnicities, 10 to 20 Years old				
County	**2006**	**2008**	**2010**	**2012**
Florida	17	18	18.4	20.59
Alachua		21.23	19.93	23.56
Baker	18.35	19.9	21.33	22.59
Bay	17.33	18.53	19.64	19.71
Bradford	21.39	16.09	21.97	22.1
Brevard	17.77	17.95	20.5	19.92
Broward	13.02	15.3	16.2	17.77
Calhoun	19.62	21.06	24.04	20.64
Charlotte	14.65	15.83	18.51	21.96
Citrus	18.6	17.98	19.06	21.98
Clay	19.05	18.9	19.15	19.97
Collier	11.42	13.92	12.07	12.77
Columbia	18.39	20.04	23.76	21.91
DeSoto	13.36	12.15	15.77	16.78
Dixie	21.07	21.95	22.48	27.56
Duval	18.86	18.67	19.9	22.97
Escambia	19.2	20.22	19.1	22.24
Flagler	16.05	16.8	19.89	22.93
Franklin	20.45	24.98	30.8	25.64
Gadsden	18.99	16.94	17.58	21.91
Gilchrist	21.94	20.29	22.39	22.23
Glades	12.57	13.93	16.87	19.51
Gulf	22.28	22.69	21.42	17.52
Hamilton	16.43	20.6	24.91	16.02
Hardee	16.06	13.89	16.43	18
Hendry	16.14	17.15	20.51	19.44
Hernando	19.51	20.53	21.96	25.65
Highlands	16.78	21.68	20.11	21.13
Hillsborough	18.95	21.15	18.39	23.17
Holmes	15.93	15.62	17.36	18.91
Indian River	16.94	17.36	14.7	18.36
Jackson	18.74	21.98	21.76	22.24
Jefferson	14.5	16.69	15.33	22.79
Lafayette	17.02	13.42	23.97	19.94
Lake	18	18.23	18.55	19.52

Source: Florida Department of Health, Bureau of Epidemiology, June 2016

Appendix 3.1 Prevalence of Lifetime Asthma in Middle and High School student in Florida ***(continued)***

Percent of middle and high school students who have ever been told they have asthma Percent by County, All Genders, All Races, All Ethnicities, 10 to 20 Years old				
County	**2006**	**2008**	**2010**	**2012**
Florida	17	18	18.4	20.59
Lee	14.53	17.23	16.47	17.83
Leon	16.47	17.97	21.17	19.53
Levy	19.29	19.26	21.82	21.18
Liberty	19.58	22.46	29.14	26.81
Madison	15.65	16.18	20.29	16.11
Manatee	17.4	15.74	18.25	20.02
Marion	17.3	19	20.23	20.53
Martin	15.41	16.21	17.3	16.74
Miami-Dade	16.63	17.12	18.71	21.45
Monroe	13.35	15.62	17.08	17.8
Nassau	13.77	20.15	17.33	21.13
Okaloosa		20.14	18.53	21.04
Okeechobee	15.51	18.38		20.22
Orange	14.83	17.95	17.92	20.48
Osceola	15.27	19.86	22.19	
Palm Beach	16.04	16.29	16.08	19.37
Pasco	17.25	18.58	20.9	22.14
Pinellas	16.69	20.89	16.73	21.02
Polk	18.35	20.51	20.51	22.7
Putnam	20.06	22.36	20.17	21.98
Santa Rosa	15.2	18.57	20.07	21.62
Sarasota	15.38		19.51	19.46
Seminole	16.56	17.36	18.37	19.86
St. Johns	14.88	18.03	19.64	19.75
St. Lucie	15.25	16.89	16.78	21.54
Sumter	16.51	19.96	19.36	22.76
Suwannee	18.82	18.9	18.76	20.3
Taylor	19.01	22.05	22.62	24.69
Union	20.28	20.71	20.24	23.31
Volusia	17.63	17.53	19.12	22.22
Wakulla	18.63	21.79	21.37	25.85
Walton	13.71	16.66	19.17	18.79
Washington	18.01	15.16	16.39	14.94

Source: Florida Department of Health, Bureau of Epidemiology, June 2016

Appendix 3.2 Crude rate of asthma emergency department visits per 10,000 for Florida

Crude rate of asthma emergency department visits per 10,000
Rate by County, All Genders, All Races, All Ethnicities, 0 to 17 Years old

County	2005	2006	2007	2008	2009	2010	2011	2012	2013	2014
Florida	86.01	85.28	83.83	83.56	98.31	103.78	104.39	115.44	116.05	118.77
Alachua	65.56	64.5	75.2	70.47	69.08	68.16	80.62	95.3	92.35	94.91
Baker	94.92	97	114.07	90.41	85.01	77.73	77.24	92.42	102.87	101.09
Bay	65.58	76.86	68.33	67.99	70.46	80.37	76.23	95.61	77.23	92.32
Bradford	121.46	97.74	102.09	94.87	110.75	95.98	95.27	126.26	123.51	80.83
Brevard	52.54	57.62	54.76	58.54	66.48	57.49	63.18	75.87	77.64	84.6
Broward	95.32	97.4	97.48	97.38	111.28	123.82	129.27	156.7	146.42	145.04
Calhoun	28.77	60.64	70.09	76.73	102.11	80.91	100.32	122.56	117.66	98.37
Charlotte	64.7	48.96	51.99	45.37	70.49	62.11	75.97	78.51	80.83	94.7
Citrus	54.04	45.67	56.17	44.4	52.31	41.97	47.61	55.48	50.88	65.66
Clay	52.82	45.74	53.31	53.59	67.17	53.17	49.6	63.16	75.36	88
Collier	54.3	59.58	54.07	60.34	66.83	66.1	65.34	71.47	81.73	86.26
Columbia	69.49	74.39	80.33	61.31	65.85	58.62	74.21	101.08	101.21	111.57
DeSoto	127.31	122.02	101.63	94.87	73.06	68.97	50.45	63.18	75.32	65.31
Dixie	24.46	36.04	35.93	29.69	*	22.51	29.3	26.02	49.65	23.29
Duval	114.01	105.4	109.76	110.49	121.17	109.55	112.05	132.86	151.22	151.81
Escambia	125.75	126.16	125.15	124.65	139.69	142.67	133	140.62	142.94	153.52
Flagler	70.95	104.42	90.69	91.46	91.16	107.91	104.86	88.84	86.86	89.37
Franklin	131.39	124.2	128.63	132.68	192.8	205.97	129.87	108.3	114.4	71.83
Gadsden	61.92	62.68	89.02	85.5	135.32	179.64	264.66	256.23	298.15	275.94
Gilchrist	38.1	38.38	29.58	24.43	30.1	29.33	26.89	*	27.51	50.18
Glades	46.86	62.45	79.65	62.4	62.57	74.1	45.85	*	49.27	53.15
Gulf	44.28	27.56	24.36	42.25	46.56	111.55	68.63	81.09	86.31	123.46
Hamilton	80.36	73.23	75.93	53.59	66.79	38.1	94.34	131.16	127.31	122.07
Hardee	111.02	89.12	74.18	89.59	85.65	113.99	103.74	118.77	107.51	82.1
Hendry	96.86	108.19	117.82	104.81	133.17	117.11	123.75	113.7	169.8	136.22
Hernando	57.06	59.85	66.27	59.12	72.91	55.89	73.35	86.98	107.2	99.82
Highlands	88.86	79.62	68.45	74.2	99.44	85.94	84.42	94.7	127.81	110.16
Hillsborough	85.93	75.04	72.42	72.35	86.29	90.38	85.58	90	89.77	101.99
Holmes	42.36	42.71	33.53	53.81	36.5	69.16	86.54	48.42	82.51	41.56
Indian River	63.74	64.84	76.08	52.89	78.25	73.35	72.69	83.04	69.04	102.86
Jackson	75.28	80.78	66.77	73.17	78.56	87.94	120.54	111.94	138.64	152.4
Jefferson	52.5	41.61	52.14	41.54	55.54	44.63	71.35	108.1	129.81	136.45
Lafayette	69.14	43.21	87.72	68.07	*	28.69	29.34	44.37	49.56	33.06
Lake	61.83	60.53	63.09	59.48	66.71	68.36	66.22	72.87	61.9	68.73

Source: EPHT , Florida Agency for Health Care Administration June 2016

Appendix 3.2 Crude rate of asthma emergency department visits per 10,000 for Florida…*continued*

Crude rate of asthma emergency department visits per 10,000 Rate by County, All Genders, All Races, All Ethnicities, 0 to 17 Years old										
County	**2005**	**2006**	**2007**	**2008**	**2009**	**2010**	**2011**	**2012**	**2013**	**2014**
Lee	70.61	75.51	71.04	75.53	94.59	107.1	113.91	118.85	106.45	104.33
Leon	59.57	53.7	58.34	58.36	73.37	67.17	77.58	82.29	100.91	110.22
Levy	76.34	60.86	63.28	41.95	57.76	49.2	64.95	79.6	67.84	107.79
Liberty	59.95	164.51	91.95	67.64	119.39	127.98	140.52	107.43	122.69	148.28
Madison	91.2	110.47	228.28	174.26	178.19	214.72	233.96	205.9	225.73	156.49
Manatee	82.09	60.73	55.58	57.57	65.54	75.51	67.67	80.84	91.37	90.87
Marion	117.31	84.82	80.77	85.93	120.96	136.75	119.54	132.32	120.78	111.4
Martin	45.31	44.62	38.82	33.59	48.74	60.1	58.58	51.43	50.82	60.15
Miami-Dade	116.05	122.81	104.12	102.03	121.91	147.97	146.4	155.13	144.86	142.03
Monroe	40.15	63.79	44.68	62.31	87.11	84.46	92.87	81.78	82.07	108.73
Nassau	61.74	53.05	61.6	81.04	78.83	67.01	73.8	81.55	86.18	93
Okaloosa	51.35	55.83	55.56	58.73	63.25	66.22	97.46	122.38	143.51	129.36
Okeechobee	90.7	60.35	90.78	79.83	78.03	84.75	96.81	87.09	107.21	93.59
Orange	68.76	66.34	78.93	79.03	103.11	114.24	112.91	122.68	132.66	128.28
Osceola	90.43	98.48	91.84	79.18	92.62	103.81	110.01	148.92	163.48	203.67
Palm Beach	66.03	60.49	68.56	76.9	94.37	101.16	96.91	102.71	109.53	110.06
Pasco	54.1	46.58	52.85	50.93	61.21	58.46	62.99	73.52	72.48	71.9
Pinellas	90.17	93.71	82.55	80.72	101.28	101.41	101.72	108.26	99.29	103.6
Polk	116.29	140.85	147.18	153.85	151.83	144.38	143	153.76	157.83	169.9
Putnam	109.17	80.35	89.49	84.22	124.96	93.89	109.12	147.06	165.1	134.93
Santa Rosa	60.48	57.17	56.74	56.81	67.59	60.28	64.76	73.19	66.27	68.38
Sarasota	69	62.69	59.56	56.21	67.93	74.57	66.42	67.3	63.68	57.15
Seminole	51.4	50.74	48.14	55.03	61.36	69.2	60.18	64.34	66.17	71.16
St. Johns	51.12	43.83	43.19	53.54	68.93	53.94	46.88	53.6	57.32	44.49
St. Lucie	68.54	58.05	57.89	56.37	75.64	78.66	81.59	88.07	94.98	103.71
Sumter	77.36	50.15	52.3	38.49	52.42	105.93	73.96	82.52	73.05	81.63
Suwannee	54.63	70.99	74.67	94.46	82	79.29	127.86	137.13	122.79	111.16
Taylor	89.9	82.73	101.93	113.09	97.58	86.96	89.82	71.28	87.5	61.08
Union	102.96	128.5	99.14	135.35	152.48	97.22	78.26	106.33	109.44	156.34
Volusia	134.32	141	143.32	121.92	138.1	136.02	129.62	140	130.85	157.54
Wakulla	35.84	40.7	42.02	44.16	37.09	29.46	39.9	50.03	68.22	97.41
Walton	70.38	55.23	61.73	39.74	78.45	63.1	69.53	83.68	87.54	75.68
Washington	76.44	73.04	74.93	64.87	69.82	80.41	83.58	59.61	110.79	68.88

Source: EPHT , Florida Agency for Health Care Administration June 2016

Appendix 3.3 Lifetime Adolescent Asthma Prevalence by Race/Ethnicity, FYTS 2006 – 2012

Year	Non - Hispanic White		Non - Hispanic Black		Hispanic		Other	
	Mean	95% CI	Mean	95% CI	Mean	95% CI	Mean	95% CI
2006	16.2	(15.7 - 16.7)	17.7	(16.7 - 18.7)	17.7	(16.7 - 18.8)	18.8	(17.3 - 19.9)
2008	16.8	(16.2 - 17.4)	20.2	(19.2 - 21.3)	18.2	(17.2 - 19.3)	18.5	(17.2 - 19.9)
2010	17.4	(16.8 - 18.0)	20.1	(18.9 - 21.3)	18.6	(17.7 - 19.6)	18.5	(17.3 - 19.7)
2012	18.9	(18.3 - 19.4)	21.8	(20.7 - 22.9)	21.8	(20.9 - 22.8)	21.6	(20.1 - 23.1)

Source: FAP 2014: *Asthma burden 2013.*

Appendix 3.4 Lifetime Adolescent Asthma Prevalence, YRBS 2011

	Florida		US	
	Mean	95% CI	Mean	95% CI
Total	21.7	(20.5 - 22.9)	23.0	(21.7 - 24.3)
Male	23.4	(22.0 - 24.8)	23.2	(21.8 - 24.6)
Female	20.0	(18.4 - 21.7)	22.8	(21.2 - 24.5)
Non-Hispanic White	19.7	(17.9 - 21.7)	22.8	(21.2 - 24.5)
Non-Hispanic Black	22.0	(19.6 - 24.5)	26.8	(24.1 - 29.6)
Hispanic	24.5	(22.3 - 27.0)	20.3	(17.9 - 23.0)

Source: FAP2014: *Asthma burden 2013.*

Appendix 3.5 Current Adolescent Asthma Prevalence, YRBS 2011

	Florida		US Median	
	Mean	95% CI	Mean	95% CI
Total	10.2	(9.5 - 11.0)	11.9	(10.9 - 12.9)
Male	9.7	(8.7 - 10.9)	10.4	(9.4 - 11.4)
Female	10.8	(9.9 - 11.9)	13.5	(12.1 - 15.1)
Non-Hispanic White	9.8	(8.6 - 11.1)	12.4	(11.2 - 13.8)
Non-Hispanic Black	11.7	(9.8 - 13.8)	13.5	(11.7 - 15.6)
Hispanic	9.5	(8.1 - 11.2)	9.1	(7.4 - 11.1)

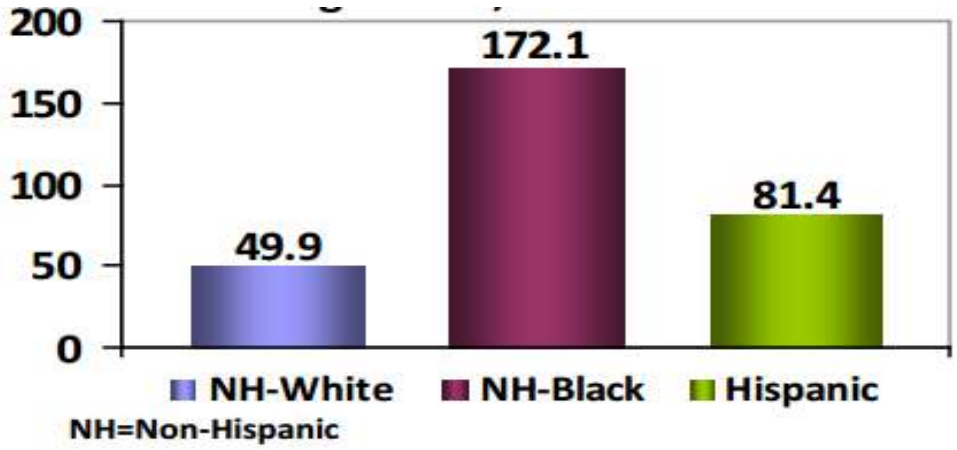
Source: FAP 2014: *Asthma burden 2013.*

Appendix 3.6 Current Adolescent Asthma Prevalence, YRBS 2007 – 2011

Year	Florida		US Median	
	Mean	95% CI	Mean	95% CI
2007	9.4	(8.2 - 10.6)	10.9	(10.1 - 11.9)
2009	9.0	(8.4 - 9.6)	10.8	(9.9 - 11.7)
2011	10.2	(9.5 - 10.9)	11.9	(10.9 - 12.9)

Source: FAP 2014: *Asthma burden 2013.*

Appendix 3.7 Asthma ED Rates per 10,000 Ages 5-17, 2011 AHCA



Source: FAP2014: *Asthma burden 2013.*

Appendix 4: Ambient $PM_{2.5}$ (in µg/m^3) concentration by Florida Counties

Average ambient concentrations of particulate matter ($PM_{2.5}$ per ug/m^3)										
Count by County										
County	**2002**	**2003**	**2004**	**2005**	**2006**	**2007**	**2008**	**2009**	**2010**	**2011**
Alachua	9.88	9.68	9.45	9.74	9.34	10.63	7.93	7.2	7.8	8.3
Bay	10.64	10.77	11.71	11.36	12.01	10.93	9.9	8.9		
Brevard	7.56	7.45	8.62	8.32	8.99	7.29	7.43	6.5	6.9	6.5
Broward	8.03	8.16	8.38	8.32	8.51	8.19	7.69	7	7	6.8
Citrus	8.63	8.68	9.42	9.16	9.27	8.59	7.81	6.9	7.7	7.6
Duval	10.19	9.8	10.86	10.66	10.17	10.42	9.24	8.3	9.4	9
Escambia	10.95	11.18	12.84	12.04	11.61	10.88	9.47	8.4	9.1	9.5
Hillsborough	10.75	10.48	11.46	11.12	9.99	10.52	8.51	7.9	8.2	7.8
Lee	7.81	7.98	8.59	8.7	8.28	8.22	7.07	6.6	7	7.2
Leon	12.92	11.83	13.15	13.11	12.55	12.31	10.64	9.7	9.8	10.3
Manatee	8.91	8.42	9.11	8.91	8.74	8.52				
Marion	9.82	9.3	10.62	10.5	9.51	10.01				
Miami-Dade	9.13	9.4	9.59	9.69	9.53	8.93	8.1	7.6	7.8	7.4
Okeechobee								7	7.5	7.3
Orange	9.69	9.44	10.05	9.79	9.33	8.99	7.52			
Palm Beach	7.31	8.1	8.13	8.07	8.17	7.3	6.56	6.1	6.4	6.4
Pinellas	10.34	9.33	10.27	10.42	9.55	9.68	8.05	7.4	8.2	7.6
Polk	10.09	9.19	10.34	9.86	9.51	9.36	8.16	7.3	7.8	7.5
Santa Rosa		9.89								
Sarasota	8.86	8.58	8.9	9.26	8.9	8.11	6.9	6.6	7.2	6.9
Seminole	8.85	8.56	9.98	10	9.55	9.47	7.98	7.4	7.7	7.6
St. Lucie	8.01	8.29	7.74	9.05	8.92	7.82	7.64	7.7	7.9	
Volusia	8.75	8.52	9.74	9.21	9.01	9.8			7.5	7.6

Source : Environmental Public Health Tracking program, Florida Department of Health, http://www.floridatracking.com/HealthTrackFL/report.aspx, accessed on 06-06-2016

Appendix 5: Prevalence Of Asthma In Florida Gender And Ethnicity And County

	Male				Female				All Races
County	**White**	**Black**	**Hispanic**	**Other**	**White**	**Black**	**Hispanic**	**Other**	
Florida									
Alachua County	2.97	15.43	5.24	4.83	2.21	8.38	0.61	1.09	5.46
Baker County	3.32	13.30	0.00	0.00	2.60	6.62	0.00	0.00	3.57
Bay County	2.53	12.75	0.90	5.19	3.05	7.94	0.86	0.00	3.68
Bradford County	2.68	20.09	0.00	17.54	1.07	12.63	0.00	0.00	4.59
Brevard County	2.85	12.08	3.36	3.53	1.79	6.64	2.75	1.36	3.41
Broward County	4.76	16.54	7.11	5.17	3.22	9.27	3.75	2.36	7.39
Calhoun County	2.69	0.00	0.00	0.00	3.01	0.00	0.00	0.00	2.17
Charlotte County	5.62	8.64	6.26	0.00	3.58	8.27	3.03	3.28	4.77
Citrus County	3.42	20.62	2.18	0.00	1.20	6.73	0.00	0.00	2.57
Clay County	4.41	10.71	2.51	4.42	1.61	8.22	3.08	2.81	3.79
Collier County	4.07	9.30	7.89	16.67	1.76	6.06	4.46	9.23	5.18
Columbia County	4.11	14.41	2.51	2.99	2.22	6.35	4.55	3.07	4.66
DeSoto County	1.38	4.69	3.13	15.15	6.36	2.99	0.79	0.00	3.16
Dixie County	2.34	0.00	0.00	0.00	0.87	0.00	0.00	0.00	1.41
Duval County	4.33	24.47	6.21	3.88	2.70	13.64	2.05	1.79	9.28
Escambia County	5.65	30.18	5.90	2.90	2.17	17.10	3.85	0.00	9.98
Flagler County	4.13	6.89	1.79	2.98	2.42	6.48	2.78	0.00	3.53
Franklin County	7.73	28.57	0.00	0.00	1.42	0.00	0.00	0.00	5.13
Gadsden County	8.70	22.57	19.02	11.11	1.29	20.09	2.52	0.00	16.60
Gilchrist County	1.44	0.00	8.06	0.00	1.49	0.00	7.69	0.00	1.87
Glades County	2.14	28.57	2.45	0.00	8.16	15.15	6.02	8.62	5.71
Gulf County	3.24	13.51	0.00	0.00	2.17	0.00	0.00	0.00	3.11
Hamilton County	7.25	10.96	0.00	0.00	5.47	4.90	0.00	0.00	5.90
Hardee County	5.64	8.77	4.42	0.00	3.90	5.21	2.92	0.00	4.08
Hendry County	12.43	33.87	5.47	9.90	0.93	17.67	6.08	10.53	8.74

Appendix 5: Prevalence Of Asthma In Florida Gender And Ethnicity And County *(Continued)*

	Male				Female				All Races
County	**White**	**Black**	**Hispanic**	**Other**	**White**	**Black**	**Hispanic**	**Other**	
Florida									
Hernando County	3.02	20.47	5.43	0.00	1.89	5.96	1.27	0.00	3.17
Highlands County	5.10	18.84	5.05	0.00	1.89	3.50	2.15	4.33	4.61
Hillsborough County	2.83	14.02	7.86	2.52	1.89	7.62	3.67	1.34	5.02
Holmes County	9.24	21.74	0.00	11.63	3.81	20.00	0.00	0.00	6.85
Indian River County	2.24	14.07	7.36	0.00	1.18	6.76	0.91	6.83	3.59
Jackson County	3.68	33.45	0.00	0.00	2.88	11.40	6.10	0.00	8.12
Jefferson County	3.55	5.62	9.62	0.00	0.00	3.73	15.15	0.00	3.66
Lafayette County	3.19	0.00	0.00	0.00	0.00	0.00	0.00	0.00	1.20
Lake County	3.84	21.56	5.22	4.73	2.31	8.02	3.07	1.66	4.91
Lee County	4.50	15.77	7.83	4.39	3.33	8.86	5.77	1.06	5.83
Leon County	2.93	16.05	0.69	2.18	1.95	12.84	0.74	0.54	6.75
Levy County	1.72	25.32	0.00	6.76	1.58	2.06	0.00	7.14	3.18
Liberty County	5.92	21.74	0.00	0.00	6.69	0.00	0.00	0.00	6.08
Madison County	11.82	40.32	0.00	0.00	5.18	37.41	0.00	0.00	21.12
Manatee County	3.60	10.09	4.40	2.57	1.22	8.49	1.87	0.00	3.46
Marion County	4.95	20.67	7.78	3.79	3.95	10.37	4.20	3.80	6.63
Martin County	3.24	9.28	5.69	2.02	0.93	6.58	2.75	2.06	2.96
Miami-Dade County	2.85	13.03	10.30	10.30	1.45	7.80	5.94	5.89	7.71
Monroe County	5.30	39.60	6.40	5.32	2.38	13.66	3.98	0.00	6.19
Nassau County	3.74	13.57	0.00	3.65	2.70	4.02	0.00	0.00	3.42
Okaloosa County	4.48	22.18	9.71	7.42	3.37	15.91	1.81	2.20	5.87
Okeechobee County	8.15	10.71	5.26	4.63	5.41	6.80	2.65	9.09	5.88
Orange County	4.82	18.60	10.34	4.83	2.54	11.15	6.07	2.17	7.85
Osceola County	4.74	14.95	12.36	6.71	3.03	5.82	7.86	2.12	7.93

Appendix 5: Prevalence Of Asthma In Florida Gender And Ethnicity And County *(Continued)*

	Male				Female				All Races
County	**White**	**Black**	**Hispanic**	**Other**	**White**	**Black**	**Hispanic**	**Other**	
Florida									
Palm_Beach County	3.78	15.99	4.91	8.10	1.72	9.63	2.84	2.62	5.71
Pasco County	3.57	9.68	3.63	2.77	2.36	12.36	2.69	1.97	3.43
Pinellas County	3.95	26.24	5.27	4.72	3.40	14.18	2.95	3.22	6.46
Polk County	6.15	26.35	10.03	2.61	3.72	12.61	5.40	1.17	8.25
Putnam County	3.41	14.07	2.55	10.20	2.63	8.73	2.70	3.65	4.93
Santa_Rosa County	6.21	16.20	0.00	1.68	3.58	9.09	2.16	0.00	4.78
Sarasota County	3.25	16.70	8.45	3.11	1.83	11.84	3.71	1.78	4.08
Seminole County	2.53	21.79	6.87	4.85	1.46	11.06	3.82	1.04	4.60
St. Johns County	2.13	23.60	2.66	2.18	1.47	9.42	1.38	0.76	2.78
St_Lucie County	3.35	13.14	5.30	4.63	1.39	7.27	2.03	3.77	4.86
Sumter County	4.67	18.45	3.70	5.10	0.84	7.49	1.97	5.26	4.42
Suwannee County	5.87	34.69	3.19	24.19	1.72	9.47	5.66	0.00	6.27
Taylor County	6.23	19.02	0.00	0.00	0.75	12.95	0.00	0.00	5.73
Union County	5.86	5.88	0.00	0.00	3.95	5.75	0.00	0.00	4.59
Volusia County	6.62	34.51	10.02	4.28	3.54	15.33	4.58	1.25	8.12
Wakulla County	2.92	15.71	0.00	0.00	1.27	3.09	8.93	0.00	2.99
Walton County	5.14	10.58	3.03	3.45	1.53	14.04	0.00	0.00	3.77
Washington County	4.95	23.12	0.00	0.00	1.25	13.33	11.90	0.00	5.27

Appendix 6: Prevalence Of Asthma Exacerbation In Florida Gender And Ethnicity And County

	Male				Female				All Races
County	**White**	**Black**	**Hispanic**	**Other**	**White**	**Black**	**Hispanic**	**Other**	
Florida									
Alachua County	2.97	15.43	5.24	4.83	2.21	8.38	0.61	1.09	5.46
Baker County	3.32	13.30	0.00	0.00	2.60	6.62	0.00	0.00	3.57
Bay County	2.53	12.75	0.90	5.19	3.05	7.94	0.86	0.00	3.68
Bradford County	2.68	20.09	0.00	17.54	1.07	12.63	0.00	0.00	4.59
Brevard County	2.85	12.08	3.36	3.53	1.79	6.64	2.75	1.36	3.41
Broward County	4.76	16.54	7.11	5.17	3.22	9.27	3.75	2.36	7.39
Calhoun County	2.69	0.00	0.00	0.00	3.01	0.00	0.00	0.00	2.17
Charlotte County	5.62	8.64	6.26	0.00	3.58	8.27	3.03	3.28	4.77
Citrus County	3.42	20.62	2.18	0.00	1.20	6.73	0.00	0.00	2.57
Clay County	4.41	10.71	2.51	4.42	1.61	8.22	3.08	2.81	3.79
Collier County	4.07	9.30	7.89	16.67	1.76	6.06	4.46	9.23	5.18
Columbia County	4.11	14.41	2.51	2.99	2.22	6.35	4.55	3.07	4.66
DeSoto County	1.38	4.69	3.13	15.15	6.36	2.99	0.79	0.00	3.16
Dixie County	2.34	0.00	0.00	0.00	0.87	0.00	0.00	0.00	1.41
Duval County	4.33	24.47	6.21	3.88	2.70	13.64	2.05	1.79	9.28
Escambia County	5.65	30.18	5.90	2.90	2.17	17.10	3.85	0.00	9.98
Flagler County	4.13	6.89	1.79	2.98	2.42	6.48	2.78	0.00	3.53
Franklin County	7.73	28.57	0.00	0.00	1.42	0.00	0.00	0.00	5.13
Gadsden County	8.70	22.57	19.02	11.11	1.29	20.09	2.52	0.00	16.60
Gilchrist County	1.44	0.00	8.06	0.00	1.49	0.00	7.69	0.00	1.87
Glades County	2.14	28.57	2.45	0.00	8.16	15.15	6.02	8.62	5.71
Gulf County	3.24	13.51	0.00	0.00	2.17	0.00	0.00	0.00	3.11
Hamilton County	7.25	10.96	0.00	0.00	5.47	4.90	0.00	0.00	5.90
Hardee County	5.64	8.77	4.42	0.00	3.90	5.21	2.92	0.00	4.08
Hendry County	12.43	33.87	5.47	9.90	0.93	17.67	6.08	10.53	8.74

Appendix 6: Prevalence Of Asthma Exacerbation In Florida Gender And Ethnicity And County *(Continued)*

	Male				Female				All Races
County	**White**	**Black**	**Hispanic**	**Other**	**White**	**Black**	**Hispanic**	**Other**	
Florida									
Hernando County	3.02	20.47	5.43	0.00	1.89	5.96	1.27	0.00	3.17
Highlands County	5.10	18.84	5.05	0.00	1.89	3.50	2.15	4.33	4.61
Hillsborough County	2.83	14.02	7.86	2.52	1.89	7.62	3.67	1.34	5.02
Holmes County	9.24	21.74	0.00	11.63	3.81	20.00	0.00	0.00	6.85
Indian River County	2.24	14.07	7.36	0.00	1.18	6.76	0.91	6.83	3.59
Jackson County	3.68	33.45	0.00	0.00	2.88	11.40	6.10	0.00	8.12
Jefferson County	3.55	5.62	9.62	0.00	0.00	3.73	15.15	0.00	3.66
Lafayette County	3.19	0.00	0.00	0.00	0.00	0.00	0.00	0.00	1.20
Lake County	3.84	21.56	5.22	4.73	2.31	8.02	3.07	1.66	4.91
Lee County	4.50	15.77	7.83	4.39	3.33	8.86	5.77	1.06	5.83
Leon County	2.93	16.05	0.69	2.18	1.95	12.84	0.74	0.54	6.75
Levy County	1.72	25.32	0.00	6.76	1.58	2.06	0.00	7.14	3.18
Liberty County	5.92	21.74	0.00	0.00	6.69	0.00	0.00	0.00	6.08
Madison County	11.82	40.32	0.00	0.00	5.18	37.41	0.00	0.00	21.12
Manatee County	3.60	10.09	4.40	2.57	1.22	8.49	1.87	0.00	3.46
Marion County	4.95	20.67	7.78	3.79	3.95	10.37	4.20	3.80	6.63
Martin County	3.24	9.28	5.69	2.02	0.93	6.58	2.75	2.06	2.96
Miami-Dade County	2.85	13.03	10.30	10.30	1.45	7.80	5.94	5.89	7.71
Monroe County	5.30	39.60	6.40	5.32	2.38	13.66	3.98	0.00	6.19
Nassau County	3.74	13.57	0.00	3.65	2.70	4.02	0.00	0.00	3.42
Okaloosa County	4.48	22.18	9.71	7.42	3.37	15.91	1.81	2.20	5.87
Okeechobee County	8.15	10.71	5.26	4.63	5.41	6.80	2.65	9.09	5.88
Orange County	4.82	18.60	10.34	4.83	2.54	11.15	6.07	2.17	7.85
Osceola County	4.74	14.95	12.36	6.71	3.03	5.82	7.86	2.12	7.93

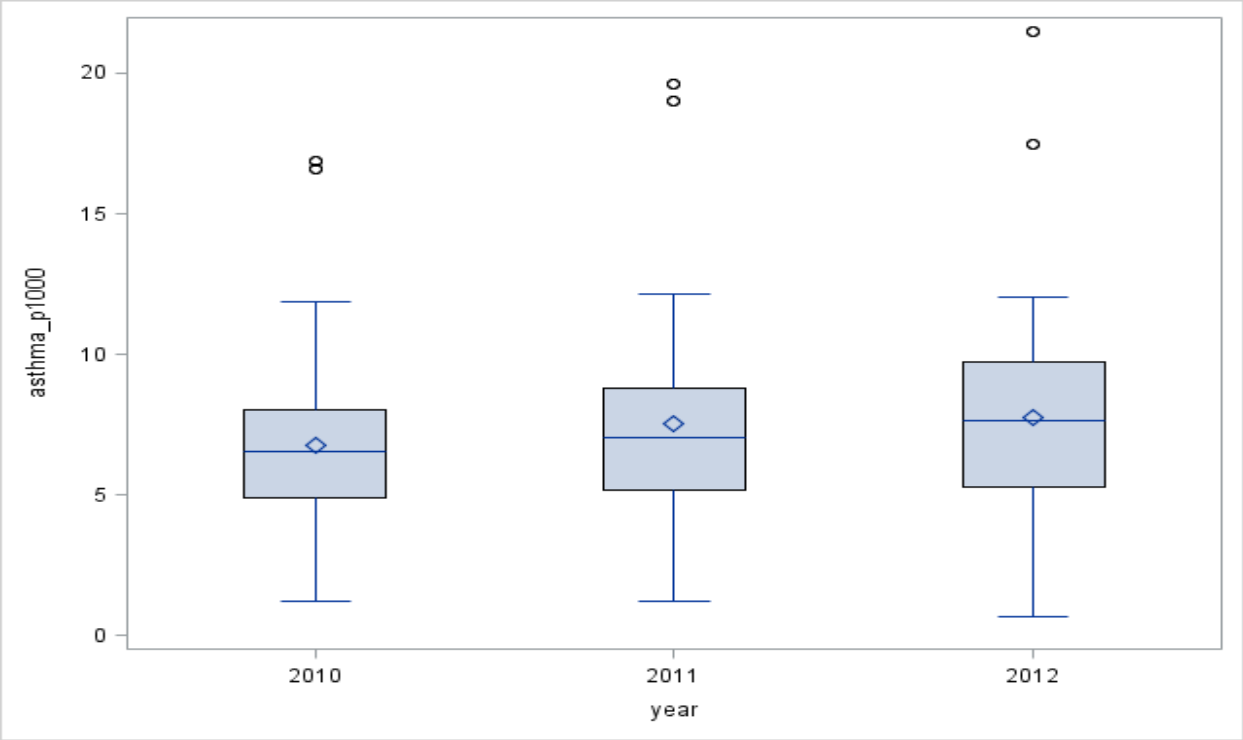
Appendix 6: Prevalence Of Asthma Exacerbation In Florida Gender And Ethnicity And County *(Continued)*

	Male				Female				All Races
County	**White**	**Black**	**Hispanic**	**Other**	**White**	**Black**	**Hispanic**	**Other**	
Florida									
Palm_Beach County	3.78	15.99	4.91	8.10	1.72	9.63	2.84	2.62	5.71
Pasco County	3.57	9.68	3.63	2.77	2.36	12.36	2.69	1.97	3.43
Pinellas County	3.95	26.24	5.27	4.72	3.40	14.18	2.95	3.22	6.46
Polk County	6.15	26.35	10.03	2.61	3.72	12.61	5.40	1.17	8.25
Putnam County	3.41	14.07	2.55	10.20	2.63	8.73	2.70	3.65	4.93
Santa_Rosa County	6.21	16.20	0.00	1.68	3.58	9.09	2.16	0.00	4.78
Sarasota County	3.25	16.70	8.45	3.11	1.83	11.84	3.71	1.78	4.08
Seminole County	2.53	21.79	6.87	4.85	1.46	11.06	3.82	1.04	4.60
St. Johns County	2.13	23.60	2.66	2.18	1.47	9.42	1.38	0.76	2.78
St_Lucie County	3.35	13.14	5.30	4.63	1.39	7.27	2.03	3.77	4.86
Sumter County	4.67	18.45	3.70	5.10	0.84	7.49	1.97	5.26	4.42
Suwannee County	5.87	34.69	3.19	24.19	1.72	9.47	5.66	0.00	6.27
Taylor County	6.23	19.02	0.00	0.00	0.75	12.95	0.00	0.00	5.73
Union County	5.86	5.88	0.00	0.00	3.95	5.75	0.00	0.00	4.59
Volusia County	6.62	34.51	10.02	4.28	3.54	15.33	4.58	1.25	8.12
Wakulla County	2.92	15.71	0.00	0.00	1.27	3.09	8.93	0.00	2.99
Walton County	5.14	10.58	3.03	3.45	1.53	14.04	0.00	0.00	3.77
Washington County	4.95	23.12	0.00	0.00	1.25	13.33	11.90	0.00	5.27

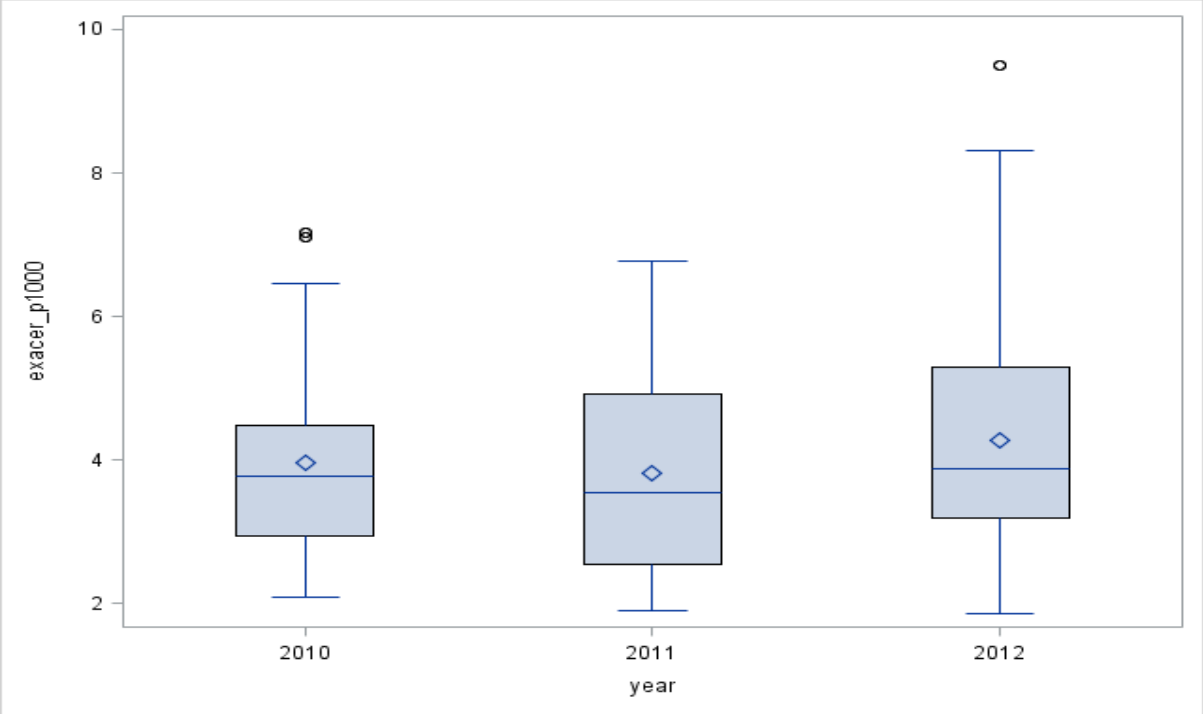
Appendix 7: Summary Statistics of Comparison ED Visits by Rural and Urban

Appendix 7.1: Distribution of Asthma ED visits per 1000 for Urban Florida Counties

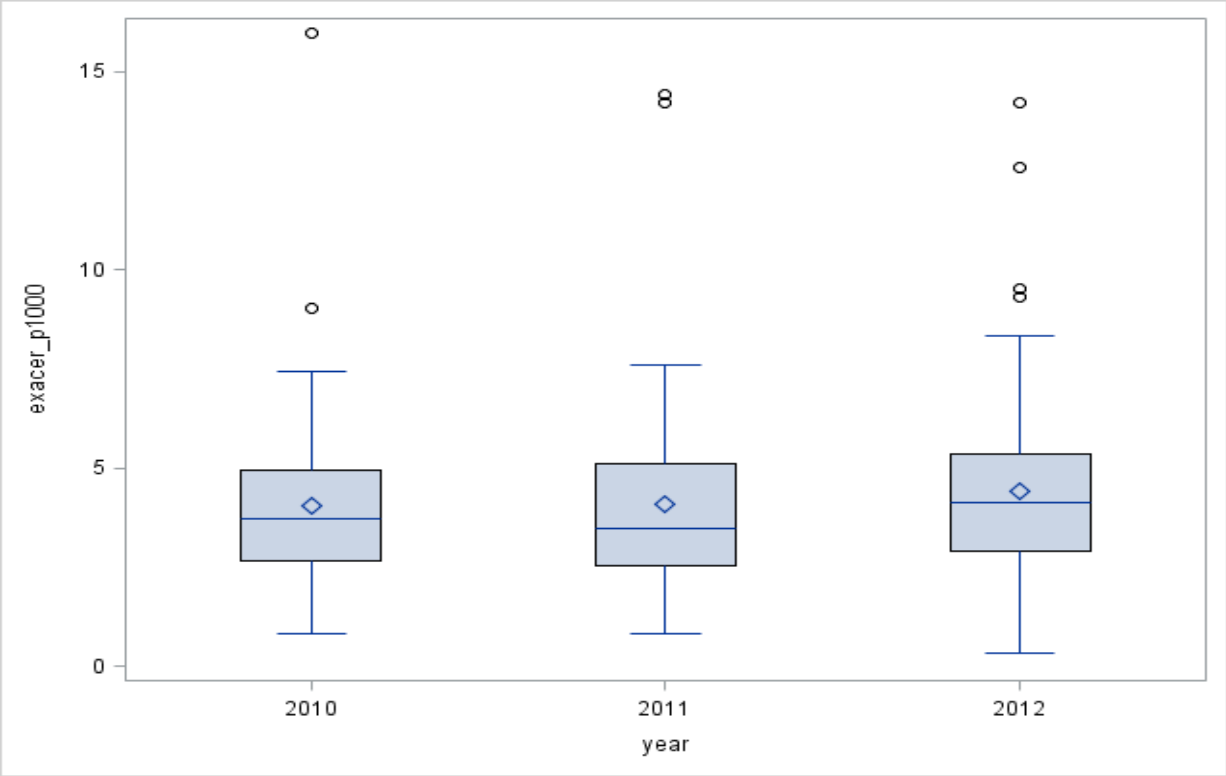
Appendix 7.2: Distribution of Asthma ED visits per 1000 for Rural Florida Counties Rural



Appendix 7.3: Distribution of Asthma Exacerbation ED visits per 1000 for Urban Florida Counties

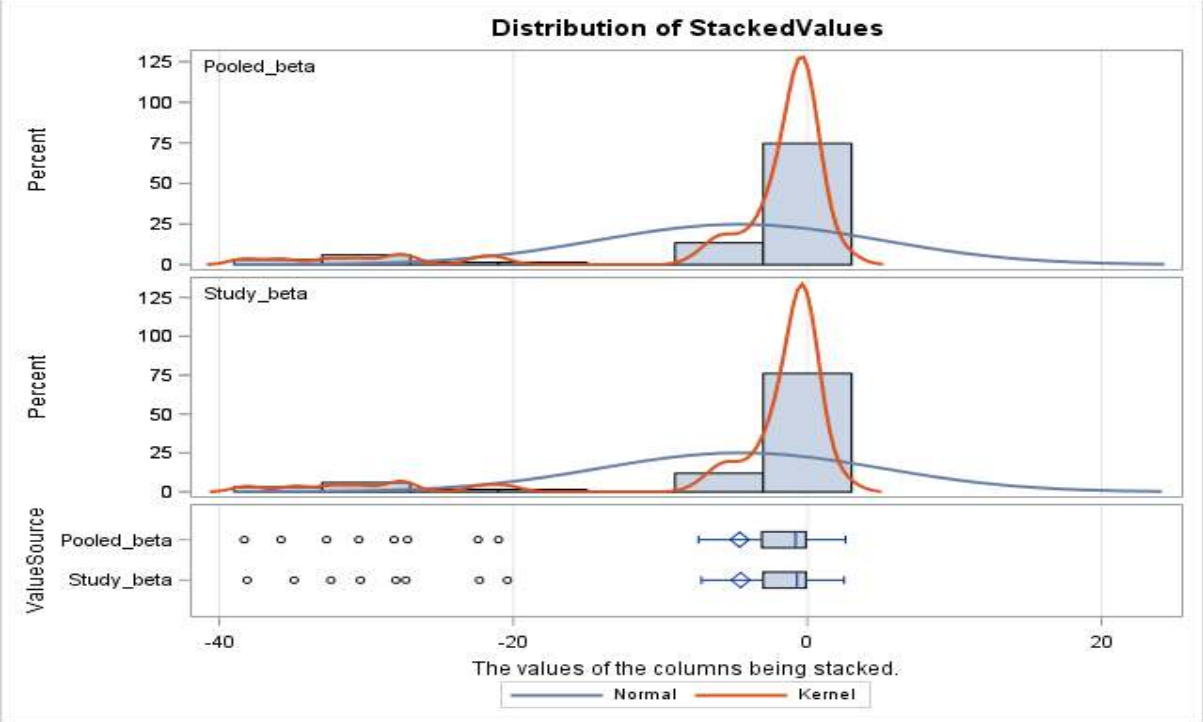


Appendix 7.4: Distribution of Asthma Exacerbation ED visits per 1000 for Rural Florida Counties Rural

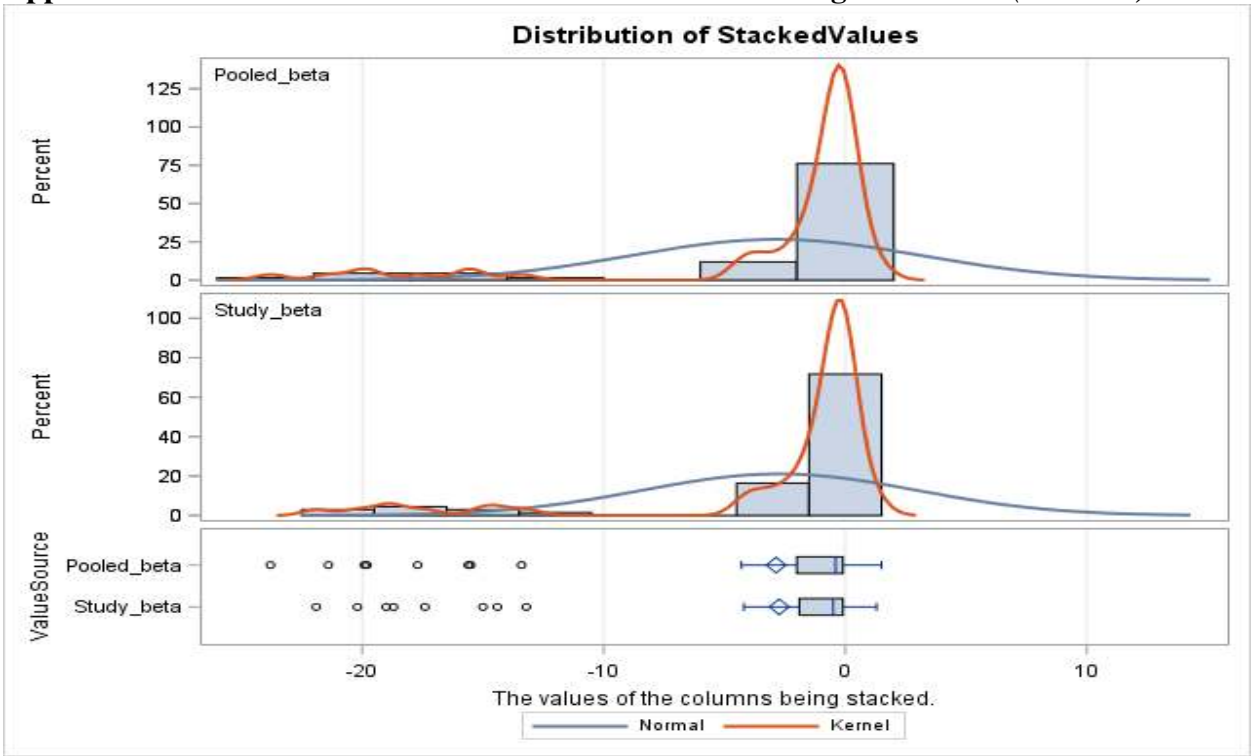


Appendix 8: Summary Statistics of Comparison in BenMAP

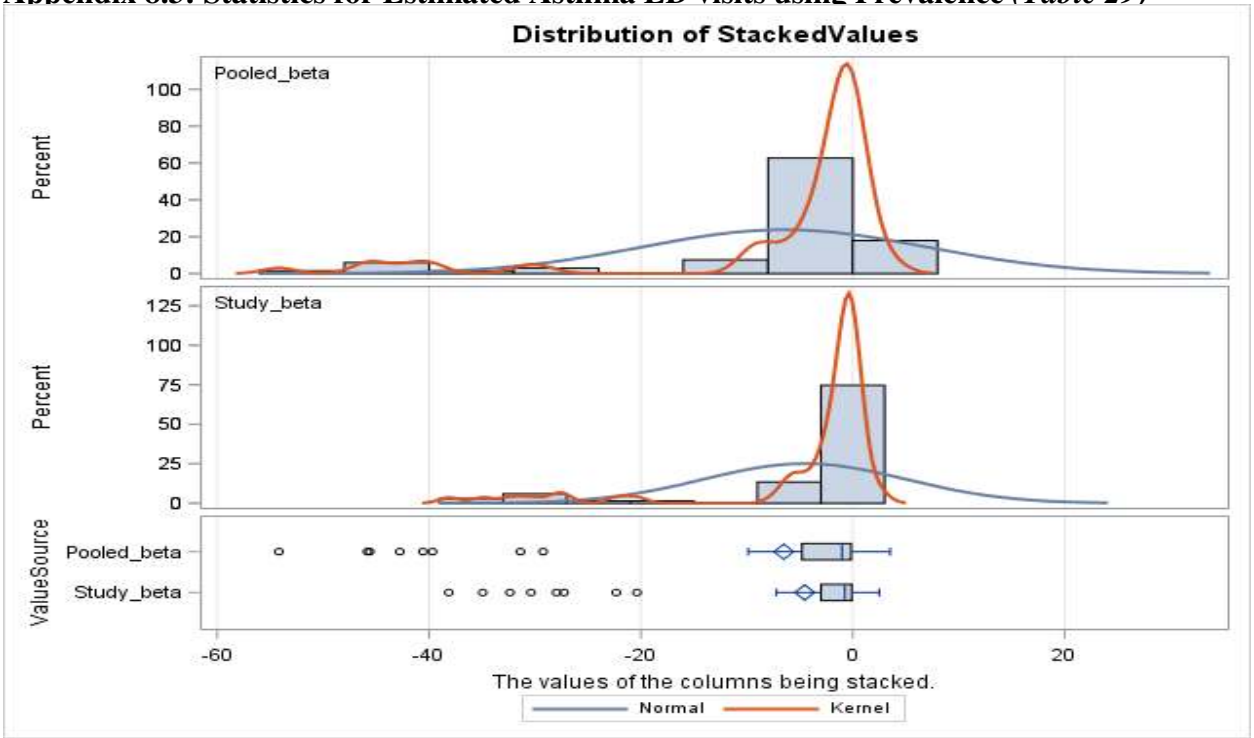
Appendix 8.1: Statistics for Estimated Asthma ED visits using Prevalence *(Table 27)*



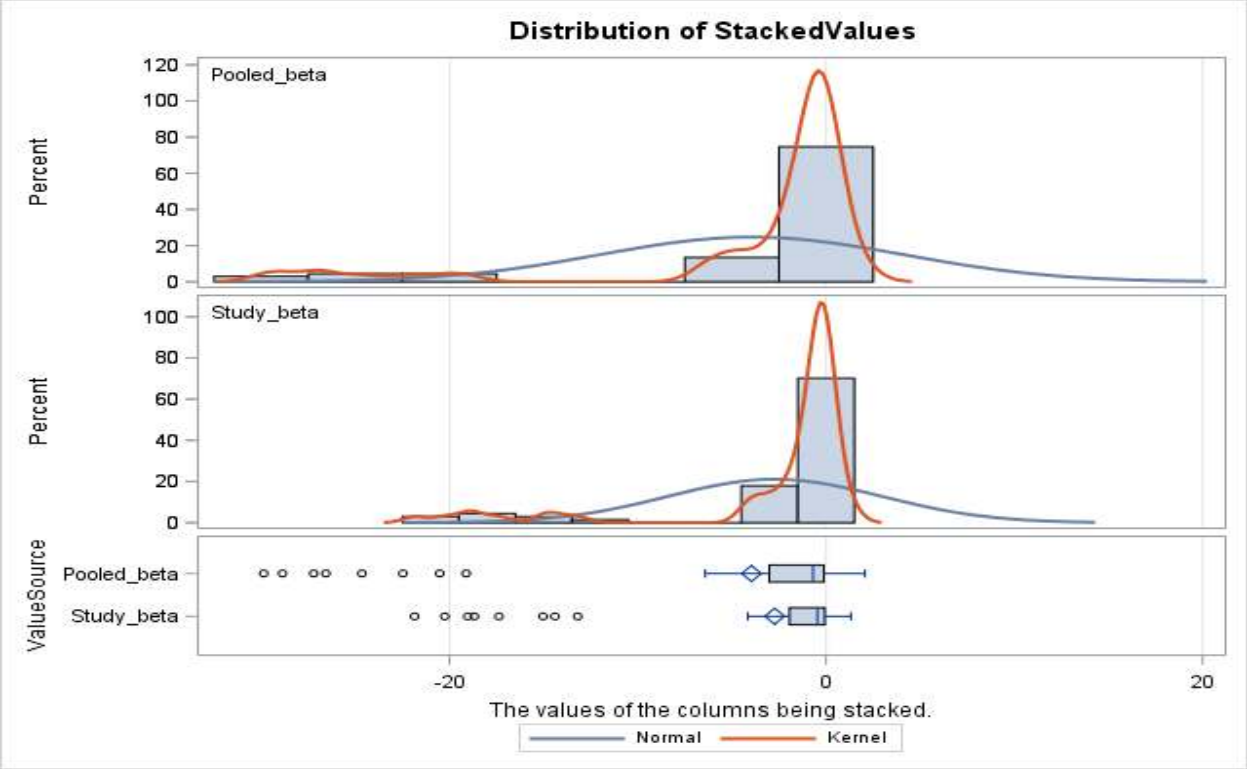
Appendix 8.2: Statistics for Estimated Asthma ED visits using Prevalence *(Table 28)*



Appendix 8.3: Statistics for Estimated Asthma ED visits using Prevalence *(Table 29)*



Appendix 8.4 :Statistics for Estimated Asthma Exacerbation ED visits using Prevalence *(Table 30)*



Appendix 9: USF-IRB Approval Letter

USF
UNIVERSITY OF SOUTH FLORIDA

RESEARCH INTEGRITY AND COMPLIANCE
Institutional Review Boards, FWA No. 00001669
12901 Bruce B. Downs Blvd., MDC035 • Tampa, FL 33612-4799
(813) 974-5638 • FAX(813)974-7091

8/17/2015

Shabnam Mehra
Thomas Mason
Environmental and Occupational Health
4202 East Fowler Avenue
Tampa, FL 33620

RE: **Expedited Approval for Initial Review**
IRB#: Pro00023392
Title: Estimating The Impact of Select Criteria Pollutants on Childhood Asthma in Florida

Study Approval Period: 8/17/2015 to 8/17/2016

Dear Shabnam Mehra and Thomas Mason:

On 8/17/2015, the Institutional Review Board (IRB) reviewed and **APPROVED** the above application and all documents contained within, including those outlined below.

Approved Item(s):
Protocol Document(s):
ICPPA 2015
ICPPA- ACHA Proposal

It was the determination of the IRB that your study qualified for expedited review which includes activities that (1) present no more than minimal risk to human subjects, and (2) involve only procedures listed in one or more of the categories outlined below. The IRB may review research through the expedited review procedure authorized by 45CFR46.110 and 21 CFR 56.110. The research proposed in this study is categorized under the following expedited review category:

(5) Research involving materials (data, documents, records, or specimens) that have been collected, or will be collected solely for nonresearch purposes (such as medical treatment or diagnosis).

Your study qualifies for a waiver of the requirements for the informed consent process as outlined in the federal regulations at 45CFR46.116 (d) which states that an IRB may approve a consent procedure which does not include, or which alters, some or all of the elements of

informed consent, or waive the requirements to obtain informed consent provided the IRB finds and documents that (1) the research involves no more than minimal risk to the subjects; (2) the waiver or alteration will not adversely affect the rights and welfare of the subjects; (3) the research could not practicably be carried out without the waiver or alteration; and (4) whenever appropriate, the subjects will be provided with additional pertinent information after participation.

Your study qualifies for a waiver of the requirement for signed authorization as outlined in the HIPAA Privacy Rule regulations at 45CFR164.512(i) which states that an IRB may approve a waiver or alteration of the authorization requirement provided that the following criteria are met (1) the PHI use or disclosure involves no more than a minimal risk to the privacy of individuals; (2) the research could not practicably be conducted without the requested waiver or alteration; and (3) the research could not practicably be conducted without access to and use of the PHI. A waiver of HIPAA Authorization is granted for this secondary data analysis. This waiver allows the study team to obtain and use PHI included in the data set that will be provided by the Florida Agency for Healthcare Administration (AHCA).

Per CFR 45 Part 46, Subpart D, this research involving children was approved under the minimal risk category 45 CFR 46.404: Research not involving greater than minimal risk.

As the principal investigator of this study, it is your responsibility to conduct this study in accordance with IRB policies and procedures and as approved by the IRB. Any changes to the approved research must be submitted to the IRB for review and approval via an amendment. Additionally, all unanticipated problems must be reported to the USF IRB within five (5) calendar days.

We appreciate your dedication to the ethical conduct of human subject research at the University of South Florida and your continued commitment to human research protections. If you have any questions regarding this matter, please call 813-974-5638.

Sincerely,

V Jorgensen MD

E. Verena Jorgensen, M.D., Chairperson
USF Institutional Review Board