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## **The Effects of Outdoor Air Pollutants on the Costs of Pediatric Asthma Hospitalizations in the United States, 1999-2007**

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## **Abstract**

**Background—**Acute exposure to outdoor air pollutants has been associated with increased pediatric asthma morbidity. However, the impact of sub-chronic exposures is largely unknown.

**Objective—**To examine the association between sub-chronic exposure to six outdoor air pollutants  $(PM_{2.5}, PM_{10}, O_3, NO_2, SO_2, CO)$  and pediatric asthma hospitalization length of stay, charges, and costs.

**Methods—**We linked pediatric asthma hospitalization discharge data from a nationally representative dataset, the 1999-2007 Nationwide Inpatient Sample, with outdoor air pollution data from the Environmental Protection Agency. Hospitals with no air quality data within 10 miles were excluded. Our predictor was the average concentration of six pollutants near the hospital during the month of admission. We conducted bivariate analyses using Spearman correlations and multivariable analyses using Poisson regression for length of stay and linear regression for logtransformed charges and costs, controlling for patient demographics, hospital characteristics, and month of admission.

**Results—**In unadjusted analyses, all six pollutants had minimal correlation with the three outcomes ( $rh \propto 0.1$ , p<0.001). In multivariable analyses, a 1-unit ( $\mu g/m^3$ ) increase in monthly  $PM<sub>2</sub>$ , led to a \$123 increase in charges (95% CI \$40-249) and a \$47 increase in costs (95% CI \$15-93). No other pollutants were significant predictors of charges or costs, or length of stay.

**Conclusion—**Sub-chronic PM<sub>2.5</sub> exposure is associated with increased costs for pediatric asthma hospitalizations. Policy changes to reduce outdoor sub-chronic pollutant exposure may lead to improved asthma outcomes as well as substantial savings in healthcare spending.

## **Background**

Asthma is one of the most common chronic illnesses of children in the United States (US), causing a significant health and economic burden. Morbidity from this disease results in many preventable hospital admissions and considerable use of healthcare dollars.<sup>1-4</sup> Asthma prevalence is highest among children living in inner cities.<sup>5-7</sup> The development and severity of asthma is multi-factorial, but ambient air pollutants are known risk factors.<sup>8,9</sup> The economic burden from air pollutants and asthma is estimated to be substantial,<sup>3</sup> but the only previous analysis relied on expert judgment about attributable burden of disease. No

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epidemiological studies have been conducted to quantify costs attributable to individual pollutants.

In the US, air pollutant levels are regulated by the Environmental Protection Agency (EPA), which has set standards for six criteria pollutants, including particulate matter, ozone  $(O_3)$ , nitrogen oxides ( $NO<sub>2</sub>$ ), sulfur oxides ( $SO<sub>2</sub>$ ), carbon monoxide (CO), and lead. Particulate matter can be further classified into aerodynamic diameter  $10\mu$ m (PM<sub>10</sub>) or 2.5 $\mu$ m  $(PM_{2.5})$ .

Increasing evidence supports the association between air pollutants and asthma morbidity, including worsening asthma symptoms,  $10-13$  increased emergency department visits,  $14-18$ and decreased lung function.<sup>19-21</sup> This evidence is primarily from studies of exposures in limited geographic areas,  $10, 17, 20, 22-23$  although some national multi-city studies exist as well.<sup>19, 24-26</sup> Of the six EPA criteria pollutants,  $PM_2$ , and  $O_3$  have most often been associated with asthma morbidity.10, 12, 13, 27-29

The existing literature on air pollutants and asthma has primarily focused on acute exposures; data regarding more chronic exposures are limited.<sup>22,24</sup> In general, exposure duration of less than 14 days is acute, more than 14 days up to one year is sub-chronic, and greater than one year is chronic.<sup>30</sup> Higher chronic ozone exposure is associated with increased risk of asthma hospitalization, $22$  and higher chronic exposure to both ozone and particulate matter is associated with increased risk of current asthma and symptom exacerbation.<sup>24</sup> Both sub-chronic and chronic PM<sub>2.5</sub> exposure have been associated with increased risk of hospitalization for infant bronchiolitis, another pediatric respiratory illness.<sup>31</sup>

The objective of this exploratory study was to examine the association between sub-chronic exposure to air pollutants and length of stay, total charges, and total costs for pediatric asthma hospitalizations. To accomplish this, we linked two nationally representative datasets: the Nationwide Inpatient Sample (NIS), an annual survey of hospital discharge data, and the Environmental Protection Agency (EPA) Aerometric Information Retrieval System (AIRS), containing US air pollutant data.

The NIS allows quantification of hospitalization costs, an important step towards evaluating the economic impact made by air pollutants on pediatric asthma. Patient data in the NIS is de-identified, and the dataset contains no information on individual residence. Therefore, merging with the EPA air pollutant data occurs at the level of the hospital, not the patient. Although this is an important deficiency to consider, the NIS represents the largest dataset available to measure increases in asthma hospitalization costs associated with air pollutant levels. We therefore proceeded to perform descriptive, bivariate, and multivariable analysis of the 1999-2007 NIS to quantify the economic impact of air pollutants on pediatric asthma hospitalizations.

## **Methods**

## **Study Design**

This was a multi-year cross-sectional study, including hospitalizations between 1999 and 2007 for children ages 2-17 with a primary diagnosis of asthma (ICD-9 code 493.XX). Children under 2 were excluded because wheezing at this age is often transient and diagnosis of asthma is difficult.<sup>32</sup> Hospitalizations with no corresponding air pollutant data were excluded. Weights accounting for sampling design were included in the NIS; these weights were incorporated into all analyses except descriptive pollutant data. Analyses were conducted using SAS-callable SUDAAN software version 10.0.1 (Research Triangle

Institute, NC) to take into account the complex sampling design used by NIS. This research involved previously collected, de-identified data and was exempt from review by the Institutional Review Board of Mount Sinai School of Medicine.

#### **Databases**

The NIS is the largest all-payer inpatient care database in the US, containing data from approximately 8 million hospitalizations annually.33 Details regarding sample design, data collection and weighting are described elsewhere.<sup>33</sup> The survey includes diagnostic codes and basic patient demographics, as well as length of stay and total hospital charges.

The EPA AIRS contains pollutant data recorded at defined intervals, ranging from hourly to every few days,<sup>34</sup> available to the public for download.<sup>35</sup> We obtained text files for six air pollutants (PM<sub>2.5</sub>, PM<sub>10</sub>, O<sub>3</sub>, NO<sub>2</sub>, SO<sub>2</sub>, CO) from 1999 through 2007 to merge with the NIS hospitalization data. To assess for any systematic missing data, we compared the number of measurements available per month, day and hour for each pollutant for each year to the total number of measurements possible; we observed no systematic patterns of missing data.

#### **Main outcomes: hospitalization data**

Outcomes in this study included length of stay, total charges, and total costs for pediatric asthma hospitalizations. The length of stay was provided in days. Total charges represent the amount billed for each hospitalization. Charges were controlled for inflation by adjusting to 2005 dollars using the Healthcare Consumer Price Index from the Bureau of Labor.<sup>36</sup> Costs represent the amount of money actually paid to the hospital, generally significantly less than the charges. To estimate costs, charges were converted using hospital-specific cost-to-charge ratio files in a dataset accompanying the NIS. These cost-to-charge ratio files are constructed using all-payer inpatient cost and charge information from the Centers for Medicare and Medicaid Services.<sup>37</sup>

Cost-to-charge ratios were not available for data from 1999–2000; these years were excluded when analyzing cost. Overall, many hospitals did not have cost-to-charge ratios available, and therefore cost analyses represent only a fraction of all hospitalizations; also, as explained above, costs are only an estimate based on a ratio assigned to the hospital overall and not specific to the visit. The majority of our analyses were conducted to explore the nature of the relationship between pollutants and healthcare spending. For these analyses, we used charges as the outcome, allowing a greater number of hospitalizations to be included and more accuracy in our results. However, for analyses used to estimate potential healthcare savings in real healthcare dollars, we used costs as the outcome; charges as the outcome for this purpose would over-represent any potential savings.

#### **Main predictors: air pollutants**

The predictors of interest were average monthly air pollutant levels. Because the NIS does not contain identifying information about patient residence, air pollutant levels were determined for a defined area surrounding the hospital.

For all NIS hospitals with available addresses, we determined the latitude and longitude, which were verified using a geocoding website<sup>38</sup> and Google maps. The latitude and longitude for the AIRS monitors are available on a publicly available website.<sup>39</sup> We determined the distance between NIS hospitals and AIRS monitors using the following equation, calculating the hypotenuse of a right triangle formed by the two locations: D=  $\cos^{-1}(\sin(\text{lat1})^*)\sin(\text{lat2})+\cos(\text{lat1})^* \cos(\text{lat2})^* \cos(\text{lon2}-\text{lon1}))^* 6371^* 0.62137$  where D is distance in miles between a hospital and a monitor, lat1 and lat2 are latitudes of first and second locations respectively and lon1 and lon2 are longitudes of those locations.

To calculate the monthly average air pollutant levels, we averaged data from all monitors located within 10 miles of the hospital. We then linked the NIS hospital data to average monthly levels for all six pollutants. Two-month averages (which included the month of admission as well as the month prior) as well as three-, four-, and five-month averages were also calculated and linked to the hospital data.

## **Statistical Analyses**

We used Spearman correlations to examine the unadjusted association between all six pollutants and the three outcomes: length of stay, charges, and costs. Recognizing that pollutants arising from similar sources (e.g. motor vehicles) can be highly correlated, 40,41 we performed correlations among the pollutants to determine which ones to include in multivariable analyses. Any two pollutants with  $rho$  absolute value of 0.3 were not included together in multivariable models, and we prioritized the inclusion of  $PM<sub>2.5</sub>$  and  $O<sub>3</sub>$ , the pollutants with the strongest evidence linked to adverse asthma outcomes.

Early in our analysis, we found an extremely narrow distribution of LOS among pediatric asthma hospitalizations, which are coded as ordinal numbers of days rather than hours. With the caveat that the limited distribution of LOS might make detection of pollutant effects unlikely, we used Poisson regression to account for the skewed distribution of the variable. For total charges and costs, we  $log_{10}$ -transformed the variables. We included air pollutants in linear regression models based on results of correlation analysis. We also included covariates associated with asthma outcomes and/or hospital charges: age, race (white, black, Hispanic, or other), gender, income, insurance (Medicare, Medicaid, private, self-pay, no charge, or other), hospital region of the country (Northeast, South, Midwest, or West), teaching status of the hospital, and month of admission. We used the median household income quartile for patient's zip code as a proxy for income.

Any pollutant found to be a significant predictor was also categorized by quartiles to examine the effects at different levels of exposure. For air pollutants found to be significant predictors, we ran stratified models for different seasons and different age groups (2-5 years, 6-12 years, 13-17 years). For the linear regressions, we retransformed the results from  $log_{10}$ to the original scale using the smearing factor technique described by Duan.<sup>42</sup>

## **The Effect of Chronicity**

For pollutants that were significant predictors of hospitalization charges, we examined the effect of increased exposure chronicity on outcomes. We built linear regression models with  $\log_{10}$  charges as the outcome and average PM<sub>2.5</sub> levels over differing lengths of exposure (2-5 months) as the predictor in four separate models. The strength of the association between  $PM_{2.5}$  levels of increasing duration and  $log_{10}$  total charges were compared.

#### **Appendicitis Outcomes**

To validate findings between air pollutants and asthma outcomes, we ran similar multivariable models for appendicitis, a frequent cause of pediatric hospitalization that should have no association with change in air pollutant levels. We hypothesized that there would be no significant relationships found.

## **Results**

Between 1999 and 2007, there were 70,052,217 hospital admissions in the NIS database; 206,562 were for children ages 2-17 with a primary diagnosis of asthma. Hospital admissions with no corresponding air pollutant data were excluded for a final sample of 66,256 hospitalizations. Compared to all pediatric asthma hospitalizations, discharges included in the analysis had more patients who were black or Hispanic, were more likely to be in teaching hospitals, almost exclusively located in urban settings, and less likely to be located in the South (Table 1). These findings reflect the fact that air pollutant monitors are primarily located in urban areas, where air pollutant levels are highest. Race was unreported for a large percentage of patients as many states do not report this data to  $NIS$ .<sup>43,44</sup>

The mean total charges for pediatric asthma hospitalizations was \$7,341, with a maximum charge of \$994,726. The median charges and interquartile range were \$4972 and \$5227 respectively. The mean total cost was \$3,235, with a maximum cost of \$441,001. The median costs and interquartile range were \$2338 and \$2147 respectively. The descriptive data for air pollutant levels are shown in Table 2, including the current EPA criteria.

In unadjusted analyses, all six pollutants had minimal correlation with the three outcomes: LOS, total charges, and total costs ( $rh\infty$ 0.1 and  $p<0.001$  for all analyses). Among the different air pollutants,  $PM_{2.5}$  and  $PM_{10}$  were most highly correlated (*rho*=0.5). SO<sub>2</sub> and CO were negatively correlated with  $O_3$  (*rho*= −0.4), and PM<sub>2.5</sub> and CO were positively correlated (*rho*=0.4). As mentioned above, we prioritized PM<sub>2.5</sub> and O<sub>3</sub> for inclusion in final models because of known relationships with asthma morbidity. Therefore,  $PM_{2.5}$ ,  $O_3$ , and NO2 were further examined in three-pollutant multivariable analyses.

In multivariable analyses, none of the three pollutants were significant predictors of LOS (Table 3). However,  $PM_{2.5}$  remained a significant predictor of both charges and costs. A 1unit ( $\mu$ g/m<sup>3</sup>) increase in monthly PM<sub>2.5</sub> led to a \$123 increase in charges (95% CI: +\$40 to +\$249) and a \$47 increase in costs (95% CI: +\$15 to +\$93). O<sub>3</sub> (+\$4797, 95% CI: -\$10,111 to  $+$ \$6,463,445) and NO<sub>2</sub> ( $+$ \$88, 95% CI:  $-$ \$162 to  $+$ \$483) were not significant predictors of charges. Other significant predictors of charges included older age, female gender, and hospital location in the west (p<0.0001 for all associations).

When the model for costs was run with  $PM_{2,5}$  as a variable categorized by quartile, the lowest quartile of exposure led to an average decrease of \$918 (95% CI: +\$559 to +\$1105) in costs compared to the highest quartile of exposure. Stratified models examining  $PM<sub>2.5</sub>$ and total charges showed evidence of effect modification by season and age (Table 4). The relationship was strongest in the summer and weakest in the winter. The oldest age group had the strongest association between PM<sub>2.5</sub> and charges (+\$240, 95% CI: +\$29 to +\$658).

## **The Effect of Chronicity**

When increasing durations of  $PM<sub>2.5</sub>$  exposure were used as the predictor in the linear regression model for asthma total charges, the relationship between  $PM_{2.5}$  and charges continued to become stronger and more significant, with average  $PM_{2.5}$  during the 5 months preceding hospitalization having the strongest association (+\$222, 95% CI: +\$100 to +\$404, Figure 1).

#### **Appendicitis Outcomes**

Linear regression models with the outcomes of total charges and costs for appendicitis hospitalizations including the same predictors as in the other models did not show a significant association with  $PM<sub>2.5</sub>$ . Although some variables were significant predictors, demonstrating the potential variability in appendicitis charges,  $PM_{2.5}$  was not significantly associated with the outcome, contrary to the findings with asthma hospitalizations.

## **Discussion**

In this national study, we found a significant association between average  $PM_{2.5}$  levels around hospitals and pediatric asthma hospitalization total charges and costs, but not LOS. The relationship was found to be the strongest in the summer and among older children. The association was stronger with an increasing chronicity of exposure. We found no association between PM<sub>2.5</sub> and appendicitis outcomes.

Substantial health care dollars are spent each year on asthma, $1,3$  and controlling levels of PM<sub>2.5</sub> may result in considerable savings. We found that a 1-unit increase in PM<sub>2.5</sub> increased pediatric asthma hospitalization costs on average by \$47. There are approximately 200,000 pediatric asthma hospitalizations in the US annually, about 85% of which are in urban areas.45 Extrapolating our findings to all urban pediatric asthma hospitalizations, reducing the average level of  $PM<sub>2.5</sub>$  by 1 unit could save about \$8 million annually. We also found that decreasing  $PM<sub>2.5</sub>$  from the highest to lowest quartile decreased costs on average by \$918 per hospitalization. Extrapolating this to the 25% of urban hospitals in the highest quartile, decreasing  $PM<sub>2.5</sub>$  to the lowest quartile could save about \$39 million. This amount represents only a small fraction of potential healthcare savings resulting from reduced PM<sub>2.5</sub> levels, especially given other well-known adverse health outcomes associated with this pollutant, such as cardiac mortality.46,47 These figures are also conservative as they represent total costs, not charges.

Interestingly, increased charges were not found with other air pollutants, such as  $O_3$ , known to have adverse effects on asthma outcomes. One explanation is that, as shown in previous studies, ambient  $O_3$  levels measured at central monitoring sites may not be representative of personal exposure, in contrast to  $PM_{2.5}$  for which ambient levels are highly correlated with personal exposure.48-50 Inaccurate represention of personal exposure biases the results towards the null; perhaps this is why no relationship is seen with other pollutants. Another potential explanation is that the effects of sub-chronic exposure to  $O_3$  are different than acute exposure. Although the acute effects of  $O_3$  on asthma are well known, sub-chronic exposure to  $O_3$  may not play a significant role in adverse asthma outcomes, at least not enough to translate into increased hospitalization costs and charges.

We hypothesized that higher  $PM_{2.5}$  levels lead to increased asthma severity and thus increased charges, in part mediated by increased LOS. However, we found no significant association with LOS. Because LOS is measured in units of whole days, it would take quite a significant increase in severity to result in another full day of hospitalization and as mentioned above, the narrow distribution of LOS limited our analytic capacity.

The associations between charges and  $PM<sub>2.5</sub>$  showed effect modification by season and age, with results most significant for summer months and older children. Previous studies have shown that ambient  $PM<sub>2.5</sub>$  concentrations are most highly correlated to personal exposure during summer months when people spend the most time outdoors.<sup>48</sup> Older children also tend to spend more time outdoors. In both cases, the stronger associations found may be because personal exposure is being more closely approximated by ambient  $PM_{2.5}$  levels during warmer months and in older children. Although asthma severity and overall hospitalization charges may be highest in the winter,  $PM<sub>2.5</sub>$  levels may not be as strongly associated with charges because children have relatively lower exposure compared to warmer seasons.

There is little known about sub-chronic effects of  $PM_{2.5}$  on asthma outcomes. We found that the chronicity of PM<sub>2.5</sub> exposure played an important role in the association with costs and charges: an increasing duration of exposure showed stronger and more significant associations. That is, long-term exposure to  $PM_{2.5}$  had more of an effect than short-term exposure. These results are supported by a recent study showing that children with higher exposure to  $PM_{2.5}$  for one year were more likely to be diagnosed with asthma and have asthma attacks.<sup>24</sup> One plausible explanation is that similar to its role in cardiac mortality,<sup>46</sup>  $PM<sub>2.5</sub>$  leads to increased inflammation over time and consequently increased severity when children have asthma attacks. Our findings raise some interesting questions and highlight the importance of continued research into the associations between sub-chronic  $PM_{2.5}$  exposure and asthma morbidity.

An important limitation of this study is that the exposure measurement is crude. We have used ambient pollutant concentrations near the hospitals because information about personal exposures is unavailable. Prior studies have shown that ambient  $PM_{2.5}$  levels measured at central monitoring sites are highly correlated with average personal  $PM_{2.5}$  exposure. <sup>48</sup> In contrast, ambient  $O_3$ ,  $NO_2$ , and  $SO_2$  concentrations are weakly correlated with personal exposures to these gases.<sup>48-50</sup> This supports the fact that our  $PM_{2.5}$  exposure measure represents personal exposure fairly accurately, provided that the majority of patients live in the vicinity of the hospital. There are inevitably some incorrect exposure assessments; however, these misclassifications would tend to bias the results towards the null, and we found clear and consistent relationships with  $PM_{2.5}$  in spite of these misclassifications. The lack of association with the other pollutants may be due to incorrect exposure assessment. An alternate explanation, as mentioned above, is that sub-chronic exposure to the other pollutants may not play a role in asthma severity.

It is important to note that all four regions of the country were not equally represented. Although the South represented 38% of all hospitalizations, but only 6% of included hospitalizations. Because fewer hospitalizations in the South occur in urban areas  $(75%)$  compared with other regions  $(>=85%)$ , a greater proportion did not have corresponding pollutant data, limiting the sample of Southern hospitalizations compared with other regions. Correspondingly, the Northeast and West are over-represented in our analyses.

Another limitation is that the NIS does not contain details regarding management during hospitalization, e.g. medications used and intensive care. As discussed above, the proposed mechanism of the association is increased asthma severity resulting in increased use of hospital resources. Without detailed information about hospital stays, we are unable to examine the specific etiologies of the increased charges.

An additional potential limitation is residual confounding by socioeconomic status. It is known that asthma severity is worse in urban, inner-city areas, which are also the areas where  $PM_{2.5}$  levels tend to be the highest. In multivariable analyses, we did our best to control for income and insurance as markers of socioeconomic status. Also, urban areas are where other air pollutant levels are highest, and no associations were found with pollutants besides  $PM<sub>2.5</sub>$ . The clear and consistent associations found between increasing durations of PM<sub>2.5</sub> and hospital charges make it likely that the relationships seen are specific to PM<sub>2.5</sub> and not due to confounding by socioeconomic status.

A major strength is that our results are based on a large amount of data nationally representative of urban area hospitals. Although these results cannot be translated to nonurban areas, as stated above, 85% of pediatric asthma hospitalizations do occur in urban areas.45 Another important strength is that secondary analysis examining the relationship

between  $PM_{2.5}$  and appendicitis hospitalization charges showed no significant association, validating our results that the association is for a diagnosis with a biologically plausible connection with  $PM_{2.5}$  and not a spurious finding. We also have results in costs, not just charges. It is a strength that our study provides results in actual healthcare dollars spent.

Our results should be interpreted with caution because the exposure assessment is inherently with limitations. Other studies have employed more complex methods to assess personal exposure to air pollutants, such as modeling multiple point source data into an aggregate exposure metric. However, no other studies have examined healthcare costs associated with specific air pollutants or the health effects of sub-chronic air pollutant exposure on pediatric asthma – two important areas of future research. In spite of the deficiencies noted, our findings merit further study using more rigorous exposure assessment and datasets containing detailed information regarding healthcare utilization.

Sub-chronic  $PM<sub>2.5</sub>$  exposure is associated with increased costs for pediatric asthma hospitalizations. Studies such as this one should stimulate further research in this area and incite regulatory agencies such as the EPA to consider sub-chronic pollutant levels when setting standards for air pollutants. Our results provide economic data to reinforce the need for ongoing efforts to reduce levels of air pollutants in this country. Policy changes to improve air quality may lead to improved asthma outcomes as well as substantial savings in healthcare spending.

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## **Figure 1.**

Total charges and costs of pediatrics asthma hospitalizations for increasing durations of particulate matter exposure.  $PM_{2.5}$  is particulate matter with diameter less than 2.5 micrometers.

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## **Table 1 Characteristics of Patients and Hospitals among Pediatric Asthma Hospitalizations in the Nationwide Inpatient Sample, 1999-2007**



1 Hospitalizations with data available for all six pollutants were included in bivariate and multivariable analyses

Table 2<br>**Pollutant levels corresponding to Pediatric Asthma Hospitalizations from the Nationwide Inpatient Sample 1999-2007 and United States Air Pollutant levels corresponding to Pediatric Asthma Hospitalizations from the Nationwide Inpatient Sample 1999-2007 and United States Air Quality Standards Quality Standards**



I<br>abbreviations: abbreviations:

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PM2.5=particulate matter of diameter<2.5microns; PM2.5=particulate matter of diameter<2.5microns;

PM10=particulate matter of diameter<10microns; PM10=particulate matter of diameter<10microns;

ppm=parts per million ppm=parts per million  $2_{\rm http://www.epa.gov/air/criterion.html}$ <http://www.epa.gov/air/criteria.html>

 $^3$  To attain this standard, the 3-year average of the weighted annual mean PM2.5 concentrations from single or multiple monitors must not exceed 15.0  $\mu$ g/m<sup>3</sup>  $\tau_{\rm D}$  attain this standard, the 3-year average of the weighted annual mean PM2.5 concentrations from single or multiple monitors must not exceed 15.0 μg/m<sup>3</sup>

 $^{4}$ To attain this standard, the 3-year average of the 98<sup>th</sup> percentile of 24-hour concentrations at each monitor within an area must not exceed 35 µg/ m<sup>3</sup> (effective December 17, 2006) To attain this standard, the 3-year average of the 98<sup>th</sup> percentile of 24-hour concentrations at each monitor within an area must not exceed 35 μg/ m<sup>3</sup> (effective December 17, 2006)

 $5$  ot to be exceeded more than once per year on average over  $3$  years Not to be exceeded more than once per year on average over 3 years

 $\sigma_{\rm D}$  attain this standard, the 3-year average of the fourth-highest daily maximum 8-hour average ozone concentrations measured at each monitor within an area over each year must not exceed 0.075 ppm To attain this standard, the 3-year average of the fourth-highest daily maximum 8-hour average ozone concentrations measured at each monitor within an area over each year must not exceed 0.075 ppm (effective May 27, 2008) (effective May 27, 2008)

The standard is attained when the expected number of days per calendar year with maximum hourly average concentrations above  $0.12$  ppm is The standard is attained when the expected number of days per calendar year with maximum hourly average concentrations above 0.12 ppm is <sup>1</sup>

 ${}^2$ To attain this standard, the 3-year average of the 98th percentile of the daily maximum 1-hour average at each monitor within an area must not exceed 0.1ppm or 100 ppb (effective January 22, 2010) To attain this standard, the 3-year average of the 98th percentile of the daily maximum 1-hour average at each monitor within an area must not exceed 0.1ppm or 100 ppb (effective January 22, 2010)

 $9$  Not to be exceeded more than once per year Not to be exceeded more than once per year

 $10<sub>T</sub>$  attain this standard, the 3-year average of the 99th percentile of the daily maximum 1-hour average at each monitor within an area must not exceed 0.075 ppm or 75 ppb (effective June 2, 2010)  $^{10}$ To attain this standard, the 3-year average of the 99th percentile of the daily maximum 1-hour average at each monitor within an area must not exceed 0.075 ppm or 75 ppb (effective June 2, 2010)

## **Table 3 Multivariable results for Pediatric Asthma Hospitalization Length of Stay, Charges, and Costs as predicted by Changes in Levels of Fine Particulate Matter***<sup>1</sup>*



Increment changes for 1-unit (ug/m3) increase in particulate matter of diameter <2.5ug (PM2.5)

<sup>2</sup> Average level of PM<sub>2.5</sub> within a 10-mile radius of the hospital

3 Increment percent change calculated from exponentiating Poisson beta coefficient

4 Increment change in dollars calculated using Duan transformation for linear regression

p-value 0.1

\*

\*\* $p$ -value  $0.001$ 

\*\*\*p-value 0.0001

5 avg=average

#### **Table 4**

## **Pediatric Asthma Hospitalization Charges in Association with Fine Particulate Matter Levels, Stratified by Age and Season**



<sup>1</sup>Increment change in hospitalization charges for 1-unit (ug/m3) increase in particulate matter of diameter <2.5ug (PM<sub>2.5</sub>) calculated using Duan transformation for linear regression

\* p-value<0.05

\*\* p-value<0.005

\*\*\*p-value<0.0005