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Pm2.5 And Ambient Air Pollution: Effects On Medicaid Spending And Hospitalizations

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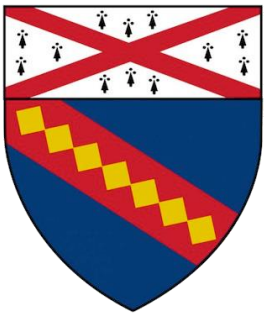

PM2.5 and Ambient Air Pollution: Effects on Medicaid Spending and Hospitalizations

A cost estimation derived from Medicaid charges in the state of Texas, 2010-2011.

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Acute PM2.5 Exposure and Medicare Hospitalizations in Texas

Abstract

Rationale: Fine particulate matter (<2.5 um), or PM2.5, and Ozone has been linked to a number of respiratory and cardiovascular conditions and is a known trigger for acute events. Though previous studies have addressed these correlations, few have examined PM2.5's acute effects on inpatient admissions and health care costs, by utilizing narrower time intervals than other research projects.

Objectives: To identify whether trends in hospitalization spending in short time-intervals is associated with PM2.5 measures, and to create a predictive model for spending based on two major categories of outcomes: selected CV conditions and respiratory conditions known to be associated with PM2.5 exposure that we will identify by Medicaid charge codes. We also attempt to model inpatient admissions altogether as an alternative outcome.

Methods: We link Medicaid charge information for all procedures in Texas to daily air-quality data sourced from 63 EPA sites in the state and fit a longitudinal mixed model to extrapolate costs and risks of additional inpatient stays due to respiratory conditions from particulate matter readings. Outcomes are identified by APR-DRG codes listed in the Blue Ribbon Medicaid set and exposure measurements are sourced from the EPA's monitoring stations' data mart. Our study covered September 2010 to August 2011. We also adjust for other potential covariates and exposures like Ozone in later models.

Measurements and Main Results: We find positive association between environmental PM2.5 and healthcare spending. Our simpler multilevel linear model gave us these cost estimates: rates of Respiratory and CV charges to Medicaid increase $3.95 million dollars to each increase of ug/m^3 in PM2.5, under our simplest per capita model. A more sophisticated secondary-model states that ozone is the more significant predictor of costs/charges, and one of our models produced an annual $600,000 increase per additional 0.01 ppm of exposure over the year. We also find that modelling counts of hospitalizations fit our model better as an outcome.

***Conclusion*:** The models suggest a positive association between Medicaid spending and PM2.5 or Ozone exposure. These measurements can be utilized to predict morbidities based off of air quality, as well as estimate the impact to Medicaid in terms of its financial strain. This information can be utilized in resource allocation in terms of hospital staffing and local medical needs, as well as on larger scale, aiding policy decisions.

Introduction

Air Quality and fine particulate matter exposure affects peoples' respiratory and cardiovascular health. His impact can be measured in admission rates and increases in medical costs. On an aggregate level, the CDC estimates that, inducing costly conditions like asthma that costs the US up to $56 Billion per annum* (CDC 2011). This increase in costs is in part due to hospital/ER admissions, and for our purposes we will isolate the spending trends in Medicaid-dependents of Texas, whose 3.5 million members account for $25 billion in annual healthcare spending. Regulatory authorities in Texas consider PM2.5 level at acceptable at 12 $\mu g/m^3$ average daily exposure over the year, compared to the 10 ug/m^3 standard set by the WHO and other international standards (WHO 2015). In the context of guiding future policy decisions, this type of research might provide some sort of financial guideline for resource allocation and policy reconsideration.

Background and Prior research

Although previous studies have found an association between air quality and health care spending, these studies had some limitations. Some analyses and bundled air measurements by month, which provided poor temporal resolution in modeling fine responses to air quality changes, and the resulting economic burden. Some analyses established a relationship between conditions and particulate matter in for a narrow number outcomes, like only measuring mortality's association without accounting for nonlethal morbidities (Katsouyanni 1998). Looking at all hospital treatments at the daily level might provide better resolution to find more subtle associations.

Research has been executed utilizing Medicare charge data joining datasets with EPA exposure data to do analyses over a wide geographical area, with separate models for different regions, (Peng 2008). These models tracked and measured hospitalizations compared to daily readings aggregated to the county level, providing both a more accurate assessment of the burden of the disease, as well as its geographical distribution. " (Peng 2008). Though originally evaluating PM10 exposure, larger particles that are more likely to be filtered out and caught before doing damage, they found that after controlling for PM2.5 the PM10 exposure associated become much more attenuated, and that the ultrafine PM2.5 particles were more likely to be causally associated with admissions for respiratory and cardiovascular diseases. These authors convincingly demonstrated higher Risk Ratios in respiratory and cardiovascular conditions in geographical areas with higher ambient particulate matter. Other analyses undertook similar analysis but utilizing indirect measurements like distance from roads as a proxy over direct air readings, requiring yet another set of assumptions that affect the precision of individual exposure estimates (Gilbert 2003).

Contribution

Through monitoring daily air-readings and inpatient admissions to the zip-code level, this paper adds to this literature by using more granular geographic areas to measure these associations. I will refine the geography of exposures by linking environmental air quality measurements to patient rather than hospital data. Also, rather than simply tracking risk ratios, I will provide a predictive estimate to the social burden of these direct costs—by providing cost estimates for the medical procedures for conditions that are generally accepted to be associated with PM2.5, and how it is influenced by daily AQI environmental readings.

This analysis is restricted to the state of Texas and uses patient level charge data to estimate fluctuations in spending on cardiovascular and respiratory diseases and how it is associated with changes in air quality. By utilizing the EPA's daily PM2.5 readings from 58 measuring stations in Texas (data from the AIR QUALITY SYSTEM DATA MART database spanning years 2010-2014), we can get a finer level of geospatial AND temporal precision that is required when measuring acute responses and costs.

These measurements can be utilized to predict morbidities based off of air quality, as well as estimate the impact to Medicaid in terms of its financial strain. This information can be utilized in resource allocation in terms of hospital staffing and local medical needs, as well as on larger scale, aiding policy decisions.

Table 1: Descriptive Information of our overall Texas Medicaid Sample and Associated Measurements

SITE:EPA	Number CV/Resp Charges*	Mean Cost per charge*	Std Dev	MeanPM*	Std Dev	Total Cost CV/breathing*
1	110	4608.79	4049.1	9.966	5.124	$506,966.90
2	155	5158.88	6125.93	9.632	5.149	$799,626.40
3	1438	5074.01	3454.16	10.114	5.063	$7,296,426.38
4	131	4673.9	11943.67	9.847	4.973	$612,280.90
6	91	3835.7	2690.48	10.563	5.220	$349,048.70
7	2	4120.83	2075.1	10.237	5.196	$8,241.66
~~8~~	~~437~~	~~3435.05~~	~~2202.77~~	.	.	~~$1,501,116.85~~
9	31	3562.31	1452.92	6.637	4.676	$110,431.61
10	1123	3846.65	4565.87	10.867	5.266	$4,319,787.95
11	38	3682.29	2566.37	10.987	4.552	$139,927.02
12	6	2670.4	694.12177	6.664	5.525	$16,022.40
13	2035	6126.57	9309.88	11.104	3.551	$12,467,569.95
14	535	6091.99	5528.57	10.130	4.065	$3,259,214.65
~~15~~	~~187~~	~~7425.72~~	~~11007.56~~	.	.	~~$1,388,609.64~~
16	427	5606.25	5549.68	8.754	4.551	$2,393,868.75
17	737	3874.02	3474.99	10.216	5.532	$2,855,152.74
18	378	3668.06	4296.19	9.246	4.361	$1,386,526.68
19	156	4979.67	7376.79	8.452	4.095	$776,828.52
20	79	4264.88	2152.24	9.254	3.841	$336,925.52
21	10	3891.75	2677.07	10.124	4.161	$38,917.50
22	640	5560.48	18306.18	10.857	5.194	$3,558,707.20
24	81	4824.61	3239.75	12.450	7.660	$390,793.41
26	463	5126.45	17496.44	8.618	4.362	$2,373,546.35
27	305	9866.57	13393.34	9.641	3.945	$3,009,303.85
28	154	4809.52	2690.86	11.570	4.820	$740,666.08
29	199	6306.67	16385.42	11.386	4.400	$1,255,027.33
31	3536	5866.44	10604.19	10.951	4.229	$20,743,731.84
33	13	4815.13	1502.26	12.255	4.234	$62,596.69
34	206	5015.63	3412.86	10.616	4.299	$1,033,219.78
35	554	5057.39	10431.91	10.792	4.545	$2,801,794.06
36	196	5188.51	3782.59	9.408	3.498	$1,016,947.96
37	852	4752.77	4790.44	10.625	7.681	$4,049,360.04
38	3521	3191.47	3178.75	11.510	5.603	$11,237,165.87
40	263	3624.41	3607.11	10.318	6.288	$953,219.83
41	49	19533.52	92830.08	9.140	3.966	$957,142.48
42	581	3724.28	2820.3	8.644	4.220	$2,163,806.68
43	778	5098.99	7388.92	10.125	4.027	$3,967,014.22
44	54	6028.77	7571.08	10.768	4.674	$325,553.58
45	549	3823.5	5487.15	9.507	6.355	$2,099,101.50
46	666	4320.83	9834.33	8.736	3.883	$2,877,672.78
47	349	3661.44	2566.51	10.274	6.028	$1,277,842.56
48	733	5272.56	18006.95	12.944	5.397	$3,864,786.48
49	470	4282.16	4075.77	9.950	4.299	$2,012,615.20
50	54	4401.8	3576.8	10.096	4.497	$237,697.20

~~51~~	~~581~~	~~5026.23~~	~~4041.3~~	~~.~~	~~.~~	~~$2,920,239.63~~
52	198	4115.11	2443.61	9.268	3.820	$814,791.78
~~53~~	~~150~~	~~3295~~	~~2984.02~~	~~.~~	~~.~~	~~$494,250.00~~
54	182	4495.89	10712.26	9.705	4.184	$818,251.98
55	723	4663.74	12117.53	7.094	4.027	$3,371,884.02
~~56~~	~~242~~	~~5190.72~~	~~5544.81~~	~~.~~	~~.~~	~~$1,256,154.24~~
57	1170	5354.69	8681.7	10.488	4.142	$6,264,987.30
58	376	7092.81	20000.42	9.614	4.456	$2,666,896.56
59	1039	6246.86	7400.63	9.612	4.885	$6,490,487.54
60	490	6567.42	44787.68	8.445	4.490	$3,218,035.80
62	712	3441.29	5867.9	12.235	6.064	$2,450,198.48
63	585	4086.27	5183.53	7.991	3.900	$2,390,467.95
TOTAL						$146,729,448.97

SITE:EPA represents the catchment zone generated with an EPA monitoring site with a center. Blanks are sites where we were not able to derive information, and were shuffled into the next closest site in subsequent steps. Amongst the 13 sites we ended up discarding, 5 were discarded due to sparse air readings (strikeout).

TABLE 2: Data Overview-Aggregate Data

DATA SOURCE	**Value ± SD**	**p-value**
Mean charges	49437.14 ± 65628.48	P<0.0001*
Mean Ozone (ppm)		P<0.0001*
Mean PM2.5 (ug/m^3)	8.9727 ± 3.0706	P<0.0001*
Mean # Medicaid	62699 ± 84809	--
Mean Pop	429217 ± 581472	--

P-values derived from ANOVA for tests comparing one EPA-Site Catchment area's weekly readings to another, suggesting fundamental differences caused by hidden secondary covariates between zones.
*p-values are lifted from ANOVA comparing by groups organized by EPA-site, and are significant on a $p < 0.05$ under a Bonferonni correction.

Datasets and Sources

We utilized 2011-2012 Medicaid charge information from the Texas 'Blue Ribbon' dataset as our source of healthcare cost information and place of residency associated with each patient. We utilize this information and geocode cases of asthma/COPD to the nearest EPA catchment site. These places of residency are paired to one of the **three EPA 63 monitoring** sites which offer daily* average mean readings of PM2.5 (particulate matter) exposure, which we utilize as an environmental proxy for an individuals' exposure.

In this model, we have divided Texas into 55 geographic areas (after dropping 8 sites due to data scarcity), whose centroid is one of the associated EPA sites. All respiratory cases in the blue ribbon set are paired with one of these geographic locations, contributing to that sites' daily repeated measures as an outcome (Table 1). We utilize EPA sites' readings for the date, utilizing the 3 day moving window average of PM2.5 for that site.

TABLE 3: Data Overview

DATA SOURCE	**Description**	**Relevant variables**
BLUE RIBBON	SOURCE OF MEDICAID DATA	CHARGE Costs, Area Code associated with residency, Diagnosis Related group
EPA-QS	Daily/Weekly PM2.5 readings	Longitude/latitude of measuring sites, pm2.5 level
Census	Population controls useful for establishing rate outcome.	Population per geographic area
Centroid Locations	Geocoding/geoprocessing Our link between our Blue Ribbon Set and our EPA set.	Longitude and Latitude associated with area codes.

Blue Ribbon Project (HCUP data)–Our charge information and proxy for 'direct' respiratory costs was sourced from the Texas-based Medicaid dataset managed by state Health and Human Services. Out dataset contains two years' worth of charge information from 2011-2012, identified by DRG. However, unlike other datasets it is lacking in SES type information.

We were able to isolate certain categories of charges from form information. The dataset categorized the type of inpatient admission via APR-DRGs (2009), looking at known respiratory outcomes from the literature (Chung 2005).

EPA-AIR QUALITY SYSTEM DATA MART DATABASE: The source of air quality measurements are collected from the EPA monitoring stations and downloaded rom their Air Quality System Data Mart system, sampled from 2010 to 2014. The AQS database-provided specific daily averages taken at variable intervals across the year. Data was provided in the form of average PM2.5 and Ozone readings for the day. It includes specific measurements by type of air pollutant. We utilized readings from the 58 air quality monitoring stations in Texas. We also drew the boundaries of our catchment zones from each EPA-site longitude and latitude. The centroids of area codes are assigned to the nearest EPA Air quality site as its landmark, and all the area codes designated to the same EPA monitoring site are assigned to the same zone/catchment area.

Census Data: 2010 Census information helped estimate the population of each catchment area by ZCTA (analog for zip code) and a ZTCA crosswalk file that allowed us to translate raw area codes to the census ZCTAs. By combining these datasets we can generate per-capita cost figures and hospitalization rates. An open source population centroid dataset was utilized to translate/mask the individual's area code to a 'zone' of these using the SAS 9.4 package Geocoding features to receive longitude and latitude data, to filter only for datasets close by to the EPA or state air-quality monitoring stations.

We repeated this process for the different OZONE monitoring stations also derived from the AQS EPA system, which had its own sets of sites and longitude and latitude information.

METHODOLOGY: DATA CATCHMENT/ SELECTION CRITERIA

Methods: Measurements of Exposures and Outcome Identification

Like previous studies we utilize environmental measurements closest to individuals' place of residence as a proxy for Medicaid patients' exposures to PM2.5 or ozone. We did not account for indoor exposures or personal air monitoring readings. This method, however, gave us the advantage of being able to fit longitudinal repeated-measures models to elucidate the relationship between spikes in PM2.5 air concentration and causally-linked health outcomes like asthma and COPD on a very large scale.

Outcomes were identified from the hospital blue ribbon dataset using commonly known 2011 APR-DRGs (diagnosis related groups) that are indications of the type of condition a patient is admitted under. From the literature we focused on respiratory admissions whose primary diagnosis was Asthma, COPD, Respiratory Inflammation, pulmonary embolisms, Bronchiolitis, all conditions whose associations with PM2.5 are well documented (Dominici 2006). We looked up codes with the accompanying data-dictionary for the Medicaid charges that specify the type of primary condition the patient was afflicted with.

We also focused on certain cardiovascular conditions whose relationship to acute PM2.5 is also supported within the literature (CDC 2011). Heart Failure, Cardiac Arrest, myocardial infection, along with a few other acute conditions have strong positive associations with localized PM2.5 exposure (Talbott 2014). We also listed some conditions associated with Ozone exposure, as ozone is associated with our primary outcome asthma. The rest of the exposures are listed in **Table 3** (Lin 2002).

We subset outcomes for more refined analysis based on Respiratory Only categories (left column, Table 3), Cardiovascular Conditions (right column, Table 3), and all-combined. In increase the percentage of cases, as we would've ended up with many strata with zero values, we ended up utilizing the combined conditions listed in, all associated with PM2.5 (Karr 2006).

Table 4: Medicare-DRG Classifications used to identify charges for conditions associated with PM2.5

Respiratory Conditions:	**Cardiovascular Conditions**
Pulmonary edema	Myocardial Infarction
Pulmonary embolism	Heart Failure
Major Inflammation	Cardiac Arrest
Bronchiolitis	Angina Pectoris
COPD	Cardiomyopathy
Asthma	Cardiac Arrhythmia / Hypertension

Methods: Geocoding and Group Allocation

Allocation of individuals was done utilizing charge data from the Blue Ribbon file. The dataset had a zip code of home residence that we were able to utilize. From this we are able to pair people by area code to their mean weighted population centroid location via longitude and latitude. This is similar to the method employed by previous studies linking hospitals to the county level measurements (Peng 2008), and likely provides a more realistic measurement than others that utilized even more indirect measurements like distance from roads as a proxy.

Geocoding of study sites was executed by taking longitude and latitude measurements for all 63 EPA air monitoring stations and utilized them as the center of circular zones that would be utilized to allocate charges to. With fields provided by both datasets, we can geocode the individual charges to specific EPA sites by min distance. Map them via the area-code information provided by Blue Ribbon charge dataset, pairing individuals to an EPA monitoring site as its anchor point. Since we only had area code resolution, we utilized a publically available mean-population centroid dataset.

SAS handled all geo-coding measurements, utilizing built-in operators like a geo-distancing function to calculate distances. We determined which EPA site most representative of the individual patients' exposure by calculating the closest site. For each of the 400k observations available in the Blue Ribbon charge data, SAS calculated distances (from the patient's area code centroid location) to an array of 58 longitude-latitudes associated with the air monitor locations.

In effect, our model treated each geographic circle as the lowest level of measurement for our outcome (hospitalizations/spending per site), utilizing the monitoring station as both the center and the source of that area's daily-ish PM2.5 / AQI readings.

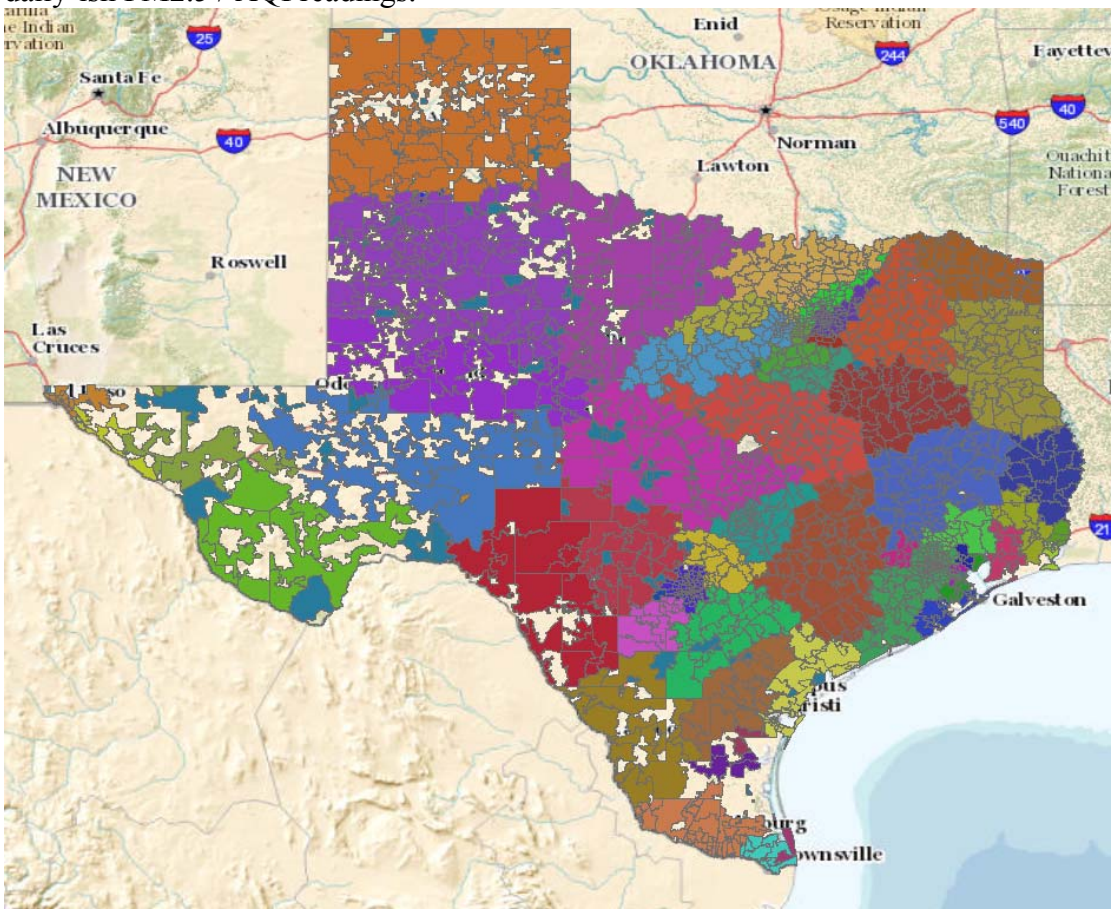


Figure 1: Aggregated Catchment Zones.
A map of ZCTAs (smallest borders) with each color representing a cluster of catchment zones for readability.

Figure 2: Structure of Aggregated Data

	Daily Counts from September 1- August 31st				
EPA-Monitoring Area	Day 1	Day2	Day3	…	Day365
1	obs1 (= COST + PM2.5)	Obs2			Obs365
2					
3			…		
…					
63	OBS 1	Obs2			Obs365

*Structured for hierarchical repeated measures analysis.
Individual charge data was distilled to number of counts (/or cost accumulated) for respiratory conditions per day, along with a paired pm2.5 reading from that specific EPA monitoring site/catchment area.

Data Imputation:

The data was complex: the 63 EPA sites were not homogenous in the amount of readings a year available. Due to wanting to properly utilize all charge information available and not just drop the hospital information associated with missing air-reading days, we utilized splining and imputation methods. Regression Splines with Longitudinal Data was the basis of our imputation methods.

Also utilized a simpler linear-spline method via SAS.

These modeling and estimation procedures had been utilized previously for similarly structured data to justify our use of LOESS or interpolation for mixed data (Jo 2007). We fit these measurements as the primary identifier when we were missing a true reading.

We had to drop 8 sites due to the lack of charges. Another 10 zones were also dropped because of scarcity of air data and were allocated to the next closest source. The variations in distance of the population to its matched site might provide some sort of measurement bias. We did not utilize imputation for the ozone records, as fortunately, though there were fewer sites, had daily readings for all of them.

Methods: Modeling

We executed a basic descriptive statistical analysis for an overview of the air-readings and charge information associated with each catchment area (Table 1) along with an ANOVA test comparing differences in weekly readings between zones.

Model Description

Primarily, all our models are mixed longitudinal models with repeated measures. We treated each catchment area associated with an EPA site as the smallest individual subject that provides the mean PM2.5 reading for the day aligned with the daily medical expenses. These daily repetitive readings lend itself to multilevel repeated measures model (Figure 2).

The Daily PM2.5 was our fixed independent variable component of our mixed model, as our primary association of interest. The random effects components of our mixed model consists of a random intercept associated with each EPA-catchment zone, to reflect the differences associated with each of the microenvironments and populations housed within. As with other time series, we utilized a covariance structure that was autoregressive, as subsequent air quality readings should not be independent from one another.

Outcomes and Exposure Estimation-imputation and the problem of time

Each EPA-site was the unit of analysis, with 3 outcomes: 1.) Cost associated with the day 2.) Number of procedures executed 3.) Charge amounts associated with the inpatients for the day. Daily PM2.5 and Cost calculations as the *nested, repeated measurement.* We also remodeled different outcomes based off of binned, aggregated value with weeks as the lowest unit of outcome/exposure. Table 2 and Table 3 illustrate the various model fits and time frame of exposures and outcomes that we utilized.

Delving into the literature, previous studies also considered multiple models for PM2.5 exposure (Peng 2008). Some papers and models associated the same day air quality readings with the fixed lag-time models, more traditional, and with the correlation structure previous values still have some sort of influence on the model. Peng and Bell's 2008 paper with Medicaid data utilized various lag-times, 0 day, 1 day, 2 day, while we will utilize a 3 day rolling average window. Some papers emphasize a model of gradual sensitization before an asthma attack **(Lin 2002).** I wanted the model to capture a larger window of exposures than a single lag-time(especially since acute can be triggered by an 'accumulation' consistent high levels of PM exposure), and especially given my imputation methods, a 3 day moving average is appropriate so that results will 'dilute' the effect of missing an extra outlier. However, this will likely drag associations closer to the null. Later, through data aggregation we utilize a weekly average to measure ozone and PM2.5 exposure.

Model Progression

We will fit multiple models, simple outcomes isolating PM2.5 and later fits that include known covariates that affect our outcome like Ozone. We will also try alternative time-frames in terms of exposure measurements: not only are we going to utilize three day moving averages, but we will estimate our outcomes using broader binned weekly measurements of PM2.5 and ozone.

Results and Estimates

The statistically significant p-values derived from ANOVA for tests comparing one EPA-Site Catchment area's weekly readings to another, suggesting fundamental differences caused by hidden secondary covariates when comparing different areas of the state in Table 2. Of course, this ignores some of the temporal component, but is useful for understanding that there is indeed a difference in the effect of air quality between groups and spending. The statistically significant p-values derived from ANOVA suggest differences in average per capita costs and air exposure.

Daily PM2.5 as a Linear Predictor of Actual Amount Paid:
Table 5 contains the parameters to calculate amount of money associated with each increase in ug/m^3 in our mixed model. Models have varying outcomes (by site by site or per person, which enables different controls) and (varying lag times and measurement intervals) for the independent variable measurements. In our mixed model with a 3-day rolling average of PM2.5 ambient concentration as a predictor, we were able to find statistically significant values for our daily costs as an outcome per geographical site($p<0.001$), and marginally significant outcome with the per capita outcome ($p=0.0737$, Table 5). This true cost outcome is the actual amount paid out by Medicaid for the hospitalizations in the Blue Ribbon set. We find that site specific estimates produce an estimated

$50 * 365 * 515.68\left(\frac{\$}{\frac{ug}{m^3}}\right)$ = $9,411,160, in additional state Medicaid spending per additional year.

Utilizing the individual per capita spending as an outcome and again extrapolating these results to a statewide estimate, a 1 PPM increase in annual PM2.5 exposure to the Texas population(25MM) is estimated to have this economic impact:

25.26MM People *365* $0.0004190\left(\frac{\$}{\frac{ug}{m^3}}\right)$ = $3,863,138 for Texas over the year.

TABLE 5: Model Parameter for mixed model with TRUE-Cost as outcome.

Model-Outcome	Outcome Time Frame	Exposure Window	PM2.5-B1$\left(\frac{\$}{\frac{ug}{m^3}}\right)$	Covariates	p
$ per **site**	Per day	3-day rolling average PM2.5	515.68	*	**<.0001**
$ per **site**	Per week	Weekly Average	158.09	*	0.2466
$ per capita	Per day	3-day rolling average PM2.5	4.190E-4	* +local pop	0.0747
$ per capita	Per week	Weekly Average	0.01217	* +local pop	<.0001

Parameters to calculate amount of money associated with each increase in ug/m^3Mixed Linear Models with Repeated Measures. Models have varying outcomes (by site by site or per person, which enables different controls) and (varying lag times and measurement intervals) for the independent variable measurements.
*Adjusted for Census Population in catchment Area, and proportion of Medicaid participants

Daily PM2.5 as a Linear Predictor of Charges:
TABLE 6 shows parameters to calculate amount of charges, not amount paid, associated with each increase in ug/m^3 in our mixed linear models. Models have varying outcomes (by site by site or per person, which enables different controls) but not have fixed weekly exposure variables for the independent variable measurements.

In our mixed model we had fit PM2.5 concentration, measured in ug/m^3, against the daily outcome of hospital charges. It is a secondary measurement subject to inflation of costs and variance. But unlike 'amount paid', charges however are not negatively biased downwards by payments still due or costs that have been waived. We utilized an alpha =0.05 to test against the hypothesis that a predictor included is statistically significant (P<0.0001). We found that per site, a 1 ug/m3 increase in the 3-day window PM2.5 average would result in an estimated $1179 increase in charges per day (Table 3). This parameter is statistically significant. Translated to: a number that covers the year and the annual financial burden (as we have 50 catchment zones and 365 days in year):

$50 * 365 * 1178.85 \left(\frac{\$}{\frac{ug}{m^3}}\right)$ = *$*21,514,012.5 increase in Medicaid charges to Texas per 1ug/m^3 of PM2.5

We fit an alternative model which accounted for the population within each of our designed catchment zones, and it produced a positive linear association between pm2.5 and Medicaid charges (P<0.0001, Figure 3). Again, translating these results to a statewide estimate, a 1 PPM increase in annual PM2.5 exposure to the Texas population is estimated to have this economic impact:

25.26MM patients *365* $0.002664\left(\frac{\$}{\frac{ug}{m^3}}\right)$ = *$*24,892,416 for Texas over the year.

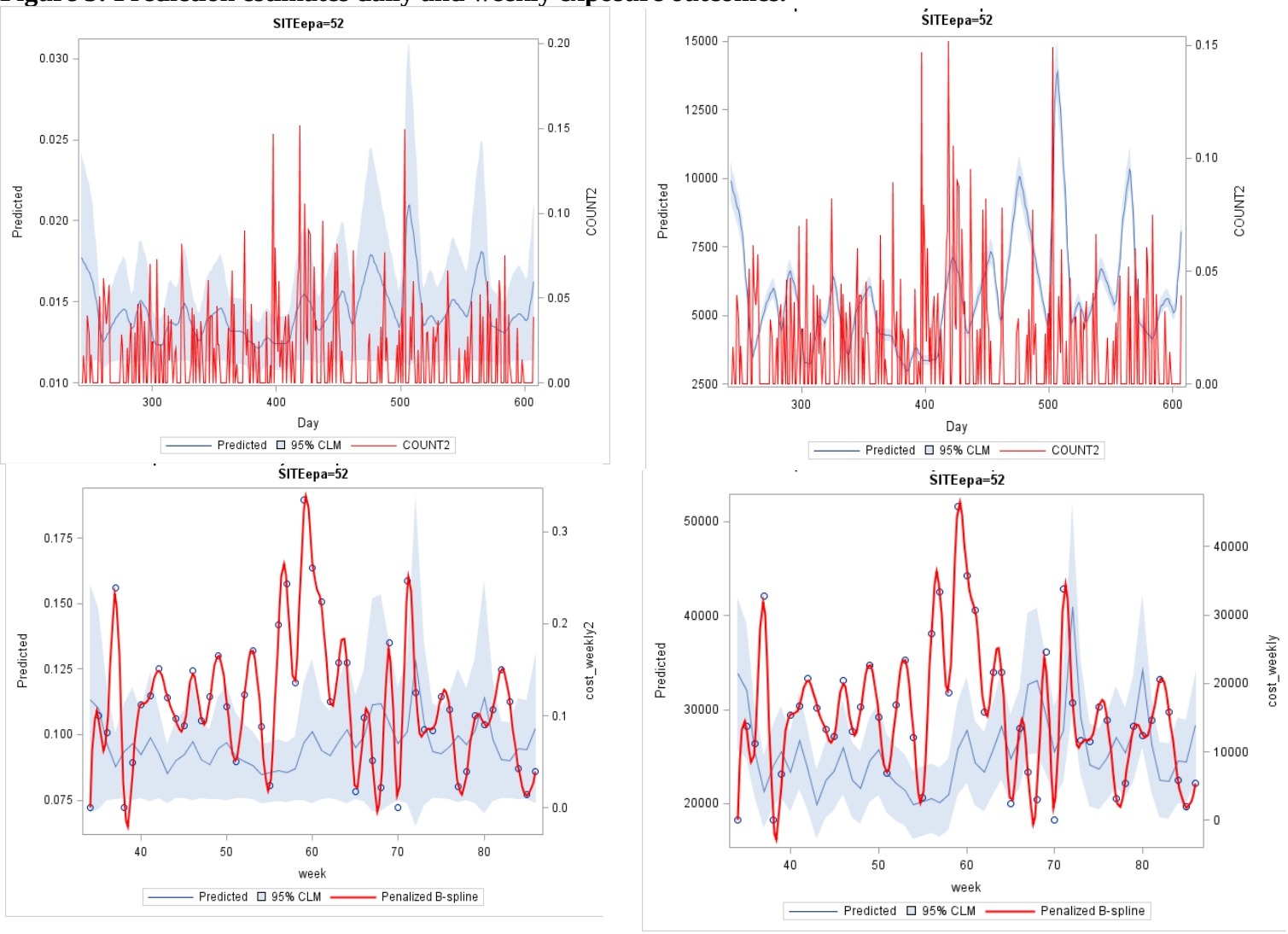
Alternative Models: Weekly PM2.5 and Ozone as a Linear Predictor of Amount Paid:
Table 7 contains Mixed Model regression parameters of alternative fits weekly exposure averages for models with Ozone and PM2.5. Models have exposure variables measured as a weekly average for the independent variable measurements, but differ. Additional covariates are illustrated by the plus sign. Also transformed outcome variable by taking square root of the per capital cost. We find that our mixed model with $ Per Capita Per Week as an outcome, when fixed with a time effect, had no significant air exposure parameters, with PM2.5 having a p-value of 0.84 and Ozone at 0.15.

In our model with $ per capita per week as an outcome (Table 7), each 1ug/m3 increase in PM2.5 results in a **$**2.181E-3, and each 0.01 increase in ppm results in a $0.024503 per capita, which roughly translates to $612575.

From our model, we took the square root transformation of $ per capita per week, and found that PM2.5 is a non-statistically significant predictor, but ozone is borderline significant (p=0.0582). For each 0.01 increase in ozone ppm increases square root per-capita cost per week by 0.006871, which translates to an additional $621824 to Texas Medicaid a year.

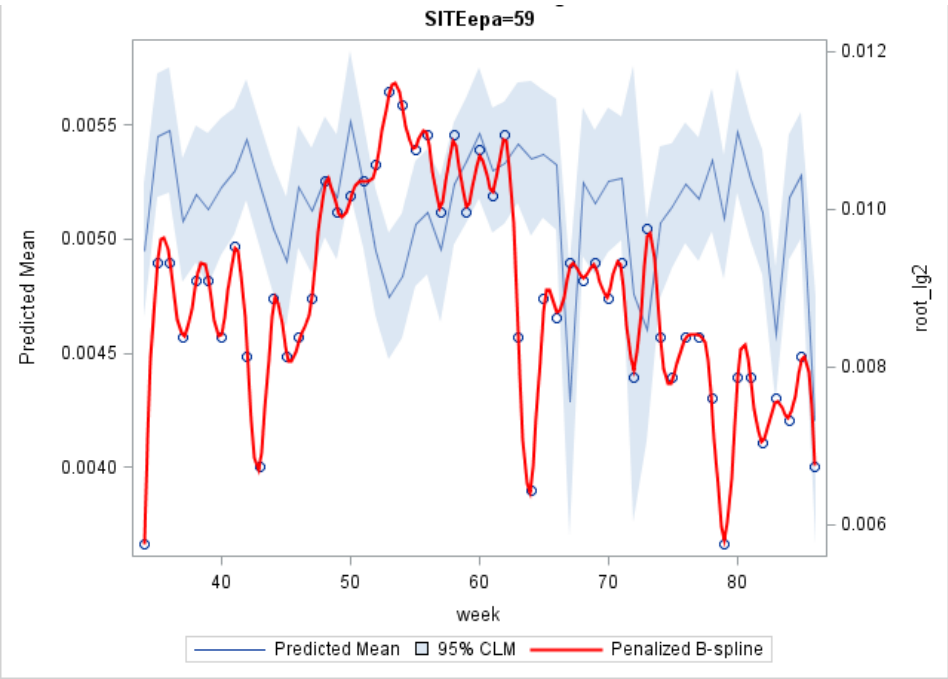
Count as an outcome:
We also modelled count as a secondary outcome. We find that both PM2.5 and Ozone are statistically significant predictors at p=0.0334 and p<0.0001 respectively. For each increase in ug/m^3, the weekly per capita hospitalization count increases 0.002181, and for each 0.01 ppm increase in ozone, it is increased by 0.005351.

Figure 3: Prediction estimates daily and weekly exposure outcomes.



Clockwise from top left to right, daily per capita predicted costs are charted against actual costs, daily costs per catchment area predicted cost vs Actual readings, Predicted weekly costs per capita vs actual, and Predicted weekly region costs vs actual ones. PM2.5 is the only exposure variable.

Top row displays our daily readings for our outcomes. Bottom row utilized our weekly binned averages.



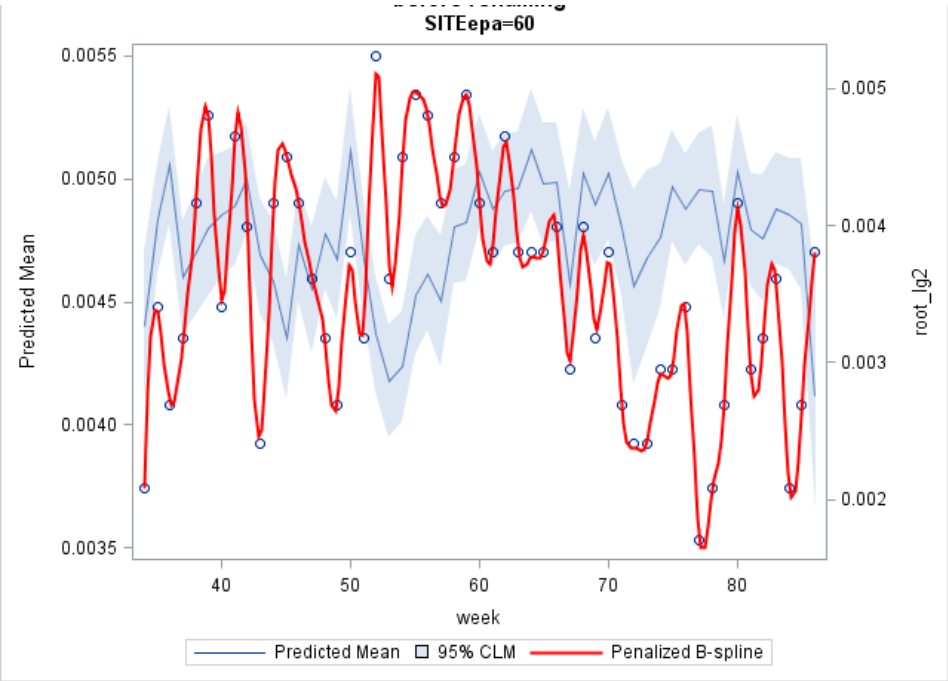
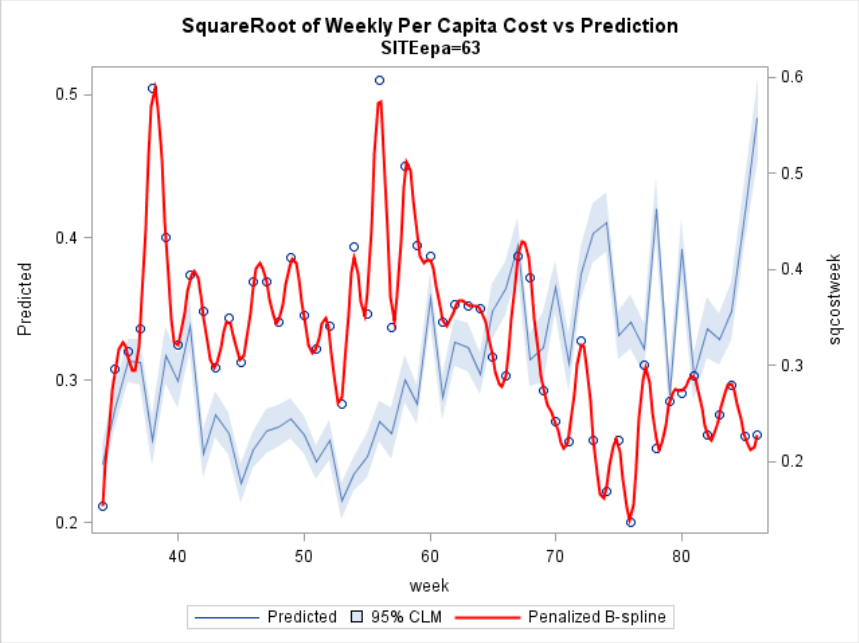


Figure 4.Weekly per Capita Count Outcome, square root transformed. Predictive Models versus actual data.
Our mixed model generated a count of hospitalizations per capita. Two sites were chosen for readability. The shaded blue line is the predicted values, and the colored area is the 95% confidence interval

The red series is a splined fit of the actual outcome variables, detecting a series of cost measurements.

Note: Left axis refers to the prediction. Scales are not equal and just meant to illustrate trends.

Covariates include PM2.5 and Ozone in these model



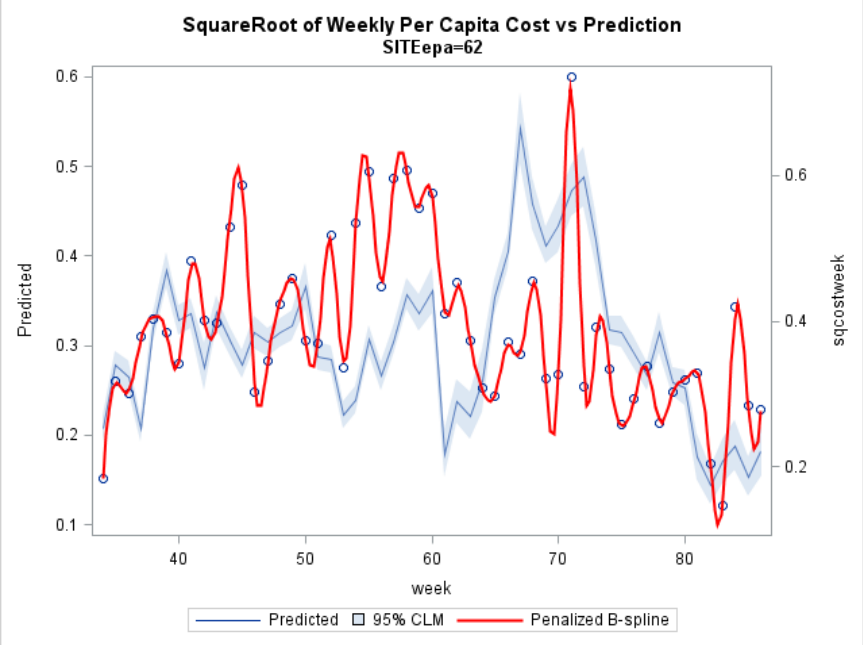


Figure 5: Square-root transformed per-capita spending and its predictive model.

Depicts the predicted mean of square root of cost per capita per week in sites, compared with the raw, tabulated cost per capita. These costs are measured by actual amount paid. Observations are binned by weekly units.

The blue series represents the models prediction along with the 95% CLM.

The red spline fit are the actual cost readings per capita per week.

Model includes both PM2.5, Ozone.

Note: Scales are not equal and just meant to illustrate trends.

TABLE 6: Model Parameters with Charges as an Outcome

Model-Outcome	Exposure Window	PM2.5-B1	Covariates	p
$ per site Per day	3-day AVG PM2.5 Interpolation	1178.85 $\left(\frac{\$}{\frac{ug}{m^3}}\right)$	*	<.0001
$ per person Per Day	3-day AVG PM2.5	2.664E-3 $\left(\frac{\$}{\frac{ug}{m^3}}\right)$	*	<.0001
$ per person Per Week	Weekly Average	2.181E-3$\left(\frac{\$}{\frac{ug}{m^3}}\right)$	* + OZONE	<.0334

Parameters to calculate amount of money associated with each increase in ug/m^3Mixed Linear Models with Repeated Measures. Models have varying outcomes (by site by site or per person, which enables different controls) and (varying lag-times and measurement intervals) for the independent variable measurements.
*Adjusted for Census Population in catchment Area, and proportion of Medicaid participants.

TABLE 7: Model Parameters with Ozone Fits

Model - Outcome	Exposure Window	Covariates *	PM2.5 }{	Ozone -
$ per capita Per Week	Weekly Average	+ Ozone	2.181E-3 (P<.0334)	2.4503 (p<.0001)
$ per capita Per Week	Weekly Average	+Ozone +Fixed Time Covariates	0.000573 (0.8462)	0.8080 (p=0.1506)
$\sqrt{\frac{\$}{\text{person}}}$ Per week	Weekly Average	+ Ozone	0.008587 (p<.0001)	4.0555 (P<.0001)
$\sqrt{\frac{\$}{\text{person}}}$ Per week	Weekly Average	+Ozone +Fixed Time Covariates	-0.00042 (p=0.7428)	0.6871 (p=0.0582)

Mixed Model regression parameters. Models may have varying outcomes (by site or per person, which enables different controls) all have weekly exposure variables for the independent variable measurements. Additional covariates are illustrated by the plus sign. Transformed outcome also present in last rows. Models refer to regression fits in Figure 7
*Adjusted for Census Population in catchment Area, and proportion of Medicaid patients.
}{ Parameter estimate measured in $\left(\frac{\$}{\frac{ug}{m^3}}\right)$
- Parameter estimate measured in ppm for Ozone

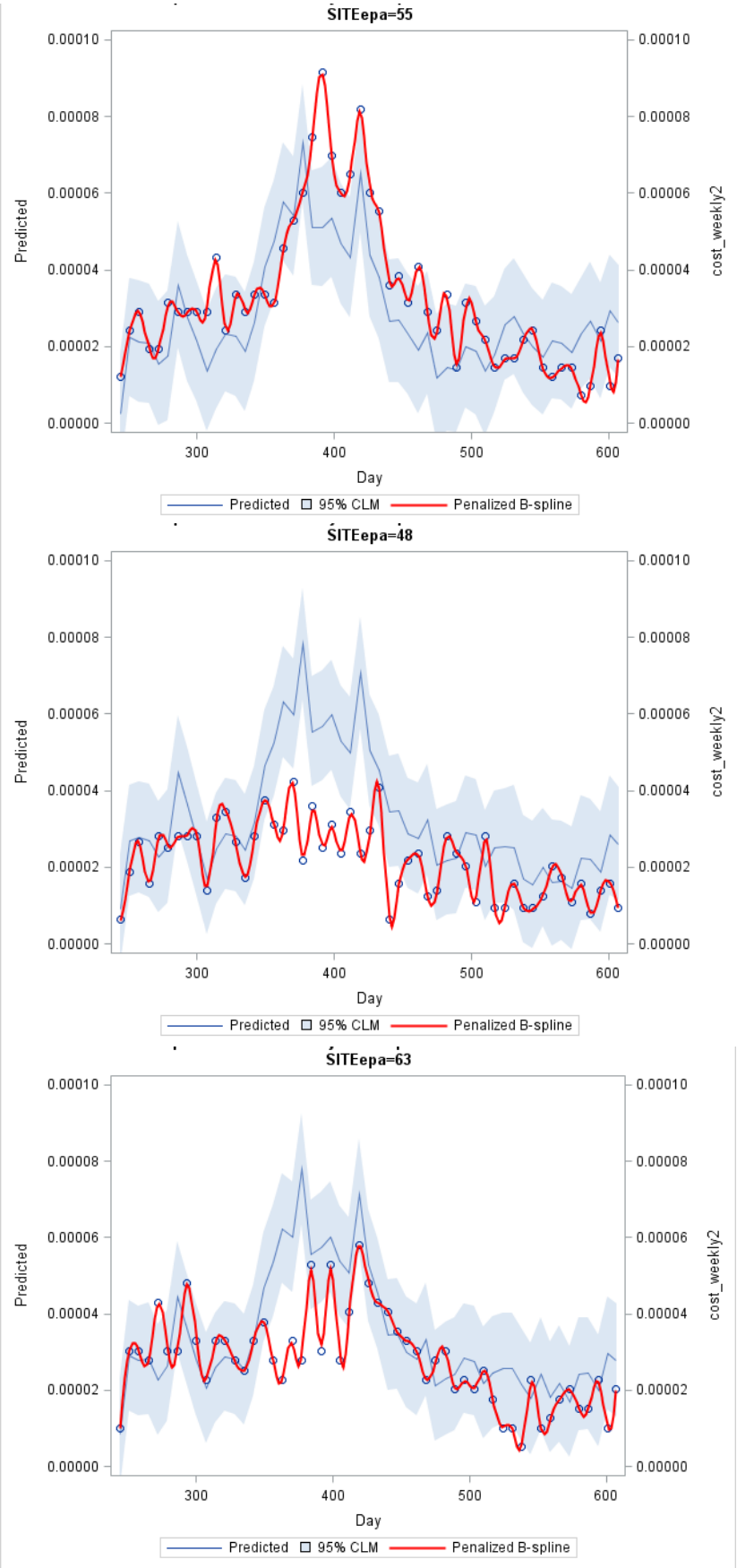


Figure 6- Per-week count of inpatients predicted controlling for OZONE + Fixed Time Effect

The following three graphs describe the Per Capita Number of hospitalizations vs the Predictive Model. Again, scales are different and meant to highlight the predictive validity association with our major covariates. Major independent variables in this predictive model include both PM2.5 & OZONE, as well as a fixed time effect.

The blue series represents the models prediction along with the 95% CLM.

The red spline fit are the actual cost readings per capita per week.

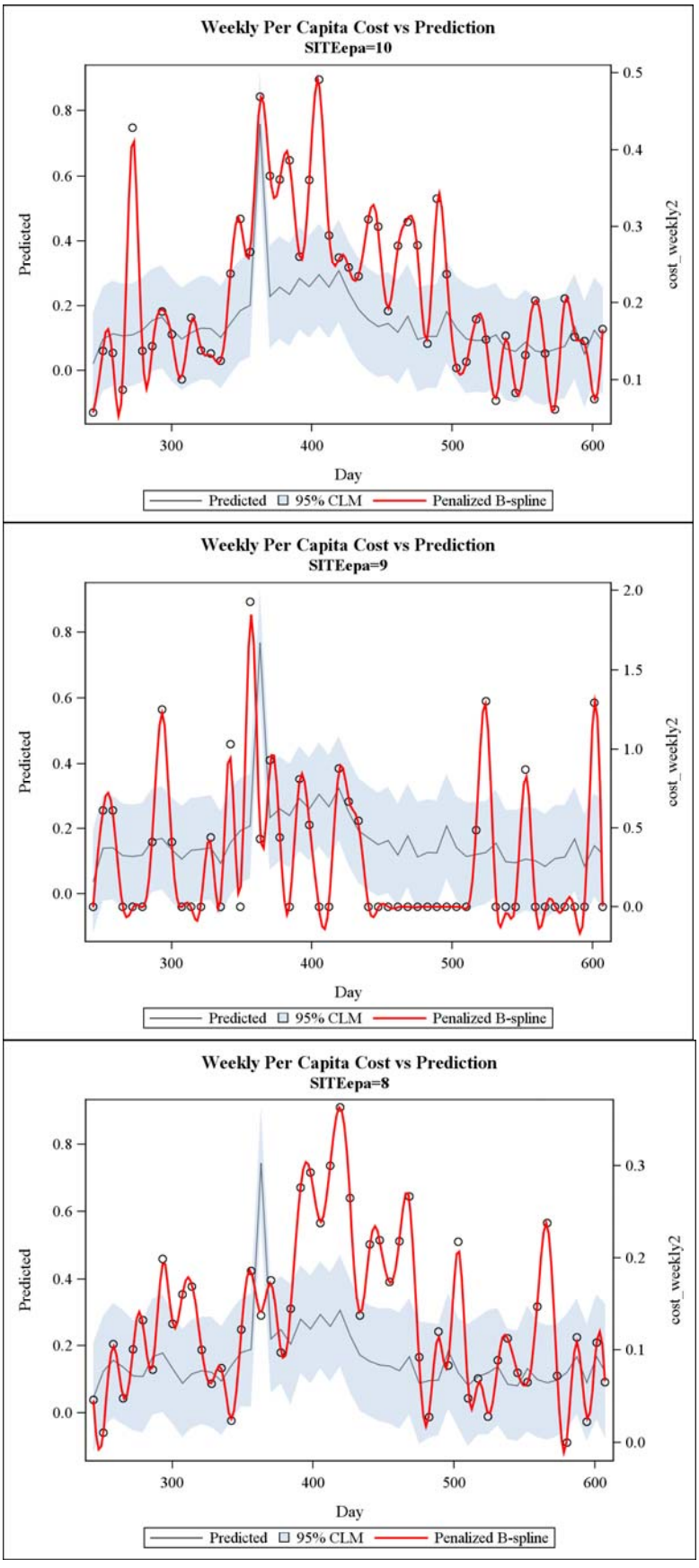


Figure 7-Model Prediction of Per-Week Per-Capita costs with OZONE + Fixed Time Effect

The following three graphs describe the Per Capita Cost vs the Predictive Model. Again, scales are different and meant to highlight the predictive validity association with our major covariates. Major independent variables in this predictive model include both PM2.5 & OZONE, as well as a fixed time effect. PM2.5 is not significant at all in the model.

The blue series represents the models prediction along with the 95% CLM.

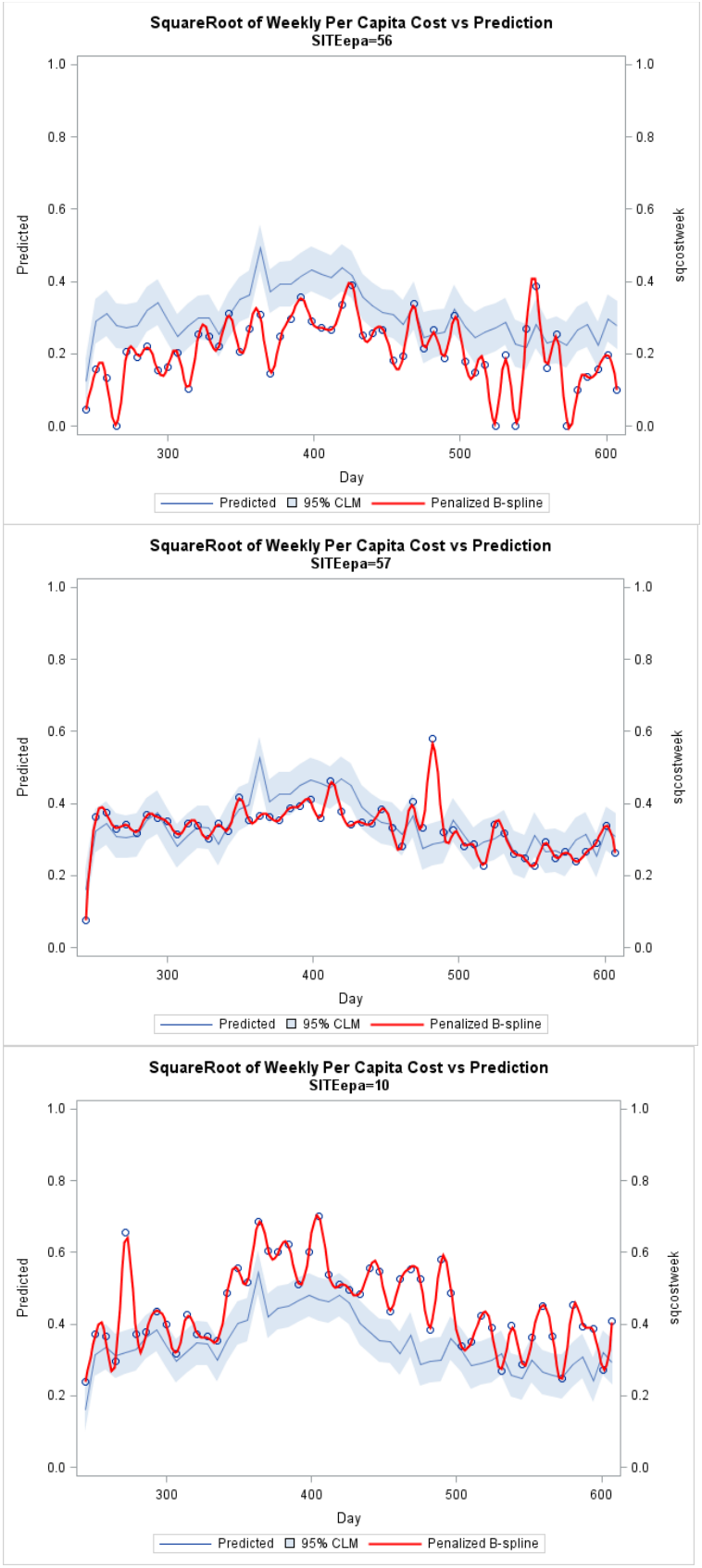
The red spline fit are the actual cost readings per capita per week.

Figure 8-Square Root Transformed Weekly Cost Per Capita. With OZONE + Fixed Time Effect additional covariates

The following three graphs describe the Per Capita Cost vs the Predictive Model. Again, scales are different and meant to highlight the predictive validity association with our major covariates. Major independent variables in this predictive model include both PM2.5 & OZONE, as well as a fixed time effect. PM2.5 is not significant in this model (Table 7) but OZONE is marginally significant.

The blue series represents the models prediction along with the 95% CLM.

The red spline fit are the actual cost readings per capita per week.



Discussion

Estimating financial impacts:

The project had a number of different models designed with a different purpose in mind. We began studying costs by utilizing the charge amounts as a proxy outcome, as some bills were left unpaid. But due to the extreme ratio of actual-cost:charge that exist in Texas (as high as 100:1), it resulted in an over dispersion of values and an almost certain overestimation of costs. Instead we elected to utilize the 'amount paid' as an outcome which is far more accurate, and actually reflects the amount of money going to providers.

In Figure 3, we have mapped out the cost calculations and have estimates in the PM2.5 parameter $20,000,000/year range in Medicaid charges alone that can be attributed to a PM2.5. And under the guise of evaluating the cost of the US's more lax standards, at 12 ug vs the 10 ug recommended by the WHO, this translates to a $40,000,000/year increase to Medicaid. However, this model can actually underestimate cardiovascular + respiratory costs associated with our gas exposures, as we are only utilizing the primary diagnosis codes in the Blue Ribbon's Medicaid charge information, leaving out secondary conditions that may contain some of the conditions of interest listed out in Table 4.

For our later models we decided only to focus on the binned, weekly exposures instead of taking advantage of the daily outcomes that we could generate in our model. The day-to-day level of resolution generated some 'noisy' graphs and slight over dispersion for the lower descriptive values, and the weekly exposures actually circumvented some of that. These estimations provided far more conservative estimations in the $600,000 range per additional increase of ug/m^3 in PM or 0.01 ppm increase in ozone.

Rationale for multiple model generation:

We have two broad classes of models that we fit: a predictive model that attempts to isolate the effect of PM2.5 (Table 4) and a better fitted descriptive model (Table 5). We found a statistically significant relationship between PM2.5 exposure and hospitalization costs, after controlling for the population size of each catchment sector and the proportion of Medicaid enrollees in the area. We found a similar proportion of Medicaid recipients in all areas. The main reason we did not utilize time fixed effects in our model is to attempt to isolate the effect of the contaminants. Though the predictive model would definitely perform better with the fixed time effects, there is a danger of over-fitting; what might actually be attributed to the variation in gas exposure might be reflected by the time effect, eliminating the chance of detecting a subtle effect of air quality on acute conditions/hospital admissions. As well, over fitting would weaken the models' usefulness and applicability to other years as seasonal disruptive events might account for many of the 'fixed effect' intercepts.

Validation of our predictive model and modelling progression:

Figure 3 output listed the expected change in cost per additional unit of PM2.5 exposure in a 3day moving window. Plotting diagnostics like a residual plot and QQplots (not pictured) we noticed a heteroscedasticity and an over-dispersion when looking at lower predicted values against the residuals. There is a fan shape with variation highest at the lowest predictive values and becoming. It does look relatively normally distributed otherwise despite the shifting variance. Modelling count data as an outcome (or the number of hospitalizations or medical charges themselves) in **Figures 4, 6** actually solves some of this

heteroscedasticity and the residual plot was much improved; likely because we used charges as our cost outcome, which has a huge variation in values and in inflation (cost to charge ratios can balloon from 2x to orders of magnitude greater). Another solution was transforming our outcome variable by taking the square root, which turns out to be effective. As mentioned previously, binning results by weekly exposures and cost-outcomes produced less noisy graphs, as in figure 3, and we adjusted exposure levels to be weekly means to reflect the changes to our measurement outcome from the daily values.

In regards to the more parsimonious models, notice that Figures 3-6 have predictive models that mimic the shape of cost or count trends, if not the proper magnitude. This problem might be due to some seasonal effect that we're not monitoring, or some other time-dependent covariate that we do not have data on (possibly another air exposure, or climate conditions that might affect exposure or the manifestation). In subsequent I hope to integrate temperature readings which likely affect rates of respiratory/cardiovascular inpatient admissions (Peng 2002).

The predictor preserves the correct directionality in change, but the predictions are often off in the magnitude of change. Most likely our model does not capture all the necessary time sensitive-predictors; either as confounders, or effect modifiers most likely. The bursts in amplitude at certain points give evidence there is probably and synergistic effect-modifier that is lurking in the population.

Another possibility is that each individual has a specific environmental exposure threshold that when crossed, greatly increases their risk in a non-linear fashion, though support for this model is tenuous according to some research. As a result, we began to add on additional covariates like ozone effects and fixed-time parameters (for exploratory purposes, year to year fixed time effects will differ and doing so might weaken the applicability of my model to other years and other states). However, as a result the most accurate predictive models, seen in Figure 7, has PM2.5 association almost completely attenuated, while leaving ozone's parameter marginally significant (p=.0582).

In Figure 7 we notice an interesting spike present in all the graphs at ~ Day 380. After checking diagnostics to make sure it wasn't an outlier or error in readings resulted in this odd artifact, we found that there was a spike in costs and hospitalization in most of the regional catchment sites in this time frame. It was not an error or artifact of the data; this sudden spike was evidence of a disruptive climate event. This date translates to late January of 2011 and coincides with a major storm, the 2011 North American Blizzard. It was a category 5 storm that hit North America and affected multiple states, even Texas, causing rolling blackouts, water treatment shutdowns, and school district cancellations due to road hazards of ice and snow. Temperature dropped into the single digits, halting infrastructure, (NWS 2011). It is noted that extreme changes in temperature often trigger negative outcomes that include cardiac issues like heart failure and arrest, as well as increasing the rate of asthma attacks (Huynen 2001). The effect on asthma and respiratory conditions is twofold: not only does the cold induce exercise induced asthma through a mechanism of water/heat loss and inflammation, but it also keeps children, an at-risk subgroup, indoors where there are more allergens (Medina 2006; Carlsen 2012).

Count as an outcome:

We also modelled count as a secondary outcome-and predictably, inpatient counts were easier to fit. Prediction graphs are listed in figures 4-6, and notice how the variations in magnitude of spikes are not as dramatic as in charges (which has an additional cause of variation caused by price differences between conditions). We find that both PM2.5 and Ozone are statistically significant predictors at $p=0.0334$ and $p<0.0001$ respectively. For each increase in ug/m^3, the weekly per capita hospitalization count increases 0.002181, and for each 0.01 ppm increase in ozone, it is increased by 0.005351.

Potential Issues and Limitations:

There are fundamental issues with how we measure exposure and outcome that was caused by the limitations of the data. As for our exposures: PM2.5 is a catchall, only referring to the size of particles and not the composition. Previous research have noted that the specific source of PM2.5 affects the risk ratio for a health outcome. And since we neither looked at the composition of the exposure nor modelled against single outcomes, we could be missing out on cleaner correlations. Of course, these issues are due to sample size and arriving at a zero-inflated over dispersed model, that might require some other modelling techniques like a zero-inflated, mixed negative binomial regression , but it doesn't quite preserve the strength 'repeated' structure, instead modelling each reading as a nested random effect of the individual (Liu 2007).

Our research relies on multiple assumptions in its design. One is that the bootstrapped values are accurate; that the distribution of the 3.5 million Medicaid enrollees is distributed similarly to how charges are distributed for the year. Our methods also assume that area code centroids an adequate estimate of peoples' true residency location in regard to EPA-sites, and that these allocations to EPA-sites are adequate for modeling purposes. However, there is the possibility that there'd be differential misclassification bias in terms of environmental exposure: Those people who are living much further away from the nearest EPA site will have values more likely to deviate from their true exposure. The distance from the nearest EPA site might also harbor some confounding effects, as this population likely live further from major population centers or differ in ways from the populations who live closer. We also assume two things in terms of exposures: The nearest EPA-air quality site is an adequate measurement of environmental exposure of the place of residency, and that the composition of particulate matter within the state is relatively constant from one monitoring station delineated zone to another.

As well, we assume that the random effects component of our model enough to address multivariate confounders: like variable proportions of SES, Age, Smoking (and other indoor exposures). We operated under similar assumptions to "Statistical Analysis of Repeated Measures data" (Littell 1998). Variables I seek to include in future analysis are temperature/ humidity, variables that are broadly considered significant modifiers of cardiovascular AND respiratory outcomes (Carlsen 2012), and the specific components of our exposure variable, PM2.5. As previously mentioned, the specific components of its composition was not considered state to state, and the chemical profile will likely have differing effects. It is likely incorrect to broadly assume that all PM2.5 has the same effect on the populace merely due to its similar size.

Our design also lends itself to the possibility of selection bias—the inpatient charges utilized were derived from Medicaid only (particularly fee-for-service). Even when restricting my extrapolations and conclusions to a Medicaid only group introduces possible confounding by multivariate factors as well (SES, age distributions), since generally only poor, old, and infants comprise the main beneficiaries of Texas Medicaid/CHIP and the distributions of these people amongst my sites might not be entirely uncorrelated/independent. According to the Kaiser Health foundation, 4.6 million of the 25 MM residents of the state are Medicaid beneficiaries (KFF 2013).

Also, selection bias might arise due to how we allocated EPA sites when we removed certain ones due to missing data. Reallocation of catchment zone borders were caused by dropped EPA-sites that were filtered out due to missing data and sparse measurements (regarding both charge information and environmental) which would've led to excessive imputation and inaccurate models. Hopefully, the individuals that would've been associated with these catchment are adequately represented by the next closest site and new catchment zone we had paired them to. How this potential bias caused by our methodology and the lack of broader population from which to derive charges from, including private and Medicare patients, remains unanswered and require additional analysis.

In response, the next time I might to get a broader pool of charge information, including the private payers and Medicare charges, and have set catchment zones of a predetermined radius, similar to some of the other studies on the subject (Peng 2008), while excluding those subjects who don't live within the minimum distance of one of the EPA measurement sites.

Finally, in terms of exposure assessment, utilizing environmental level measurements as a proxy for individual level exposures lacks a lot of individual exposure information—particularly indoor exposure. We assume that utilizing air monitoring stations are a good proxy for their outdoor exposure, but there is no guarantee that they spend most of their time 1.) in the outdoors 2.) close to their place of residence. Another issue pertinent issue when the nearest EPA air-monitoring site is much further than some patients to their nearest site, leading to some questions of validity.

If I am restricting analysis to hospitals within a certain radius from certain air quality stations, I am excluding a lot of potential data-points and possibly in a biased manner that will skew my results. Another potential source of bias is the presence of specialty hospitals clustering individuals with the respiratory conditions of interest. However, hopefully through randomization I can minimize potential confounding from the multitude of potential modifiers. However, specifics information on measurement equipment for each site were not available, leading to some unknowns as to the precision of the measurements. Despite these issues, I feel there are useful impacts that might be gleaned through a single year cross-sectional relational model.

Conclusions and Future Directions:

With additional data, I hope to adjust for additional confounders from the census as well as adjust for additional exposure, using EPA sites. We can reevaluate the effect of other gasses like SO2 or ground level ozone. As well, utilizing the census information once again, looking at population densities associated with each area code and urban vs rural designations, we might be able to account for the difference in PM2.5 composition. Researchers mention that urban vs rural particulate matter exposure differ widely (Peng 2008). Hopefully with these additional covariates we can improve the accuracy of our estimates and account for more of the variation. There are also indirect burdens that are more difficult to quantify, due to elevated stress and discomfort, and lost productivity. However, being able to quantify the financial and medical burden of this air exposure in hard numbers is a fine start, especially in the light of policy standards for acceptable exposures and when pushing for new public health initiatives.

Also I'd like to establish alternative models: like a moving average, hazard model that might prove useful for medical organizations to forecast burdens to medical systems as a function of climate and air quality. As well, utilizing more years' worth of data I can undertake more model validation steps to determine predictive power and refine the model. More years' worth of data also enables more complex modelling techniques, by allowing more 'seasonal' repetitions, enabling use of an auto-regressive moving average (ARIMA) forecasting model to extrapolate future trends.

Policy makers and environmental regulators should consider the evidence about the associations between cardiovascular/respiratory diseases with air quality when setting environmental standards for industry or evaluating population level health interventions. The immediate costs of upgrading to 'clean-air technology' and other air quality interventions might be offset by the 'hidden burden' of pollution, opening up additional potentially worthwhile investments for the public safety. Though I will be unable to monitor the indirect costs, analyses such as this one can give a glimpse to a slice of the true costs of pollution.

In the meantime, these direct predictions can used in decision-making and policy adjustments, utilized in relation to evaluate costs/morbidities associated with new public-works/manufacturing/energy projects. Some ACOs like Kaiser are interested in utilizing these environmental models to predict rates of asthma, heatstroke, and other CV conditions. With some additional controls, techniques, and data to improve the accuracy of these models, predictive tools like these may be useful when predicting burden on facilities and local staff and during 'spikes' in demand, to prevent short Medical staff shortages and facility overflow; they are not merely limited to higher level policy decisions.

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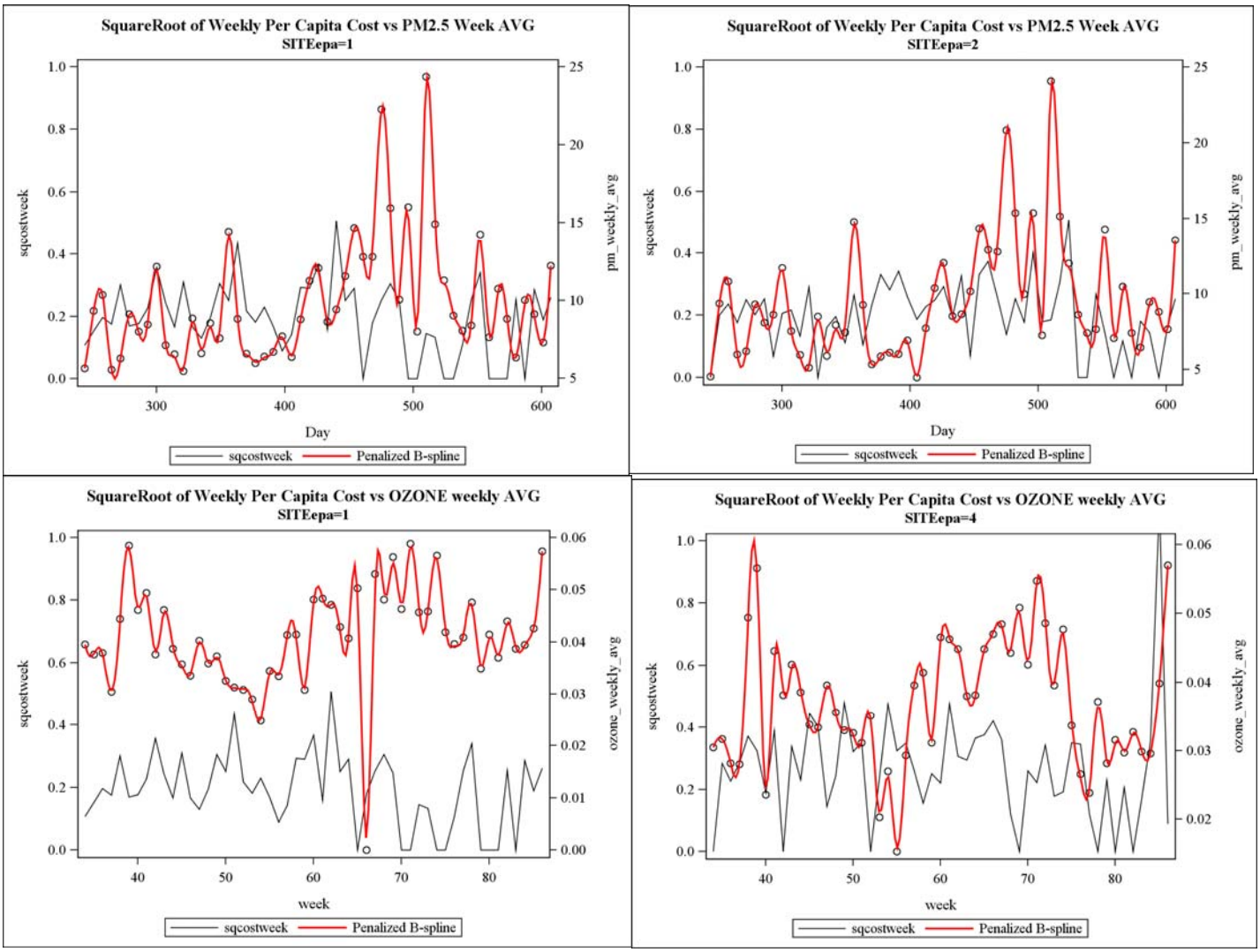
Appendix



Appendix Figure A: Air Quality Measurements against Transformed Spending

Above two rows: Square Root of Weekly Cost per capita (Blue) against pm2.5 weekly averages. Bottom two Rows: same outcome, but against mean ozone readings (red). For briefness, only included sites 1 and 4.