Distribution Agreement

In presenting this thesis or dissertation as a partial fulfillment of the requirements for an advanced degree from Emory University, I hereby grant to Emory University and its agents the non-exclusive license to archive, make accessible, and display my thesis or dissertation in whole or in part in all forms of media, now or hereafter known, including display on the world wide web. I understand that I may select some access restrictions as part of the online submission of this thesis or dissertation. I retain all ownership rights to the copyright of the thesis or dissertation. I also retain the right to use in future works (such as articles or books) all or part of this thesis or dissertation.

Signature:

_____ _____

Xueying Zhang Date

The Application of Satellite Remote Sensing in Time-series Study of Associations between $PM_{2.5}$ and Pediatric Asthma/Wheeze Emergency Department Visits in Metropolitan Atlanta

By

Xueying Zhang

MSPH

Department of Epidemiology

Matthew J. Strickland

Committee Chair

Yang Liu

Committee Member

The Application of Satellite Remote Sensing in Time-series Study of Associations between $PM_{2.5}$ and Pediatric Asthma/Wheeze Emergency Department Visits in Metropolitan Atlanta

By

Xueying Zhang

B.S., Anhui Medical University, China, 2011

Thesis Committee Chair: Matthew J. Strickland, PhD

An abstract of

A thesis submitted to the Faculty of the
Rollins School of Public Health of Emory University

In partial fulfillment of the requirements for the degree of
Master of Science in Public Health
in Epidemiology

2013

Abstract

The Application of Satellite Remote Sensing in Time-series Study of Associations between $PM_{2.5}$ and Pediatric Asthma/Wheeze Emergency Department Visits in Metropolitan Atlanta

By Xueying Zhang

An increase in emergency department visits for respiratory diseases was observed in association with the short-term exposure of ambient air pollution. This association was investigated among children from 5 to 17 years old due to their immature and sensitive respiratory system. Traditional air pollution studies have limitations of collecting data with inadequate coverage and coarse resolution. The satellite remote sensing (RS) technique may help resolve this problem. This study examined the association between RS modeled air pollution and pediatric asthma/wheeze emergency department (ED) visits in metropolitan Atlanta during 2001 through 2007. After controlling for meteorological and holiday effects, a 10 $\mu g/m^3$ increase in three-day moving average $PM_{2.5}$ was positively associated with ED visits, rate ratio (RR) = 1.026, 95% confidence interval (CI) = 1.014, 1.040. The seasonal specific RR is 1.024 (95% CI =0.998, 1.031) for warm season (May through October), and 1.043 (95% CI = 1.023, 1.064) for cold season (November through April). Compared to previous SOPHIA studies, our study obtained slightly different RR with a small range of differences from 0 to 2.9%, and the effects of two seasons were somewhat inconsistent with past SOPHIA studies. In conclusion, the modeled ambient $PM_{2.5}$ exposure is associated with exacerbation of pediatric asthma and wheeze in Atlanta; RS provides high resolution exposure data, which will contribute to the precision of epidemiological studies.

Length: 221 words

The Application of Satellite Remote Sensing in Time-series Study of Associations between $PM_{2.5}$ and Pediatric Asthma/Wheeze Emergency Department Visits in Metropolitan Atlanta

By

Xueying Zhang

B.S., Anhui Medical University, China, 2011

Thesis Committee Chair: Matthew J. Strickland

A thesis submitted to the Faculty of the

Rollins School of Public Health of Emory University

In partial fulfillment of the requirements for the degree of
Master of Science in Public Health
in Epidemiology

2013

ACKNOWLEDGEMENTS

I would like to express the deepest appreciation to my thesis advisor, Professor Matthew Strickland, for his dedicated instructions; without his guidance and persistent help this thesis would not have been completed.

I would like to thank my field advisor, Professor Yang Liu, who has the attitude and the substance of a genius: it is him introduced satellite remote sensing technique to me and let me explore its value in an application of public health. I'm enjoying work in his group and learn from him as well as his team members.

Finally, I also need to thank Professor Mitchel Klein, for his great help in the study dataset generation; Dr. Xuefei Hu's instructions of the knowledge related to satellite remote sensing technique.

TABLE OF CONTENTS

LIST OF TABLES AND FIGURES

BACKGROUND

Particulate matter is regarded as one of three most important pollutants in ambient air, with ozone (O_3) and nitrogen oxides (NO_x) (Burtscher and Schuepp 2012). The toxicology of particulate particles is strongly associated with their size (Dockery et al. 1993). Due to its tiny sizes, most particulate matter with aerodynamic diameter lower than 2.5 μm ($PM_{2.5}$) are able to penetrate the bronchioles into epithelia and lead damages to lung endothelial cells(Giere et al. 2006). Animal and human toxicological studies supported that hypothesis (Konczol et al. 2012; Konczol et al. 2013; Li et al. 2003; Zanini et al. 2013). Nowadays, the world-wide increase of air $PM_{2.5}$ has become a global fast-growing health threat. In the 8 years' follow-up of Harvard Six Cities Study (Laden et al. 2006), a significant raise of mortality was observed with each 10 $\mu g/m^3$ increase in ambient $PM_{2.5}$ (RR= 1.14, P-value= 0.0003). Within their study results, cardiovascular mortality was positively and significantly associated with daily average $PM_{2.5}$ concentrations; this associations were also positive but weak with respect to lung cancer mortality and respiratory diseases mortality (P-value=0.1 for lung cancer, P-value= 0.63 for respiratory diseases). Correspondingly, in a recent acute study conducted by Andrew W. Correia, with every 10 $\mu g/m^3$ decrease in the $PM_{2.5}$ concentrations, the average life expectancy will increase by 0.35 years in the U.S. (SD = 0.16 years, P = 0.033)(Correia et al. 2013). Considering the high risk of human health, $PM_{2.5}$ is under tracked by the U.S. government currently (Nel 2005). The U.S. Environmental Protection Agency (EPA) has built different air pollution surveillance systems with which gave great convenience to researchers. With these surveillances, researchers can extract the air pollution data they were interested under different study purposes. For example, Environmental Protection Agency Aerometric Information Retrieval System [AIRS](Laden et al. 2006), air quality data for CDC National Environmental Public Health Tracking Network(Wilhelm et al. 2008), and Air Quality System Technology Transfer Network(EPA) 2009), etc. These

surveillance systems included daily or even hourly $PM_{2.5}$ data in a large geographical range. Besides, some studies had their own $PM_{2.5}$ measurements. From the exposure data collected with either surveillance systems or researchers themselves, we know the regional and temporal emission, composition, and kinetics of $PM_{2.5}$, which were extremely various in spatially and temporally.(Wallace et al. 2003) So the adverse health effects related to the $PM_{2.5}$ were also dynamical at the dimension of locations and time. When facing of this complex situation, epidemiologists took advantages of flexible methology to represent air pollution as study exposure and designed different studies to investigate the regional and temporal association between $PM_{2.5}$ and adverse health outcomes. (Tecer et al. 2008) People in difference ages have distinguished sensitivities toward air pollution exposure. And children's high sensitivity of air pollution is determined by their continuing process of lung growth, incomplete metabolic systems, immature host defenses and activity patterns.(World Health Organization 2005) Then recent epidemiological researches focus on the health effects of air pollution on potentially sensitive population sub-groups such as children in order to better understand the mechanisms of action and identify the most susceptible population groups for the purpose of protecting and improving children's health(Darrow et al. 2012; Samoli et al. 2011; Schwartz and Neas 2000; Silverman and Ito 2010; Strickland et al. 2010). In their findings, the positive and obvious associations between $PM_{2.5}$ and children's exacerbation of respiratory problems were in an agreement with the established toxicological studies (Baccarelli and Kaufman 2011; Karakatsani et al. 2012; Samoli et al. 2011). More pediatric asthma and wheeze cases occurred if PM2.5 concentrations increase. The study strategies of obtaining the valuable results were varied with researcher's study designs, for example, the regression models used in analyzing the associations were built with different exposure of interests and other covariates in different studies' regions. The regional effects were studied based on where

the exposure and outcome data were collected from, researchers fitted regressions models based on their study purpose and selected covariates based the characters of their data. And the temporal effects from $PM_{2.5}$ exposure were studied separately from long-term effect to short-term effect. The associations between $PM_{2.5}$ and health outcomes maybe confounded by people's living locations, seasons and meteorological factors (Mustafic et al. 2012; Samet et al. 2000). In the studies of long-term effect, birth cohorts were frequently used to analyze how the air pollution give occurrences to the new cases of asthma among young children (Gruzieva et al. 2013; Jung et al. 2012). The birth cohort is defined as a cohort which the study population being born at a particular same time(Kenneth J. Rothman 2008). By this way, the effect of exposure in the cohort that exposed on a special time could be compared with people who have not exposed during that time. The adverse health effect could be the over-one rate ratio or mortality. The short-term effect was studied with different epidemiological methods. As the air pollution exposure is easy to identify with existed surveillance systems and advanced technique, we can link today's health data with this day's air pollution data, to assess whether a short, harsh exposure will also lead to the changes of diseases' risks. For example, the risk of heart failure hospital visits increased 1.28% (increases of 95% confidence interval, 0.78%-1.78%) with every 10 $\mu g/m^3$ increase of $PM_{2.5}$ in the same day(Dominici et al. 2006). In hospital-based study, the health outcomes are usually recorded as binary variables (e.g. 1= asthma, 0=not) in an individual patient's hospital record, but in a population scale, the degree of how serious health problems it is will be reflected by the daily counts of emergency room (ED) visits. Because of the Nation's rapid demographic changes, rising numbers of uninsured patients and difficulties in making appointment, EDs has become more popular because of their convenience and accessibility (Neelon et al. 2013). Otherwise, for diseases as acute as asthma, ED patient is an ideal source population for epidemiological studies. The hospital ED visits are

considered as Poisson distributed. In the study of binary outcomes in a rare situation and the baseline number is constant, Poisson regression is usually recommended (Zou 2004). Poisson regression model is one type of generalized linear models (GLM)(P. McCullagh 1989). The GLM is expressed through the link function, which is constructed by a known monotonic function that transforms the expectation of the responses to a scale on which they are set to link with independent variable. The Poisson regression model corresponded to the GLM with log link. The Poisson regression can be used to estimate the relative risk on time scale, so that with it we can calculate the risk of primary outcome's exacerbation comparing between different levels of exposures, adjusting for potential temporal and spatial factors. Studies involved with Poisson regression were shown various levels of associations between short-term air pollution exposure and childhood asthma's exacerbations (Strickland et al. 2011; Wu et al. 2012). Case Cross-over design is another common type of GLM model. In case-crossover design, the cases are compared with themselves between periods of exposure and non-exposure, or different exposure levels. Therefore, self-matching of cases avoids the selection bias in selecting study controls then increase efficiency(Maclure 1991). According to the Faststats report(CDC), in present, about 26 million people in U.S. has asthma and one third of them are children under 18 (7.1 millions)(Centers for Disease Control and Prevention 2012). The national mortality of asthma was 0.3 per 100,000 among children 5-14 in 2010(Centers for Disease Control and Prevention 2010). Approximately 297,000 (12%) children aged 0-17 years old have asthma in Georgia. In average, 113 asthma deaths per year in the whole state from 2001 to 2007 (The Georgia Department of Community Health 2010). The Atlanta metropolitan area's air pollution and its association with pediatric asthma has been studied previously, include time-series analysis, the impact from Olympic Games, etc. (Friedman et al. 2001; Tolbert et al. 2000) Details on previous time-series studies were discussed in later sections. City of Atlanta is

a good candidate for study the adverse health effects caused by air pollution because of its established hospital admissions network, large diverse population, distinguished cold season and warm season, and severe air pollution bring by the fast growing urban construction. What is more, regional effects may also confound the results in large-scale study. So by studying with a single city, we can avoid the bias caused by the variation of exposure-response effects among cities. In modern air quality relevant epidemiological studies, the pollution data were usually collected by EPA or other ground air pollution monitoring networks. Therefore, the coverage of pollution data was limited to the areas where EPA ground monitors located. As to operate and maintain such networks are very expensive (Liu et al. 2005), we need a cost-effective, easy-to-do way to obtain valid air pollution data. Moreover, the preponderance of health studies suggests that there is no PM exposure threshold over which adverse health effects will occur (Chow et al. 2006). What is more, even within a city, air quality was a mixed situation of the intensity and spatial distribution of human activity and emission, the distribution of pollutants were also decided by the regional emission, urban planning and the regional meteorological factors(Kurtzweg 1973). Then to understand the effects of fine particles on exacerbation of children's respiratory syndrome, it is necessary to obtain $PM_{2.5}$ concentrations on a board-wide basis, with a high spatial resolution.

As a approach of collecting data without a direct contact, the techniques of satellite remote sensing (RS) have the potential to revolutionize the discipline of epidemiology and its application in epidemiologic study.(Hay 2000) In the study of air pollution, RS helps increasing the coverage of pollution measurements. At the places where air quality data is not available from on-site monitors, RS models can be used to estimate pollutants' concentrations. (Liu et al. 2005) For example, aerosol optical depth (AOD) data retrieved from the Moderate Resolution Imaging Spectroradiometer (MODIS) sensor were used to estimate ground-based particulate matters collected by US Environmental Protection

Agency (EPA) monitoring networks (Engel-Cox et al. 2004). In the approach of assessing data in a small domain, satellite observations of AOD, a measurement of light extinction by aerosols in the total atmospheric column (Stohl et al. 2006), are used to calculate domain-level concentrations of fine particulate matter ($PM_{2.5}$). With this technique, we can obtain a point estimate of $PM_{2.5}$ concentrations, and take use of it to investigate whether there is a significant association between $PM_{2.5}$ exposure and childhood respiratory syndrome ED visits in this small area. We can also perform an analysis adjusting for other considerable variables in the predictive model, such as metrological factors. As another air pollution related respiratory syndrome, pediatric wheeze was also considered as an adverse health outcome of air pollution. The first study objective is to evaluate the association between pediatric asthma and wheeze ED visits and ambient air pollutions with predictive Poisson Regression Model. The quantitative association between pediatric asthma/wheeze and air pollution has been studied by different epidemiologists. However, few of them use highly modeled $PM_{2.5}$ concentrations (Jerrett et al. 2005). The literature review helped verifying the validation of modeled $PM_{2.5}$ concentrations. Akinbami conduct a study with a sample (N=34,073) from 2001–2004 National Health Interview Survey and the 2-month average air pollutant data from EPA Aerometric Information Retrieval System (AIRS).(Akinbami et al. 2010) Their study reported that for $PM_{2.5}$, every increase of 5 $\mu m/m^3$ caused the rate of pediatric asthma was insignificantly increased by 2%, with 95% confidence interval (0.97, 1.06); Restrepo had studied the association between asthma hospital admissions and ambient air pollutant in New York City. (Restrepo et al. 2006) In their findings in New York City, one-hour maximum $PM_{2.5}$ was significantly associated with daily hospital asthma admissions (RR = 1.055, 95% CI = 1.008, 1.103).Moreover, the similar positive association between $PM_{2.5}$ and pediatric asthma/wheeze was also found in Lewis' study.(Lewis et al. 2004) They built a cohort of 298 children with asthma and record

their daily respiratory symptoms for 14 days of each season over 11 seasons from 1999 to 2002. The single pollutant logistic regression models they fit shown that children living in the Southwest area of Detroit had elevated odds of wheeze in response to an interquartile range change in $PM_{2.5}$, while this association was not observed among children living on the Eastside of Detroit. Therefore, the geographic differences of exposure-outcome responses were important when we are going to compare the results among studies with different databases. And the $PM_{2.5}$ increase the risk of pediatric asthma in various degrees for separated cities even that their studies analyzed the associations with same model design (Stieb et al. 2009). Therefore, in our study, the modeling consequences were compared with previous studies with same health database (SOPHIA). This is also a good way to evaluate the consistency between ground $PM_{2.5}$ concentrations and modeled $PM_{2.5}$ concentrations, which is the second study objective. In addition to Lewis' study, the well-established link between the asthma ED visits to $PM_{2.5}$ pollution may also not constantly cross the metropolitan Atlanta area.

DATA SOURCES

1. Emergency Department (ED) Visits

We obtain children's asthma and wheeze emergency department visits data from 40 cooperated hospitals in metropolitan Atlanta recorded during 2001–2007, conducted as a part of the Study of Particles and Health in Atlanta (SOPHIA)(Tolbert et al. 2000). Using the International Classification of Diseases, 9th Revision, we defined emergency department visits for pediatric asthma as all visits with a code for asthma (493.0–493.9) or wheeze (786.09 before October 1, 1998; 786.07 after October 1, 1998) among children aged 5 to 17 years(Centers for Disease Control and Prevention 1979). We also identified emergency department visits for acute respiratory infections (codes 460.0–466.0) that did not have a code for from metropolitan Atlanta hospitals during 2001–2007(Strickland et al. 2010). To calculate rate ratios (RRs) among ED visits, we considered each visit was independent. Such as, the ED visits counts were the number of admissions, no distinguish between primary visit and secondary visit.

In our study, the participants were originally recorded with the zip code of their living address. To lower analysis bias caused by over-flowed zero values in the dataset, and also for the convenience of analysis, we regroup the 192 zip code districts to 101 grid cells with same totally number of visits counts as the zip code-based dataset. Those grid cells are in a same size, which were 12km x 12km squares. The geographic transformation strategy was that: If there are one or more zip code areas whose central points located in one grid cell, then the all ED visits in those zip code areas were aggregated into this grid call. The distribution of grid cells is presented in figure 1.

2. $PM_{2.5}$ data

The original $PM_{2.5}$ ground measurements are the 24-h average EPA $PM_{2.5}$ concentrations collected from the federal reference monitors (FRM) are downloaded from the EPA's Air Quality System Technology Transfer Network, contained data from 2001 to 2007. The AOD data we used were provided by NASA from MODIS satellite, MAIAC AOD products (Emili et al. 2011). A linear mixed effect model with day-specific random intercepts, AOD slopes, temperature slopes, and wind speed slopes was developed because AOD, temperature, and wind speed are all time-varying parameters. Then the model that used to spatially allocate stationary $PM_{2.5}$ sources to a regional grid-cell as:

$$PM_{2.5} = (\beta_0 + \mu) + (\beta_1 + \nu)AOD + (\beta_2 + \kappa)Temperature + (\beta_3 + \sigma)Wind\ Speed + \beta_4 Elevation + \beta_5 Major\ Roads + \beta_6 Forest\ Cover + \beta_7 Po\,\text{int}\ Emissions + \varepsilon$$

$$(\mu\ \nu\ \kappa\ \sigma) \sim [(0\,0\,0\,0), \Sigma]$$

METHODS

The first place study purpose was designed to examine short-term effects of $PM_{2.5}$ on the exacerbations of pediatric asthma/wheeze and, in particular, to build predictive model to estimate the effects' variability with exposure of interests and considerable covariates. The pediatric asthma/wheeze emergency department visits by zip code were matched with our exposure estimates for each 12 km x 12km grid cell it fell into. The statistical analyses of these time series data involved two steps. Firstly, a statistical model was evolved time-series to describe the temporal patterns of the ED visits. In the time-series analysis, we analyzed whether the childhood asthma will get exacerbate after short-term exposure of $PM_{2.5}$ by using a statistical model which was constructed with terms of seasonal and temporal pattern of ED visits, temperature and weekend effect. Based on the design of case-crossover study, the association between $PM_{2.5}$ and childhood asthma emergency department visits were evaluated with Poisson generalized linear models. Due to the many zero values in the data, the mean of study outcomes was larger than variances. And this over-dispersion may cause big concerns when using Poisson regression, which is called inflates type I error (Soyseth et al. 2012).So we put a "pscale" option in the model to reduce the type I error. Since the $PM_{2.5}$ in our study was a continuous parameter, the exponential of the regression coefficient stands for the rate ratio of any observation on a given day with one unit higher air pollution level compared with a day with a lower air pollution level, adjusting for confounders(Schwartz and Morris 1995). The dependent variable was the grid-specific daily count of childhood asthma visits. In analysis, we used both grid-specific daily $PM_{2.5}$ concentrations and three-day moving average $PM_{2.5}$ (the average of concentrations today [lag 0], yesterday [lag 1], and 2 days ago [lag 2]) as the exposure of interest. (Strickland et al. 2010) However, 45.1% observations were missing values in grid specific $PM_{2.5}$. And the Pearson Correlation Analysis shown that the grid specific $PM_{2.5}$ and three-day moving average

were highly correlated (Correlation Coefficient = 0.9, P-value<0.0001). Otherwise other potential indicators were also considered to replace the three-day moving average as the main predictor in models. For example, on the line graph of daily average ED visits and daily $PM_{2.5}$ concentrations, the line of ED visits was correlated very well with one day's lag of $PM_{2.5}$ concentrations (Insert figure). So we made a hypothesis that the $PM_{2.5}$ with one day's lag may be another good predictor of exposure. However, without enough valid days, the spearman correlation test and Poisson regression model disproved this hypothesis. To avoid the statistical fallacy caused by over-inflated missing values, the regression models were using three-day moving average as a the main exposure. We added 13 individual variables as predictive variables in the statistical model analysis. These variables include temperature, dew point, day, day of week, holiday, month, year, which were tested in previous SOPHIA studies(Strickland et al. 2011; Tolbert et al. 2000). Interactions between months and daily maximum temperature spline were considered. The following terms were included in the full model: today's maximum temperature, squared and cubic terms; 2-day average minimum temperature (yesterday and the day before yesterday), squared and cubic terms; 3-day moving average dew point's linear, squared and cubic term; day of year; month; year; day of week and weekend effect; interactions between month and year, month and the linear, squared, cubic terms of maximum temperature. Separated models are set for warm season (May to October) and cold season (November to April). When operating seasonal models, the spline "day of year" changes to "day of season". Meteorological variables were collected in Atlanta Hartsfield-Jackson International Airport. The covariates' definitions are followed by table 1.

$$LN(\text{Childhood Asthma \& Wheeze ED Visits}) = \beta_0 + \beta_1 \text{Exposure} + \beta_{21} \text{Max Temperature} + \beta_{22} \text{Max Temperature2} + \beta_{23} \text{Max Temperature3} + \beta_{31} \text{Two-Day Average Min Temperature} + \beta_{32}$$

*Two-Day Min Termpreature2+ β_{33} Two-Day Min Termperature3+β_{41} Three-Day Average Dew Point+β_{42} Three-Day Average Dew Point^2+β_{43} Three-Day Average Dew Point^3+β_5 Day of Year+β_6 Month+β_7 Year+β_8 Weekday_Holiday+β_9 Month*Year+β_{10} Month*Weekday _Holiday +β_{111} Month*Max Temperature+β_{112} Month*Max Temperature^2+β_{113} Month*Max Temperature^3+ β_{12} Grid + E*

The literature review helped verifying the validation of modeled $PM_{2.5}$ concentrations. To make the satellite modeled data mostly comparable with the previous study results, the selected studies for comparisons were also based on SOPHIA data. All quantitative analysis were conduct in SAS 9.3, the statistical significant standard was uniformly 0.05 in our study.

RESULTS

As the main exposure of interests, $PM_{2.5}$ concentrations that generated with satellite remote sensing technique was reviewed about the temporal and regional distribution patterns (table 2, figure 2). The predicted $PM_{2.5}$ concentrations were normally distributed and slightly right skewed (figure 3). The RS data was available only in "cloud free" days, so the only 54.9% observations have valid predicted $PM_{2.5}$ data, according to the calculation results of valid N/ (7 years x 365 days x 101 grid cells + 1 Feb 29th). Warm season's (May through October) average predicted grid $PM_{2.5}$ was significantly higher than cold season's (November through next year's April) (P-value < 0.0001). According to the one-way ANOVA test, at least one pair of the comparisons between two years' predicted grid $PM_{2.5}$ concentrations was significant (P-value<0.0001). The distribution of three-day moving average $PM_{2.5}$ concentrations, as well as its seasonal pattern and yearly pattern were similar to the predicted $PM_{2.5}$ with large amount of missing data (figure 4). These two formats of $PM_{2.5}$ concentrations were highly correlated (Spearman Correlation Coefficient = 0.90, P-value <0.0001). And the typical lifetime of $PM_{2.5}$ is larger than 3 days.(Peter H.McMurry) So we can use the three-day moving average to replace the predicted grid $PM_{2.5}$ as the main predictor in the following analysis.

Table 3 contains a descriptive analysis of the daily pediatric asthma/wheeze ED visits for both warm and cold seasons across all 101 grid cells. 66.2 % daily grid-specific pediatric asthma/wheeze observations were 0 values. So the ED visits distributions were extremely right skewed, daily grid means was lower than its standard deviation. (Kimes et al.) The grid cell's average daily pediatric asthma/wheeze was less than one (mean=0.77, SD=1.65); Cold season has significantly more ED visits than warm season (P-value < 0.0001), and the numbers of ED visits were also shown an increasing

tendency from 2001 to 2007 , but this increase might due to the growth of total population in Atlanta city.

Geographic distributions of $PM_{2.5}$ were shown by table 4 and figure 2. Because of the population density and traffic emission, the $PM_{2.5}$ concentrations are high in the central area of city. Grid cell with ID "41" had an abnormal high pollution concentration due to the coal-fired power plant within the area. As similar as the distribution of $PM_{2.5}$, the daily asthma/wheeze visits were also squeezed in the grid cells close to the downtown Atlanta (figure 5). But it should be cautious to interpret the distribution since the population is not evenly distributed as well.

Table 5 presents the estimated percent increase in ED visits for a 10 mg/m^3 increase for both short term $PM_{2.5}$ by cause of admission and associated 95% confidence intervals through the whole study area. Overall, for every 10 mg/m^3 increase of predicted $PM_{2.5}$ exposure there is a 1.015 times increase of rate in pediatric asthma/wheeze (95% CI = 1.000 to 1.029). The cold season resulted in stronger association between predicted $PM_{2.5}$ and pediatric asthma/wheeze visits, which is 2.4% increase of rate with every 10 $\mu g/m^3$ $PM_{2.5}$ concentrations. With same change of predicted $PM_{2.5}$, the change of rate in cold season was 1.5 times greater than the rate's change in warm season. The significant (alpha=0.05) effect were observed in the models built with predicted $PM_{2.5}$, lag 2 day's effect and three-day moving average with overall data and predicted $PM_{2.5}$, lag1 day's effect in cold season. Detailed discussion followed in the next section.

DISCUSSION

Satellite remote sensing gives epidemiology advantages to fit log-linear model with high-resolution data coverage.(Fan et al. 1999; Houlahan et al. 2000) In this study, we have identified significant effects of ambient $PM_{2.5}$ in a large, multi-year metropolitan study of air pollution and ED visits. Unique features of this study include application of satellite remote sensing technique in generating the $PM_{2.5}$ concentrations, and transferring the zip code level hospital visits data to multiple grid cells, which covered about 200,000 visits in total from 2001 to 2007 in Atlanta; We assessed the association between short-term exposure to ambient $PM_{2.5}$ and grid cell specific exacerbation of pediatric asthma/wheeze. A positive association between asthma/wheeze hospital emergency department visits and $PM_{2.5}$'s concentrations were identified. But this association was less consistent with Atlanta's warm season and cold season. To evaluate our study effects, we compare our own study results with the studies using same data source (SOPHIA) to test the validation of modeled $PM_{2.5}$.

In 2000, Tolbert modeled multiple air pollutants, which in include $PM_{2.5}$, coarse PM, PM10 ozone, NO_2 , SO_2 , CO, polar VOCs, 10-100 nm particulate matter count and surface area, and $PM_{2.5}$'s components, with more than 2 million ED visits in Atlanta for the period January 1, 1993-August 31, 2000, using Generalized Linear Model with Poisson regression.(Tolbert et al. 2000) In their results, the rate of adult asthma increased 0.9% with daily average $PM_{2.5}$ increase by 10 $\mu g/m^3$, but this association is not statistical significant (P-value = 0.81). Peel's SOPHIA study received similar effects with Tolbert's study. They selected 5 pollutants (PM, ozone, NO2, CO, and SO2) and link it with given day's ED visits data. For $PM_{2.5}$, the data was only selected from 1 August 1998 to 31 August 2000. 4 million emergency department visits from 31 hospitals in Atlanta were analyzed in the model. The $PM_{2.5}$ concentrations were positive but also

insignificantly associated with overall asthma ED visits (RR=1.005, 95% CI= 0.977, 1.033).(Peel 2005) In Strickland's 2010 study, over 90,000 ED visits of pediatric asthma were retrieved from SOPHIA and analyzed with ambient air pollutant concentrations.(Strickland et al. 2010) The models used by Strickland had controlled for meteorological factors, seasonal asthma trends, month, weekend and holiday. The main exposure of interest in their model was three-day model average of ground $PM_{2.5}$. With a interquartile range increase of main exposure, the rate of pediatric asthma increase by 2.0% for the overall data, 4.3% for the warm season (May through October), 0.5% for the cold season (November through April). However, the results of associations' Wald Chi-square Test were not constant cross the seasons. The 95% CI of rate ratio in cold season included null but the other two were not. Strickland also considered lag effect of exposure. In their study results, with an interquartile range increase of three-day moving average $PM_{2.5}$ with 1 day's lag, the rate of pediatric asthma ED visits increase by 2.4%, 95% CI ranges from 1.004 to 1.045. When it is turn to our study, smaller effects were found. Our model was uniformly use grid cell's daily pediatric asthma/wheeze ED visits as the dependent variables. The dependent variables were moderated separately with grid cell $PM_{2.5}$, 1 day lag, 2 day lag, 3 day lag, IQR of grid cell $PM_{2.5}$, three-day moving average $PM_{2.5}$ and its IQR as study exposure of interest. In our study, the effects of $PM_{2.5}$ on pediatric asthma/wheeze are positive with all modeling results. RR=1.025 in the predicted $PM_{2.5}$ model, which means the rate of pediatric asthma/wheeze increases 2.5% if the $PM_{2.5}$ increase 10 $\mu g/m^3$. This raise of $PM_{2.5}$ concentrations causes 1.6% increase of rate in warm season, and 2.4% increase of rate in cold season. When using three-day moving average as the main exposure, our model gave us very close, positive results to Strickland's: the rate increases 2.6% with every 10 $\mu g/m^3$ increase of three-day moving average $PM_{2.5}$ in overall, the effects change by 1.4% in warm season and by 4.3% in cold season. The interquartile range increase of main exposure leads to 2.3% increase of rate,

1.1% for warm season and 2.5% for cold season. Only the overall RR and cold season RR get statistical significant results in analysis. Nevertheless, there are some conflicts between our results and Strickland's study. In their study, the $PM_{2.5}$ cause stronger effect in cold season rather than warm season. But the RR in warm season in our study is lower than the RR in cold reason. The conflict indicates that except for more adjustments of models, the measurement limitation in remote sensing technique should be considered in this situation.

In general, our results in a harmony with those in previous SOPHIA studies. Quantitatively, the effect's estimates are difficult to compare because we assessed the pollutants with in different ways, and in our study, the ambient $PM_{2.5}$ concentrations have been assigned to grid cell level but the others didn't. We observed several positive, statistical significant associations between ambient $PM_{2.5}$ concentrations and the rate of pediatric asthma/wheeze in Atlanta. Those findings are likely to be confounded by some temporal asthma risk factors such as pollen if high pollen days in corresponds with high pollution days. Second-hand smoke maybe a risk factor of asthma, exposure to environmental tobacco smoke has been linked to more frequent exacerbation of asthma in children who have diagnosed with the diseases with established study results.(Chilmonczyk et al. 1993) Besides, age could be an effect modifier in respiratory diseases studies. So our study population was restricted to specific age groups (children) and pollutant ($PM_{2.5}$), to reduce the bias from tobacco use and age distribution. We cannot exclude that all the unmeasured confounders to some extent may bias the associations. The meteorological factors we adjusted in model were primarily decided by literature review and private communication with SOPHIA scholars. So that there is a limitation of our study that since we should consider the variation of doe-responses effects across the city, but the demographical factors can confounded the effect between $PM_{2.5}$ and pediatric asthma /wheeze. Atlanta is a city with a high immigration rate.

Because of the genetic distinguishes among different races, children in different races have different sensitivities to air pollution. For example, Non-Hispanic black children were more likely to have ever been diagnosed with asthma (18%) than Hispanics(Centers for Disease Control and Prevention 2012) Moreover, socioeconomic status (SES) is another factor that commonly considered in pediatric asthma studies. (Kozyrskyj et al. 2010; Pope et al. 2009) For children already diagnosed with asthma, the utilization rate of prophylactic medications, hospitalization rate and the rate of readmission were higher among children with low family income than high family income children in earlier time's studies (Auger et al. 2013; Braaback et al. 2011; Kozyrskyj et al. 2010). Family income is also help in explaining the increased prevalence of asthma among Black compared with White children, adjusting for other individual disparities. (Akinbami et al. 2009; Gold and Wright 2005) Furthermore, the ethnicities and household income were not evenly distributed in our study area, according to the data provide by U.S. census bureau.(Bureau 2010) Being lack of appropriate method to obtain accurate demographic information for each grid cells, we put the indicator variable of locations in the epidemiological model and create the case-crossover model, to adjust the demographic characteristics inner grid cells. However, with the reasons above, we need considered demographic variations in the future's study. Another limitation exits in the model selection. In our study, 66.2% of ED visits daily observations were 0 values (N= 170789). So the standard deviation of ED visits (1.65) is larger than its mean (0.77), which is not a perfect Poisson distribution.

CONCLUSION

In conclusion, ambient $PM_{2.5}$ exposure is associated with exacerbation in pediatric asthma and wheeze in Atlanta. Findings from quantitative models support previously published reports that studied with time-series analysis, with same or not same data source. Cold season is a risk factor of pediatric asthma and wheeze. The results obtain with satellite moderated exposures are very similar to the modeling results from ground $PM_{2.5}$ measurements. But we get a stronger effect between ambient $PM_{2.5}$ and pediatric respiratory syndrome. The previous SOPHIA studies were pooled all the data to a city scale, but we can adjust the model to a smaller domain. The satellite remote sensing is expected to be used in more environmental epidemiology studied in the future.

REFERENCES

Akinbami, L.J., Lynch, C.D., Parker, J.D., & Woodruff, T.J. (2010). The association between childhood asthma prevalence and monitored air pollutants in metropolitan areas, United States, 2001-2004. *Environmental Research, 110*, 294-301

Akinbami, L.J., Moorman, J.E., Garbe, P.L., & Sondik, E.J. (2009). Status of Childhood Asthma in the United States, 1980-2007. *Pediatrics, 123*, S131-S145

Auger, K.A., Kahn, R.S., Davis, M.M., Beck, A.F., & Simmons, J.M. (2013). Medical Home Quality and Readmission Risk for Children Hospitalized With Asthma Exacerbations. *Pediatrics, 131*, 64-70

Baccarelli, A., & Kaufman, J.D. (2011). Ambient Particulate Air Pollution, Environmental Tobacco Smoking, and Childhood Asthma: Interactions and Biological Mechanisms. *American Journal of Respiratory and Critical Care Medicine, 184*, 1325-1327

Braaback, L., Vogt, H., & Hjern, A. (2011). Migration and asthma medication in international adoptees and immigrant families in Sweden. *Clinical and Experimental Allergy, 41*, 1108-1115

Bureau, U.S.C. (2010). 2010 U.S. Census Survey

Burtscher, H., & Schuepp, K. (2012). The occurrence of ultrafine particles in the specific environment of children. *Paediatric Respiratory Reviews, 13*, 89-94

CDC FastStats. *http://www.cdc.gov/nchs/fastats/*

Centers for Disease Control and Prevention, C. (1979). Clinical Modification (ICD-9-CM) Hyattsville, MD. *International Classification of Diseases, 9th Revision*

Centers for Disease Control and Prevention, C. (2010). National Mortality tables, 2010

Centers for Disease Control and Prevention, C. (2012). Summary Health Statistics for U.S. Children: National Health Interview Survey, 2011

Chilmonczyk, B.A., Salmun, L.M., Megathlin, K.N., Neveux, L.M., Palomaki, G.E., Knight, G.J., Pulkkinen, A.J., & Haddow, J.E. (1993). Association Between Exposure to Environmental Tobacco Smoke and Exacerbations of Asthma in Children. *New England Journal of Medicine, 328*, 1665-1669

Chow, J.C., Watson, J.G., Mauderly, J.L., Costa, D.L., Wyzga, R.E., Vedal, S., Hidy, G.M., Altshuler, S.L., Marrack, D., Heuss, J.M., Wolff, G.T., Pope, C.A., & Dockery, D.W. (2006). Health effects of fine particulate air pollution: Lines that connect. *Journal of the Air & Waste Management Association, 56*, 1368-1380

Correia, A.W., Pope, C.A., Dockery, D.W., Wang, Y., Ezzati, M., & Dominici, F. (2013). Effect of Air Pollution Control on Life Expectancy in the United States An Analysis of 545 US Counties for the Period from 2000 to 2007. *Epidemiology, 24*, 23-31

Darrow, L.A., Hess, J., Rogers, C.A., Tolbert, P.E., Klein, M., & Sarnat, S.E. (2012). Ambient pollen concentrations and emergency department visits for asthma and wheeze. *Journal of Allergy and Clinical Immunology, 130*, 630

Dockery, D.W., Pope, C.A., Xu, X.P., Spengler, J.D., Ware, J.H., Fay, M.E., Ferris, B.G., & Speizer, F.E. (1993). An Association Between Air Pollution and Mortality in 6 United States Cities. *New England Journal of Medicine, 329*, 1753-1759

Dominici, F., Peng, R.D., Bell, M.L., Pham, L., McDermott, A., Zeger, S.L., & Samet, J.M. (2006). Fine particulate air pollution and hospital admission for cardiovascular and respiratory diseases. *Jama-Journal of the American Medical Association, 295*, 1127-1134

Emili, E., Lyapustin, A., Wang, Y., Popp, C., Korkin, S., Zebisch, M., Wunderle, S., & Petitta, M. (2011). High spatial resolution aerosol retrieval with MAIAC: Application to mountain regions. *Journal of Geophysical Research-Atmospheres, 116*

Engel-Cox, J.A., Holloman, C.H., Coutant, B.W., & Hoff, R.M. (2004). Qualitative and quantitative evaluation of MODIS satellite sensor data for regional and urban scale air quality. *Atmospheric Environment, 38*, 2495-2509

EPA), U.S.E.P.A.U. (2009). Technology Transfer Network (TTN): Air Quality System (AQS)

Fan, X.T., Thompson, B., & Wang, L. (1999). Effects of Sample Size, Estimation Methods, and Model Specification on Structural Equation Modeling Fit Indexes. *Structural Equation Modeling-a Multidisciplinary Journal, 6*, 56-83

Friedman, M.S., Powell, K.E., Hutwagner, L., Graham, L.M., & Teague, W.G. (2001). Impact of changes in transportation and commuting behaviors during the 1996 Summer Olympic Games in Atlanta on air quality and childhood asthma. *Jama-Journal of the American Medical Association, 285*, 897-905

Giere, R., Blackford, M., & Smith, K. (2006). TEM study of PM2.5 emitted from coal and tire combustion in a thermal power station. *Environmental Science & Technology, 40*, 6235-6240

Gold, D.R., & Wright, R. (2005). Population disparities in asthma. *Annual Review of Public Health* (pp. 89-113). Palo Alto: Annual Reviews

Gruzieva, O., Bergstrom, A., Hulchiy, O., Kull, I., Lind, T., Melen, E., Moskalenko, V., Pershagen, G., & Bellander, T. (2013). Exposure to Air Pollution from Traffic and Childhood Asthma Until 12 Years of Age. *Epidemiology, 24*, 54-61

Hay, S.I. (2000). An overview of remote sensing and geodesy for epidemiology and public health application. *Advances in Parasitology, Vol 47, 47*, 1-35

Houlahan, J.E., Findlay, C.S., Schmidt, B.R., Meyer, A.H., & Kuzmin, S.L. (2000). Quantitative evidence for global amphibian population declines. *Nature, 404*, 752-755

Jerrett, M., Arain, A., Kanaroglou, P., Beckerman, B., Potoglou, D., Sahsuvaroglu, T., Morrison, J., & Giovis, C. (2005). A review and evaluation of intraurban air pollution exposure models. *Journal of Exposure Analysis and Environmental Epidemiology, 15*, 185-204

Jung, K.H., Hsu, S.I., Yan, B.Z., Moors, K., Chillrud, S.N., Ross, J., Wang, S., Perzanowski, M.S., Kinney, P.L., Whyatt, R.M., Perera, F.P., & Miller, R.L. (2012). Childhood exposure to fine particulate matter and black carbon and the development of new wheeze between ages 5 and 7 in an urban prospective cohort. *Environment International, 45*, 44-50

Karakatsani, A., Analitis, A., Perifanou, D., Ayres, J.G., Harrison, R.M., Kotronarou, A., Kavouras, I.G., Pekkanen, J., Hameri, K., Kos, G.P.A., de Hartog, J.J., Hoek, G., & Katsouyanni, K. (2012). Particulate matter air pollution and respiratory symptoms in individuals having either asthma or chronic obstructive pulmonary disease: a European multicentre panel study. *Environmental Health, 11*

Kenneth J. Rothman, S.G., Timothy L. Lash (2008). Modern Epidemiology, 3th Edition. *Lippincott Williams & Wilkins*

Kimes, D., Ullah, A., Levine, E., Nelson, R., Timmins, S., Weiss, S., Bollinger, M.E., & Blaisdell, C. (2004). Relationships between pediatric asthma and socioeconomic/urban variables in Baltimore, Maryland. *Health & Place, 10*, 141-152

Konczol, M., Goldenberg, E., Ebeling, S., Schafer, B., Garcia-Kaufer, M., Gminski, R., Grobety, B., Rothen-Rutishauser, B., Merfort, I., Giere, R., & Mersch-Sundermann, V. (2012). Cellular Uptake and Toxic Effects of Fine and Ultrafine Metal-Sulfate Particles in Human A549 Lung Epithelial Cells. *Chemical Research in Toxicology, 25*, 2687-2703

Konczol, M., Weiss, A., Gminski, R., Merfort, I., & Mersch-Sundermann, V. (2013). Oxidative stress and inflammatory response to printer toner particles in human epithelial A549 lung cells. *Toxicology Letters, 216*, 171-180

Kozyrskyj, A.L., Kendall, G.E., Jacoby, P., Sly, P.D., & Zubrick, S.R. (2010). Association Between Socioeconomic Status and the Development of Asthma: Analyses of Income Trajectories. *American Journal of Public Health, 100*, 540-546

Kurtzweg, J.A. (1973). URBAN PLANNING AND AIR POLLUTION CONTROL - REVIEW OF SELECTED RECENT RESEARCH. *Journal of the American Institute of Planners, 39*, 82-92

Laden, F., Schwartz, J., Speizer, F.E., & Dockery, D.W. (2006). Reduction in fine particulate air pollution and mortality - Extended follow-up of the Harvard six cities study. *American Journal of Respiratory and Critical Care Medicine, 173*, 667-672

Lewis, T., Robins, T., Mentz, G., Yip, F., Dvonch, J.T., Keeler, G., Ramirez, E., Parker, E., & Israel, B. (2004). Association of ambient air pollution with presence of respiratory symptoms among children with asthma. *Epidemiology, 15*, S37-S37

Li, N., Sioutas, C., Cho, A., Schmitz, D., Misra, C., Sempf, J., Wang, M.Y., Oberley, T., Froines, J., & Nel, A. (2003). Ultrafine particulate pollutants induce oxidative stress and mitochondrial damage. *Environmental Health Perspectives, 111*, 455-460

Liu, Y., Sarnat, J.A., Kilaru, A., Jacob, D.J., & Koutrakis, P. (2005). Estimating ground-level PM2.5 in the eastern united states using satellite remote sensing. *Environmental Science & Technology, 39*, 3269-3278

Maclure, M. (1991). The Case-Crossover Desgin - A Method for Studying Transient Effects on the Risk of Acute Events. *American Journal of Epidemiology, 133*, 144-153

Mustafic, H., Jabre, P., Caussin, C., Murad, M.H., Escolano, S., Tafflet, M., Perier, M.C., Marijon, E., Vernerey, D., Empana, J.P., & Jouven, X. (2012). Main Air Pollutants and Myocardial Infarction A Systematic Review and Meta-analysis. *Jama-Journal of the American Medical Association, 307*, 713-721

Neelon, B., Ghosh, P., & Loebs, P.F. (2013). A spatial Poisson hurdle model for exploring geographic variation in emergency department visits. *Journal of the Royal Statistical Society Series a-Statistics in Society, 176*, 389-413

Nel, A. (2005). Air pollution-related illness: Effects of particles. *Science, 308*, 804-806

P. McCullagh, J.A.N. (1989). Generalized Linear Model

Peel, J.L. (2005). Ambient Air Pollution and Respiratory Emergency Department Visits. *Epidemiology, 16*, 164-174

Peter H.McMurry, M.F.S., James S. Vickery Particulate Matter Science for Policy Makers: A NARSTO Assessment, 119-120

Pope, C.A., Ezzati, M., & Dockery, D.W. (2009). Fine-Particulate Air Pollution and Life Expectancy in the United States. *New England Journal of Medicine, 360*, 376-386

Restrepo, C., Simonoff, J., Thurston, G., & Zimmerman, R. (2006). Asthma hospital admissions and ambient air pollution concentrations in New York City. *Epidemiology, 17*, S266-S266

Samet, J.M., Dominici, F., Curriero, F.C., Coursac, I., & Zeger, S.L. (2000). Fine particulate air pollution and mortality in 20 US Cities, 1987-1994. *New England Journal of Medicine, 343*, 1742-1749

Samoli, E., Nastos, P.T., Paliatsos, A.G., Katsouyanni, K., & Priftis, K.N. (2011). Acute effects of air pollution on pediatric asthma exacerbation: Evidence of association and effect modification. *Environmental Research, 111*, 418-424

Schwartz, J., & Morris, R. (1995). Air-pollution and Hospital Admissons for Cardiovascular Diseases in Detroit, Michigan. *American Journal of Epidemiology, 142*, 23-35

Schwartz, J., & Neas, L.M. (2000). Fine particles are more strongly associated than coarse particles with acute respiratory health effects in schoolchildren. *Epidemiology, 11*, 6-10

Silverman, R.A., & Ito, K. (2010). Age-related association of fine particles and ozone with severe acute asthma in New York City. *Journal of Allergy and Clinical Immunology, 125*, 367-373

Soyseth, V., Johnsen, H.L., Bugge, M.D., & Kongerud, J. (2012). The association between symptoms and exposure is stronger in dropouts than in non-dropouts among employees in Norwegian smelters: a five-year follow-up study. *International Archives of Occupational and Environmental Health, 85*, 27-33

Stieb, D.M., Szyszkowicz, M., Rowe, B.H., & Leech, J.A. (2009). Air pollution and emergency department visits for cardiac and respiratory conditions: a multi-city time-series analysis. *Environmental Health, 8*

Stohl, A., Andrews, E., Burkhart, J.F., Forster, C., Herber, A., Hoch, S.W., Kowal, D., Lunder, C., Mefford, T., Ogren, J.A., Sharma, S., Spichtinger, N., Stebel, K., Stone, R., Strom, J., Torseth, K., Wehrli, C., & Yttri, K.E. (2006). Pan-Arctic enhancements of light absorbing aerosol concentrations due to North American boreal forest fires during summer 2004. *Journal of Geophysical Research-Atmospheres, 111*

Strickland, M.J., Darrow, L.A., Klein, M., Flanders, W.D., Sarnat, J.A., Waller, L.A., Sarnat, S.E., Mulholland, J.A., & Tolbert, P.E. (2010). Short-term Associations between Ambient Air Pollutants and Pediatric Asthma Emergency Department Visits. *American Journal of Respiratory and Critical Care Medicine, 182*, 307-316

Strickland, M.J., Darrow, L.A., Mulholland, J.A., Klein, M., Flanders, W.D., Winquist, A., & Tolbert, P.E. (2011). Implications of different approaches for characterizing ambient air pollutant concentrations within the urban airshed for time-series studies and health benefits analyses. *Environmental Health, 10*

Tecer, L.H., Alagha, O., Karaca, F., Tuncel, G., & Eldes, N. (2008). Particulate matter (PM2.5, PM10-2.5, and PM10) and children's hospital admissions for asthma and respiratory diseases: A

bidirectional case-crossover study. *Journal of Toxicology and Environmental Health-Part a-Current Issues, 71*, 512-520

The Georgia Department of Community Health, G. (2010). 2010 Georgia Data Summary

Tolbert, P.E., Klein, M., Metzger, K.B., Peel, J., Flanders, W.D., Todd, K., Mulholland, J.A., Ryan, P.B., & Frumkin, H. (2000). Interim results of the study of particulates and health in Atlanta (SOPHIA). *Journal of Exposure Analysis and Environmental Epidemiology, 10*, 446-460

Wallace, L.A., Mitchell, H., O'Connor, G.T., Neas, L., Lippmann, M., Kattan, M., Koenig, J., Stout, J.W., Vaughn, B.J., Wallace, D., Walter, M., Adams, K., & Liu, L.J.S. (2003). Particle concentrations in inner-city homes of children with asthma: The effect of smoking, cooking, and outdoor pollution. *Environmental Health Perspectives, 111*, 1265-1272

Wilhelm, M., Meng, Y.Y., Rull, R.P., English, P., Balmes, J., & Ritz, B. (2008). Environmental Public Health Tracking of childhood asthma using California Health Interview Survey, traffic, and outdoor air pollution data. *Environmental Health Perspectives, 116*, 1254-1260

World Health Organization, E.C.f.E.a.H.B.O. (2005). Effects of Air Pollution on Children's Health and Development. *World Health Organization*

Wu, A.C., Gregory, M., Kymes, S., Lambert, D., Edler, J., Stwalley, D., & Fuhlbrigge, A.L. (2012). Modeling asthma exacerbations through lung function in children. *Journal of Allergy and Clinical Immunology, 130*, 1065-1070

Zanini, D., Schmatz, R., Pelinson, L.P., Pimentel, V.C., da Costa, P., Cardoso, A.M., Martins, C.C., Schetinger, C.C., Baldissareli, J., Araujo, M.D., Oliveira, L., Chiesa, J., Morsch, V.M., Leal, D.B.R., & Schetinger, M.R.C. (2013). Ectoenzymes and cholinesterase activity and biomarkers of oxidative stress in patients with lung cancer. *Molecular and Cellular Biochemistry, 374*, 137-148

Zou, G.Y. (2004). A modified Poisson regression approach to prospective studies with binary data. *American Journal of Epidemiology, 159*, 702-706

TABLES

Table 1: Definitions of Adjusted Predictors (Except for the $PM_{2.5}$) in Poisson Log-linear Regression Model

	Type	Data Source	Variable Description
Maximum Temperature	Continuous	USEPA	Today's Maximum Temperature in Celsius
Two-Day Minimum Temperature	Continuous	USEPA	(Today 's Minimum Temperature +yesterday's minimum Temperature)/No. of valid data in today and yesterday
Three-Day Average Dew Point	Continuous	USEPA	(Today 's dew point+ lag 1 day's dew point + lag 2 day's dew point)/No. of valid data in today and past two days.
Day of Year	Continuous	-	Number of days in a given study year earlier then the study day (include the study day).
Day of Season	Continuous	-	Number of days in a given study season earlier then the study day (include the study day).
Weekday & Holiday	Continuous/Categorical	-	1=Sunday, 2= Monday, 3=Tuesday, 4=Wednesday, 5=Thursday, 6=Friday, 7=Saturday and 8=Official Holidays from Federal Government

Table 2: Descriptive Analysis of Grid $PM_{2.5}$ and Its Three-Day Moving Average from 2001 to 2007

PM2.5 concentrations		Overall			Warm Season			Cold Season		
		Valid* N	Mean [SD]	25th–75th percentile	Valid* N	Mean [SD]	25th–75th percentile	Valid* N	Mean [SD]	25th–75th percentile
Daily Grid $PM_{2.5}$ (RS)		141735	15.21 [6.98]	9.90 - 19.40	69025	18.50 [7.06]	13.50 - 22.60	72710	12.08 [5.27]	8.25-15.00
Three-day Moving Average		214358	15.09 [6.39]	10.17 - 19.02	103742	18.50 [6.35]	14.20 – 22.30	110616	11.89 [4.50]	8.50 – 14.43
Daily Grid $PM_{2.5}$ (RS) by Year	2001	17916	15.74 [7.41]	10.20 – 20.20	7888	17.81 [7.76]	11.30 – 23.00	10028	14.12 [6.69]	9.60 – 17.00
	2002	15473	15.71 [6.60]	10.70 – 20.20	7207	20.11 [6.03]	16.10 – 23.70	8266	11.87 [4.29]	8.80 – 14.20
	2003	19874	14.22 [6.56]	9.65 – 18.00	9837	17.45 [6.33]	13.35 – 20.05	10037	11.05 [5.06]	7.60 – 13.50
	2004	19502	14.42 [6.30]	9.60 – 18.05	8998	18.08 [6.17]	14.12 – 21.35	10504	11.28 [4.44]	7.83 – 14.20
	2005	21289	16.06 [7.35]	10.40 – 20.30	10854	20.09 [7.11]	15.20 – 23.83	10435	11.86 [4.82]	8.40 – 14.70
	2006	24131	15.34 [6.50]	10.20 – 19.90	12542	18.09 [6.68]	13.10 – 22.95	11589	12.37 [4.76]	8.60 -15.40
	2007	23550	15.04 [7.71]	8.90 – 19.30	11699	18.14 [8.22]	12.20 – 22.90	11851	11.98 [5.72]	7.57 – 15.45
Three-day Moving Average by Year	2001	28069	15.81 [6.77]	10.65 – 19.80	12703	18.49 [7.17]	12.12 – 23.25	15366	13.60 [5.52]	9.75 – 16.65
	2002	26161	15.48 [6.05]	10.65 – 19.78	12041	19.77 [5.41]	16.55 – 23.23	14120	11.82 [3.72]	9.35 – 13.90
	2003	30961	14.41 [6.17]	9.70 – 17.70	15476	17.85 [5.81]	14.40 – 20.35	15215	10.92 [4.26]	8.00 – 12.90
	2004	29965	14.40 [5.86]	9.67 – 17.90	13967	18.09 [5.60]	14.75 – 20.95	15998	11.18 [3.84]	8.25 – 13.80
	2005	32030	15.86 [6.73]	10.80 – 19.90	15806	19.98 [6.39]	15.60 – 23.20	16224	11.83 [4.11]	8.70 – 14.20
	2006	34197	15.01 [5.88]	10.55 – 19.10	17225	17.72 [6.14]	12.61 – 22.20	16972	12.26 [4.05]	9.30 – 14.77
	2007	33245	14.78 [6.97]	9.10 – 19.03	16524	17.95 [7.13]	12.80 – $2_{2.5}$0	16721	11.64 [5.16]	7.80 – 14.60

Table 3: Descriptive Analysis of Pediatric ED Visits from 2001 to 2007

ED visits		Overall		Warm Season		Cold Season	
		Valid N	Mean [SD]	Valid* N	Mean [SD]	Valid* N	Mean [SD]
Daily Grid ED visits		258156	0.77 [1.65]	130088	0.70 [1.55]	128068	0.85 [1.74]
Daily Grid ED visits by Year	2001	36865	0.60 [1.30]	18584	0.54 [1.23]	18281	0.67 [1.36]
	2002	36865	0.69 [1.49]	18584	0.62 [1.38]	18281	0.76 [1.59]
	2003	36865	0.71 [1.45]	18584	0.63 [1.32]	18281	0.80 [1.56]
	2004	36966	0.70 [1.41]	18584	0.64 [1.33]	18382	0.77 [1.48]
	2005	36865	0.89 [1.93]	18584	0.83 [1.86]	18281	0.96 [1.99]
	2006	36865	0.88 [1.87]	18584	0.80 [1.74]	18281	0.96 [1.99]
	2007	36865	0.95 [1.96]	18584	0.87 [1.84]	18281	1.04 [2.06]

Table 4: Geographic Distribution of Mean and Standard Deviation of Three-Day Moving Average Ambient PM2.5 Concentrations

Grid Cell ID	Three-day moving average		Grid Cell ID	Three-day moving average		Grid Cell ID	Three-day moving average		Grid Cell ID	Three-day moving average		Grid Cell ID	Three-day moving average	
	Mean	SD		Mean	SD		Mean	SD		Mean	SD		Mean	SD
1	15.81	6.35	**21**	15.41	6.39	**41**	27.48	6.48	**61**	14.63	6.37	**81**	14.90	6.30
2	15.47	6.23	**22**	14.93	5.99	**42**	15.25	6.53	**62**	15.12	6.35	**82**	15.22	6.44
3	16.21	6.64	**23**	14.77	6.05	**43**	14.70	6.48	**63**	15.50	6.52	**83**	14.85	6.22
4	14.85	5.99	**24**	14.70	5.80	**44**	15.02	6.40	**64**	14.60	6.10	**84**	14.91	6.39
5	14.96	6.21	**25**	15.80	6.54	**45**	14.77	6.47	**65**	14.75	6.48	**85**	14.58	5.91
6	14.92	5.90	**26**	15.27	6.42	**46**	14.36	6.20	**66**	14.91	6.32	**86**	16.06	6.49
7	14.59	6.05	**27**	15.63	6.51	**47**	15.01	6.51	**67**	14.67	6.26	**87**	15.75	6.51
8	15.37	6.24	**28**	15.65	6.63	**48**	14.00	5.98	**68**	14.60	6.25	**88**	15.77	6.64
9	14.62	5.99	**29**	15.93	6.48	**49**	14.87	6.45	**69**	14.56	5.94	**89**	15.31	6.60
10	15.81	6.40	**30**	15.56	6.56	**50**	13.66	5.85	**70**	14.98	6.28	**90**	14.92	6.00
11	15.37	6.23	**31**	14.97	6.33	**51**	14.67	6.34	**71**	14.69	6.29	**91**	14.63	5.92
12	14.85	5.81	**32**	14.96	6.37	**52**	15.06	6.39	**72**	14.53	5.94	**92**	15.14	6.15
13	15.35	6.38	**33**	15.44	6.50	**53**	14.72	6.27	**73**	15.44	6.46	**93**	13.97	5.91
14	15.45	6.29	**34**	14.15	6.01	**54**	14.62	6.20	**74**	14.64	5.92	**94**	14.78	5.90
15	14.71	6.30	**35**	14.78	6.32	**55**	13.46	6.04	**75**	14.72	6.01	**95**	15.01	5.98
16	14.85	6.20	**36**	14.68	6.36	**56**	14.92	6.44	**76**	14.99	6.10	**96**	14.93	5.95
17	14.98	6.16	**37**	14.45	6.25	**57**	14.85	6.31	**77**	14.72	6.37	**97**	14.76	5.94
18	15.42	6.22	**38**	14.66	6.07	**58**	14.50	6.37	**78**	17.80	6.39	**98**	14.84	5.79
19	15.27	6.26	**39**	14.67	6.30	**59**	14.23	6.31	**79**	14.89	6.33	**99**	14.98	5.99
20	15.63	6.34	**40**	14.98	6.16	**60**	14.38	6.40	**80**	14.93	6.41	**100**	15.08	5.96
												101	14.18	6.16

Table 5: Rate Ratio and 95% Confidence Intervals from Poisson Log-Linear Regression Models for Increases in Three-Day Moving Average Ambient $PM_{2.5}$ Concentrations.

Main Exposure	Overall	Warm Season	Cold Season
	RR (95% CI)	RR (95% CI)	RR (95% CI)
Grid-specific $PM_{2.5}$ 10 ug/m^3	1.015 (1.000, 1.029)	1.016 (0.997, 1.035)	1.024 (1.002, 1.047)
Lag 1 day	1.012 (0.999, 1.025)	1.001 (0.983, 1.019)	1.029 (1.008, 1.050)
Lag 2 day	1.016 (1.002, 1.030)	1.016 (0.997, 1.035	1.019 (0.998, 1.040)
Lag 3 day	0.011 (0.998, 1.024)	1.006 (0.989, 1.024)	1.010 (0.990, 1.003)
IQR	1.014 (1.001, 1.028)	1.014 (0.997, 1.031)	1.016 (1.001, 1.032)
Three-day moving average 10 µg/m^3 (continuous)	1.026 (1.014, 1.040)	1.014 (0.998, 1.031)	1.043 (1.023, 1.064)
IQR	1.023 (1.012, 1.035)	1.011 (0.998, 1.025)	1.025 (1.014, 1.037)

FIGURES

Figure 1: Geographic Distribution of 101 Study Grid Cells

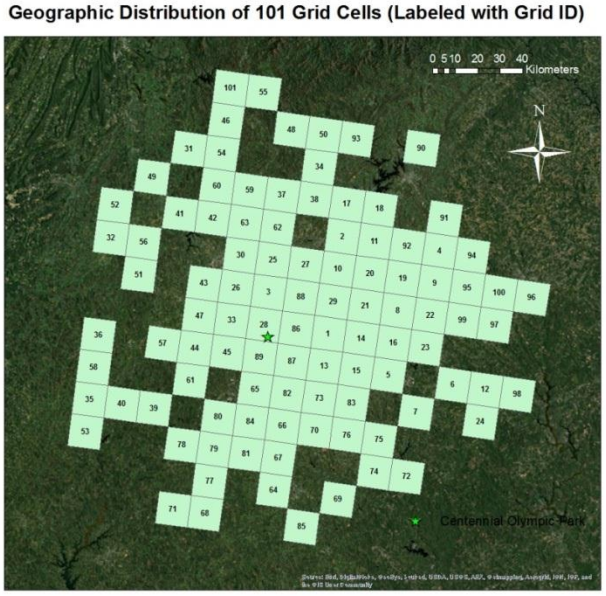

Figure 2: Distribution of Daily Grid Cells $PM_{2.5}$ Concentrations from 2001 to 2007

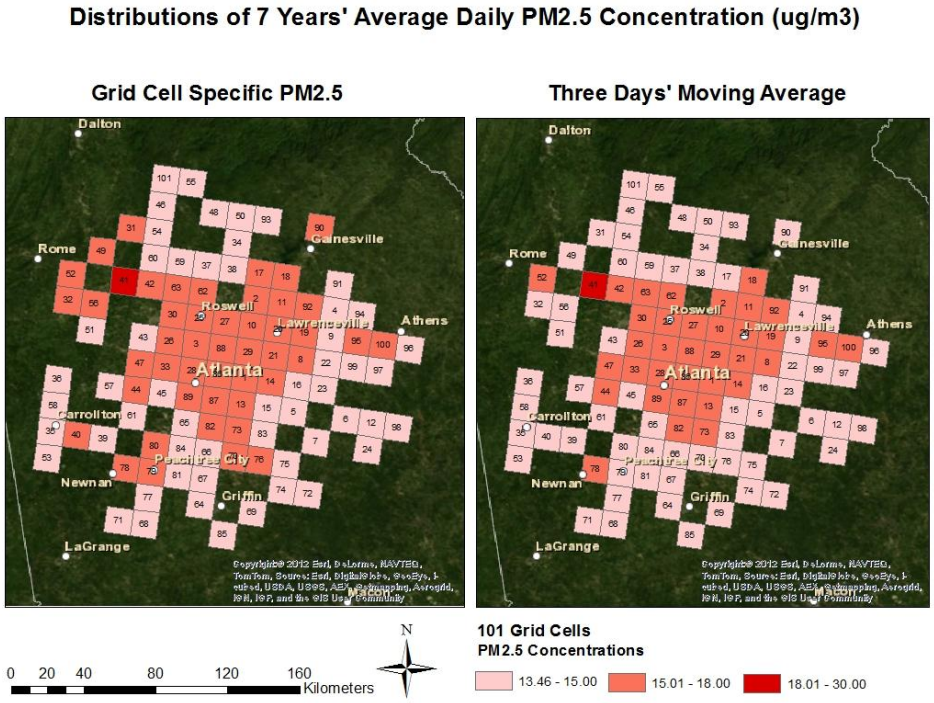

Figure 3

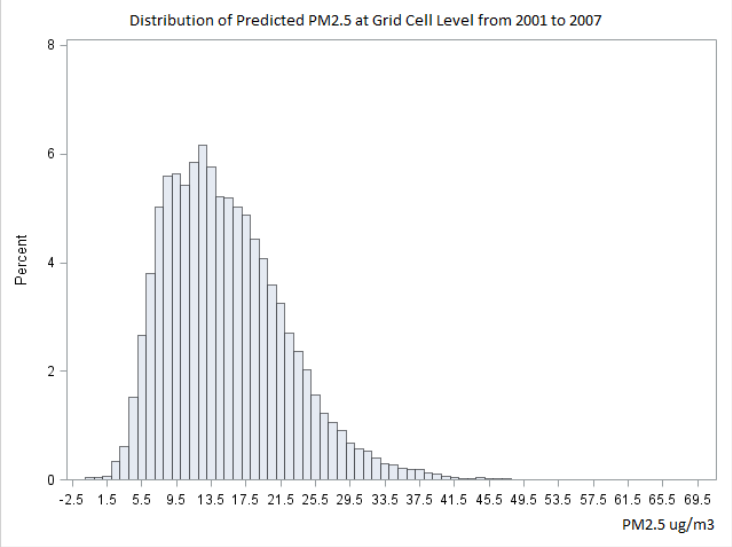

Figure 4

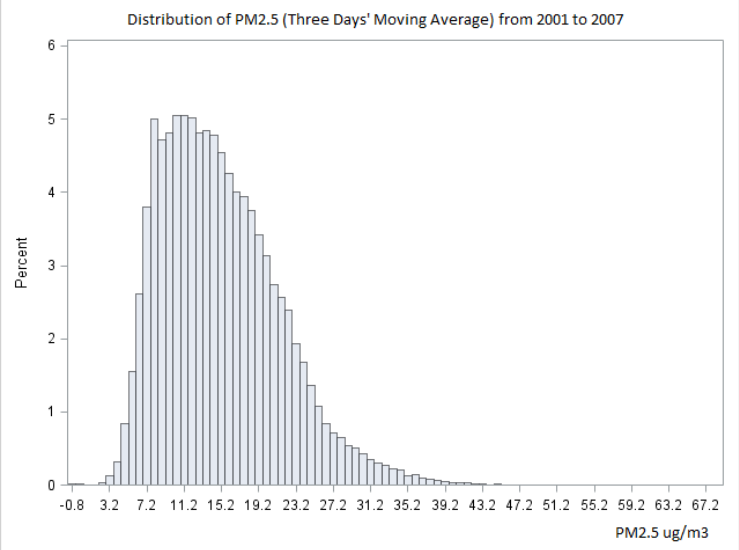

Figure 5:

Distribution of 7 Years' Daily Average Pediatric Asthma and Wheeze ED Visits in Atlanta