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Assessing the impact of fine particulate matter ($PM_{2.5}$) on respiratory-cardiovascular chronic diseases in the New York City Metropolitan area using Hierarchical Bayesian Model estimates

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ABSTRACT

An enhanced research paradigm is presented to address the spatial and temporal gaps in fine particulate matter ($PM_{2.5}$) measurements and generate realistic and representative concentration fields for use in epidemiological studies of human exposure to ambient air particulate concentrations. The general approach for research designed to analyze health impacts of exposure to $PM_{2.5}$ is to use concentration data from the nearest ground-based air quality monitor(s), which typically have missing data on the temporal and spatial scales due to filter sampling schedules and monitor placement, respectively. To circumvent these data gaps, this research project uses a Hierarchical Bayesian Model (HBM) to generate estimates of $PM_{2.5}$ in areas with and without air quality monitors by combining $PM_{2.5}$ concentrations measured by monitors, $PM_{2.5}$ concentration estimates derived from satellite aerosol optical depth (AOD) data, and Community-Multiscale Air Quality (CMAQ) model predictions of $PM_{2.5}$ concentrations. This methodology represents a substantial step forward in the approach for developing representative $PM_{2.5}$ concentration datasets to correlate with in-patient hospitalizations and emergency room visits data for asthma and inpatient hospitalizations for myocardial infarction (MI) and heart failure (HF) using case-crossover analysis. There were two key objective of this current study. First was to show that the inputs to the HBM could be expanded to include AOD data in addition to data from $PM_{2.5}$ monitors and predictions from CMAQ. The second objective was to determine if inclusion of AOD surfaces in HBM model algorithms results in $PM_{2.5}$ air pollutant concentration surfaces which more accurately predict hospital admittance and emergency room visits for MI, asthma, and HF. This study focuses on the New York City, NY metropolitan and surrounding areas during the 2004–2006 time period, in order to compare the health outcome impacts with those from previous studies and focus on any benefits derived from the changes in the HBM model surfaces. Consistent with previous studies, the results show high $PM_{2.5}$ exposure is associated with increased risk of asthma, myocardial infarction and heart failure. The estimates derived from concentration surfaces that incorporate AOD had a similar model fit and estimate of risk as compared to those derived from combining monitor and CMAQ data alone. Thus, this study demonstrates that estimates of $PM_{2.5}$ concentrations from satellite data can be used to supplement $PM_{2.5}$ monitor data in the estimates of risk associated with three common health outcomes. Results from this study were inconclusive regarding the potential benefits derived from adding AOD data to the HBM, as the addition of the satellite data did not significantly increase model performance. However, this study was limited to one metropolitan area over a short two-year time period. The use of next-generation, high temporal and spatial resolution satellite AOD data from geostationary and polar-orbiting satellites is expected to improve predictions in epidemiological studies in areas with fewer pollutant monitors or over wider geographic areas.
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1. Introduction

Fine particulate matter ($PM_{2.5}$), defined as particles with aerodynamic diameters $\leq 2.5\ \mu m$, has been shown to influence the frequency and severity of respiratory and cardiovascular

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diseases (e.g., Rom and Samet, 2006; Pope et al., 2004; Peters et al., 2001; Norris et al., 1999). $PM_{2.5}$ also increases inflammatory proteins and heart rate variability (HRV) in healthy volunteers (Samet et al., 2009). A common goal of public health programs in the United States is a reduction in the frequency and severity of such diseases (Talbot et al., 2009). In order to track the effects of an ambient air pollutant, such as $PM_{2.5}$, on public health, accurate measurements of the air pollutant are necessary in both time and space. $PM_{2.5}$ concentrations measured by the United States Environmental Protection Agency's (U.S. EPA's) national ground-based ambient air pollutant network provide a foundation for air pollution monitoring. The locations of individual $PM_{2.5}$ monitors across the nation are determined primarily by the requirements of state and local air pollution control agencies based on federal regulatory requirements for monitoring National Ambient Air Quality Standards (NAAQS) non-compliance areas and high-priority metropolitan areas. As a result, the national distribution of monitors does not follow a uniform or a probabilistically-based sampling plan, which would ensure some degree of optimal coverage for all areas of the country. Instead, most $PM_{2.5}$ monitors are located in urban and suburban areas, and consequently, there are significant gaps in coverage, particularly in rural regions. In addition, a subset of $PM_{2.5}$ monitors make measurements only every 3 or 6 days. As a result, there are substantial gaps in temporal coverage of $PM_{2.5}$ concentrations measurements across the nation as well.

Previous studies have addressed the spatial and temporal gaps in data from air pollution monitors in order to reduce characterization errors and more accurately predict the association of concentration data and health outcomes for epidemiological research (e.g., Goldman et al., 2010; Sarnat et al., 2010). One approach to augment the limited amount of available ambient air monitoring data is to combine these data with air quality model predictions using a statistically-based model, such as a Hierarchical Bayesian Model (HBM; McMillan et al., 2010). The HBM uses observed monitor concentration values and so-called surrogate concentration values, such as air quality model output and remotely-sensed data, to predict the “true” concentration surface values. The HBM gives more weight to highly accurate monitoring data in areas where monitoring data exist, and relies on bias-adjusted surrogate data in non-monitored areas. This approach provides the ability to predict important pollution gradients and uncertainties that might otherwise be unknown if only using interpolation results based solely on air quality monitoring data. The results derived from the HBM are useful for studies focused on health outcomes across large regions.

Recognizing the potential of the HBM technique to address spatial and temporal gaps in ambient $PM_{2.5}$ monitoring data, the Centers for Disease Control and Prevention (CDC) and U. S. EPA sponsored the development of an HBM as part of the Public Health Air Surveillance Evaluation (PHASE) project, which ran from 2004 to 2006 (CDC, 2016). PHASE was designed to identify spatial and temporal interpolation tools that can be used to generate daily surrogate measures of exposure to ambient air pollution, and relate those measures to available public health data. This initial version of the $PM_{2.5}$ HBM combined U.S. EPA $PM_{2.5}$ monitoring data and $PM_{2.5}$ predictions from the Community Multi-scale Air Quality (CMAQ) model (McMillan et al., 2010). The output from the PHASE HBM was incorporated into the CDC National Environmental Public Health Tracking Network (NEPHTN) for use by national, state, and local epidemiologists (Vaidyanathan et al., 2013).

While the initial PHASE-based version of HBM, which incorporated $PM_{2.5}$ monitor data and CMAQ $PM_{2.5}$ concentration predictions, represented a step forward in terms of generating $PM_{2.5}$ concentration fields that are accurate in time and space, it did not take advantage of remotely-sensed data. Remote sensing data, such as measurements of aerosol optical depth (AOD) taken by the MODerate resolution Imaging Spectroradiometer (MODIS) instruments on NASA's Terra and Aqua satellites, can provide information about $PM_{2.5}$ concentrations in areas where ground-based monitors do not exist. Satellite AOD is a unitless measure of the scattering and absorption of sunlight by particulate matter in a vertical column of the atmosphere between the satellite and Earth's surface. AOD is related to $PM_{2.5}$ concentrations, and many studies have shown that AOD can be used to estimate ground-level $PM_{2.5}$ concentrations (e.g., Hoff and Christopher, 2009; van Donkelaar et al., 2010; Weber et al., 2010). Although satellite AOD data do not represent the exact surface concentrations of $PM_{2.5}$, they capture the spatial distribution of the pollutant field in a way that monitor point measurements cannot (Liu et al., 2009; Gutierrez, 2010). In recent years, there has been interest in using satellite AOD data to assess the health effects of exposure to air pollutants (e.g., Kloog et al., 2011). However, previous studies have been limited to using satellite modeled data to conduct ecological studies of human health effects (e.g., Anderson et al., 2012a). This study sought to use satellite data to assess health effects of $PM_{2.5}$ using individual-level health data in a case-crossover analysis.

Satellite AOD represent physical observations of ambient $PM_{2.5}$ and therefore have the potential to supplement $PM_{2.5}$ monitor data and CMAQ model output combined by the HBM. A key objective of this study was to determine if the inclusion of MODIS AOD surfaces into the HBM results in $PM_{2.5}$ concentration surfaces that are more accurately able to predict associations and risks related to hospital admittance and emergency department (ED) visits for specific health outcomes at the individual patient level. The initial PHASE-based HBM was modified to allow for the incorporation of MODIS AOD-based estimates of surface $PM_{2.5}$ concentrations in addition to the basic components of $PM_{2.5}$ monitor data and CMAQ model output. $PM_{2.5}$ concentration surfaces generated by the HBM were compared to data on visits to the ED for asthma and inpatient hospitalizations for acute myocardial infarction (MI) and heart failure (HF). Case-crossover analyses were conducted to estimate the impact of short-term variations in $PM_{2.5}$ concentrations on the health effect outcomes using the methodology described in Haley et al. (2009). The case-crossover method has been used in recent years to assess the association of transient environmental exposures on acute health events (e.g., Schwartz, 2004). This design has been shown to yield similar associations as the traditional Poisson time series and the Cox Regression analysis (Peters et al., 2006; Fosbøl et al., 2014), with the added advantage of controlling for individual level confounding factors, trend and seasonality, and allowing assessment of effect-modification (Carracedo-Martínez et al., 2010). In this design, study subjects serve as their own controls, and the study is not subject to confounding by between-subject time-invariant unknown or unmeasured factors.

The New York City Metropolitan area of New York State was selected for this study based on geographic location, prior research on remote sensing of air pollution in those locations and availability of the New York State Department of Health (NYSDOH) to participate. The study area included New York City and the surrounding NY counties of Nassau, Suffolk, Westchester and Rockland (Fig. 1). Although the study area represents a limited geographic region, it was selected to allow for direct comparison to previous research. To facilitate comparison of results to the work of the PHASE project, the study period for this project covered the years 2004 through 2006. The expected outcome was that $PM_{2.5}$ concentration surfaces, as generated with the addition of satellite AOD data, would be more accurate for predicting asthma, acute MI, and heart failure health outcomes compared to surfaces that only incorporated $PM_{2.5}$ monitor data and CMAQ model output.

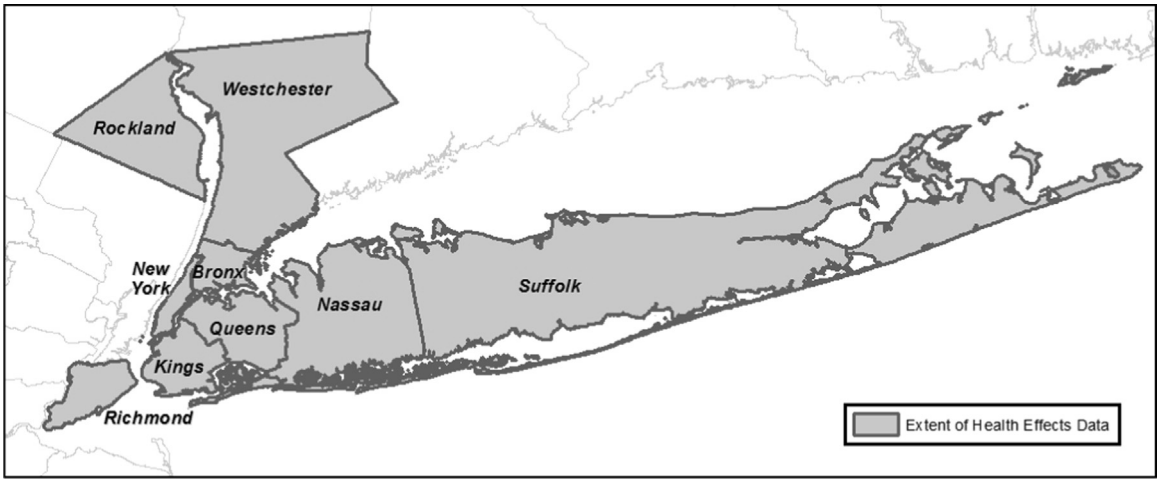

Fig. 1. Map of New York City study region.

2. Data and methods

2.1. Hierarchical Bayesian Model

The following sections outline the input data and methodology for developing estimated $PM_{2.5}$ concentrations from the HBM.

2.1.1. $PM_{2.5}$ concentration monitor data

Daily average $PM_{2.5}$ monitor data from Federal Reference Method (FRM) monitors for the entire study period of 2004–2006 were downloaded from U.S. EPA's Air Quality System (AQS) database (U.S. EPA, 2016). Each 24-h monitor observation was mapped to a 12 km × 12 km resolution CMAQ grid cell to facilitate integration into the HBM. Data from a total of 83 monitoring locations were used to generate the $PM_{2.5}$ input for the HBM. The HBM was run on an area larger than the study region to reduce potential edge effects on the estimated $PM_{2.5}$ in the study region and to include observations that may directly influence pollution concentrations in NYC. Specifically for the NYC study region, there were 33 FRM $PM_{2.5}$ monitors operating during the study period located in 19 CMAQ grid cells (Fig. 2). The FRM sites in New York are located in places that will likely have high concentrations of $PM_{2.5}$ and large monitoring scales (i.e., sites that see pollution effects from numerous, widespread sources). This ensures that the public is not exposed to higher ambient $PM_{2.5}$ concentrations than the concentrations from the FRM network reported for their area. For the NYC study region, these sites are generally co-located with school buildings in populated, urban areas. The analysis of $PM_{2.5}$ concentration from FRM monitors is filter-based, and the filters are collected on either a 3-day or a 6-day schedule, leading to missing data on the days when the filters are collected for analysis and subsequent reporting to the AQS database. When more than one daily monitor observation fell within a CMAQ grid cell, the values were averaged for that day. The resulting HBM input surface is an array of average $PM_{2.5}$ observations that have the dimensions of $day \times lon \times lat$, where *lat* and *lon* are the CMAQ grid centroid locations and *day* is an indicator of day of the year. Those grid cells that do not contain monitor readings on a given day are considered to have "missing" values.

2.1.2. CMAQ air quality model $PM_{2.5}$ concentration predictions

The CMAQ modeling system (CMAS, 2016) incorporates key physical and chemical transformations associated with the dispersion of air pollution at various geographic scales (Foley et al., 2010). CMAQ was designed to approach air quality modeling in a systematic manner by including state-of-the-science capabilities for modeling multiple air quality issues, including tropospheric ozone, air toxics, acid deposition, and fine particles (Hamilton

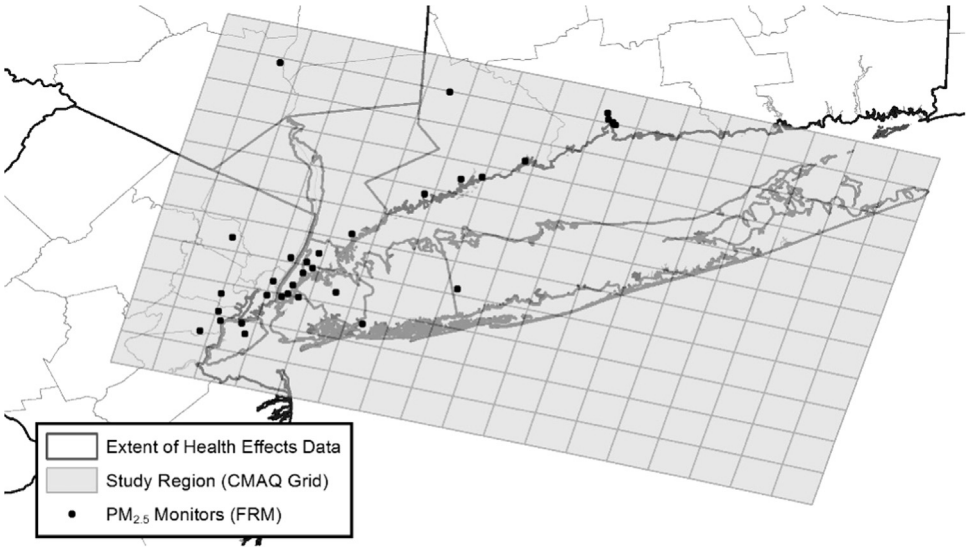

Fig. 2. Map of $PM_{2.5}$ monitors and CMAQ grid cell locations in the NYC study area.

Table 1
HBM model runs.

Model	Surface 1	Surface 2	Surface 3
A (Baseline)	Monitors	CMAQ	–
B	Monitors	AOD (missing data) Aqua/Terra	–
C	Monitors	AOD (missing data) Aqua/Terra	CMAQ
D	Monitors	AOD (kriged) Aqua/Terra	–
E	Monitors	AOD (kriged) Aqua/Terra	CMAQ

et al., 2009). CMAQ incorporates emission estimates from U.S. EPA's Office of Air Quality Planning and Standards (OAQPS) current emission inventories, observed emissions from major utility stacks, and modeled estimates of natural emissions from biogenic and agricultural sources from U.S. EPA's Biogenic Emissions Inventory System (BEIS). CMAQ relies on meteorological predictions from a mesoscale model, such as the Weather Research and Forecasting (WRF) or 5th generation Mesoscale Model (MM5). Emissions and meteorology data are processed in CMAQ algorithms, which simulate physical and chemical atmospheric processes to provide estimated concentrations of air pollutants.

Traditionally, CMAQ has been used to predict air quality across a regional or national domain at a resolution of 12 km × 12 km, and then to simulate the effects of various changes in emission levels for policymaking purposes. The CMAQ model has been used to identify locations of potential disproportionate air pollution exposure and health effects (Hamilton et al., 2009) and is the basis of the NOAA-EPA National Air Quality Forecasting Capability (NAQFC), which provides operational ozone forecasts and developmental $PM_{2.5}$ forecast guidance.

The daily average $PM_{2.5}$ concentrations predicted by CMAQ were subset to the grid cells that cover the study area (Fig. 2). The dimensions of the HBM input surface are the same as for the monitor data; however, the CMAQ input data do not contain missing values.

2.1.3. Satellite AOD-based estimates of $PM_{2.5}$ concentrations

AOD measured by the MODIS instrument onboard NASA's Terra and Aqua satellites were downloaded for the years 2004–2006 from the NASA Level 1 and Atmosphere Archive and Distribution System (LAADS) (NASA, 2016). The MODIS Terra and Aqua satellites have different overpass times, so the resulting data consisted of two observations collected per day, per location. The resolution of the MODIS Collection 5 AOD data product is 10 km × 10 km, therefore the data were re-mapped to the CMAQ 12 km grid for inclusion in the HBM. For each CMAQ grid cell (represented by a polygon), the AOD values that overlapped a specific grid cell were extracted, and the approximate fraction of each AOD grid cell that was covered by the CMAQ polygon was calculated. Those overlapping AOD values were used to calculate the weighted-mean AOD for each CMAQ grid cell, using only those AOD values that had fractions greater than 25% (i.e., more than 25% of the 10 km × 10 km AOD grid cell fell within the CMAQ grid polygon). This remapping procedure was applied separately for the AOD data from the Terra and Aqua satellites.

Drawing upon the methodologies of Zhang et al. (2009) and Weber et al. (2010), the relationship between the dimensionless AOD measurements from Aqua and Terra and 24-h average $PM_{2.5}$ concentrations for this region were calculated using linear regression methods to account for the spatial, seasonal and instrument dependence on the relationship between the total-column satellite observation of AOD and ground-level concentration. Since the relationships have been shown to have a seasonal dependence in the Eastern U.S., largely due to changes in the atmospheric mixing height, each season was calculated independently. The relationships were then used to convert AOD to estimated $PM_{2.5}$ concentrations in $\mu g/m^3$.

Two AOD input surfaces were generated to investigate the effect of missing data on the HBM predictions. The first allowed for missing values in the AOD input surface. In this case, the two observations of estimated $PM_{2.5}$ from Aqua and Terra were averaged using a weighted mean based on the standard error of the linear regression model fit for each day. If one of the satellites did not contain data for a given day and grid cell, then the data from the other satellite was used. If a grid cell contained no data from either satellite, then the data for that grid cell was considered missing. The second input surface took into account the spatial correlation of AOD by predicting data at grid cells that had no AOD observations through the use of ordinary kriging to create continuous AOD surfaces. The observations were then converted to estimated $PM_{2.5}$ values and combined across platforms, as described above.

2.1.4. HBM overview

For this study, the HBM was modified from the initial version used in the PHASE project (Battelle, 2011). The updated HBM can process input surfaces with missing data and can incorporate multiple input datasets simultaneously. These modifications allow the user to specify as many input surfaces (e.g., monitor, model, AOD) as necessary or desired to model the concentration "response" surface (e.g., selected combination[s] of monitor, model, AOD input surfaces). Table 1 enumerates the model runs that were performed and the corresponding input $PM_{2.5}$ surfaces. For each of the models, a daily, gridded surface of HBM-estimated $PM_{2.5}$ was generated and provided to NYSDOH for the epidemiologic analyses of the three selected respiratory-cardiovascular chronic diseases.

The "baseline" method, Model A, is currently used by CDC and U.S. EPA in the NEPHTN (CDC, 2016) to model $PM_{2.5}$ concentrations as a combination of the observed concentrations from the $PM_{2.5}$ monitoring network and estimated concentrations from CMAQ. An example of the model results and associated inputs for August 5, 2005 is provided in Fig. 3. The top panel for each Model is the estimated combined surface, while the bottom panel shows the input data. The HBM-combined surface is representative of 24-h average $PM_{2.5}$ concentrations. The high AOD observations for this day correlate with a regional haze event that was observed in the Mid-Atlantic region (Mubenga, 2005).

2.2. Case-crossover analysis

Time-stratified, case-crossover analyses were used to assess the effect of $PM_{2.5}$ on the risk of asthma ED visits and hospitalizations and acute MI and heart failure inpatient hospitalizations (Janes et al., 2005; Lu and Zeger, 2007; Mittleman, 2005). This method compares the air quality just before someone enters the ED or the hospital as an inpatient, with the air quality at reference times, before and after, within the same pre-specified stratum of time, when the person is not hospitalized or in the ED. Since each case serves as its own control, many important slowly varying personal characteristics that could be confounders, such as socioeconomic factors and smoking, are controlled by design.

2.2.1. Health outcome data

Data on outpatient emergency department and inpatient cases for asthma, and inpatient hospitalizations for MI and HF were obtained from the New York State Planning and Research Cooperative System (SPARCS) (NYSDOH, 2016). This system contains billing and medical abstract information from all hospitals in New York State except Federal and Veterans Administration hospitals. NYSDOH Institutional Review Board and Data Protection Review Board approvals were obtained to access individually identifying information such as address, date of birth, and date of hospital visit. All cases from January 1, 2004 to December 31, 2006 for

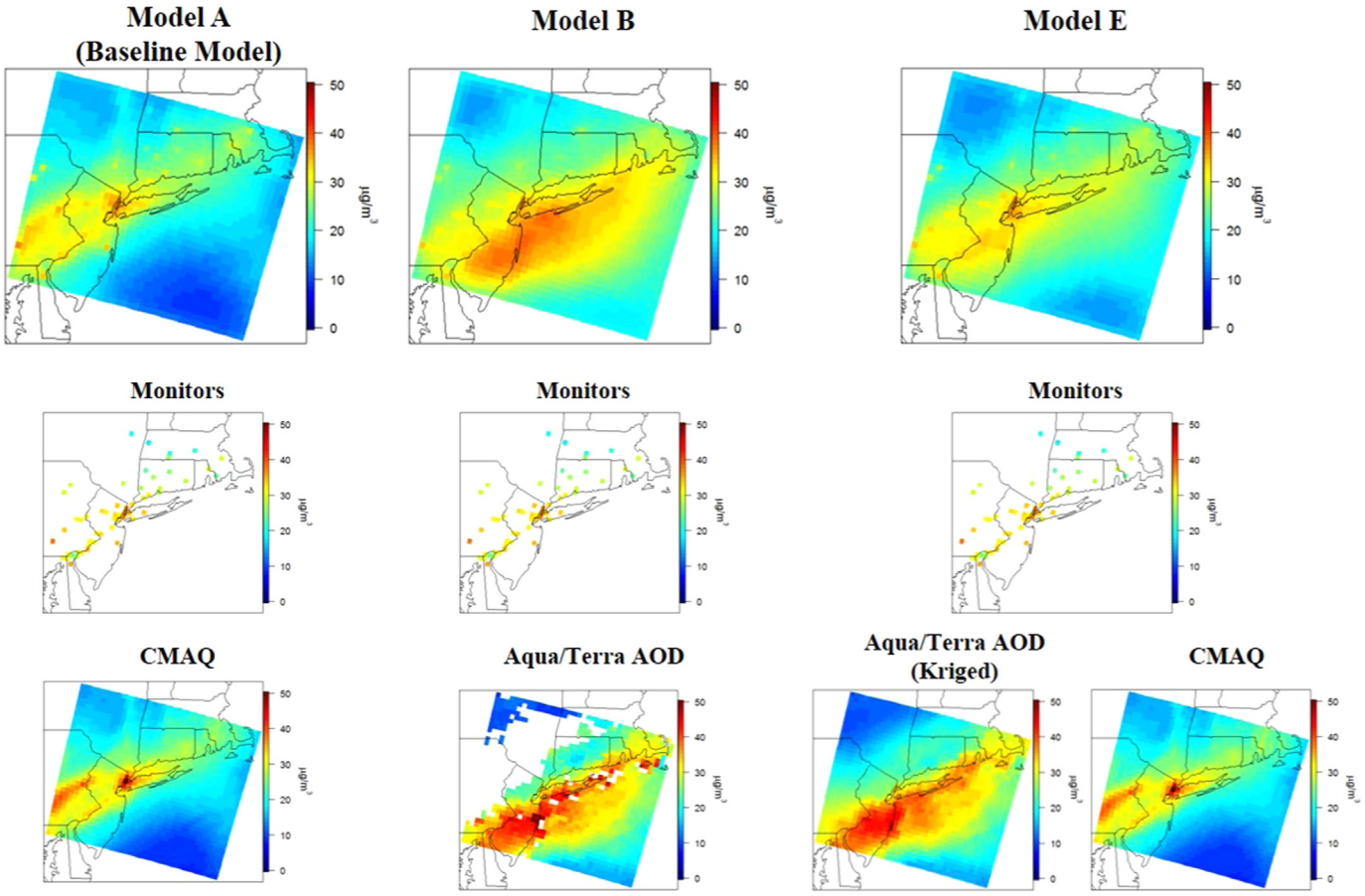

Fig. 3. Examples of input and combined $PM_{2.5}$ datasets for the New York City study area on August 5, 2005.

inpatient admissions were included in the initial analysis dataset for MI and HF. Cases where the patient was less than 35 years old were excluded for the cardiac outcomes because the pathophysiology of heart disease in younger patients is expected to be different than the pathophysiology of heart disease in older patients. Any recurrences within a period of 15 days from the original episode were excluded, with recurrences after that period considered as a new events. As some cases of asthma may be discharged directly from the ED, additional data were included to capture those cases. ED data in New York State has been available since 2005. Therefore, ED and inpatient cases for asthma were included from January 1, 2005 to December 31, 2006. Since asthma is an episodic disease affecting all ages, no age-based exclusions were made, and only recurrences within 7 days after the original episode were excluded.

Health outcome visits were assigned the mean population-weighted centroid corresponding to the patient's home postal ZIP code. These centroids were calculated using 2006 ZIP code boundaries obtained from TeleAtlas®, and census block population totals were obtained from the 2010. U.S. Census. Some ZIP codes in New York are only represented with a single latitude and longitude as opposed to a polygon representing a boundary; patients residing in these ZIP codes were assigned the geographic coordinates of the point location. The study area included a total of 633 ZIP codes, which included 201-point ZIP codes. Each case was mapped to an air pollution grid cell based on the population-weighted or point ZIP code coordinates.

2.2.2. Meteorological data

Daily average meteorological data, including temperature, humidity, and wind speed, were obtained from the National Climatic Data Center (NCDC) at the ZIP-code level (NCDC, 2016). The nearest station to each CMAQ grid centroid with weather data for that day was used. This study focused on the apparent temperature (AT), as it directly relates to the human body's ability to cool itself in hot conditions and the rate of heat transfer from a human body to the atmosphere in cold conditions. It is measured in degrees Fahrenheit (°F), and is derived from either (a) ambient temperature and wind (wind chill), or (b) ambient temperature and relative humidity. In this study, if the measured ambient temperature in a particular grid cell fell to 50°F or less, the wind chill was used in that grid cell for the AT (NOAA, 2016). When the ambient temperature in a grid cell rose above 80°F, the heat index was used as the measure of AT (Rothfusz, 1990). For temperatures between 51°F and 80°F, the AT corresponded to the measured ambient air temperature.

2.2.3. Census data

To assess effect modification due to socioeconomic status, ZIP code level data were obtained from the 2000. U.S. Census for the percent of population living under poverty for each ZCTA (U.S. Census, 2002). Each health outcome record was assigned a value for socioeconomic status based on the zip code of residence. Table 2 shows the total county-level population for the study area.

2.2.4. Case-crossover methodology

A time-stratified bidirectional method of control selection was used for the case-crossover analysis (Carracedo-Martinez et al., 2010). Each health outcome case was linked to the $PM_{2.5}$ exposure data resulting from all of the HBM $PM_{2.5}$ concentration surfaces listed in Table 1 based on the CMAQ grid cell that the patient's ZIP code of residence was assigned. One-month strata were used to

Table 2
County-level total population from 2000 Census.

County	Total population
Richmond County	468,730
New York County	1,585,873
Nassau County	1,339,532
Rockland County	311,687
Kings County	2,504,700
Westchester County	949,113
Queens County	2,230,722
Suffolk County	1,493,350
Bronx County	1,385,108

compare cases with control days of 7, 14, and 21 days before or after the case within the same stratum. Three control days were selected for every case day sampled. The comparisons were also made on the same day of the week to control for the possible contribution of differences in personal activity patterns. The time stratified design is not subject to bias resulting from time trends because there is no pattern in the placement of referents relative to the index time. Season and day of the week were also controlled for by restricting referents to the same day of the week, month and year as the index day. The time stratified design is considered a localizable, ignorable design and therefore yields unbiased estimates with conditional logistic regression analysis (Janes et al., 2005).

Lagged values were created for all epidemiologic exposure models to assess immediate (same day, i.e., lag 0) and delayed (previous 4 days exposure, i.e., lags 1, 2, and 3, and 4, respectively) effects of $PM_{2.5}$ on health outcomes. For the meteorological time-varying covariate of apparent temperature, the potential confounding role was evaluated as lag values of 0 and 1 day, with both a linear and a quadratic term to account for potential non-linear relationships between apparent temperature and health outcomes. Confounding due to major holidays, including the day of a holiday and the day after a holiday, as well as confounding by season (i.e., spring/summer vs fall/winter) were also evaluated. The dates for major holidays were obtained from the list of Federal holidays provided by the U. S. Government Office of Personnel Management (U.S. OPM, 2016).

2.2.5. *Epidemiologic model building*

A priori, different metrics were constructed to evaluate the temporal relationship between the different HBM $PM_{2.5}$ concentration surfaces and the time when an individual presented to a hospital ED or was admitted to the hospital as an inpatient. For asthma, ED cases and inpatient admissions were combined for analysis. For MI and HF, only inpatient admissions were considered in the analysis. The outcome measures were same day exposure: 1, 2, 3 and 4 days before the exposure day, and cumulative 2, 3, 4, and 5-day mean exposure estimates. Using purposeful selection with forward selection (Hosmer and Lemeshaw, 2000), the linear and quadratic terms were retained for lag 0 and lag 1, AT, season, holiday, and day after holiday dates in the preliminary model.

Once the confounding was resolved, all the interaction terms were added that were decided *a priori* based on evidence from disease pathology and prior literature. Effect modification was assessed for season, race, sex, age, rural/urban status, poverty, insurance and pre-existing diabetes or hypertension. Significance of the interaction terms was evaluated at $p < 0.20$ level, and the final model was selected, containing all relevant confounders and interaction terms, which provided the best fit according to the Akaike Information Criterion Correction (AIC_C) value for all models used. The AIC_C provides a measure of the quality of the statistical models used for a given input dataset based on the number of estimated model parameters, the maximum value of the likelihood function for a model, and sample size. The model selected with the lowest AIC_C value is the model that minimizes the "information losses" in the statistical analysis. Odds ratios estimates were calculated for a change of 10 μg/day in daily mean $PM_{2.5}$ values using conditional logistic regression with Proc PHREG in SAS™ statistical software Version 9.1. The coefficients for referent group and interactive terms were then linearly combined to assess effect modification in models with significant effect modifiers.

3. Results and discussion

There were two key objectives of this study. The first was to demonstrate that the HBM could be expanded to include input surfaces from satellite AOD data, in addition to $PM_{2.5}$ monitor data and CMAQ predictions of $PM_{2.5}$. The use of the three methodologies (monitor, model, AOD) to assess the $PM_{2.5}$ concentrations in the New York City geographical area represents an attempt to find the "true" but unmeasurable $PM_{2.5}$ concentration values. The CMAQ-based concentration values improve the spatial extent of $PM_{2.5}$, but assume uniform concentrations throughout each grid cell, which is unrealistic. The AOD-based concentration values provide spatial coverage, while yielding values based on actual physical measurements via remote sensing (satellites), which are converted to estimated concentration values. As shown in Fig. 3, the combination of these two methods provides a varying, non-uniform spatial estimate of $PM_{2.5}$ concentrations compared to using data from only monitors or from monitors and CMAQ. In this way, this study has successfully shown that the HBM model can incorporate the addition of AOD data and generate realistic $PM_{2.5}$ concentration surfaces. Subsequently, these HBM-generated $PM_{2.5}$ concentration surfaces were compared with data on inpatient hospitalizations and emergency room visits for asthma and inpatient hospitalizations for MI and HF using case-crossover analysis.

The average $PM_{2.5}$ values for the study period in New York City and surrounding areas according to the baseline HBM (Model A) were 10.02 ($\pm$ 7.45) μg/m^3 (Table 3). The other HBM combined surfaces had similar averages over the study period with B and E having slightly higher averages of 12.03 and 12.91 μg/m^3, respectively. Though these results are not valid for regulatory purposes, they can be compared to the $PM_{2.5}$ NAAQS to evaluate potential health effects from ambient $PM_{2.5}$ conditions. The average concentration values of all of the HBM $PM_{2.5}$ combined surfaces across the three-year study period are below the secondary annual $PM_{2.5}$ NAAQS of 15 μg/m^3. However, two of the HBM combined surfaces have an average concentration value that is above the primary annual NAAQS value of 12 μg/m^3. The maximum daily concentration averages for each HBM combined surface all exceed the 24-h NAAQS value of 35 μg/m^3, which highlights the fact that there were areas in the study region where ambient $PM_{2.5}$ concentrations posed an acute health risk.

Table 3
Descriptive statistics for 24-h average $PM_{2.5}$ datasets modeled with the HBM in the New York City area (67,952 12 km grid cells) averaged over the period 2004–2006 (μg/m^3).

Model	Mean	Std dev	Minimum	Maximum
A (Baseline)	10.02	7.45	0.02	57.62
B	12.03	5.65	0.48	49.85
C	10.09	6.85	0.02	55.50
D	10.51	5.98	0.03	51.06
E	12.91	5.55	0.54	52.66

Table 4
Descriptive statistics for Myocardial Infarction, Heart Failure and Asthma Cases in New York City and surrounding areas.

Primary diagnosis	Myocardial infarction[a]	Heart failure[a]	Asthma[b]
ICD9 code	***410***	***428***	***493***
Number of cases	60,939	114,137	236,568
Number of control days	182,817	342,411	709,704
Mean age (in years)	70.22	74	27.29
Average percentage of adults living below poverty	15.38	18.22	25.66
Male gender (percent)	55.6	46.2	46.44
Additional diagnoses			
Diabetes (percent)	35.43	43.23	3.76
Hypertension (percent)	55.89	54.82	6.6
Race			
Whites (percent)	62.88	55.22	21.92
Blacks (percent)	14.86	25.97	41.25
Payment source			
Medicare (percent)	51.18	61.35	7.14
Medicaid (percent)	9.26	11.45	20.26
Mean $PM_{2.5}$ for Baseline model on case days in $\mu g/m^3$	12.44	12.78	13.08
Mean $PM_{2.5}$ for Baseline model on control days in $\mu g/m^3$	12.32	12.66	12.98

[a] Excludes cases <35 years of age and recurrences within 15 days of initial occurrence. Admission dates from 1st Jan 2004–31st December 2006.
[b] Excludes recurrences <7 days. Inpatient and ED cases from 1st January 2005–31st December 2006 were combined.

Table 4 provides descriptive statistics for the cases and population of patients for the three health outcomes investigated in this study, MI, HF and asthma. Overall, there were 60,939 inpatient cases of MI after excluding cases with missing ZIP code information, cases where the patient was aged less than 35 years and cases that represented recurrences within 15 days of original episode. After these exclusions, the average age for the cases was 70 years, approximately 56% male and 63% White. There were 114,137 inpatient cases for heart failure after making exclusions similar to those for the MI analysis. The average age was 74 years but there was a lower percentage of males (46.2%) and White race (55.2%) as compared to those in the MI dataset. There were a total of 236,568 inpatient and ED cases presenting for asthma during the study period from Jan 1, 2005 to Dec 312,006 with exclusions only for those cases representing a recurrence within 7 days of initial occurrence. The average age for asthma patients was 27 years and they were more likely to be Black (41.25%) and female (54%).

Figs. 4–6 provide Odds Ratios (OR) and 95% Confidence Intervals (CI) for a 10-μg increase in $PM_{2.5}$ association with inpatient admissions for MI and HF and ED and inpatient admissions for asthma in the study area, respectively. The 5 HBM $PM_{2.5}$ combined surfaces (A through E, each for asthma, MI, and HF resulting in 15 total concentration surfaces/fields examined) yielded similar results. Model A (baseline model) and Model C had a consistently better fit based on AIC_C criterion and had a slightly better precision (based on the standard error of estimates).

For MI, there was a significant increased risk for exposure on the same day (Lag 0) for the Baseline model (OR = 1.018; 95% CI: 1.005, 1.032), Model C (OR = 1.017; 95% CI: 1.003, 1.031) and Model D (OR = 1.017; 95% CI: 1.001, 1.034) (Fig. 4). Results for exposure on previous 4 days and for cumulative exposure showed slightly elevated risk but did not approach statistical significance. No significant effect modification due to season, poverty, pre-existing diabetes or hypertension, insurance status, race, age-group or gender was found. Similarly, an elevated risk of heart failure at lag 0 for the Baseline model (OR = 1.017; 95% CI: 1.008, 1.027), Model C (OR = 1.017; 95% CI: 1.006, 1.020) and Model D (OR = 1.018; 95% CI: 1.006, 1.031) was observed (Fig. 5). There was no significant effect modification for the association between $PM_{2.5}$ and heart failure by any of the covariates listed for MI.

In multivariate models for asthma, highest significant risks were found for lag 1 (Baseline Model OR = 1.028; 95% CI: 1.021, 1.034) and cumulative 0–4 (Baseline Model OR = 1.055; 95% CI: 1.044, 1.066) (Fig. 6). For all of the examined models, exposures up to 4 days before were associated with increased risk of asthma with decreasing strength of association for the higher lags. A cumulative effect of exposure was also apparent with a distributed lag effect for exposures up to 4 days.

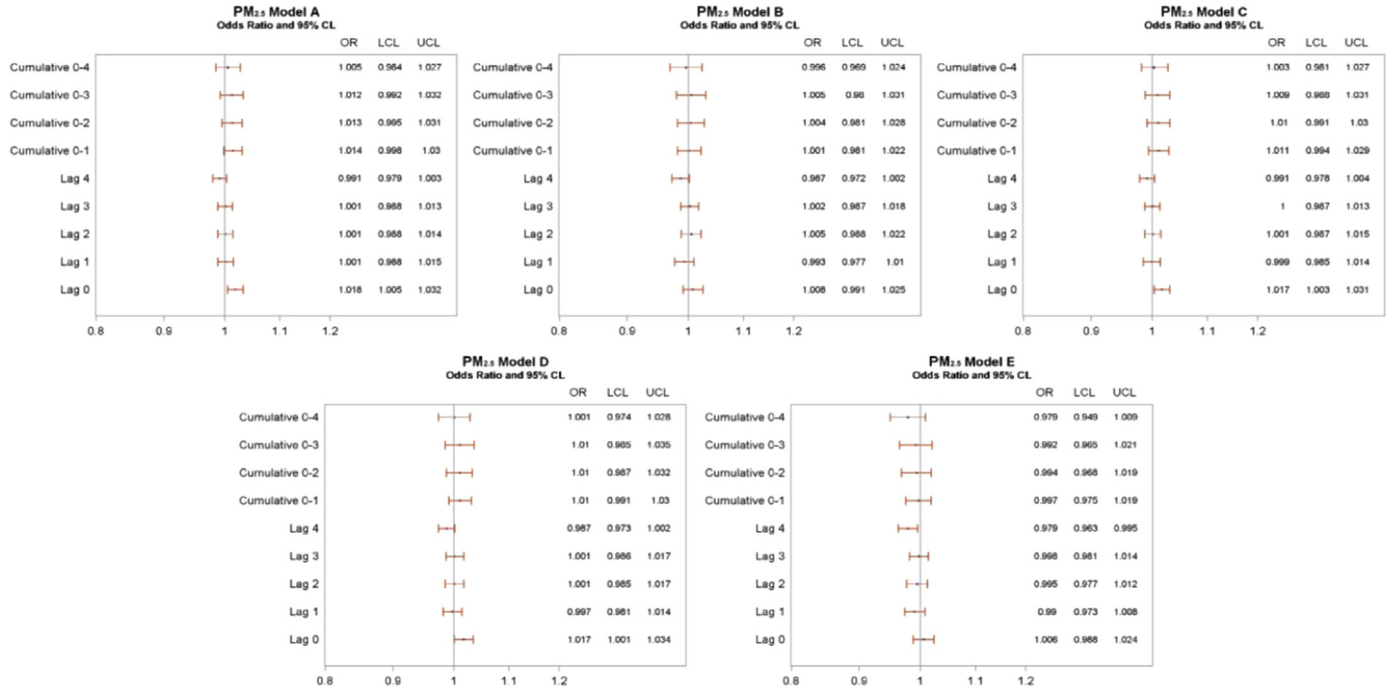

Fig. 4. Case crossover analysis of association of MI (myocardial infarction) hospitalization cases with a 10 unit change in $PM_{2.5}$ using various air pollution models in New York City Study area 2004–2006.

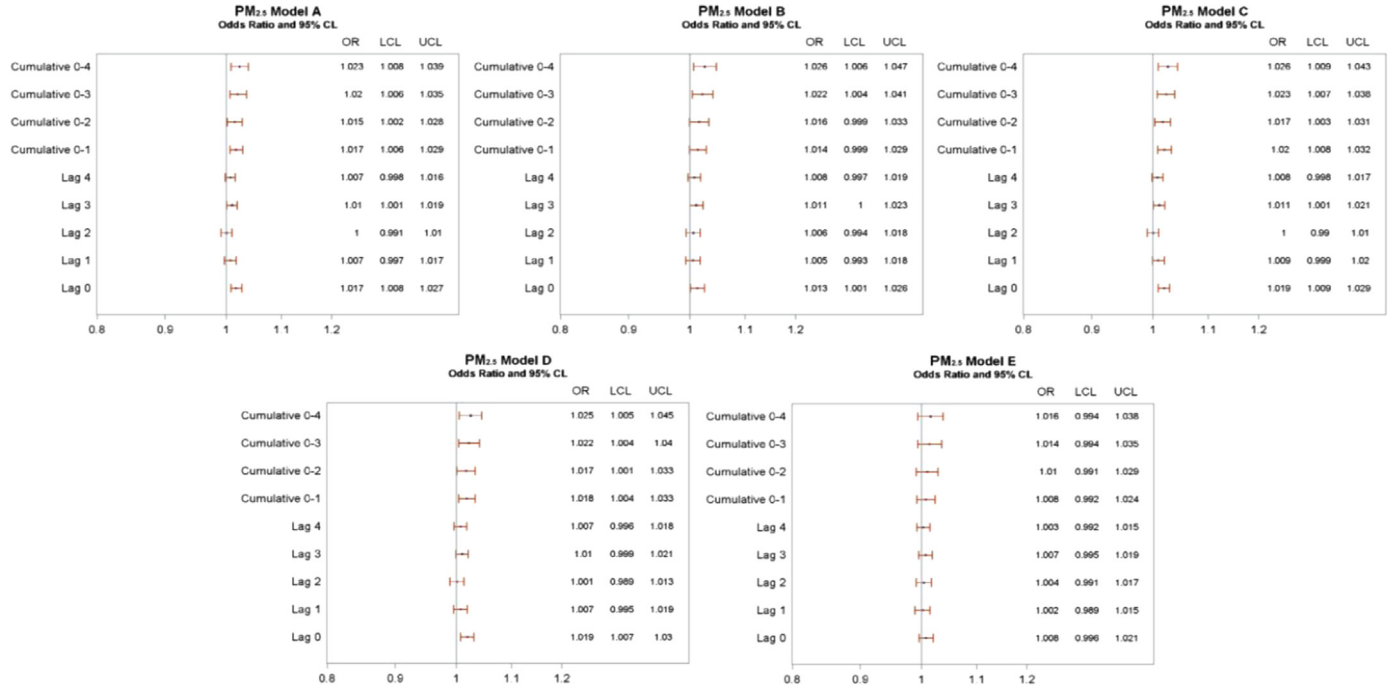

Fig. 5. Case crossover analysis of association of HF (heart failure) hospitalization cases with a 10 unit change in $PM_{2.5}$ using various air pollution models in New York City Study area 2004–2006.

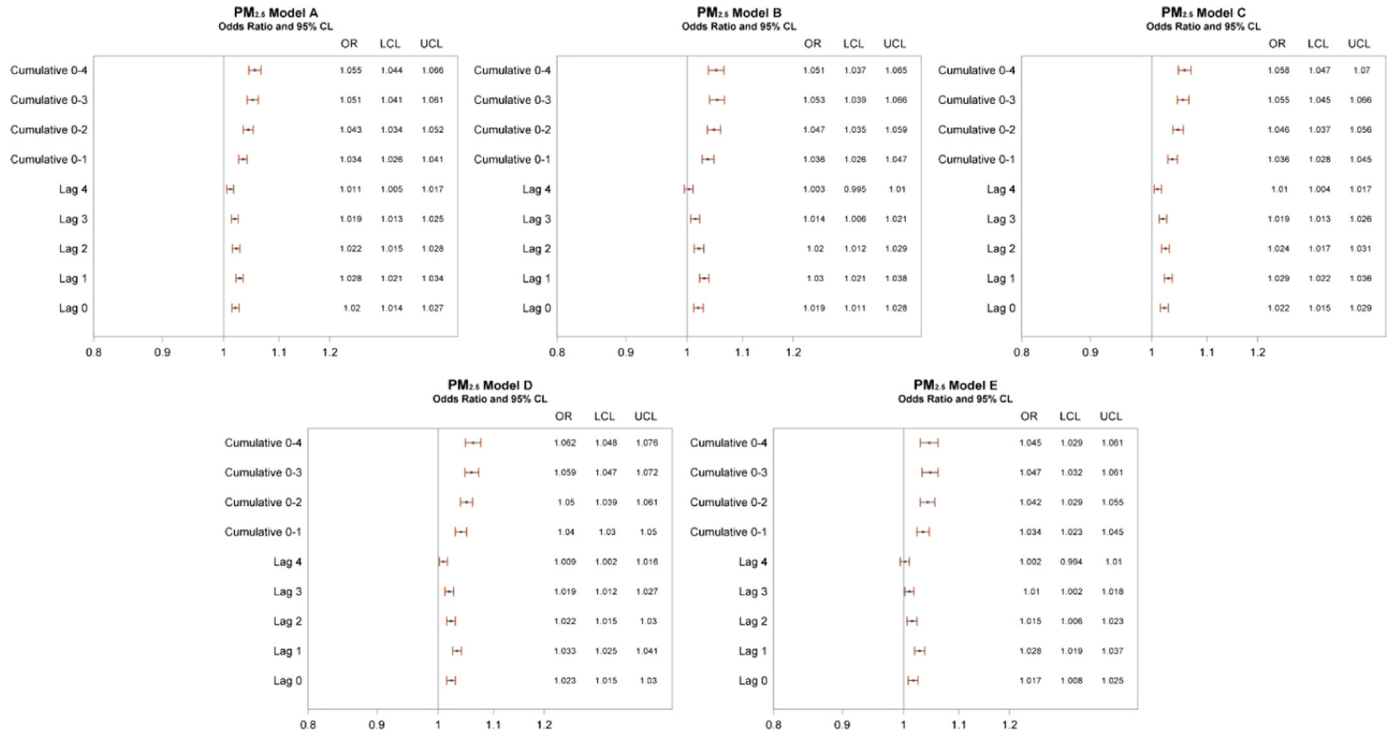

Fig. 6. Case crossover analysis of association of asthma hospitalization and ED cases with a 10 unit change in $PM_{2.5}$ using various air pollution models in New York City Study area 2005–2006.

On assessment of effect modification, significant multiplicative interactions with gender, inpatient/ED status and age for exposure at lag 1 were found (Table 5). Females who were aged ≤ 14 who were admitted with a primary diagnosis of asthma had the highest risk associated with $PM_{2.5}$ exposure on the previous day. In contrast males >14 years of age who presented with asthma at the ED had the lowest risk of these four categories. Similarly for cumulative exposure to $PM_{2.5}$, significant multiplicative interaction was found for age and exposure model (Table 6). Young children admitted for asthma had the highest risk associated with a cumulative exposure in the previous 3 days (Baseline Model OR = 1.093 (95% CI (1.070, 1.116)) while the lowest increased risk was among older individuals who presented at the ED (Baseline Model OR = 1.031 (95% CI (1.018, 1.044)).

Sensitivity analyses were conducted by restricting analysis to those inpatient heart disease cases that were transferred from the

Table 5
Effect modification results for case-crossover analyses: Odds ratios for Asthma with 10-unit change in $PM_{2.5}$ (lag1).

Model A						
	Inpatients			ED patients		
Females	Marginal OR	95% CI		Marginal OR	95% CI	
Age ≤ 14	1.069	1.053	1.086	1.046	1.033	1.059
Age > 14	1.038	1.024	1.052	1.015	1.005	1.025
Males						
Age ≤ 14	1.055	1.039	1.071	1.032	1.021	1.044
Age > 14	1.024	1.009	1.04	1.002	0.99	1.013
AIC:	699,015.75					
Model C						
	Inpatients			ED patients		
Females	Marginal OR	95% CI		Marginal OR	95% CI	
Age ≤ 14	1.073	1.055	1.092	1.049	1.036	1.063
Age > 14	1.039	1.025	1.054	1.016	1.006	1.027
Males						
Age ≤ 14	1.058	1.041	1.075	1.034	1.022	1.046
Age > 14	1.024	1.008	1.041	1.001	0.989	1.014
AIC:	699,019.14					

Multivariate adjusted for app temp (lag0 and lag1, linear) and (lag0 and lag1, quadratic) holidays, day after holidays and season

Table 6
Effect modification results for case-crossover analyses: odds ratios for asthma with 10-unit change in $PM_{2.5}$ concentration (cumulative average of days 0–3): .

Model A						
	Inpatients			ED patients		
	Marginal OR	95% CI		Marginal OR	95% CI	
Age ≤ 14	1.093	1.07	1.116	1.069	1.054	1.085
Age > 14	1.053	1.034	1.074	1.031	1.018	1.044
AIC:	698,983.2					
Model C						
	Inpatients			ED patients		
	Marginal OR	95% CI		Marginal OR	95% CI	
Age ≤ 14	1.098	1.074	1.123	1.074	1.057	1.091
Age > 14	1.058	1.037	1.08	1.034	1.02	1.049
AIC:	698,985.3					

Multivariate adjusted for apparent temperature (lag0 and lag1, linear) and (lag0 and lag1, quadratic) holidays, day after holidays and season

ED and by including only those recurrences that occurred after 28 days. However, the results for all models remained virtually unchanged with these restrictions. For asthma, additional analysis after excluding young children less than 2 years yielded results similar to those without the additional age restriction.

Thus, the results from this study confirm previous findings (e.g., Talbot et al., 2009; Haley et al., 2009), indicating that as $PM_{2.5}$ concentrations increase, the risk of asthma attacks, HF and MI also increase (e.g., Anderson et al., 2012b). The results also show that females, children less than 14 years old, and patients requiring hospitalization were at higher risk of asthma attacks after $PM_{2.5}$ exposure. Other studies have found positive associations between $PM_{2.5}$ and respiratory health in children (Schwartz and Neas, 2000) and females (Paulu and Smith, 2008).

The results of the study are inconclusive regarding the second objective, which was to determine if addition of AOD data to the HBM results in $PM_{2.5}$ air pollutant concentration surfaces that more accurately predict hospital admittance and emergency room visits for MI, asthma, and HF. Only minor differences in the reliability and precision of estimates based on model fit and standard errors of the estimates were found across the five (5) $PM_{2.5}$ exposure models tested in the analysis. In general, Model C (which combined data from three sources, monitors, CMAQ, and AOD [with missing data]) had results very similar to the Model A (baseline model using monitors and CMAQ) in terms of model fit, point estimate and precision. Model D (which combined monitor data with kriged AOD data) also had similar results in terms of point estimates, precision and model fit when compared to the baseline model. Models B and E had a somewhat lower precision than Models A, C and D. Thus, the model surfaces that incorporated AOD data were not definitively more accurate for predicting asthma, acute MI, and heart failure health outcomes compared to surfaces that only incorporated $PM_{2.5}$ monitor data and CMAQ model output. Given the limited geographical area and the narrow time period of this study (2004–2006), it is difficult to make any generalizations regarding the results of this study, however. This study represented the first test of incorporating AOD data into the HBM. HBM surfaces that include AOD data, in addition to $PM_{2.5}$ monitor data and CMAQ estimates of $PM_{2.5}$, are expected approach the "true" ambient $PM_{2.5}$ concentration values. It is expected that HBM surfaces that incorporate AOD data will be more useful for epidemiological studies over a longer time period and/or for a wider geographic area.

4. Conclusions

This study used ambient concentrations of $PM_{2.5}$ as a proxy for an individual's actual exposure to fine particulates. Actual exposures may be influenced by the extent to which ambient $PM_{2.5}$ concentrations infiltrate into indoor air spaces (such as automobiles, homes, schools, and work places) and the activity patterns of individuals (such as outdoor exercise, walking, commuting, etc.). MI and HF are indicators of underlying chronic heart disease. Chronic heart disease and asthma are long term medical conditions that progress at varying rates over a lifetime with many other environmental, biomedical and social factors. The study examined one event out of this continuum of disease to assess whether a high exposure to $PM_{2.5}$ may act as a trigger or precipitating factor. The cause of a specific disease episode may be defined as a precipitating event, prevailing condition, or specific factor that is necessary for disease occurrence, when it occurs, assuming all other factors or potential contributing conditions have no impact. Using this standard definition of disease causation, one or more events, conditions, or factors may be simultaneous precipitating cause(s) of a disease incident. In other words, $PM_{2.5}$ exposure in this scenario may be a contributing factor among others, but there is no way to determine if it is included as one of the minimum set of factors responsible for the observed increases in asthma, HF and MI. In order to link $PM_{2.5}$ exposure directly to asthma, HF and MI diseases, it must be shown to be the one of the necessary contributing factors to each disease, thereby demonstrating that there exists a complete causal mechanism explaining these disease occurrences. Applying the sufficient cause model (Rothman and Greenland, 2005; Rothman, 1976) to this situation, $PM_{2.5}$ exposure (at some point in time) must be a necessary causal factor in the disease models for asthma, HF and MI. The best way to illustrate this would be to examine baseline cases of asthma, HF and MI where there is no $PM_{2.5}$ exposure is present. If those cases subsequently experienced $PM_{2.5}$ exposure, without changes in other factors, and the incidence and severity of asthma, HF and MI increased for those particular cases, the scenario would clearly demonstrate that $PM_{2.5}$ exposure is the final causal component that explains these health outcomes.

Overall, the results of this study indicate that for one metropolitan area over a two year time period for asthma and a three year time period for MI and HF, estimates of $PM_{2.5}$ concentrations from satellite data can be used to supplement $PM_{2.5}$ monitor data in the estimates of risk associated with three common health outcomes, but the addition of the satellite data does not significantly increase model performance. Instead, the $PM_{2.5}$

concentration fields generated for this study using $PM_{2.5}$ monitor data, CMAQ $PM_{2.5}$ predictions, and satellite-based $PM_{2.5}$ estimates yielded health association estimates that were comparable to those calculated using a combination of only monitor and model data. However, this study was restricted to one urban area with a relatively dense monitoring network covering the high-population portions of the study region. The use of satellite AOD data should improve predictions in epidemiological studies in areas with fewer pollutant monitors or over wider geographic areas. The next steps for this project will be to repeat the experiment for a different metropolitan area (i.e., Baltimore, Maryland) to see if the same results hold. In addition, the release advent of next generation, high spatial and temporal resolution satellite AOD data, such as the MODIS 3 km-resolution AOD or AOD from the upcoming GOES-R geostationary satellite (Schmit et al., 2005), which will launch in October 2016, may hold the key to providing added value to $PM_{2.5}$ concentration fields.

Disclaimer

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