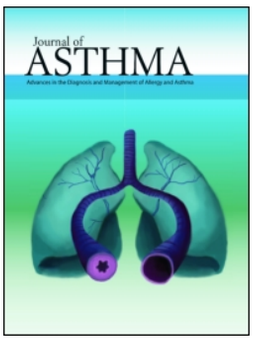

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RUNNING TITLE: Asthma in Children Residing Near Air Pollution Sites

Asthma Prevalence and Control among Schoolchildren Residing near Outdoor Air Pollution Sites

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ABSTRACT:

Introduction: Outdoor air pollution (OAP) contributes to poor asthma outcomes and remains a public health concern in Pittsburgh. The purpose of this study was to determine the prevalence of childhood asthma and its rate of control among Pittsburgh schoolchildren residing near OAP sites.

Methods: Participants were recruited from schools near OAP sites. Asthma prevalence and control were assessed using a validated survey. Demographics and socioeconomic status were collected by survey, BMI was calculated, second-hand smoke (SHS) exposure was assessed by salivary cotinine levels, and OAP was assessed by mobile platform monitoring. Multivariate analysis adjusted for confounders.

Results: In 1,202 Pittsburgh elementary school students surveyed, 50.9% were female, average age was 8.5 years (SD=1.9), 52.2% were African American and 60.6% had public health insurance. SHS exposure was relatively high at 33.9%, 17.1% of students were obese, and 70% had exposure to particulate matter ($PM_{2.5}$) greater than the World Health Organization (WHO) standard of 10 $\mu g/m^3$. Overall prevalence of asthma was 22.5% with $PM_{2.5}$, nitric oxide (NO_x), sulfur (S) and zinc (Zn) significantly related to odds of asthma. Among the 270 children previously diagnosed with asthma, 59.3% were not well controlled with $PM_{2.5}$, black carbon (BC), and silicon (Si) significantly related to odds of uncontrolled asthma.

Conclusions: These results demonstrate that asthma prevalence and poor disease control are significantly elevated in Pittsburgh schoolchildren exposed to high levels of OAP. Future efforts need to focus on primary prevention of asthma by reducing exposure to OAP in at risk populations.

Keywords: Epidemiology, Pediatrics, Morbidity and Mortality

ABBREVIATIONS:

OAP – outdoor air pollution

AAFA- Asthma and Allergy Foundation of America

ACHD – Allegheny County Health Department

$PM_{2.5}$ - particulate matter less than 2.5 microns in diameter

WHO – World Health Organization

NO_x – nitrogen oxides

S- sulfur

Zn – zinc

BC – black carbon

Si – Silica

BMI -body mass index

CDC- Centers for Disease Control

K – potassium

Cr – chromium

Fe – iron

LUR – land use regression

EPA – Environmental Protection Agency

INTRODUCTION:

Asthma is the leading chronic disease among children and affects approximately 6.2 million US children.(1, 2) The annual cost of asthma in the US is estimated to be approximately 82 billion dollars.(3) Poor outcomes persist despite access to care and improved treatments, and include 14.2 million outpatient visits, 1.8 million emergency department visits, and 439,000 hospitalizations annually.(4) Additionally, childhood asthma accounts for approximately 14 million missed school days annually and impacts on learning, academic achievement, participation in physical activity and peer interactions.(2) Demographic factors associated with poor childhood asthma outcomes include gender, race and poverty.(5) Environmental factors include, but are not limited to, respiratory infections, exposure to allergens, outdoor air pollution (OAP) and second-hand smoke (SHS), obesity, nutrition and psychological stress.(5)

Pittsburgh is a challenging city in which to live with asthma due to both the high disease prevalence and exposure to high levels of outdoor air pollution. Each year, the Asthma and Allergy Foundation of America (AAFA) releases an asthma capital report which looks at factors in cities across the US that contribute to asthma rates and disease management. They rank the cities based on asthma prevalence, emergency department visits and mortality (deaths). They also look at risk factors that contribute to these outcomes, including poverty, exposure to air pollution, access to asthma specialists, pollen counts, asthma medicine use, tobacco policies and the rate of uninsured residents. In 2019, AAFA ranked Pittsburgh as the 54th (out of 100) overall worst US city in which to live with asthma (6). Pittsburgh's rank was based on its high prevalence of asthma. Additionally, it ranked 6th worst among the 100 cities for exposure to high levels of outdoor air pollution. Similarly, the American Lung Association State of the Air 2020 report gave Pittsburgh a failing grade for both 24 hour short-term and annual mean PM2.5 exposure levels, and ranked the city as the 8th (out of 204) and the 16th (out of 216) worst for annual mean and short-term PM2.5 exposure, respectively (7).

Pittsburgh is in Allegheny County and the Allegheny County Health Department (ACHD) recently reported that an overall prevalence of adult asthma of 15.1%, which is much higher than the reported national and state rates of 9-10% (8). ACHD's report cited higher asthma prevalence among minority adults and adults with less education and lower socioeconomic status. Additionally, ACHD reported an asthma prevalence of 22.1% among 1,609 county teenagers they surveyed in 2014 (9). However, ACHD did not report on asthma prevalence in younger children. Additionally, ACHD did not report on the rate of poor asthma control or the impact of local exposure to high levels of air pollution on asthma prevalence and disease control. Consequently, the objectives of this study were to the prevalence of asthma and its rate of control among Pittsburgh elementary schoolchildren residing near point sources of outdoor air pollution.

METHODS:

Overview:

This study was approved by the Institutional Review Board at Allegheny General Hospital and informed assent/consent was obtained prior to participation. The study population was recruited from local elementary school students residing near point sources of outdoor air pollution in Allegheny County, Pennsylvania from January 2014 through December 2016. Parents/caregivers completed a written survey that included address, age, gender, race, and type of health insurance, where public health insurance was used as a marker of lower socioeconomic status. They also completed a written validated asthma detection survey.(10) At school visits, heights and weights were measured for the calculation of body mass index (BMI), and saliva samples were collected for assessment of SHS exposure.(11, 12) Linear distance of each participant's residence from the nearest OAP site was calculated and each residence was classified as being not in the path, partially in the path or fully in the path of the prevailing wind direction from the nearest OAP site. Annual mean levels of OAP were assessed using a LUR model developed from mobile air sampling data.(13)

Study Population:

Fifteen schools agreed to participate. In thirteen of these schools, administrators contacted the principal investigator to participate following dissemination of an IRB approved press release regarding this study. All thirteen chose to participate because of the proximity of their buildings to OAP sites. In the remaining two schools, the principal investigator contacted the administrators to ascertain their interest due to the proximity of their buildings to OAP sites. Total enrollment at participating schools was 2,488; 1,207 (42.4%) provided informed consent/ascent; and 1,202 completed the asthma survey. OAP sites included US Steel Edgar Thompson Works in Braddock (10

schools; n=773), DTE Energy Shenango Coke Works (2 schools; n=146), US Steel Clairton Cokes Works (1 school; n=212), NRG Cheswick Generating Station (1 school; n=24), and the Monroeville Pennsylvania Turnpike Junction (1 school; n=47).

Validated asthma detection survey:

The pediatric asthma survey included four questions focused on the following areas: prior physician diagnosed asthma; repeated asthma episodes in the past year; frequency of cough, chest tightness, trouble breathing and/or wheezing during play/exercise (rarely, never, sometimes, often, or most of the time); and frequency of these symptoms during the morning/daytime in the past 4 weeks (never, <2 days/week, ≥2 days/week but not every day, every day, more than once a day on most days). Estimates of asthma prevalence were based on reports of physician diagnosed asthma; and in those reporting physician diagnosed asthma, uncontrolled disease was defined by repeated episodes of asthma and/or exercise/play limitations often or most of the time and/or daytime symptoms ≥2 days/week. The scoring algorithm identifies the presence of asthma with a sensitivity of 83.4% and a specificity of 85.4%.(10)

BMI:

Heights and weights were collected using a portable stadiometer (model #213; SECA, Chino, CA) and a Fit Scan body fat/body water monitor (model #BF-679F; Tanita, Arlington Heights, IL), respectively. Measurers were trained in standard techniques and were blinded to the clinical status of participants. The Centers for Disease Control (CDC) calculator was used to calculate the BMI-for-age percentile.(14) Obesity was defined as a BMI at or above the 95th percentile for children of the same age and sex.

Second Hand Smoke (SHS) Exposure:

Saliva was collected using Salivette (Sartedt, Numbretch, Germany) which consists of a polypropylene tube with synthetic swab. For saliva collection, the patient removed the swab from the polypropylene tube, placed it in their mouth and chewed it for about 60 seconds to stimulate salivation. The swab with the absorbed saliva was then returned to the polypropylene tube, capped immediately, and stored on ice until frozen at -20°C within 4 hours of collection. At a later date, saliva samples were then thawed and batch assayed in duplicate for cotinine concentrations using a quantitative enzyme immunoassay kit (Salimetrics LLC, State College, Pennsylvania).(12) The technician performing the assay was blinded to the clinical status of subjects. The lower limit of detection of this assay was 0.15 ng/ml. The intra-assay and inter-assay coefficient of variation were 6.4% and 6.6%, respectively. Subjects were classified as being exposed to SHS if their salivary cotinine level was ≥1.00 ng/ml.

Relationship between Residences and Sources of Pollution:

A technician blinded to each participant's clinical status used www.LatLong.net and www.SunEarthTools.com to calculate the linear distance and bearing of each participant's residence from the nearest OAP site. The bearing was then used to classify each residence as being directly (northeast to east southeast), partially (east southeast to south and north northwest to northeast), or not in the path (south to north northwest) of the nearest OAP site based on the prevailing (west to west southwest) wind direction.(15)

Exposure to Outdoor Air Pollution (OAP):

Annual mean levels of OAP exposure were estimated at the home address of each participant. Pollutants included nitrogen oxides (NO_x), fine particulate matter ($PM_{2.5}$), and the following $PM_{2.5}$ components: black carbon (BC), potassium (K), sulfur (S), chromium (Cr), iron (Fe), silicon (Si), and zinc (Zn). NO_x and $PM_{2.5}$ have well-known associations with asthma and other respiratory conditions. The $PM_{2.5}$ components were selected because they are markers of sources such as vehicular emissions (BC, Fe), electricity generating power plants (S), industrial emissions (Fe, Zn, Cr), dust (Si), and biomass burning (K).

Data collection and spatial modeling were previously described in Li et al, and briefly summarized here.(13) OAP concentrations were measured at 70 sites in Allegheny County, Pennsylvania. Measurements were performed in three periods in both summer and winter seasons to cover different times of day: mornings (5am-11am), afternoons/evenings (11am-9pm), and overnight (9pm-5am). Each site was visited six times, with each visit lasting one hour. The OAP measurements collected at each site were used to develop land use regression (LUR) models. LURs were fit to the mean concentration across all visits to each site. We used ArcGIS-10.3 (ESRI, Redlands, CA) for geospatial analysis. Land use data was collected from several sources, including the United States Department of Transportation, United States Geological Survey, the state of Pennsylvania, Allegheny County, and the Environmental Protection Agency National Emissions Inventory. LUR models were built using forward stepwise regression where each land use variable was assigned an a priori direction (e.g., higher traffic should produce higher pollutant concentrations). Predictors were added to the model if they conformed to the assumed direction and increased the adjusted R^2 by at least 1%.

Statistical Methods:

The objectives of this study were to assess the impact of environmental exposures on asthma prevalence as well as uncontrolled disease in those with asthma. Demographic characteristics (age, gender, race, and socioeconomic status) and environmental exposures (obesity, SHS and OAP) were described in relation to prevalence of asthma and uncontrolled disease. Differences in prevalence by factor levels were assessed for significance using Chi-square test. Logistic regression analyses determined the odds of asthma and uncontrolled disease across exposure variables. Forward logistic regression analyses then determined the combination of environmental exposures that increased the odds of asthma with demographic characteristics factored into the model. Variables were stepped into the model in order of significance in the unadjusted analyses and retained when 0.05 level of significance was achieved. To ensure the best fitting curvature, linear and squared terms for each exposure were assessed as well as three-point categorical variables based on interquartile range for the respective distribution (lower 25^{th}, mid-range $26\text{-}75^{th}$, and upper 75^{th} percentile of distribution). Exception, $PM_{2.5}$ was positively skewed and examined only on the categorical scale that considered the EPA annual mean threshold of 12.0 $\mu g/m^3$ and the WHO annual mean standard of 10 $\mu g/m^3$ (<10, 10-11.9, and ≥12).(16, 17) These methods were used to determine which factor levels were combined in the final model when the odds of asthma did not significantly differ ($p>0.05$). Effect modification of demographic characteristics on the relationship between each environmental exposure and odds of asthma (two-way interaction effects) were retained in the final model when significant at the 0.05 level or when an environmental exposure variable became significant due to inclusion of interactive variables. This methodology was repeated in examination of factors associated with increased odds of uncontrolled disease. Analyses were conducted using SPSS v18.0 software.

RESULTS:

Environmental Exposure and Demographic Characteristics of Population:

In 1,202 Pittsburgh elementary school students surveyed, 50.9% were female, average age was 8.5 years (SD=1.9), and the majority were African American (52.2%; Table 1). Students were predominately underserved as reflected by 60.6% on public health insurance. SHS was relatively high at 33.9%, and 17.1% of students were obese. Most students resided either partially (43.5%) or directly (38.8%) in the path of prevailing wind from the nearest OAP site. On average, the closest OAP site was 2.2(±2.2) miles (25% <1 mile) from residences and 2.1(±1.6) miles from schools. Over 70% of students had $PM_{2.5}$ exposure in excess of the WHO standard of 10 μg/m³ and 38.9% were above the EPA threshold of 12 μg/m³ (Table 2).

Asthma Prevalence and Contributory Factors:

Overall prevalence was 22.5% (±2.4), and highest among African Americans (26.8%), and those 10-12 years of age (26.7%), on public health insurance (26.0%), and with SHS (27.7%; $p<0.05$ for all; Table 1). Obesity (27.9%) and residing within one mile of an OAP site (25.5%) appeared contributory, although not statistically significant. Prevalence was significantly higher in children exposed to elevated levels of BC (24.9%), K (27.0%), Zn (26.7%), and $PM_{2.5} \geq 10.0$ μg/m³ (24.2%; $p<0.05$ for all; Table 2). In the model that adjusted for demographic characteristics, $PM_{2.5}$ values ≥ 10.0 ug/m^3 corresponded to 58% increased odds compared to $PM_{2.5}$ exposure <10.0 ug/m^3 ($p<0.05$; Table 3). Each 100th of a unit increase in Zn resulted in 67% increased odds ($p<0.05$). Higher exposure to NO_x (>75%) more than doubled the odds in obese children (OR=2.4; 95% CI 1.1-5.5; $p<0.05$); however, this association was not observed in non-obese children ($p=0.384$). Higher level exposure to S (>75%) nearly doubled the odds in children on public health insurance (OR=1.8; 95% CI 1.1-2.8; $p<0.05$); whereas, no differential effect was observed in children on private insurance ($p=0.264$). Higher level exposure to BC did not significantly increase the odds in the final adjusted model.

Prevalence of Uncontrolled Asthma and Contributory Factors:

In the 270 children previously diagnosed with asthma, a large percentage (59.3%) were uncontrolled (Table 1). Demographic characteristics did not correspond to increased prevalence, with exception of public vs. private health insurance which approached significance (64.0% vs 51.2%; $p=0.052$). Prevalence appeared marginally higher in those with increased exposure to BC (45.2% in lower 25%, 64.9% in mid-range, and 58.0% in upper 75%; $p=0.075$), but not directly to remaining environmental exposures (Table 2). However, in the adjusted analyses, females whose $PM_{2.5} \geq 10.0$ μg/m³ were nearly five times more likely to have uncontrolled asthma than females whose $PM_{2.5}$ <10.0 ug/m^3 (OR=4.74; 95% CI 1.36-16.50; $p=0.014$: Table 4); interestingly, this effect was not observed in males ($p=0.906$). SHS exposure corresponded to 7 times higher odds in obese children (OR=7.21; 95% CI 1.84-28.32; $p=0.005$); this effect was not significant in non-obese children ($p=0.334$). Mid-range exposure to BC more than doubled the odds compared to those in the lowest exposure group (≤ 25%), (OR=2.41; 95% CI 0.97-6.00; $p=0.058$). The effect of Si exposure on uncontrolled disease depended on the child's age. Mid versus low range ((≤25%) Si exposure corresponded to lower odds from ages 5-7, to minimal differential effect from ages 8-11, and then to five times the odds in children 12 years of age (OR=5.47; 95% CI 1.00-29.89: $p=0.050$).

In summary, among the 10 OAP exposures examined, six significantly related to odds of (a) asthma and/or (b) uncontrolled disease with correspondence dependent on another factor level in the final models, as described previously: (a) asthma diagnosis: $PM_{2.5}$, NO_x, S, and Zn; (b) uncontrolled asthma: $PM_{2.5}$, BC, and Si.

DISCUSSION:

The results of this study demonstrate an overall asthma prevalence of 22.5% and an uncontrolled disease rate of 59.3% among disparate children residing near OAP sites in Pittsburgh. This asthma prevalence is nearly triple the CDC reported national rate of 8.3%, but similar to rates reported in other studies of underserved children from different US cities.(1, 18-21) Additionally, this rate is nearly identical to the rate of 22.1% reported among 1,609 county teenagers surveyed by the local health department in 2014.(9)

This study documented an increased prevalence of asthma in African Americans and those with public health insurance, as well as a trend toward increased prevalence of uncontrolled asthma in those with public health insurance. Previous studies documented similar disparities in pediatric asthma outcomes.(22-25) The local county health department recently reported similar disparate results for adult asthma prevalence but did not report on prevalence of uncontrolled asthma.(8)

This study examined select environmental factors that potentially contribute to pediatric asthma outcomes. Over 70% of participants were exposed to annual mean $PM_{2.5}$ levels >10 ug/m^3, the WHO threshold to minimize health effects.(17) This rate is alarming and much higher than the 3.1% rate reported for the US.(26) The percentage exposed to SHS was 27.7% which is higher than the CDC reported national rate of approximately 20%.(27) The percentage of children classified as obese was 27.9% which is also higher than CDC reported national rate of 18.5%.(28) All of these factors have been shown to disproportionately affect vulnerable populations including children, African Americans and those of lower socioeconomic status. It is well established that OAP has a greater impact on children as compared to adults due to the higher minute ventilation rate of children.(29) Additionally, there is evidence that OAP can impact on lung growth in children.(30-32) Moreover, several recent publications have highlighted that disparate populations, including minorities and the poor, bear the greatest exposure and health effects of OAP.(33-35)

In the unadjusted analysis, asthma prevalence was increased among those with higher levels of $PM_{2.5}$, BC, K and Zn exposure, and there was a trend toward an increased rate of uncontrolled asthma in those exposed to higher levels of BC. OAP is clearly recognized as an important environmental factor that negatively impacts asthma control and morbidity.(36-38) Several, but not all, adult studies show an association between OAP and asthma prevalence.(39, 40) Fewer studies have been conducted in pediatric cohorts; however, several have shown an association between OAP and asthma incidence.(41-43)

In the unadjusted analysis, residing within one mile of an OAP site appeared contributory, although not statistically significant, to asthma prevalence. Many studies have evaluated the relationship between proximity of residence to OAP sites and asthma outcomes in adults and children.(41-45) Recent meta-analyses have shown significant associations for BC, NO_2, $PM_{2.5}$ and PM_{10} exposures and risk of asthma development. Several studies have shown associations between residence proximity to OAP sites and increased risk of asthma in adults and children.(46-48). Moreover, one of these studies showed that in utero exposure increased the risk of asthma diagnosis during early childhood.(47)

In the adjusted model, increased exposure to $PM_{2.5}$ and Zn corresponded to increased odds of asthma. Interestingly, higher exposure to NO_x was associated with increased odds of asthma in obese but not non-obese children. This is consistent with the recent finding of increased respiratory symptoms and asthma in obese children exposed to higher levels of PM_{10}, SO_2, NO_2 and ozone.(49) We found an association of higher S with increased odds of asthma in children on public as compared to private health insurance. This finding was expected since poverty is a well-recognized risk factor for pediatric asthma and poorer families tend to reside in neighborhoods adjacent to industrial OAP sites.(5, 33-35).

Our finding that mid-range BC exposure increased the odds of uncontrolled asthma is similar to that reported by others (50-52). Surprisingly, increased $PM_{2.5}$ was associated with higher prevalence of uncontrolled asthma only in females and not males. Several recent studies have shown associations between female gender and OAP related health impacts, including type 2 diabetes in adults and increased risk of abnormal lung function and allergy predisposition in children. (53-55) Mid-range Si corresponded to increased odds of uncontrolled asthma only in children over 11 years of age. Exposure to Si has been associated with the development worsening asthma symptoms, decreased lung function, and the development of chronic respiratory diseases including asthma and chronic obstructive lung disease.(56-59) Although it was not specifically investigated in this study, a possible explanation for this finding relates to chronic accumulative Si exposure that was not yet present in the younger children.

Surprisingly, SHS was not associated with an increased prevalence of asthma after adjustment for other contributory factors. This is in contrast to the results from many studies demonstrating an association between SHS and asthma outcomes.(60) A potential explanation for this discrepancy is that the current study exclusively enrolled subjects that resided near OAP sites and that factor overshadowed the impact of other environmental exposures. The current study did show an association between SHS and higher odds of uncontrolled asthma in obese, but not non-obese children. The observed interaction between SHS and obesity on uncontrolled asthma is consistent with a prior report showing an association between obesity and increased susceptibility to indoor pollutants among urban children with asthma.(61)

There are several limitations in the current study. First, the estimate of asthma prevalence is subject to potential limitations of selection bias. All schools participating were selected based on their proximity to OAP sites. Also, subjects self-selected to participate, and it cannot be ruled out that prior diagnosis of asthma contributed to participation. However, asthma prevalence was similar to that reported among teenagers and adults surveyed by the local health department during the same timeframe, as well as that reported in studies of disparate populations from other cities.(9, 18-21)

Second, the study design would have benefited by the addition of a control group that did not live in close proximity to an OAP site but matched the demographic profile of the study group. Our team consulted with an epidemiologist to identify such a population; however, none existed. All residential locations within Allegheny County whose demographic profile was similar to that of our study population resided in close proximity to OAP sites. This finding is not unexpected since recent studies have shown that minorities and those with lower socioeconomic status are much more likely to reside near OAP sites.(33-35)

Third, only select environmental factors, including OAP, SHS and obesity, were examined. It is acknowledged that other environmental factors including allergy predisposition, infection history, and psychological stress contribute to asthma outcomes.(5) Allergy and infection history were not assessed by survey since they would have been subject to recall bias. Allergy testing was not performed due to its expense and the possibility that it would deter participation. Similarly, psychological stress assessments were not performed due to their length and the possibility that their measurement would deter participation.

Finally, use of asthma controller medication was not assessed in the current study. Interestingly, several recent studies demonstrated that treatment with controller medications did not protect, but rather worsened, the effect of OAP on pediatric asthma outcomes. A prospective three-year study of 298 children from Detroit, showed that OAP elevations increased respiratory symptoms, particularly among those using inhaled glucocorticoids.(62) Similarly, the Childhood Asthma Management Program reported adverse changes in both lung function and airway hyperreactivity among 1,003 children studied over a four-year period. (63) A recent review article

summarized the evidence linking OAP exposure to glucocorticoid resistance in patients with asthma, and described the mechanisms leading to this effect, including changes to the glucocorticoid receptor, cytokines, and inflammatory pathways, as well as epigenetics. (64)

CONCLUSIONS/KEY FINDINGS

These results show that asthma prevalence and rates of uncontrolled disease are high among disparate schoolchildren residing near OAP sites. They also show that the majority of participants are exposed to higher levels of $PM_{2.5}$ than is recommended by WHO. Future efforts should focus on reducing exposure to OAP to minimize harm, particularly in vulnerable populations.

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DISCLOSURE STATEMENT: The authors report no conflicts of interest.

DATA AVAILABILITY STATEMENT: The data set is available upon reasonable request by contacting the corresponding author.

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Table 1. Demographic characteristics and exposure risks among participants described in relation to asthma prevalence and uncontrolled disease.

		Prevalence(±margin of error[a])	
	Overall	**Asthma**	**Asthma not well controlled[b] (In 270 with prior diagnosis)**
Demographic Characteristics:	**N=1202**	**22.5(±2.4)%** **270/1202**	**59.3(±5.9)%** **160/270**
Gender:			
Male	49.1%	24.6(±3.5)%	57.9(±8.1)%
Female	50.9%	20.5(±3.2)%	60.8(±8.6)%
Age years*,mean(SD)	8.5(1.9) years		
5-6 years	19.1%	17.7(±5.2)%	62.2(±15.8)%
7-9 years	42.0%	20.5(±3.5)%	62.1(±9.4)%
10-12 years	38.9%	26.7(±3.9)%	56.6(±8.6)%
Race*:			
African-American	52.2%	26.8(±3.5)%	59.5(±7.4)%
White(40.6%); Other(7.2%)	47.8%	17.7(±3.1)%	58.8(±9.6)%
Obesity(~p=0.062 in asthma prevalence)			
BMI<95%	82.9%	21.6(±2.6)%	58.3(±6.9)%
BMI≥95%(obese)	17.1%	27.9(±6.4)%	62.3(±13.2)%
Health Insurance*,¤~0.052			
Public	60.6%	26.0(±3.2)%	64.0(±11.0)%
Private	39.4%	17.2(±3.4)%	51.2(±6.9%)%
Secondhand Smoke Exposure (SHS) *			
- (cotinine<1.00 ng/ml)	66.1%	20.1±(2.9)%	58.2(±8.0)%
+(cotinine≥1.00 ng/ml)	33.9%	27.7(±4.6)%	60.2(±9.5)%
Prevailing Wind Path from Source Site to Residence:			
Not in path	17.7%	20.9(±5.7)%	65.9(±14.7)%
Partially	43.5%	22.4(±3.7)%	56.5(±9.4)%
Directly in path	38.8%	21.7(±3.9)%	60.2(±10.0)%
Linear Distance from Source Site to residence: median[IQR]	1.55 miles [IQR=0.99,2.44]		
<1.00 mile	Lower quartile	25.5(±5.1)%	59.2(±11.5)%
1.00-2.44 miles	Mid-range (26-75th%)	21.7(±3.4)%	59.2(±8.8)%
≥2.45 miles	Upper quartile	18.6(±4.6)%	60.8(±13.5)%

SD=Standard Deviation

[a]margin of error= 1.96*Standard error of the mean

[b]Asthma not well controlled defined by repeated episodes of asthma, and/or has exercise limitations, and/or daytime symptoms ≥2days/week due to cough, chest tightness, trouble breathing, and/or wheeze.

*p<0.05, Chi-square test between factor levels in prevalence of asthma

[¤]P<0.05, Chi-square test between factor levels in prevalence of uncontrolled asthma

Table 2. Outdoor air pollution levels among participants described in relation to asthma prevalence and uncontrolled disease.

			Prevalence(± margin of error[a])	
		Exposure Level in Distribution	**Asthma**	**Asthma not well controlled [b] (In 270 with prior diagnosis)**
Environmental Exposure:		**N=1202**	**22.5(±2.4)%** **270/1202**	**59.3(±5.9)%** **160/270**
$PM_{2.5}$<10 μg/m³*	*$PM_{2.5}$ Standard*	29.3%	15.7(±4.0)%	51.0(±13.9)%
10-11.9	*Threshold Levels*	31.9%	24.2(±4.5)%	64.7(±10.2)%
≥12.0		38.8%	24.6(±4.1)%	59.4(±9.4)%
BC<0.9408 μg/m³*,~p=0.075 NWC		Lowest(≤25th%)	15.2(±4.2)%	45.2(±15.2)%
0.9409-1.2153		Middle(26-75th%)	23.6(±3.5)%	64.9(±8.2)%
>1.2153		Highest(>75th%)	24.9(±5.1)%	58.0(±11.7)%
NO_2≤4.4132 ppb		Lowest(≤25th%)	20.5(±4.8)%	54.4(±13.0)%
4.4133-6.0502		Middle(26-75th%)	20.9(±3.4)%	61.7(±8.9)%
>6.0502		Highest(>75th%)	24.6(±5.1)%	60.3(±11.7)%
NO_x≤10.7715 ppb~p=0.056 asthma		Lowest(≤25th%)	16.7(±4.4)%	58.7(±14.4)%
10.7716-19.2558		Middle(26-75th%)	23.4(±3.5)%	59.2(±8.5)%
>19.2558		Highest(>75th%)	23.8(±5.0)%	60.6(±11.9)%
K≤0.0507 μg/m³*		Lowest(≤25th%)	15.9(±4.3)%	63.6(±14.4)%
0.0508-0.0648		Middle(26-75th%)	22.5(±3.5)%	61.3(±8.6)%
>0.0648		Highest(>75th%)	27.0(±5.3)%	54.8(±11.5)%
S≤0.8000 μg/m³		Lowest(≤25th%)	22.8(±4.0)%	57.7(±9.9)%
0.8001-0.9156		Middle(26-75th%)	20.0(±3.9)%	65.0(±10.5)%
>0.9156		Highest(>75th%)	23.6(±5.0)%	55.4(±12.2)%
Cr≤0.0037 μg/m³		Lowest(≤25th%)	19.3(±4.5)%	60.3(±12.7)%
0.0038-0.0048		Middle(26-75th%)	23.5(±3.8)%	57.5(±9.2)%
>0.0048		Highest(>75th%)	22.0(±5.2)%	61.1(±13.1)%
Fe≤0.0548 μg/m³		Lowest(≤25th%)	18.8(±4.6)%	59.6(±13.5)%
0.0549-0.0912		Middle(26-75th%)	21.5(±3.4)%	60.5(±8.8)%
>0.0912		Highest(>75th%)	25.5(±5.2)%	57.1(±11.7)%
Si≤0.0658 μg/m³		Lowest(≤25th%)	22.0(±4.9)%	59.0(±12.4)%
0.0659-0.1052		Middle(26-75th%)	19.7(±3.3)%	60.6(±9.2)%
>0.1052		Highest(>75th%)	26.0(±5.2)%	58.3(±11.5)%
Zn≤0.0157 μg/m³*		Lowest(≤25th%)	16.8(±4.4)%	56.5(±14.5)%
0.0158-0.0226		Middle(26-75th%)	21.9(±3.5)%	62.5(±8.7)%
>0.0226		Highest(>75th%)	26.7(±5.3)%	56.9(±11.5)%

[a]margin of error=1.96*Standard error of the mean

[b]Asthma not well controlled defined by repeated episodes of asthma, and/or has exercise limitations, and/or daytime symptoms ≥2 days/week due to cough, chest tightness, trouble breathing, and/or wheeze.

[*]p<0.05, Chi-square test between factor levels in prevalence of asthma

[¤]P<0.05, Chi-square test between factor levels in prevalence of uncontrolled asthma

Table 3. Final multivariate model describing the combination of environmental exposures that significantly increased odds of asthma, adjusted for confounding and effect modification of demographic characteristics.

	Outcome: Asthma Diagnosis **OR(95% CI);p-value**
Gender (male vs. female)	1.39(1.02,1.91);p=0.040
Race (African-American vs. White/Other)	1.75(1.22,2.52);p=0.002
Age (per one year increase)	1.10(1.01,1.20);p=0.025
Obesity (obese vs. non-obese) in those with: $NO_x \leq 19.256$ ppb $NO_x > 19.256$ ppb(highest quartile)	 1.18(0.75,1.88);p=0.475 3.55(1.65,7.65);p=0.001
Health Insurance (public vs. private) in those with: $S \leq 0.916$ μg/m^3 $S > 0.916$ μg/m^3(highest quartile)	 1.34(0.88,2.04);p=0.170 3.70(1.71, 8.01);p=0.001
$PM_{2.5} \geq 10.0$ vs.<10.0 μg/m^3	1.58(1.01, 2.48);p=0.047
Zn(μg/m^3)x100 (per unit increase)	1.67(1.15,2.42);p=0.007
Si(μg/m^3)x100 (per unit increase)	0.92(0.86,0.99);p=0.034
NO_x(>19.256 vs. ≤19.256 ppb) in those with BMI: Non-obese(<95th%) Obese(≥95th%)	 0.81(0.51,1.30);p=0.384 2.44(1.08,5.53);p=0.033
S(>0.916 vs. ≤0.916 μg/m^3) in those with: Private health insurance Public health insurance	 0.64(0.30,1.40);p=0.264 1.77(1.14,2.76);p=0.011

OR=odds ratio: odds of asthma versus reference category or per one unit increase in continuous variables.

Table 4. Final multivariate model describing the combination of environmental exposures that significantly increased odds of uncontrolled asthma in 270 children with prior diagnosis, adjusted for confounding and effect modification of demographic characteristics.

	Outcome: Uncontrolled Asthma **OR(95% CI);p-value**
Gender (male vs. female) in patients with:	
$PM_{2.5}$<10.0 μg/m^3	2.71(0.69,10.63);p=0.153
$PM_{2.5}$≥10.0 μg/m^3	0.61(0.31,1.21);p=0.160
Race (African-American vs. White/Other)	0.54(0.26,1.15);p=0.109
Age (per one year increase) in patients with:	
Si<0.0659 μg/m^3(lowest quartile)	0.65(0.41,1.04);p=0.072
Si=0.0659-0.1052 μg/m^3(mid-range)	1.25(0.97,1.61);p=0.080
Si>0.1052 μg/m^3(highest quartile)	0.81(0.60,1.09);p=0.163
Obesity (obese vs. non-obese) in patients with:	
-SHS exposure (cotinine<1.00 ng/ml)	0.25(0.09, 0.72);p=0.011
+SHS exposure (cotinine ≥1.00 ng/ml)	2.57(0.88,7.57);p=0.086
SHS exposure (+ vs. -) in patients with BMI:	
Non-obese(<95^{th}%)	0.69(0.33,1.46);p=0.334
Obese(≥95^{th}%)	7.21(1.84,28.32);p=0.005
Health Insurance (public vs. private):	1.76(0.79,3.92);p=0.164
$PM_{2.5}$≥10.0 vs. <10.0 μg/m^3:	
Female	4.74(1.36,16.50);p=0.014
Male	1.07(0.33,3.53);p=0.906
BC	
≤0.9408 μg/m^3(lowest quartile)	----
0.9409-1.2153(mid-range)	2.41(0.97,6.00);p=0.058
>1.2153(highest quartile)	1.44(0.50,4.17);p=0.506

Si(0.0659-0.1052 vs. <0.0659 μg/m^3) in those aged:	
5 years	0.06(0.01,0.69);p=0.024
6 years	0.11(0.02,0.82);p=0.031
7 years	0.65(0.41,1.04);p=0.072
8 years	0.40(0.12,1.31);p=0.131
9 years	0.77(0.30,2.01);p=0.599
10 years	1.49(0.55,4.05);p=0.439
11 years	2.85(0.79,10.34);=0.111
12 years	5.47(1.00,29.89);p=0.050

OR=odds ratio: odds of asthma versus reference category or per one unit increase in continuous variables.