



EPA Public Access

Author manuscript

Atmosphere (Basel). Author manuscript; available in PMC 2021 May 11.

About author manuscripts

Submit a manuscript

Published in final edited form as:

Atmosphere (Basel). 2020 ; 11(2): 209. doi:10.3390/atmos11020209.

Contribution of Satellite-Derived Aerosol Optical Depth PM_{2.5} Bayesian Concentration Surfaces to Respiratory-Cardiovascular Chronic Disease Hospitalizations in Baltimore, Maryland

John T. Braggio^{*1}, Eric S. Hall², Stephanie A. Weber³, Amy K. Huff⁴

¹Mt. Diablo Analytical Solutions and Reporting Institute, Walnut Creek, CA 94595, USA

²U.S. Environmental Protection Agency (EPA), Research Triangle Park, NC 27709, USA

³Battelle Memorial Institute, Columbus, OH 43201, USA

⁴I. M. Systems Group, 5825 University Research Ct, Suite 3250, College Park, MD 20740, USA

Abstract

Licensee MDPI, Basel, Switzerland. This article is an open access article distributed under the terms and conditions of the Creative Commons Attribution (CC BY) license (<http://creativecommons.org/licenses/by/4.0/>).

*Correspondence: johntbs@msn.com; Tel.: +1-510-520-5149.

Author Contributions: Conceptualization, J.T.B. and E.S.H.; methodology, J.T.B., E.S.H., S.A.W. and A.K.H.; software, E.S.H.; validation, J.T.B., E.S.H., S.A.W., and A.K.H.; formal analysis, J.T.B. and A.K.H.; investigation, J.T.B.; resources, E.S.H.; data curation, J.T.B.; writing—original draft preparation, J.T.B.; writing—review and editing, J.T.B., E.S.H., S.A.W., and A.K.H.; visualization, J.T.B.; supervision, J.T.B.; project administration, E.S.H.; funding acquisition, J.T.B. All authors have read and agreed to the published version of the manuscript.

Supplementary Materials: The following analyses are available online at <http://www.mdpi.com/2073-4433/11/2/209/s1>, Table S1: CMAQ 12 km² grids with the 17 PM_{2.5} ambient air monitors in the Baltimore study, Table S2: PMB and the four experimental aerosol optical depth (AOD)-PM_{2.5} concentration surfaces in the Baltimore study area, Table S3: Percentiles (PCTL), means and 95% confidence intervals (CIs) for PMB and the four experimental aerosol optical depth (AOD)-PM_{2.5} concentration surfaces in Baltimore and New York City, Table S4: Correlations (*r*) and percent of variance (*r*²%) between PMB and the four experimental aerosol optical depth (AOD)-PM_{2.5} concentration surfaces by monitor status (All, Yes, No) in the Baltimore study, Table S5: Lag days 0–4 for PMB and the four experimental aerosol optical depth (AOD)-PM_{2.5} concentration surface means and 95% confidence intervals (CIs) measured in µg/m³ in the Baltimore Study: All CMAQ grids, Table S6: Lag days 01, 24 and 04 for PMB and the four experimental aerosol optical depth (AOD)-PM_{2.5} concentration surface means and 95% confidence intervals (CIs) measured in µg/m³ in the Baltimore study: All CMAQ grids, Table S7: Lag days 0–4 for PMB and the four experimental aerosol optical depth (AOD)-PM_{2.5} concentration surface means and 95% confidence intervals (CIs) measured in µg/m³ in the Baltimore study: CMAQ grids with monitors, Table S8: Lag days 01, 24 and 04 for PMB and the four aerosol optical depth (AOD)-PM_{2.5} concentration surface means and 95% confidence intervals (CIs) measured in µg/m³ in the Baltimore study: CMAQ grids with monitors, Table S9: Lag days 0–4 for PMB and the four experimental aerosol optical depth (AOD)-PM_{2.5} concentration surface means and 95% confidence intervals (CIs) measured in µg/m³ in the Baltimore study: CMAQ grids without monitors, Table S10: Lag days 01, 24 and 04 for PMB and the four experimental aerosol optical depth (AOD)-PM_{2.5} concentration surface means and 95% confidence intervals (CIs) measured in µg/m³ in the Baltimore study: CMAQ grids without monitors, Figure S1: Odds ratios (ORs) and 95% confidence intervals (CIs) for PMB and the four experimental aerosol optical depth (AOD)-PM_{2.5} concentration surfaces during the warm and cold seasons at lag day 0: (A) ED asthma (top left panel), (B) IP asthma (top right panel), (C) IP MI (bottom left panel), and (D) IP HF (bottom right panel), Figure S2: Odds ratios (ORs) and 95% confidence intervals (CIs) for PMB and the four experimental aerosol optical depth (AOD)-PM_{2.5} concentration surfaces during the warm and cold seasons at lag day 1: (A) ED asthma (top left panel), (B) IP asthma (top right panel), (C) IP MI (bottom left panel) and (D) IP HF (bottom right panel), Figure S3: Odds ratios (ORs) and 95% confidence intervals (CIs) for PMB and the four experimental aerosol optical depth (AOD)-PM_{2.5} concentration surfaces during the warm and cold seasons at lag days 01: (A) ED asthma (top left panel), (B) IP asthma (top right panel), (C) IP MI (bottom left panel) and (D) IP HF (bottom right panel).

Conflicts of Interest: The authors declare no conflict of interest. The funders had no role in the design of the study; in the collection, analyses, or interpretation of the results; in the writing of the manuscript, or in the decision to publish the results.

Publisher's Disclaimer: Disclaimer: EPA: through its Office of Research and Development, participated in this data analysis project. This manuscript went through Agency review and it was approved for publication. Although this work was reviewed by the EPA, and approved for publication, it may not necessarily reflect the official Agency policy. Mention of trade names or commercial products in this manuscript does not constitute endorsement or recommendation for use by the EPA or the authors.

The fine particulate matter baseline (PMB), which includes PM_{2.5} monitor readings *fused* with Community Multiscale Air Quality (CMAQ) model predictions, using the Hierarchical Bayesian Model (HBM), is less accurate in rural areas without monitors. To address this issue, an upgraded HBM was used to form four experimental aerosol optical depth (AOD)-PM_{2.5} concentration surfaces. A case-crossover design and conditional logistic regression evaluated the contribution of the AOD-PM_{2.5} surfaces and PMB to four respiratory-cardiovascular hospital events in all 99 12 km² CMAQ grids, and in grids with and without ambient air monitors. For all four health outcomes, only two AOD-PM_{2.5} surfaces, one not kriged (PMC) and the other kriged (PMCK), had significantly higher Odds Ratios (ORs) on lag days 0, 1, and 01 than PMB in all grids, and in grids without monitors. In grids with monitors, emergency department (ED) asthma PMCK on lag days 0, 1 and 01 and inpatient (IP) heart failure (HF) PMCK ORs on lag days 01 were significantly higher than PMB ORs. Warm season ORs were significantly higher than cold season ORs. Independent confirmation of these results should include AOD-PM_{2.5} concentration surfaces with greater temporal-spatial resolution, now easily available from geostationary satellites, such as GOES-16 and GOES-17.

Keywords

PM_{2.5}; AOD-PM_{2.5}; CMAQ; case-crossover; IP hospitalizations; ED visits: asthma; myocardial infarction; heart failure; season

1. Introduction

The adverse effects of PM_{2.5} on the respiratory-cardiovascular system have been repeatedly confirmed [1–12]. Epidemiologic studies that assess exposure risk to different ambient PM_{2.5} concentration levels have relied on the U.S. Environmental Protection Agency’s (EPA) network of ground-based ambient air pollutant monitors [4,13–16]. Ambient air monitors are not equally spatially distributed, and most make measurements every 3 or 6 days [13,16–18]. There are also characterization errors in available PM_{2.5} concentration measurements due to limited instrument measurement precision and spatial heterogeneity [19].

In 2004, the U.S. Centers for Disease Control and Prevention (CDC) and EPA established and logistically supported the CDC Public Health Air Surveillance Evaluation (PHASE) project [18,20–22]. One important PHASE project outcome was the development of the first-generation HBM that statistically fused PM_{2.5} monitor concentration readings with CMAQ PM_{2.5} model predictions [23–25]. In urban areas, PMB gives more “weight” to PM_{2.5} monitor readings than CMAQ PM_{2.5} model predictions. In rural areas, CMAQ PM_{2.5} model predictions exert more influence than PM_{2.5} monitor readings on PMB, since there are fewer monitors or no monitors. Ambient air monitors are usually found in urban areas. In the last 15 years, PMB has turned out to be a more representative PM_{2.5} concentration surface, compared to the interpolation of PM_{2.5} monitor data, as a method to resolve spatial gaps between ambient air monitors [16,18,22]. CDC subsequently incorporated PMB into its Environmental Public Health Tracking (EPHT) network of state and New York City partners

[16,18,22,26]. To date, PMB has been used by federal and state epidemiologists completing EPHT projects in different parts of the US [16,18,22,26].

Within this decade, the availability and use of satellite AOD data have become more routine [6,16,27–31]. Newer generation satellite instruments measure AOD with increased temporal accuracy and finer spatial resolution [27,32–37]. AOD is a unitless measure of the scattering and absorption of visible light by aerosols (particles) in the atmosphere [38–40]. AOD data are, by definition, actual physical measurements, an improvement over CMAQ PM_{2.5} model predictions. Once AOD unitless measurements have been calibrated with actual PM_{2.5} readings from on-the-ground ambient air monitors, it is then possible to utilize the derived AOD-PM_{2.5} concentration readings to estimate *actual* ambient PM_{2.5} concentration in areas where there are no on-the-ground air monitors. The relationship between AOD measurements and on-the-ground measurements of PM_{2.5} concentration readings has been confirmed in available publications [16,27–29,39–44].

By incorporating AOD-PM_{2.5} concentration values into the currently utilized PMB, we hypothesized there would be a further improvement in the fused AOD-PM_{2.5}-PMB surface [16]. Our intention, in this preliminary work, was to test this hypothesis by using these four experimental AOD-PM_{2.5} and PMB fused concentration surfaces with linked health outcome data from Baltimore, Maryland, and New York City, New York, in a case-crossover epidemiologic study design data files analyzed by using conditional logistic regression [16]. From the beginning, our expectation was to complete the Baltimore and New York City epidemiologic studies at the same time [16]. Unexpected circumstances related to restricted access to the Maryland emergency department (ED) and inpatient hospitalization IP confidential hospital data files, delayed the completion of the Baltimore AOD-PM_{2.5} study component, and this delay made it possible for the Baltimore investigators to examine additional questions, such as completing a fine-grain evaluation of areas with and without ambient PM_{2.5} monitors. The New York City study, published in 2016, did not find differences between the four experimental AOD-PM_{2.5} concentration surfaces and PMB [16].

These were the objectives of the Baltimore study: firstly, replicate the New York City study by using all CMAQ 12 km² grids, and completing further analyses in grids with and without PM_{2.5} ambient air monitors. Secondly, determine if the four experimental AOD-PM_{2.5} concentration surfaces differed from PMB in grids with and without monitors. Thirdly, evaluate warm season versus cold season differences.

2. Methods

2.1. Description of Study Participants

This study used the same experimental methods described in the New York City publication [16]. The same procedural steps used in the two study sites are summarized in this Methods section, with more detail included on differences unique to the Baltimore study.

Figure 1 is a map of the 11 (south–north) by 9 (west–east) 12 km² CMAQ grid cells, shown in blue circles, and the 17 Federal Reference Method (FRM) PM_{2.5} ambient air monitors,

shown in red triangles. Baltimore City had 6 PM_{2.5} ambient air monitors, more than in Anne Arundel (4), Prince George's (3), Baltimore (2), Montgomery (1) and Harford (1) Counties. Other Counties in the study area did not have a single ambient air monitor. Table S1 (Supplementary Materials) includes more information on each of the 17 FRM PM_{2.5} ambient air monitors, e.g., CMAQ grid row-column identifier, site number, county name, city name, years in operation, between 2004–2006.

2.2. HBM Inputs

2.2.1. Monitor PM_{2.5}—Baltimore study area 2004–2006 24-hour average PM_{2.5} concentration data files were downloaded from the EPA Air Quality System (AQS) database [17]. The locations of the Maryland study area FRM PM_{2.5} monitors were mapped to the 12 km² CMAQ grid system prior to their incorporation into the upgraded HBM. If a grid cell contained two or more monitor concentration readings on any given day, a single daily mean was computed and retained. Grid cells without monitor measurements on a given day were set to the “missing” data condition.

2.2.2. CMAQ PM_{2.5}—The 2004–2006 Baltimore study area CMAQ PM_{2.5} model predictions were obtained from EPA's Community Modeling and Analysis System website [24,25]. CMAQ PM_{2.5} model predictions did not have missing values.

2.2.3. AOD-PM_{2.5}—Moderate Resolution Imaging Spectroradiometer (MODIS) Collection 5 AOD 2004–2006 data files, at 10 km² resolution, were downloaded from the National Aeronautics and Space Administration (NASA) Level 1 and Atmosphere Archive and Distribution System (LAADS) [45]. AOD data were available for Terra and Aqua satellites. There were two MODIS AOD observations per day, one for each satellite. Terra satellite had a late morning observation time, while the Aqua satellite had an early afternoon observation time. MODIS AOD data were re-mapped from NASA's 10 km² native grid system to CMAQ's native 12 km² grid system before the AOD data were uploaded in the upgraded HBM. Additional AOD dataset preparation details, including the technique used to estimate surface PM_{2.5} concentrations from the AOD data, have been published [16,46,47].

The HBM runs for New York City and Baltimore produced two AOD-estimated PM_{2.5} datasets: one AOD dataset with missing observations, and another dataset where the missing observations were replaced with kriged values (ordinary kriging). The original HBM required separating and sequencing the Bayesian computations into discrete hierarchical steps and then modeling at each level, before combining PM_{2.5} monitor measurements with CMAQ model predictions [48,49]. The original HBM was modified to process input surfaces with missing data and simultaneously combine multiple datasets [46]. This latter HBM upgraded feature was “critical” to permit the incorporation of AOD-PM_{2.5} input surfaces, in addition to PM_{2.5} monitor concentration readings and CMAQ PM_{2.5} model predictions.

2.3. AOD-PM_{2.5} Fused Surfaces

The upgraded HBM was used to produce PMB and the four experimental AOD-PM_{2.5} concentration surfaces, at the same time and utilizing the same statistical procedures, for

both New York City [16] and Baltimore: (1) PMB statistically fused PM_{2.5} monitor concentration measurements with CMAQ PM_{2.5} model predictions. (2) PMC, the first AOD-PM_{2.5} surface, fused PM_{2.5} monitor concentration measurements with AOD-PM_{2.5} concentration values. This PMC included missing observations resulting from satellite recording failure or cloud cover interference in the recording column, extending from a satellite to the surface of the earth. (3) PMCK fused PM_{2.5} monitor concentration measurements with kriged AOD-PM_{2.5} concentration values. Kriging eliminated missing data from the pre-kriged PMC surface. (4) PMCQ included monitor PM_{2.5} concentration readings, CMAQ PM_{2.5} model predictions and PMC (not kriged). (5) PMCKQ incorporated monitor PM_{2.5} monitor concentration readings, CMAQ PM_{2.5} model estimates and PMCK (kriged). The shared properties of the four experimental AOD-PM_{2.5} fused surfaces made it possible to evaluate differences between AOD surfaces with and without missing observations (not kriged [PMC, PMCQ] vs. kriged [PMCK, PMCKQ]) and the absence and presence of CMAQ PM_{2.5} model predictions (absent [PMC, PMCK] vs. present [PMCQ, PMCKQ]). Table S2 (Supplementary Materials) summarizes additional information about PMB and the four experimental AOD-PM_{2.5}-fused surfaces into columns that included PMB/AOD-PM_{2.5} surface name, surface description, and input surfaces used to produce each fused surface.

2.4. ED/IP Chronic Diseases

2.4.1. Subjects—The 2004–2006 Maryland electronic ED visits and IP hospitalization confidential patient files, with one hospital record per patient per hospital encounter, were obtained from the Maryland Health Services Cost Review Commission (HSCRC) [50]. All hospitals in Maryland must report, by State statute, their ED visits/IP hospital events to the HSCRC quarterly. Available temporal variables only included years (2004–2006), quarters (winter, spring, summer, fall), and days of the week (Sunday through Saturday), but excluded month. Patient’s residential information was limited to 5-digit United States Postal Service Zone Improvement Plan (ZIP) codes (Maryland Department of Planning; MDP) [51]. Each patient record contained demographic variables, primary and secondary diagnoses, entered as International Classification of Diseases, Ninth Revision, Clinical Modification (ICD-9-CM) billing codes [52]. The ICD-9-CM codes were used to select asthma (493), myocardial infarction (MI, 410) and heart failure (HF, 428), as primary discharge diagnoses, and the co-morbid conditions of diabetes mellitus (250), hypertension (401), and atherosclerosis (414, 440), as secondary diagnoses. The Maryland Department of Health (MDH) Institutional Review Board approved this Baltimore AOD-PM_{2.5} data linkage and analysis study.

2.4.2. Cases–Controls—The case-crossover design was used to form three different controls for each case [53–57]. Controls differed from cases only in the assigned exposure period. A case had one quarterly exposure value. Each control had one monthly mean exposure value for each of the four quarters: the first control had monthly mean exposure data for January, April, July, and October; the second control had monthly mean exposure data for February, May, August, and November; the third control combined monthly mean exposure data for March, June, August, and December. Each monthly mean exposure data represented a different calendar quarter (January–March, winter; April–June, spring; July–

September, summer; October–December, fall). For each of the three controls, the selected four monthly mean exposures values were averaged, and the overall mean was used as the annual background exposure estimate.

Maclure's [56] case-crossover design paper proposed using cases as their own controls but assigning a different exposure period to the same cases that were also used as controls. Since the same cases are used as controls, the cases and controls should not differ from each other on patient attributes such as gender, age, race, and insurance coverage. This is what we did in this study, because the month temporal variable was not available in the confidential hospital files obtained from the Maryland HSCRC, but was available in the New York City study [16].

The overall average for four months, with each month serving as a proxy for each of the four quarters in one year, provided an estimate of annual (background) exposure level. Since a different month was used to represent each quarter in each of the three controls, the exposure assignment algorithm used in this study is “functionally” equivalent to the case-crossover bidirectional design used in the New York City study [16,53]. In this study, cases with 1st and 2nd quarter exposure values always preceded all three controls because their mean monthly ranks ($Q_1 = 2.0$, $Q_2 = 5.0$) were always numerically smaller than the mean monthly ranks of all three controls (1st = 5.5, 2nd = 6.5, 3rd = 7.5). However, when cases had 3rd and 4th quarter exposure values they always came after all three controls, because their mean monthly ranks ($Q_3 = 8.0$, $Q_4 = 11.0$) were always numerically larger than the mean monthly ranks of all three controls.

2.5. Confounders

2.5.1. Co-Morbid Conditions (Diabetes, Hypertension, Atherosclerosis)—

Diabetes, hypertension, and atherosclerosis, when present in a patient's hospital record, can synergistically contribute to the occurrence of a patient's ED visit or IP hospitalization with a discharge diagnosis of asthma, MI, or HF. Diabetes mellitus, hypertension, and atherosclerosis by themselves have been shown to lead to an ED visit or IP hospitalization [13,16,58–62].

2.5.2. Apparent Temperature (AT and AT²)—

Ambient temperature, relative humidity, and wind speed can contribute to the occurrence of respiratory-cardiovascular chronic disease ED visits or IP hospitalizations. AT is one summary variable that includes ambient temperature, relative humidity, and wind speed. All three weather parameters were obtained from the CMAQ model [25] and made available to the Baltimore investigators by one of the co-authors (ESH). AT was computed using the formula reported on the National Oceanic and Atmospheric Administration website (NOAA) [16,63,64]. AT² was computed as the product of AT*AT, once AT was available. Both AT and AT² have been shown to influence respiratory-cardiovascular ED visits and IP hospitalizations, even in the absence of elevated ambient PM_{2.5} [13]. AT and AT² values were computed for each CMAQ grid cell, which were stratified by year, month, and day.

2.5.3. Pollen—

Recent publications have implicated ambient pollen levels in respiratory-cardiovascular chronic disease ED visits/IP hospitalizations [65–67]. The single pollen

counting station in Baltimore County, Maryland, provided the multi-year pollen readings. They were used as proxy measures for ambient pollen levels in the Baltimore study area [68].

2.5.4. Holidays—Each major holiday included the day after each holiday. The dates of recognized holidays were obtained from the U.S. Office of Personnel Management website (OPM) [69]. Each holiday was combined in a single annual dummy variable and coded as 1 (holiday and day after) or 0 (no holiday). The holiday dummy variables were entered in each of the three annual data files for 2004–2006.

2.5.5. Snowstorms—Each snowstorm was coded as a dummy variable, 1 (snowstorm) or 0 (no snowstorm), for each of the three years separately (National Centers for Environmental Information (NCEI) [70]. The snowstorm variable was a proxy for physical exertion during winter snow removal and its precipitation of a cardiovascular event, such as MI taking place, followed by an ED visit or IP hospitalization.

2.6. Effect Modifiers

2.6.1. Poverty and Population Density—Population density and poverty co-occur more often in urban areas compared to rural locations. Brochu and colleagues [71] found a significant inverse association between economic resources and ambient $PM_{2.5}$ concentration levels. Bell and Ebisu [72] found a higher poverty percent in census tracts with ambient air monitors compared to census tracts without monitors. Both are associated with barriers to healthcare access, e.g., fewer hospitals in rural than urban areas, and more frequent use of ED medical services by persons with fewer economic resources and no health insurance. Maryland Zip Code Tabulation Area (ZCTA) poverty percent and population density (subsequently converted to the \log_{10} scale) data were obtained from the US Census Bureau website (USCB) [73].

2.6.2. Season—Although Weber et al. [16] did not find warm–cold season differences, others have found season differences in the contribution of $PM_{2.5}$ to respiratory-cardiovascular chronic disease hospital events [12,59,74–79]. Warm vs. cold season differences were evaluated in this study.

2.7. File Linkage

Given that the ED visit and IP hospitalization files included temporal variables for year (2004–2006), quarter (winter, spring, summer, fall), day of week (Sunday through Saturday) and spatial variables for residential five-digit ZIP codes, it was necessary to map these ZIP codes to CMAQ grids. It was also necessary to map ZCTA polygons for poverty percent and population density \log_{10} measures to CMAQ grids.

ZIP code and ZCTA latitude–longitude centroid coordinates were entered in a Geographic Information System (GIS), which included a multi-layered map of Baltimore City/Maryland Counties and a 11 (south to north) by 9 (west to east) 12 km^2 CMAQ grid map layer of the Baltimore study area, to develop a ZIP code-ZCTA-CMAQ 12 km^2 grid polygon correspondence file. This assignment was done for each year separately [51,73,80].

Latitude-longitude centroid coordinates of 5-digit ZIP codes and ZCTAs were mapped to the interior area of a unique CMAQ 12 km² grid cell. This ZIP code/ZCTA polygon assignment to CMAQ km² grid cells was done for each of the three years separately, 2004–2006.

Base Statistical Analysis System (SAS) software, version 9.4, was used to link PMB and the four experimental AOD-PM_{2.5} concentration surfaces, de-identified ED/IP hospitalization records, confounders and effect modifiers by using the same assigned CMAQ grid identifier (1–99) and the temporal variables of year (3), quarter (4) and day of the week (7) [81].

2.7.1. Case-Crossover Analyses—Each stratum included one case and three controls. Based on the time-space grouping variables of year (three), quarter (4), day of week (7), and spatial CMAQ 12 km² grid cells (99), there were 8316 possible combinations of space–time locations that a case paired with three controls could be assigned. SAS/STAT Proportional Hazards Regression (PHREG) Procedure (Proc) was used to perform a regression analysis of stratified subpopulation’s “survival time” data. These conditional logistic regression analyses, based on the SAS/STAT PHREG Proc, use the Cox proportional hazards model, and the results explain the effect of time-dependent explanatory variables on survival times [82]. SAS/STAT PHREG Proc, version 14.3, and Base SAS, version 9.4, software programs were used to complete all conditional logistic regression analyses [57,81,82]. In these regression analyses, ties (for [censored] survival/failure times or [uncensored] event times under the Cox proportional hazards model) were set using the Breslow methodology [82,83].

2.7.2. Statistical Analyses—The Chi Square test, in Base SAS, version 9.4, was used to evaluate case-control count data differences in age categories, gender, race, co-morbid conditions and health insurance [81]. By comparing the mean of one group with the lower and upper values for the 95% Confidence Interval (CI) of the reference group mean, it was possible to determine if the two means were significantly different from each other at $p < 0.05$. A group mean was significantly different from the reference group mean if the value for the first comparison mean was either below the lower limit or above the upper limit of the 95% CI of the reference group mean ($p < 0.05$). The Means Procedure in Base SAS, version 9.4, was used to compute means and 95% CIs for poverty percent and population density [81].

2.8. Conditional Logistic Regression Analyses

2.8.1. Variable Selection—A multi-step variable evaluation procedure was used for all conditional logistic regression runs in all CMAQ grid cells, with and without ambient air monitors [57,84]. The starting statistical run only contained PMB, or one experimental AOD-PM_{2.5} concentration surface, and confounders (holidays, snowstorms, pollen, and AT and/or AT²). The index ED visit/IP hospitalization day was identified as lag day 0. The day before was lag day 1, and so on, through to lag day 4. Summary lag day measures for individual lag days of 0 through 4 were also computed. Summary lag days were obtained by taking the average of the included individual lag days. To illustrate, summary lag days 0 and 1, displayed as lag days 01. Lag days 2–4 were referenced as lag days 24. Lag days of 0 through 4 were named lag days 04. The AT² was also entered on lag days 0, 1, 01 and 04. Effect modifiers were evaluated in separate conditional logistic regression runs (diabetes

mellitus, hypertension, atherosclerosis; gender, age, race; health insurance, poverty, population density; and, season). An effect modifier was retained and included in the subsequent variable assessment runs if it had a computed probability value of $p \leq 0.20$.

2.8.2. Variable Evaluation of Effect Modifiers—This variable evaluation phase involved utilizing PMB or one of the four experimental AOD-PM_{2.5} concentration surfaces, and retained confounders on lag days of 0–4, 01, 24, and 04, in all grids and in grids with and without monitors. AT² was added on lag days of 0, 1, 01 and 04. Each retained effect modifier was evaluated in a separate run. Each effect modifier with $p \leq 0.20$ was evaluated in the final conditional logistic regression runs.

2.8.3. Final Models—A stepwise procedure, with a variable entry criterion of $p \leq 0.20$ and variable stay criterion of $p \leq 0.09$, was used to identify the most parsimonious combination of confounders, effect modifiers for each PMB and the four experimental AOD-PM_{2.5} concentration surfaces in all grids, and in grids with and without monitors. The Akaike Information Criterion (AIC) statistic was also used to confirm the selection of the “best” conditional logistic regression runs, with lower AIC values representing a better model fit [84]. The null hypothesis was rejected when $p \leq 0.05$ [85].

2.8.4. Season and Monitor—Follow-up conditional logistic regression analyses evaluated the main factors of season (S), monitor (M) and the interaction term of S*M, for PMB and the four experimental AOD-PM_{2.5} concentration surfaces, at lag days of 0, 1 and 01, in all grids, and in grids with and without monitors, for ED asthma, IP asthma, IP MI, and IP HF, in separate analyses.

3. Results

3.1. Cases and Controls

Case-control group characteristics are in Table 1 (ED and IP asthma) and Table 2 (IP MI and IP HF). As stated previously, there were three controls for each case in each of the four groups. There were more ED asthma cases (11,723) than IP asthma cases (3376), with IP MI (4745) and IP HF (6919) between two asthma case groups. ED asthma cases were significantly younger than IP asthma cases ($p \leq 0.05$). The 0–14 age category included 43.8% ED asthma cases but only 27.6% IP asthma cases. The 35+ age category contained 61.8% of the IP asthma cases but only 28.7% of the ED asthma cases. There was also a significant age category difference between IP HF cases and IP HF controls ($p \leq 0.05$), with lower percentages for cases (18.5%) and controls (19.2%) in the 35–59 age category and higher percentages for cases (44.9%) and controls (46.0%) in the 76+ age category. Other significant differences were found between grids without monitors and grids with monitors for poverty percent and population density for all case and control groups (all p 's ≤ 0.05).

3.2. AOD-PM_{2.5} Concentration Surfaces

3.2.1. PMB and AOD-PM_{2.5}—Only the PMCK PM_{2.5} concentration three-year mean of 14.38 $\mu\text{g}/\text{m}^3$ (95% CI = 14.31–14.44) was significantly higher than the PMB PM_{2.5} three-year mean of 14.19 (95% CI = 14.13–14.26) ($p \leq 0.05$). The other three experimental AOD-

PM_{2.5} concentration surface means (PMC = 13.66 (95% CI = 13.60–13.72), PMCQ = 13.79 (95% CI = 13.73–13.85), and PMCKQ = 13.91 (95% CI = 13.85–13.97) were significantly lower than the PMB mean (all p 's < 0.05). The unexpected finding was confirmation that the Baltimore PMB and four experimental AOD-PM_{2.5} concentration surfaces were *significantly higher* than the New York City PMB and four experimental AOD-PM_{2.5} concentration surfaces (PMB = 10.02; PMC = 12.03, PMCK = 10.51; PMCQ = 10.09; PMCKQ = 12.91) (all p 's < 0.05). Table S3 (Supplementary Materials) summarizes these and other descriptive statistics (percentiles) for PMB and the four experimental AOD-PM_{2.5}-fused surfaces.

3.2.2. Correlations between AOD-PM_{2.5} and PMB—The r^2 % statistic, representing the percentage of shared variance between PMB and another experimental AOD-PM_{2.5} concentration surface, was highest between PMB and PMCQ in all three grid conditions (both, 94.7%; with monitors, 97.4%; without monitors, 94.3%). This measure of shared variance was lowest between PMCK and PMB (both, 30.6%; with monitors, 62.1%; without monitors, 26.5%). The r^2 % difference (no monitors versus monitors) produced a similar ranking, with the smallest negative difference for PMCQ (–3.1%), and the largest for PMCK (–35.6%), followed by PMCKQ (–15.9%) and PMC (–32.4%) with intermediate values between the first two. Table S4 (Supplementary Materials) contains correlation analyses for PMB with each of the four experimental AOD-PM_{2.5}-fused surfaces.

3.2.3. PM_{2.5} Concentration Values—Three-year PM_{2.5} concentration means (95% CIs) were computed for all lag days, surfaces, and monitor grid conditions. All comparisons were between each AOD-PM_{2.5} concentration surface and PMB. PMCK PM_{2.5} concentration means were significantly higher than PMB PM_{2.5} concentration means in all grids and in grids without monitors for all lag days (all p 's < 0.05). The monitor PMCK PM_{2.5} concentration surface was significantly lower than the monitor PMB PM_{2.5} concentration surface at all lag days (p < 0.05). All other comparisons between PMC, PMCQ and PMCKQ PM_{2.5} concentration surfaces were also significantly lower than PMB for all lag days of 0–4, 01, 24 and 04 in all grids, in grids with monitors and in grids without monitors (all p 's < 0.05). Supplementary Materials includes three-year PM_{2.5} concentration mean values for PMB and each of the four AOD-PM_{2.5} concentration surfaces on lag days 0–4, 01, 24 and 04 in all CMAQ grids (Tables S5 and S6), in grids with monitors (Tables S7 and S8) and in grids without monitors (Tables S9 and S10).

3.3. Conditional Logistic Regression

Significant conditional logistic regression analyses for the four health outcomes, three CMAQ grid conditions, and the four experimental AOD-PM_{2.5} concentration surfaces and PMB only occurred at lag days of 0, 1 and 01 (all p 's < 0.01). Significant, but protective, population density (M) effect modifiers were found for ED asthma in all grids for PMB (lags of 01), PMCQ (lags of 0, 1 and 01) and PMCKQ (lags of 01) (all p 's < 0.01). Significant, but protective, Season (S) effect modifiers occurred for ED asthma in all grids at lags of 01 (PMB, PMC, PMCK, PMCQ and PMCKQ) and for IP HF in all grids at lags of 01 (PMB, PMC, PMCK, PMCQ, PMCKQ) (all p 's < 0.05).

Odds Ratios (ORs) and 95% Confidence Intervals (CIs) for the four health outcomes, under the three grid conditions, PMB and the four experimental AOD-PM_{2.5} concentration surfaces, are displayed graphically below, and separated into successive graphs by lag days of 0, 1 and 01. To determine if the AOD-PM_{2.5} concentration surface ORs differed from the PMB OR, each AOD-PM_{2.5} OR was compared to the PMB's OR (95% CI). If the AOD-PM_{2.5} OR was either below or above the PMB's 95% CI lower or upper limit, the outcome was significant ($p < 0.05$).

3.3.1. Lag Day 0 (Figure 2A–D)—For ED asthma (Figure 2A) in all grids (Both) and in grids without monitors (No) each of the four AOD-PM_{2.5} ORs were significantly higher than the PMB OR (all p 's < 0.05). However, in grids with monitors (Yes), only the PMCK OR was significantly higher than the PMB OR ($p < 0.05$). For IP asthma (Figure 2B), IP MI (Figure 2C) and IP HF (Figure 2D) in all grids and in grids without monitors, only PMC, PMCK and PMCKQ ORs were significantly higher than PMB ORs (all p 's < 0.05). For all four health outcomes, only PMC and PMCK had significantly higher ORs in the no monitor condition than the PMC and PMCK ORs in the monitor condition (all p 's < 0.05).

3.3.2. Lag Day 1 (Figure 3A–D)—The ED asthma (Figure 3A), IP asthma (Figure 3B), IP MI (Figure 3C) and IP HF (Figure 3D) OR results for all comparisons between each of the four AOD-PM_{2.5} concentration surfaces with the PMB ORs at lag 1 in all grids, and in grids without and with monitors were identical to the comparisons made on lag day 0 and described above (all p 's < 0.05). No monitor versus monitor OR comparisons for all four health outcomes were also the same as those previously described above on lag day 0 ($p < 0.05$).

3.3.3. Lag Day 01 (Figure 4A–D)—ED asthma (Figure 4A), IP asthma (Figure 4B), IP MI (Figure 4C) and IP HF (Figure 4D) comparisons between AOD-PM_{2.5} ORs and PMB ORs at lag days 01 were the same as the comparisons at lag day 0 and lag day 1 and described above (all p 's < 0.05). For IP asthma, IP MI, and IP HF in the no monitor grid condition PMCQ ORs were significantly higher than the PMB ORs (all p 's < 0.05). In addition, in grids with monitors for ED asthma and IP HF PMCK ORs were significantly higher than the PMB ORs (both p 's < 0.05). The no monitor OR versus the monitor OR comparisons for PMC and PMCK were the same as those previously described for lag days 0 and 1 (all p 's < 0.05). In addition, for ED asthma the no monitor PMCKQ OR was significantly higher than the monitor PMCKQ OR ($p < 0.05$).

3.4. Lag Day and Monitor

Further evidence supporting the robustness of PMC and PMCK concentration surfaces, especially in grids without monitors, is summarized in Figure 5A–D. Percentage change in ORs in grids without monitors versus ORs in grids with monitors at lag days of 0, 1 and 01 demonstrate the same pattern for ED asthma (A), IP asthma (B), IP MI (C) and HF (D): (1) PMC and PMCK concentration surfaces had positive percent change values, with the largest increase always occurring at lag days 01. (2) All PMB percent change values were negative. (3) PMCQ results resembled most the PMB outcomes, with negative percent change values at lag days of 0, 1 and 01 for IP asthma (B) and at lag days of 0 and 1 for IF HF (D), or close

to 0% change for IP MI (C) and ED asthma at lag days of 0 and 1 (A). (4) In all four panels, the PMCKQ surface had positive percent change values, thereby suggesting that kriging partly reversed the percent decrease due to the inclusion of CMAQ.

3.5. Lag Day and Season

Results of follow-up conditional logistic regression analyses evaluating warm–cold season (S) differences at lag days of 0, 1 and 01 on PMB and the four AOD-PM_{2.5} concentration surfaces support these conclusions: (1) only the cold season ORs were protective, e.g., below 1.000. (2) All warm season ORs were significantly higher than cold season ORs (all p 's < 0.05). (3) During the warm season, only ED asthma, IP MI, and IP HF PMCK ORs were significantly higher than PMB ORs (all p 's < 0.05). Figure 6 below shows the percent change between warm season and cold season ORs for the four AOD-PM_{2.5} concentration surfaces and PMB on lag days of 0, 1 and 01 for the four hospital-based health outcomes. For all four health outcomes the largest percent increase occurred at lag day of 01. Figures S1–S3 (Supplementary Materials) display warm–cold season ORs for the four AOD-PM_{2.5} experimental surfaces and PMB for ED asthma, IP asthma, IP MI, and IP HF at lag days of 0, 1 and 01, respectively.

4. Discussion

The shared objectives of the Baltimore and New York City [16] studies were to evaluate the differential contribution of the four experimental AOD-PM_{2.5} concentration surfaces relative to the current baseline, PMB, on ED asthma, IP asthma, IP MI and HF hospitalizations. Our expectation was to find the most improvement in the new experimental AOD-PM_{2.5} concentration surface, which included PMC (not kriged or kriged) fused with PMB (monitor PM_{2.5} and CMAQ PM_{2.5} model estimates). The New York City study did not find differences between PMB and the four AOD-PM_{2.5} concentration surfaces. Contrary to our expectation, these Baltimore study results suggest that PMC and PMCK, and not PMCQ or PMCKQ, are better estimates of ambient PM_{2.5}, especially in grids without monitors.

Although the same upgraded HBM was used to generate the PMB and the four AOD-PM_{2.5} concentration surfaces for Baltimore and New York City, all five Baltimore surfaces had *significantly higher* three-year mean PM_{2.5} concentration values than the New York City surfaces. Since both the Baltimore and New York City study sites analyzed the same asthma, MI and HF chronic diseases, used the case-crossover design to create three controls for each case, and used the same SAS conditional logistic regression procedure to analyze the linked exposure–health outcome files, the only remaining *major* difference was the significantly elevated ambient PM_{2.5} levels in Baltimore compared to New York City.

The three methodological differences between the two study sites included: (1) more asthma, MI, and HF cases (and associated controls) in the New York City study than in the Baltimore study. (2) Completion of separate ED asthma and IP asthma conditional logistic regression analyses in the Baltimore study than in the New York City study, because ED asthma cases (and associated controls) were significantly younger than the IP asthma cases (and associated controls). (3) In the Baltimore study, the three controls were formed by assigning a different exposure period to the cases that were also used as controls [56], while

the New York City study selected the three controls within a 28 day strata. Both test site replications attained the same case–crossover endpoint, since each case was preceded or followed by at least one control—the definition of a bidirectional case-crossover design [53,55]. It is unlikely that these methodological differences would be more important than the fact that the Baltimore study three-year mean PMB and four AOD-PM_{2.5} concentration surfaces had significantly higher concentration values than the New York City study.

This Baltimore study also found differences in the contribution of PMB and the four experimental AOD-PM_{2.5} concentration surfaces to asthma ED visits and IP asthma, IP MI, and IP HF hospitalizations in grids without monitors. In grids without monitors, PMC, PMCK and PMCKQ ORs were significantly higher than PMB ORs at lag days of 0, 1 and 01, for all four ED/IP respiratory-cardiovascular chronic diseases. In grids with monitors, only ED asthma PMCK ORs were significantly higher than PMB ORs for all three lag days of 0, 1 and 01. A similar outcome occurred for IP HF, but only on lag days of 01. Additional no monitor versus monitor analyses showed that only PMC and PMCK had significantly higher ORs in grids without monitors than in grids with monitors at all three lag days of 0, 1 and 01 for all four respiratory-cardiovascular chronic disease hospital events. The only exception was for IP asthma at lag day of 1, where the PMC no monitor versus monitor comparison was not significant.

Since two AOD-PM_{2.5} surfaces included CMAQ PM_{2.5} model estimates, PMCQ (PMC not kriged) and PMCKQ (PMCK kriged), we now know that a bias is introduced by the addition of CMAQ PM_{2.5} model estimates to the experimental AOD-PM_{2.5} concentration surfaces. This bias was expressed by shifting the PM_{2.5} concentration values of this surface, PMCQ, toward the PMB PM_{2.5} concentration values. PMCKQ resembled PMB less, a reversal that can be attributed to kriging.

Results from this Baltimore study support these conclusions: Firstly, only PMCQ ORs in all grids at lag days of 0, 1 and 01 and in grids without monitors at lag days of 0 and 1, were not significantly different from PMC ORs for IP asthma, IP MI and IP HF. Secondly, no monitor PMCQ, PMCKQ and PMB ORs were not significantly different from monitor PMCQ, PMCKQ and PMB ORs at lag days 0, 1 and 01 for ED asthma, IP asthma, IP MI, and IP HF. Thirdly, the difference in the r^2 statistic between PMCQ and PMB in grids with monitors (97.4%) and grids without monitors (94.3%) was negatively smaller (–3.1%) than it was for PMCKQ (–15.9%), PMC (–32.4%) and PMCK (–35.6%). These results suggest that PMC and PMCK could be used as a replacement for CMAQ PM_{2.5} model estimates in an upgraded HBM-generated PMB.

We also found significant season effects in the Baltimore study, which were not found in the New York City study [16]. In this study only warm season ORs were greater than 1.000, while cold season ORs were below 1.000, and therefore protective *for those affected cases*. Warm season PMCK ORs at lag days of 01 were significantly higher than cold season PMCK ORs for ED asthma, IP MI, and IP HF. Some published studies have reported significant warm season effects [12,76,78], while other publications have reported significant cold season effects [74,75,77,79]. Kuo and colleagues [78] found an association between PM_{2.5} concentration level and asthma hospitalization in children in the fall season.

One interpretation of warm season effects, based on the positive results of this study and the non-significant outcome in the New York City study [16], is higher ambient PM_{2.5} concentration levels during the warm season compared to the cold season [8].

Surprisingly, there are only *two* publications on PM_{2.5} concentration levels and respiratory-cardiovascular hospitalizations in urban Baltimore [14,15], and *no* published papers in rural areas. This study demonstrated, for the first time, that PM_{2.5} concentration level does contribute to respiratory-cardiovascular hospital events, ED visits and inpatient stays, both in Baltimore City, with its higher population density and poverty, and in other study area grids that lacked ambient air monitors, and had lower population density and poverty. By analyzing grids without monitors, we were able to demonstrate that in these rural areas PMC and PMCK were associated with ED visits and IP hospitalizations of respiratory-cardiovascular chronic conditions. Others have also found associations between ambient PM_{2.5} concentration levels and respiratory-cardiovascular hospital events in rural areas [6,71], but due to coarse PM [86]. Strickland and associates [10] reported an association between PM_{2.5} concentration and ED asthma visits, but no change due to levels of urbanicity.

There are methodological limitations and strengths to the Baltimore study. First, these results were based on 2004–2006 ED visits and IP hospitalizations that, in 2020, are more than a decade old. Baltimore ambient PM_{2.5} levels were higher in 2004–2006 than in 2020. Replicating the Baltimore study with more current HBM-generated PMB and experimental AOD-PM_{2.5} concentration surfaces and ED/IP respiratory-cardiovascular chronic diseases would go a long way to confirm the generalizability of these exploratory results. A second limitation is the smaller Baltimore study area that only included 99 CMAQ 12 km² grids. A replication of this study should enlarge the CMAQ grid study area to contain the entire state of Maryland.

Methodological strengths include the inclusion of confounders in all conditional logistic regression analyses (apparent temperature, snow storms, major holidays, pollen apparent temperature), and the evaluation of effect modifiers (poverty, population density, season). A correspondence file was used to assign ZIP code and ZCTA polygons to a *single* CMAQ grid. This procedure was used to *minimize* differences in polygon shapes found between ZIP codes (United States Postal Service) and ZCTAs (United States Census Bureau), and to uniformly complete all analyses by only using CMAQ grids. Evaluation of conditional logistic regression runs in all three CMAQ grid conditions, all grids, and in grids with and without monitors was another methodological strength of this study.

Within this decade, AOD readings with a grid resolution of 10, 5, 2, 1 and <1 km² have become easier to obtain and use [27–29,31,33–36]. Kumar and colleagues evaluated NASA MODIS Level 1 and 2 in grid dimensions of 10, 5, and 2 km² [33,34]. They concluded that AOD in 2 km² grids provided greater resolution than AOD in either 10 km² or 5 km² grids [34]. The major advantages of AOD data in 2 km² grids are: (1) higher correlation with on-the-ground ambient PM_{2.5} monitors; and, (2) a decrease in lost data due to cloud interference. Another advantage is related to “scale” effects. The higher resolution found in

smaller grids permits more *precise* AOD PM_{2.5} measurements of ambient PM_{2.5} concentrations than in larger grids [87].

Another benefit of using AOD in <10 km² grids is the possibility of replacing ZIP-code-aggregated health data with address-level health data [23,30,88–91]. Patient privacy issues require obtaining the patient’s written consent before using a person’s ED visit or IP hospitalization record in an environmental health epidemiologic study. The dual benefits of having available AOD data in <10 km² grids with address-level data include the possibility of evaluating the contribution of ambient PM_{2.5} to ED visits and IP hospitalizations for respiratory-chronic diseases in both urban and rural areas, and utilizing remote sensing data to better understand shared pathophysiological mechanisms responsible for the occurrence of asthma, MI and HF events, and the development and testing of newer population-based intervention efforts.

5. Conclusions

If these Baltimore experimental AOD-PM_{2.5} concentration surface results are confirmed, then a shift in using AOD-PM_{2.5} concentrations readings in place of CMAQ PM_{2.5} model predictions in the PMB may be warranted, particularly in light of the greater temporal (every 5 min during daylight hours) and spatial (2 km²) data now available from geostationary satellites such as GOES-16 and GOES-17.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgments:

Fred Dimmick, EPA, facilitated inter-agency collaboration on this data linkage and analysis project and two prior U.S. Centers for Disease Control and Prevention (CDC)–EPA data linkage studies; Eric S. Hall, EPA, continued inter-agency collaboration between CDC–EPA on this data linkage and analysis project and co-developer of Hierarchical Bayesian Model (HBM) used in analyses; Michelle Morara, Battelle Memorial Institute, HBM update, from two to three input surfaces. Judy Qualters, CDC, endorsement and support of this data linkage and analysis project.

Funding:

This research was funded by a National Aeronautics and Space Administration Grant (NNH11CD19C), for Earth Science Applications Feasibility Studies: Public Health, awarded to the Battelle Memorial Institute, Columbus, Ohio.

Abbreviations

AIC	Akaike Information Criterion
AOD	Aerosol Optical Depth
AQS	Air Quality System
AT	Apparent Temperature
AT²	Product of AT, AT*AT

CDC	U.S. Centers for Disease Control and Prevention
CI	95% Confidence Interval
CMAQ	Community Multiscale Air Quality Model
ED	Emergency Department
EPA	U.S. Environmental Protection Agency
EPHT	Environmental Public Health Tracking
FRM	Federal Reference Method
GIS	Geographic Information System
HBM	Hierarchical Bayesian Model
HF	Heart Failure
HSCRC	Maryland Health Services Cost Review Commission
ICD-9-CM	International Classification of Diseases, Ninth Revision, Clinical Modification
IP	Inpatient Hospitalization
MDH	Maryland Department of Health
MDP	Maryland Department of Planning
MI	Myocardial Infarction
MODIS	MODerate resolution Imaging Spectroradiometer
LAADS	Level-1 and Atmosphere Archive and Distribution System
NASA	National Aeronautics and Space Administration
NCEI	National Centers for Environmental Information
NOAA	National Oceanic and Atmospheric Administration
OPM	U.S. Office of Personnel Management
OR	Odds Ratio
PHASE	Public Health Air Surveillance Evaluation
PHREG	Proportional Hazards Regression
PM_{2.5}	Fine Particulate Matter
PMB	PM _{2.5} Baseline Model (monitor PM _{2.5} and CMAQ PM _{2.5})
PMC	AOD PM _{2.5} Model (monitor PM _{2.5} and AOD PM _{2.5})

PMCK	AOD PM _{2.5} Kriged Model (monitor PM _{2.5} and AOD PM _{2.5} Kriged)
PMCKQ	AOD PM _{2.5} Kriged and CMAQ PM _{2.5} Model (monitor PM _{2.5} and AOD PM _{2.5} Kriged and CMAQ PM _{2.5})
PMCQ	AOD PM _{2.5} and CMAQ PM _{2.5} Model (monitor PM _{2.5} and AOD PM _{2.5} and CMAQ PM _{2.5})
SAS	Statistical Analysis System
USCB	U.S. Census Bureau/Department of Commerce
ZCTA	ZIP Code Tabulation Area (US Census)
ZIP Code	Zone Improvement Plan (US Postal Service)

References

1. Anderson JO; Thundiyil JG; Stolbach A Clearing the air: A review of the effects of particulate matter air pollution on human health. *J. Med. Toxicol* 2012, 8, 166–175. [PubMed: 22194192]
2. Du Y; Xu X; Chu M; Guo Y; Wang J Air particulate matter and cardiovascular disease: The epidemiological, biomedical, and clinical evidence. *J. Thorac. Dis* 2016, 8, E8–E19. [PubMed: 26904258]
3. Egondi T; Ettarh R; Kyobutungi C; Ng N; Rocklöv J Exposure to outdoor particles (PM_{2.5}) and associated child morbidity and mortality in socially deprived neighborhoods of Nairobi, Kenya. *Atmosphere* 2018, 9, 351.
4. Garcia E; Berhane KT; Islam T; McConnel R; Urman R; Chen Z; Gilliland FD Association of changes in air quality with incident asthma in children in California, 1993–2014. *JAMA* 2019, 321, 1906–1915. [PubMed: 31112259]
5. Kirrane E; Svendsgaard D; Ross M; Buckley B; Davis A; Johns D; Kotchmar D; Long TC; Luben TJ; Smith G; et al. A comparison of risk estimates for the effect of short-term exposure to PM, NO₂ and CO on cardiovascular hospitalizations and emergency department visits: Effect size modeling of study findings. *Atmosphere* 2011, 2, 688–701.
6. Kloog I; Nordio F; Zanobetti A; Coull BA; Koutrakis P; Schwartz JD Short term effects of particle exposure on hospital admissions in the Mid-Atlantic states: A population estimate. *PLoS ONE* 2014, 9, e88578. [PubMed: 24516670]
7. Liu HY; Dunea D; Iordache S; Pohoata A A review of airborne matter effects on young children's respiratory systems and diseases. *Atmosphere* 2018, 9, 150.
8. Miller L; Xu X Ambient PM_{2.5} human health effects—Findings in China and research directions. *Atmosphere* 2018, 9, 424.
9. Peters A; Dockery DW; Muller JE; Mittleman MA Increased particulate air pollution and the triggering of myocardial infarction. *Circulation* 2001, 103, 2810–2815. [PubMed: 11401937]
10. Strickland MJ; Hao H; Hu X; Chang HH; Darrow LA; Liu Y Pediatric emergency visits and short-term changes in PM_{2.5} concentrations in the U.S. State of Georgia. *Environ. Health Perspect* 2016, 124, 690–696. [PubMed: 26452298]
11. Tétreault LF; Doucet M; Gamache P; Fournier M; Brand A; Kosatsky T; Smargiassi A Childhood exposure to the ambient air pollutants and the onset of asthma: An administrative cohort study in Québec. *Environ. Health Perspect* 2016, 124, 1276–1282. [PubMed: 26731790]
12. Wu Y; Li M; Tian Y; Cao Y; Song J; Huang Z; Wang X; Hu Y Short-term effects of ambient fine particulate air pollution on inpatient visits for myocardial infarction in Beijing, China. *Environ. Sci. Pollut. Res. Int* 2019, 26, 14178–14183. [PubMed: 30859442]
13. Haley VB; Talbot TO; Felton HD Surveillance of the short-term impact of fine particle air pollution on cardiovascular disease hospitalizations in New York State. *Environ. Health* 2009, 8, 1–42. [PubMed: 19138417]

14. Hirshon JM; Shardell M; Alles S; Powell JL; Squibb K; Ondov J; Blaisdell CJ Elevated ambient air zinc increases pediatric asthma morbidity. *Environ. Health Perspect* 2008, 116, 826–831. [PubMed: 18560541]
15. Symons JM; Wang L; Guallar E; Howell E; Dominici F; Schwab M; Ange BA; Samet J; Ondov J; Harrison D; et al. A case-crossover study of fine particulate matter air pollution and onset of congestive heart failure symptom exacerbation leading to hospitalization. *Am. J. Epidemiol* 2006, 164, 421–433. [PubMed: 16793862]
16. Weber SA; Insaf TZ; Hall ES; Talbot TO; Huff AK 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. *Environ. Res* 2016, 151, 399–409. [PubMed: 27543787]
17. EPA (U.S. Environmental Protection Agency). Air Quality System (AQS). Available online: <https://www.epa.gov/aqs> (accessed on 10 November 2019).
18. Vaidyanathan A; Dimmick WF; Kegler SR; Qualters JR Statistical air quality predictions for public health surveillance: Evaluation and generation of county level metrics of PM_{2.5} for the environmental public health tracking network. *Int. J. Health Geogr* 2013, 12, 1–13. [PubMed: 23305074]
19. Goldman GT; Mulholland JA; Russell AG; Srivastava A; Strickland MJ; Klein M; Waller LA; Tolbert PE; Edgerton ES Ambient air pollutant measurement error: Characterization and impacts in a time-series epidemiologic study in Atlanta. *Environ. Sci. Technol* 2010, 44, 7692–7698. [PubMed: 20831211]
20. Boothe V; Dimmick F; Haley V; Paulu C; Bekkedal M; Holland D; Talbot T; Smith A; Warner M; Baldrige E; et al. A review of public health air surveillance evaluation project. *Epidemiology* 2006, 17, S450–S451.
21. Boothe V; Dimmick WF; Talbot TO Relating air quality and environmental public health tracking data. *WIT Trans. Ecol. Environ* 2005, 85, 43–52.
22. Talbot TO; Haley VB; Dimmick WF; Paulu C; Talbot EO; Rager J Developing consistent data and methods to measure the public health impacts of ambient air quality for Environmental Public Health Tracking: Progress to date and future directions. *Air Q. Atmos. Health* 2009, 2, 199–206.
23. Appel KW; Napelenok SL; Foley KM; Pye HOT; Hogrefe C; Luecken DJ; Bash JO; Roselle SJ; Pleim JE; Foroutan H; et al. Description and evaluation of the Community Multiscale Air Quality (CMAQ) modeling system version 5.1. *GeoSci. Model Dev* 2017, 10, 1703–1732. [PubMed: 30147852]
24. Foley KM; Roselle SJ; Appel KW; Bhawe PV; Pleim JE; Otte TL; Mathur R; Sarwar G; Young JO; Gilliam RC; et al. Incremental testing of the Community Multiscale Air Quality (CMAQ) modeling system version 4.7. *GeoSci. Model Dev* 2010, 3, 205–226.
25. EPA. Community Modeling and Analysis System (CMAS), CMAQ. Available online: <https://www.cmascenter.org/> (accessed on 10 November 2019).
26. Kearney GD; Namulanda G; Qualters JR; Talbot EO A decade of environmental public health tracking (2002–2012): Progress and challenges. *J. Public Health Manag. Pract* 2015, 2, S23–S35.
27. Chu Y; Liu Y; Li X; Liu Z; Lu H; Lu Y; Mao Z; Chen X; Li N; Ren M; et al. A Review on predicting ground PM_{2.5} concentration using satellite aerosol optical depth. *Atmosphere* 2016, 7, 129.
28. Lee HJ; Coull BA; Bell ML; Koutrakis P Use of satellite-based aerosol optical depth and spatial clustering to predict ambient PM_{2.5} concentrations. *Environ. Res* 2012, 118, 8–15. [PubMed: 22841416]
29. Liu Y; Sarnat JA; Kilaru V; Jacob DJ; Koutrakis P Estimating ground-level PM_{2.5} in the eastern United States using satellite remote sensing. *Environ. Sci. Technol* 2005, 39, 269–3278.
30. McGuinn LA; Ward-Caviness CK; Neas LM; Schneider A; Diaz-Sanchez D; Cascio WE; Kraus WE; Hauser E; Dowdy E; Haynes C; et al. Association between satellite-based estimates of long-term PM_{2.5} exposure and coronary artery disease. *Environ. Res* 2016, 145, 9–17. [PubMed: 26613345]

31. Wang Z; Liu Y; Hu M; Pan X; Shi J; Chen F; He K; Koutrakis P; Christiani DC Acute health impacts of airborne particles estimated from satellite remote sensing. *Environ. Int* 2013, 51, 150–159. [PubMed: 23220016]
32. Huang J; Kondragunta S; Laszlo I; Liu H; Remer LA; Zhang H; Superczynski S; Ciren P; Holben BN; Petrenko M Validation and expected error estimation of Suomi-NPP VIIRS aerosol optical thickness and Ångström exponent with AERONET. *J. Geophys. Res. Atmos* 2016, 121, 7139–7160.
33. Kumar N; Chu AD; Foster AD; Peters T; Willis R Satellite remote sensing for developing time and space resolved estimates of ambient particulate in Cleveland, OH. *Aerosol. Sci. Technol* 2011, 45, 1090–1108. [PubMed: 22238503]
34. Kumar N; Liang D; Comellas A; Chu AD; Abrams T Satellite-based PM concentrations and their application to COPD in Cleveland, OH. *J. Expo. Sci. Environ. Epidemiol* 2013, 23, 637–646. [PubMed: 24045428]
35. Wang B; Chen Z High-resolution satellite-based analysis of ground-level PM_{2.5} for the City of Montreal. *Sci. Total Environ* 2016, 541, 1059–1069. [PubMed: 26473708]
36. Xie Y; Wang Y; Bilal M; Dong W Mapping daily PM_{2.5} at 500 m resolution over Beijing with improved hazy day performance. *Sci. Total Environ* 2019, 659, 410–418. [PubMed: 31096372]
37. Zhang H; Kondragunta S; Laszlo I; Liu H; Remer LA; Huang J; Superczynski S; Ciren P An enhanced VIIRS aerosol optical thickness (AOT) retrieval algorithm over land using a global surface reflectance ratio database. *J. Geophys. Res. Atmos* 2016, 121, 10717–10738.
38. Duncan BN; Prados AI; Lamsal LN; Liu Y; Streets DG; Gupta P; Hilsenrath E; Kahn RA; Nielsen JE; Beyersdorf AJ; et al. Satellite data of atmospheric pollution for U.S. air quality applications: Examples of applications, summary of data end-user resources, and answers to FAQs, and common mistakes to avoid. *Atmos. Environ* 2014, 94, 647–662.
39. Hoff RM; Christopher SA Remote sensing of particulate pollution from space: Have we reached the promised land? *J. Air Waste Manag. Assoc* 2009, 59, 645–675. [PubMed: 19603734]
40. Hidy GM; Brook JR; Chow JC; Green M; Husar RB; Lee C; Scheffe RD; Swanson A; Watson JG Remote sensing of particulate pollution from space: Have we reached the promised land? *J. Air Waste Manag. Assoc* 2009, 59, 1130–1139. [PubMed: 19842321]
41. Engel-Cox JA; Holloman CH; Coutant BW; Hoff RM Qualitative and quantitative evaluation of MODIS. Satellite sensor data for regional and urban scale quality. *Atmos. Environ* 2004, 38, 2495–2509.
42. Lee SJ; Serre ML; van Donkelaar A; Martin RV; Burnett RT; Jerrett M Comparison of geostatistical interpolation and remote sensing techniques for estimating long-term exposure to ambient PM_{2.5} concentrations across the continental United States. *Environ. Health Perspect* 2012, 120, 1727–1732. [PubMed: 23033456]
43. Van Donkelaar A; Martin RV; Brauer M; Boys BL Use of satellite observations for long-term exposure assessment of global concentrations of fine particulate matter. *Environ. Health Perspect* 2015, 123, 135–143. [PubMed: 25343779]
44. Zhang H; Hoff RM; Engel-Cox JA The relation between Moderate Resolution Imaging Spectroradiometer (MODIS) aerosol optical depth and PM_{2.5} over the United States: A geographical comparison by U.S. Environmental Protection Agency Regions. *J. Air Waste Manag. Assoc* 2009, 59, 1358–1369. [PubMed: 19947117]
45. NASA (National Aeronautics and Space Administration). Level-1 and Atmosphere Archive and Distribution System (LAADS). Available online: <https://ladsweb.modaps.eosdis.nasa.gov> (accessed on 10 November 2019).
46. McMillan NJ; Holland DM; Morara M; Feng J Combining numerical model output and particulate data using Bayesian space-time modeling. *Environmetrics* 2009, 21, 48–65.
47. Weber SA; Engel-Cox JA; Hoff RM; Prados AI; Zhang H An improved method for estimating surface fine particle concentrations using seasonally adjusted satellite aerosol optical depth. *J. Air Waste Manag. Assoc* 2010, 60, 574–585. [PubMed: 20480857]
48. Battelle Memorial Institute. User's Guide: Temporal-Spatial Ambient Concentration Estimator (T-SpACE), Version 5_4h.0.1.21; Battelle Memorial Institute: Columbus, OH, USA, 2011.

49. Hall ES Temporal-Spatial Ambient Concentrator Estimator (T-SpACE): Hierarchical Bayesian Model Software Used to Estimate Ambient Concentrations of NAAQS Air Pollutants in Support of Health Studies; EPA/600/R-18/01; U.S. Environmental Protection Agency: Washington, DC, USA, 2018. Available online: https://cfpub.epa.gov/si/si_public_record_report.cfm?Lab=NERL&dirEntryId=339714 (accessed on 10 November 2019).
50. HSCRC (Maryland Health Services Cost Review Commission). Available online: <https://hscrc.maryland.gov> (accessed on 10 November 2019).
51. MDP (Maryland Department of Planning). Maryland State Data Center. Zip Code Boundary Area Files, 2004 and 2006. Available online: http://planning.maryland.gov/MSDC/Pages/s5_map_gis.aspx (accessed on 10 November 2019).
52. CDC (U.S. Centers for Disease Control and Prevention). International Classification of Diseases, Ninth Revision, Clinical Modification (ICD-9-CM). Available online: <https://www.cdc.gov/nchs/icd/icd9cm.htm> (accessed on 10 November 2019).
53. Carracedo-Martinez E; Taracido M; Tobias A; Saez M; Figueiras A Case-crossover analysis of air pollution health effects: A systematic review of methodology and application. *Environ. Health Perspect* 2010, 118, 1173–1182. [PubMed: 20356818]
54. Jaakkola JJK Case-crossover design in air pollution epidemiology. *Eur. Respir. J* 2003, 40, 81s–85s.
55. Janes H; Sheppard L; Lumley T Case-crossover analyses of air pollution exposure data: Referent selection strategies and their implications for bias. *Epidemiology* 2005, 16, 717–726. [PubMed: 16222160]
56. Maclure M The case-crossover design: A method for studying transient effects on the risk of acute events. *Am. J. Epidemiol* 1991, 133, 144–153. [PubMed: 1985444]
57. Stokes ME; Davis CS; Koch GG *Categorical Data Analysis Using the SAS System*; SAS Institute, Inc.: Cary, NC, USA, 1995; ISBN 978-1-60764-664-8.
58. Bai Y; Sun Q Fine particulate matter air pollution and atherosclerosis: Mechanistic insights. *Biochim. Biophys. Acta* 2016, 1860, 2863–2868. [PubMed: 27156486]
59. Brook RD; Kousha T Air pollution and emergency department visits for hypertension in Edmonton and Calgary, Canada: A case-crossover study. *Am. J. Hypertens* 2015, 28, 1121–1126. [PubMed: 25663064]
60. Chen H; Burnett RT; Kwong JC; Villeneuve PJ; Goldberg MS; Brook RD; van Donkelaar A; Jerrett M; Martin RV; Brook JR; et al. Risk of incident diabetes in relation to long-term exposure to fine particulate matter in Ontario, Canada. *Environ. Health Perspect* 2013, 121, 804–810. [PubMed: 23632126]
61. Dubowsky SD; Suh H; Schwartz J; Coull BA; Gold DR Diabetes, obesity, and hypertension may enhance associations between air pollution and markers of systemic inflammation. *Environ. Health Perspect* 2006, 114, 992–998. [PubMed: 16835049]
62. Hoshino T; Hoshino A; Nishino J Assessment of associations between ischaemic attacks in patients with type 2 diabetes mellitus and air concentrations of particulate matter <2.5 µm. *J. Int. Med. Res* 2016, 44, 639–655. [PubMed: 27020595]
63. NOAA (National Oceanic and Atmospheric Administration). The New Improved “Wind Chill Index. Available online: <https://www.weather.gov/ffc/wci> (accessed on 10 November 2019).
64. Rothfus LP The Heat Index “Equation,” SR-90-23, 7/1/90. Available online: https://www.weather.gov/media/ffc/ta_htindx.PDF (accessed on 10 November 2019).
65. DellaValle CT; Triche EW; Leaderer BP; Bell ML Effects of ambient pollen concentrations on frequency and severity of asthma symptoms among asthmatic children. *Epidemiology* 2012, 23, 55–63. [PubMed: 22082997]
66. Weichenthal S; Lavigne E; Villeneuve PJ; Reeves F Airborne pollen concentrations and emergency room visits for myocardial infarction: A multicity case-crossover study in Ontario, Canada. *Am. J. Epidemiol* 2016, 183, 613–621. [PubMed: 26934896]
67. Wu HC; Sapkota A; Braggio JT Pollen Effects on Asthma, Allergic Rhinitis, and Finger Wound Emergency Department Visits between 2000–2010 in Baltimore, Maryland. In *Proceedings of the Council of State and Territorial Epidemiologists Annual Conference, Nashville, TN, USA, 25 June*

- 2014; Available online: <https://cste.confex.com/cste/2014/webprogram/Paper3178.html> (accessed on 10 November 2019).
68. Braggio JT; Brunner W; Lutzker L; Mangan A; Simms EF; Tomasallo C; Wahl RL; Yip F Analysis of 2009 pollen readings. In Proceedings of the Council of State and Territorial Epidemiologists Annual Conference, Omaha, NE, USA, 4–7 June 2012.
 69. OPM (U.S. Office of Personnel Management). Federal Holidays, 2004–2006. Available online: https://archive.opm.gov/Operating_Status_Schedules/fedhol/2004.asp (accessed on 10 November 2019).
 70. NCEI (National Centers for Environmental Information). Climate Data Online. Available online: <https://www.ncdc.noaa.gov/cdo-web/> (accessed on 10 November 2019).
 71. Brochu PJ; Yanosky JD; Pacioreck CJ; Schwartz J; Chen JT; Herrick RF; Suh HH Particulate air pollution and socioeconomic position in rural and urban areas of the northeastern United States. *Am. J. Public Health* 2011, 101, S224–S230. [PubMed: 21836114]
 72. Bell ML; Ebisu K Environmental inequality in exposures to airborne particulate matter components in the United States. *Environ. Health Perspect* 2012, 120, 1699–1704. [PubMed: 22889745]
 73. USCB (U.S. Census Bureau/Department of Commerce). 2000 Census of People and Housing, Summary File 3. Available online: <https://www.census.gov/census2000/sumfile3.html> (accessed on 10 November 2019).
 74. Bell ML; Ebisu K; Peng RD; Walker J; Samet JM; Zeger SL; Dominici F Seasonal and regional short-term effects of fine particles on hospital admissions in 202 US counties, 1999–2005. *Am. J. Epidemiol* 2008, 168, 1301–1310. [PubMed: 18854492]
 75. Chen K; Glonek G; Hansen A; Williams S; Tuke J; Salter A; Bi P The effects of air pollution on asthma hospital admissions in Adelaide, South Australia, 2003–2013: Time-series and case-crossover analyses. *Clin. Exp. Allergy* 2016, 46, 1416–1430. [PubMed: 27513706]
 76. Goldberg MS; Burnett RT; Stieb DM; Brophy JM; Daskalopoulou SS; Valois MF; Brook JR Associations between ambient air pollution and daily mortality among elderly persons in Montreal, Quebec. *Sci. Total Environ* 2013, 463–464, 931–942.
 77. Hsu WH; Hwang SA; Kinney PL; Lin S Seasonal and temperature modifications of the association between fine particulate air pollution and cardiovascular hospitalization in New York state. *Sci. Total Environ* 2016, 578, 626–632. [PubMed: 27863872]
 78. Kuo CY; Pan RH; Chan CK; Wu CY; Phan DV; Chan CL Application of a time-stratified case-crossover design to explore the effects of air pollution and season on childhood asthma hospitalization in cities of differing urban patterns: Big data analytics of government open data. *Int. J. Environ. Res. Public Health* 2018, 15, 647.
 79. Rodopoulou S; Samoli E; Chalbot MCG; Kavouras IG Air pollution and cardiovascular and respiratory emergency visits in Central Arkansas: A time series analysis. *Sci. Total Environ* 2015, 536, 872–879. [PubMed: 26232212]
 80. ESRI (Environmental Systems Research Institute). ArcGIS Desktop (ArcMap), Release 10.6.1; Environmental Systems Research Institute: Redlands, CA, USA, 7 2018.
 81. SAS (Statistical Analysis System). Base SAS; SAS Institute Inc.: Cary, NC, USA, 2018.
 82. SAS (Statistical Analysis System). SAS/STAT 14.1 User’s Guide: High-Performance Procedures; SAS Institute Inc.: Cary, NC, USA, 2015.
 83. Wang SV; Coull BA; Schwartz J; Mittleman MA; Wellenius GA Potential for bias in case-crossover studies with shared exposures analyzed using SAS. *Am. J. Epidemiol* 2011, 174, 18–124.
 84. Hosmer DW Jr.; Lemeshow S; Sturdivant RX Applied Logistic Regression, 3rd ed.; John Wiley & Sons: Hoboken, NJ, USA, 2013; ISBN 978-0-470-58247.
 85. Agresti A Categorical Data Analysis, 2nd ed.; John Wiley: Sons Hoboken, NJ, USA, 2002; ISBN 978-0-470-08289-8.
 86. Brook RD; Bard RL; Morishita M; Dvonch T; Wang L; Yang HY; Spino C; Mukherjee B; Kaplan MJ; Yalavarthi S Hemodynamic, autonomic, and vascular effects of exposure to coarse particulate matter air pollution from a rural location. *Environ. Health Perspect* 2014, 122, 624–630. [PubMed: 24618231]

87. Chudnovsky AA; Kostinski A; Lyapustin A; Koutrakis P Spatial scales of pollution from variable resolution satellite imaging. *Environ. Pollut* 2013, 172, 131–138. [PubMed: 23026774]
88. Gehring U; Wijga AH; Hoek G; Bellander T; Berdel D; Brüske I; Fuertes E; Gruzieva O; Heinrich J; Hoffmann B Exposure to air pollution and development of Asthma and rhinoconjunctivitis throughout childhood and adolescence: A population-based birth cohort study. *Lancet Respir. Med* 2015, 3, 933–942. [PubMed: 27057569]
89. Hart JE; Puett RC; Rexrode KM; Albert CM; Laden F Effect modification of long-term air pollution exposures and the risk of incident cardiovascular disease in US women. *J. Am. Heart Assoc* 2015, 4, e002301. [PubMed: 26607712]
90. Lim CC; Hayes RB; Ahn J; Shao Y; Silverman DT; Jones RR; Garcia C; Thurston GD Association between long-term exposure to ambient air pollution and diabetes mortality in the US. *Environ. Res* 2018, 165, 330–336. [PubMed: 29778967]
91. Yang WY; Zhang ZY; Thijs L; Bijlens EM; Janssen BG; Vanpoucke C; Lefebvre W; Cauwenberghs N; Wei FF; Luttun A; et al. Left ventricular function in relation to chronic residential air pollution in a general population. *Eur. J. Prev. Cardiol* 2017, 24, 1416–1428. [PubMed: 28617090]

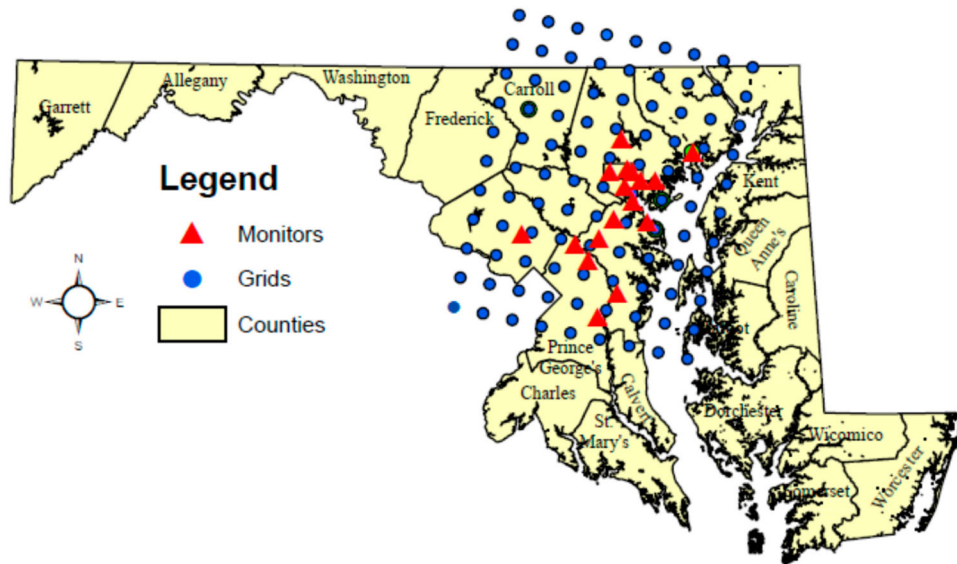


Figure 1.

Map shows Maryland's Counties and Baltimore City in the study area. The extent of the study area is defined by the 1–11 (south–north row) by 1–9 (west–east column) Community Multiscale Air Quality (CMAQ) 12 km² grids (blue circles). The 17 Federal Reference Method (FRM) PM_{2.5} ambient air monitors are shown as red triangles. Baltimore City and Maryland Counties within the CMAQ grid boundaries provided the 2004–2006 respiratory-cardiovascular chronic disease hospital events included in this data analysis study.

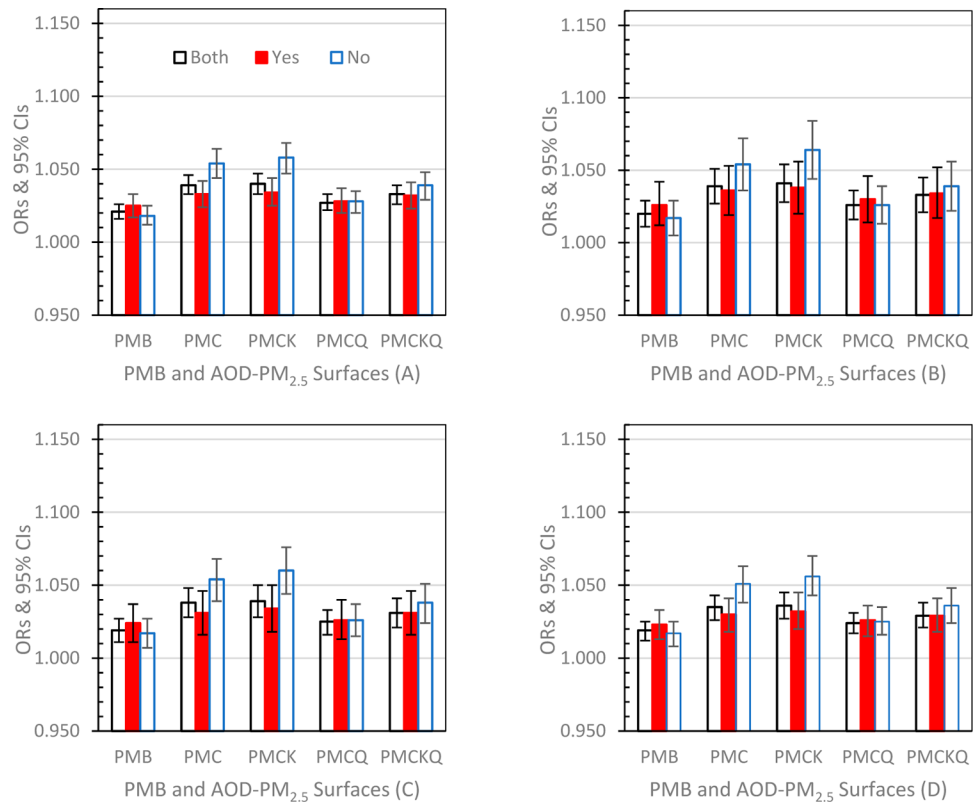


Figure 2. Odds Ratios (ORs) and 95% Confidence Intervals (CIs) for the four experimental aerosol optical depth (AOD)-particulate matter (PM)_{2.5} concentration surfaces and particulate matter baseline (PMB) under both grid conditions (Both), grids with monitors (Yes) and grids without monitors (No) at lag day 0: (A) ED asthma (top left panel), (B) IP asthma (top right panel), (C) IP MI (bottom left panel), and (D) IP HF (bottom right panel).

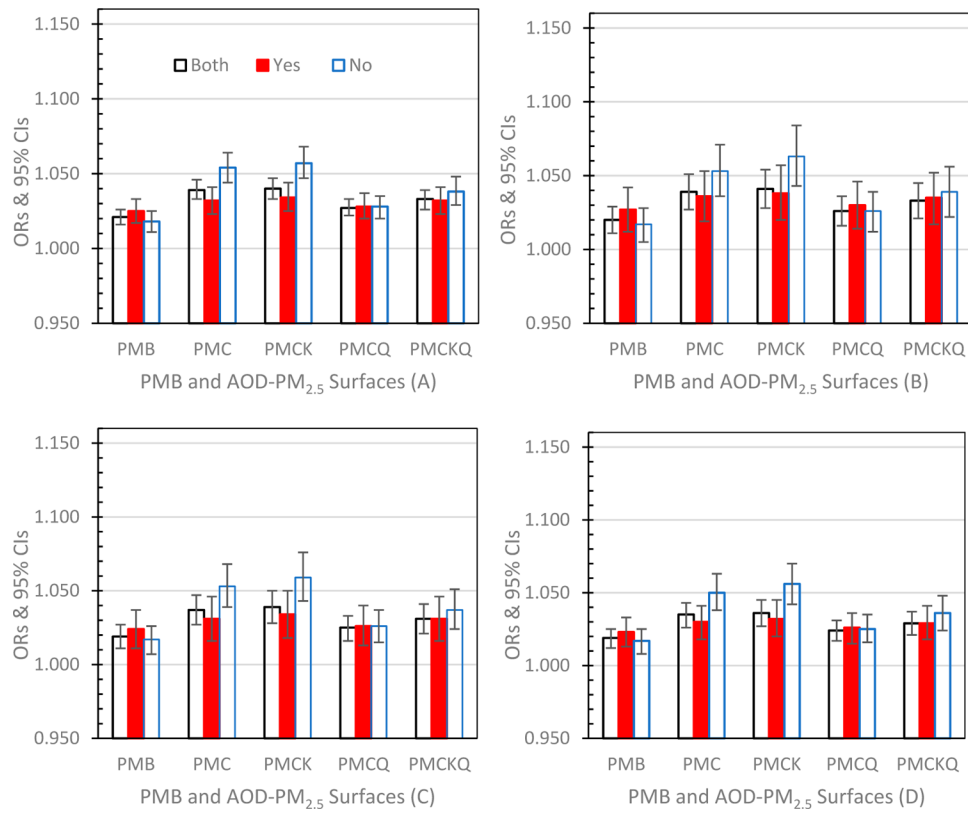


Figure 3. Odds Ratios (ORs) and 95% Confidence Intervals (CIs) for the four experimental AOD-PM_{2.5} concentration surfaces and PMB under both grid conditions (Both), grids with monitors (Yes) and grids without monitors (No) at lag day 1: (A) ED asthma (top left panel), (B) IP asthma (top right panel), (C) IP MI (bottom left panel) and (D) IP HF (bottom right panel).

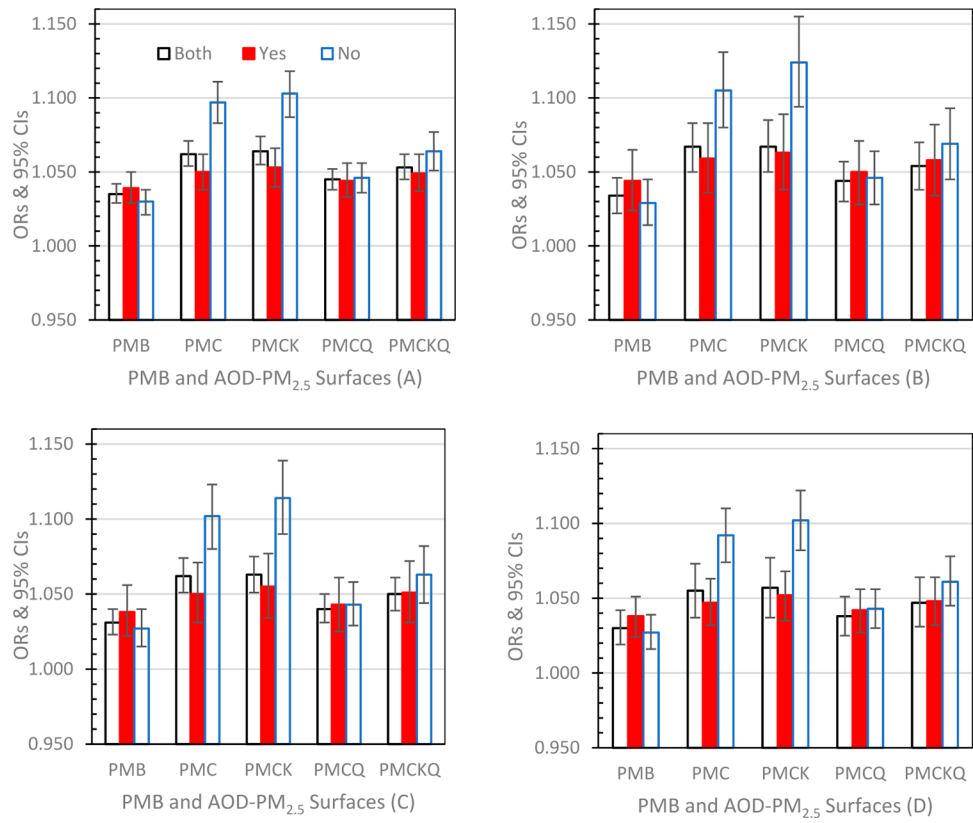


Figure 4. Odds Ratios (ORs) and 95% Confidence Intervals (CIs) for the four experimental AOD-PM_{2.5} concentration surfaces and PMB under both grid conditions (Both), grids with monitors (Yes) and grids without monitors (No) at lag days 01: (A) ED asthma (top left panel), (B) IP asthma (top right panel), (C) IP MI (bottom left panel), and (D) IP HF (bottom right panel).

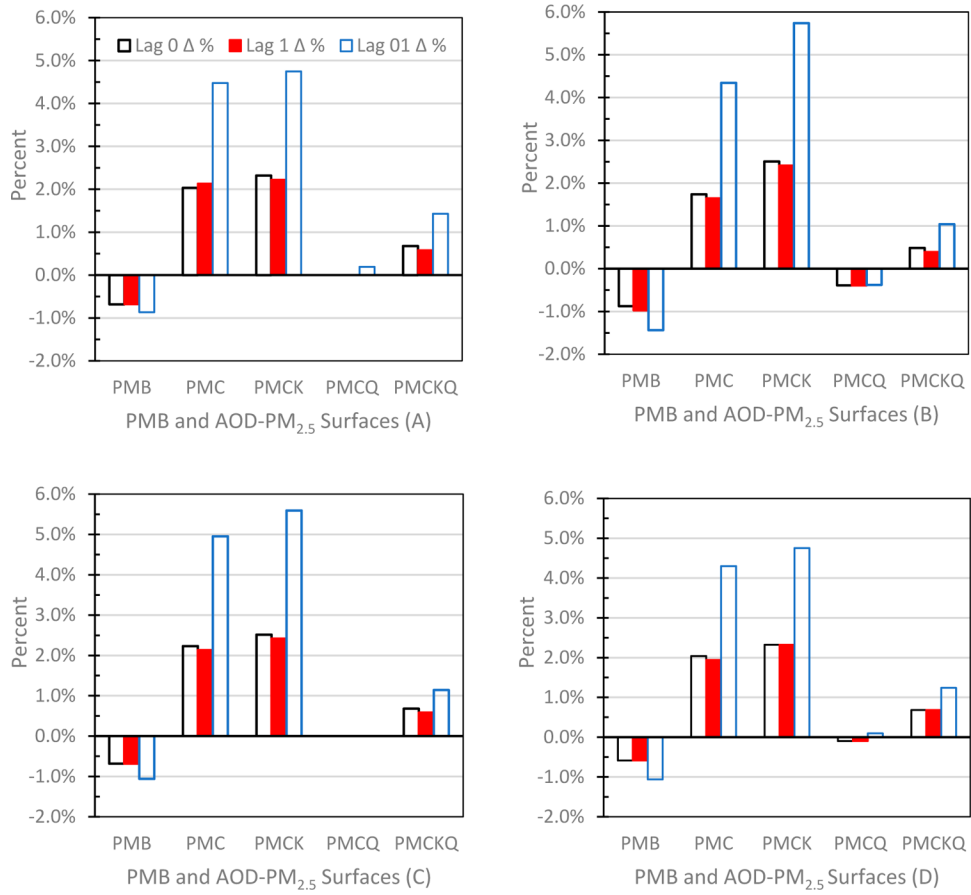


Figure 5. Percent change between no monitor and monitor Odds Ratios (ORs) for the four experimental AOD-PM_{2.5} concentration surfaces and PMB at lag days of 0, 1 and 01: **(A)** ED asthma (top left panel), **(B)** IP asthma (top right panel), **(C)** IP MI (bottom left panel), and **(D)** IP HF (bottom right panel).

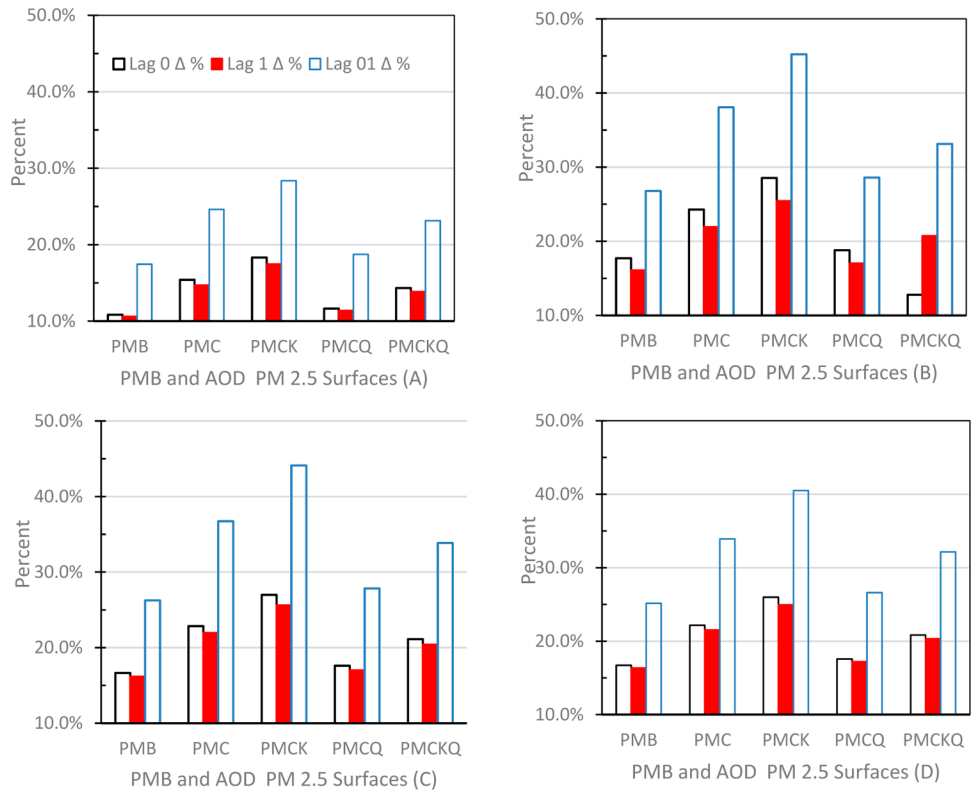


Figure 6. Percent change between warm and cold season Odds Ratios (ORs) for PMB and the four experimental aerosol optical depth (AOD)-PM_{2.5} concentration surfaces at lag days of 0, 1 and 01: **(A)** ED asthma (top left panel), **(B)** IP asthma (top right panel), **(C)** IP MI (bottom left panel) and **(D)** IP HF (bottom right panel).

Table 1.

Demographics for ED Asthma and IP Asthma Cases and Controls in the Baltimore Study.

Variables ¹⁻²	ED Asthma Cases	ED Asthma Controls	IP Asthma Cases	IP Asthma Controls
Total	11,723 (100)	35,533 (100)	3376 (100)	10,139 (100)
Age Category ³⁻⁴				
0–14 years	5131 (43.8) †	15,492 (43.6) †	930 (27.6)	2791 (27.5)
15–34 years	3223 (27.5)	9765 (27.5)	358 (10.6)	1080 (10.6)
35 years	3369 (28.7)	10,276 (28.9)	2088 (61.8)	6268 (61.8)
Gender—Female	6093 (52.0)	18,489 (52.0)	2125 (62.9)	6388 (63.0)
Male	5628 (48.0)	17,041 (48.0)	1251 (37.1)	3751 (37.0)
Race—Black	5618 (48.1)	17,078 (48.3)	1130 (33.5)	3380 (33.4)
Other	749 (6.4)	2311(6.5)	164 (4.9)	505 (5.0)
White	5305 (45.4)	15,989 (45.2)	2076 (61.6)	6236 (61.6)
Atherosclerosis—No	11675 (99.6)	35,387 (99.6)	3055 (90.5)	9176 (90.5)
Yes	48 (0.4)	146 (0.4)	321 (9.5)	963 (9.5)
Diabetes—No	11458 (97.7)	34,730 (97.7)	2842 (84.2)	8557 (84.4)
Yes	265 (2.3)	803 (2.3)	534 (15.8)	1582 (15.6)
Hypertension—No	11,111 (94.8)	33,650 (94.7)	2316 (68.6)	6950 (68.6)
Yes	612 (5.2)	1883 (5.3)	1060 (31.4)	3189 (31.4)
Insurance—No	2099 (17.9)	6409 (18.1)	207 (6.1)	623 (6.2)
Yes	9606 (82.1)	29,070 (81.9)	3164 (93.9)	9501 (93.8)
Poverty ⁵	9.6 (9.5–9.7)	9.6 (9.5–9.7)	9.4 (9.2–9.6)	9.4 (9.2–9.5)
Monitor—No	6.3 (6.3–6.4) *	6.4 (6.3–6.4) *	6.3 (6.2–6.5) *	6.3 (6.3–6.4) *
Monitor—Yes	13.7 (13.5–3.9)	13.7 (13.6–13.8)	13.6 (13.3–13.9)	13.5 (13.4–13.7)
Population (Log ₁₀) ⁶	3.3 (3.3–3.3)	3.3 (3.3–3.3)	3.2 (3.2–3.2)	3.2 (3.2–3.2)
Monitor—No	3.1 (3.1–3.1) *	3.1 (3.1–3.1) *	2.9 (2.9–2.9) *	2.9 (2.9–2.9) *
Monitor—Yes	3.6 (3.6–3.6)	3.6 (3.6–3.6)	3.5 (3.5–3.6)	3.5 (3.5–3.6)

¹ Each column displays total observations (n) and percent (%) for emergency department (ED)/inpatient (IP) asthma case-control groups.² Significance was evaluated with the Chi Square test:* = $p < 0.05$;† = $p < 0.01$.³ Significant age group differences between ED asthma cases and IP asthma cases, $p < 0.01$.⁴ Significant age group differences between ED asthma controls and IP asthma controls, $p < 0.01$.⁵ Significant differences between no monitor versus monitor within poverty, $p < 0.05$.⁶ Significant differences between no monitor versus monitor within population density (Population, L₁₀), $p < 0.05$.

Table 2.

Demographics for IP Myocardial Infarction (MI) and IP Heart Failure (HF) Cases and Controls in the Baltimore study.

Variables ¹⁻²	IP MI Cases	IP MI Controls	IP HF Cases	IP HF Controls
Total	4745 (100)	14276 (100)	6919 (100)	20,427 (100)
Age Category ³⁻⁴ 35–59 years	1477 (31.1) †	4462 (31.3) †	1279 (18.5) *	3921 (19.2)
60–75 years	1638 (34.5)	4907 (34.4)	2531 (36.6)	7119 (34.8)
76 years	1630 (34.4)	4907 (34.4)	3109 (44.9)	9387 (46.0)
Gender—Female	2041 (42.6)	6155 (42.7)	3559 (52.1)	10,808 (52.2)
Male	2749 (57.4)	8256 (57.3)	3267 (47.9)	9884 (47.8)
Race—Black	633 (13.2)	1913 (13.3)	1737 (25.5)	5292 (25.6)
Other	214 (4.5)	634 (4.4)	191 (2.8)	602 (2.9)
White	3937 (82.3)	11,843 (82.3)	4892 (71.7)	14,780 (71.5)
Atherosclerosis—No	1716 (35.8)	5163 (35.8)	3554 (52.1)	10,777 (52.1)
Yes	3074 (64.2)	9248 (64.2)	3272 (47.9)	9915 (47.9)
Diabetes—No	3363 (70.2)	10,101 (70.1)	3950 (57.9)	11,948 (57.7)
Yes	1427 (29.8)	4310 (29.9)	2876 (42.1)	8744 (42.3)
Hypertension—No	2511 (52.4)	7499 (52.0)	4022 (58.9)	12197 (59.0)
Yes	2279 (47.6)	6912 (48.0)	2804 (41.1)	8495 (41.1)
Insurance—No	149 (3.1)	443 (3.1)	111 (1.6)	324 (1.6)
Yes	4637 (96.9)	13,956 (96.9)	6710 (98.4)	20,356 (98.4)
Poverty ⁵	8.3(8.2–8.4)	8.4 (8.3–8.4)	9.1 (9.0–9.2)	9.2 (9.1–9.2)
Monitor—No	6.0 (5.9–6.1) *	6.0 (6.0–6.1) *	6.4 (6.3–6.5) *	6.4 (6.3–6.4) *
Monitor—Yes	12.2 (11.9–12.4)	12.3 (12.2–12.4)	2.8 (12.6–13.0)	12.8 (12.7–12.9)
Population (Log ₁₀) ⁶	3.1 (3.1–3.1)	3.1 (3.1–3.1)	3.2 (3.2–3.2)	3.2 (3.2–3.2)
Monitor—No	2.8 (2.8–2.8) *	2.8 (2.8–2.8) *	2.9 (2.9–2.9) *	2.9 (2.9–2.9) *
Yes	3.6 (3.5–3.6)	3.6 (3.5–3.6)	3.6 (3.6–3.6)	3.6 (3.6–3.6)

¹ Each column displays total observations (n) and percent (%) for IP MI and IP HF case-control groups.

² Significance evaluated with the Chi Square test:

* = $p < 0.05$;

† = $p < 0.01$.

³ Significant age group difference between IP HF cases and controls, $p < 0.05$.

⁴ Significant difference between no monitor versus monitor within poverty, $p < 0.05$.

⁵ Significant difference between no monitor versus monitor within population density (Population, L10), $p < 0.05$.

⁶ Significant differences between no monitor versus monitor within population density (Population, L10), $p < 0.05$.