
Relationship between visits to emergency departments for asthma and ozone exposure in greater Seattle, Washington

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Background: Air pollution is known to affect asthma symptoms in controlled and epidemiologic studies.

Objective: To determine whether ozone exposure in Seattle is associated with increased use of hospital emergency departments.

Methods: Hospital data on daily asthma cases for all ages were obtained for 1998 through 2002. Ozone and fine particulate matter ($\leq 2.5 \mu\text{m}$ in diameter) ($\text{PM}_{2.5}$) data were obtained from local air agencies. Poisson regression models were used to assess the association between asthma visits to emergency departments and air pollutants. Maximum daily 1- and 8-hour average ozone concentrations and the daily $\text{PM}_{2.5}$ concentration were used.

Results: We observed associations between both ozone metrics and emergency department visits in children. For the maximum daily 1- and 8-hour average ozone concentrations, the relative risks (RRs) were 1.08 (95% confidence interval [CI], 1.00–1.18) and 1.11 (95% CI, 1.02–1.21), respectively, at 3 days' lag. Weaker but significant associations were also observed for adults. For the maximum daily 1-hour average ozone concentration, the RR was 1.06 (95% CI, 1.01–1.11) at 4 days' lag, and for the maximum daily 8-hour average ozone concentration, the RR was 1.06 (95% CI, 1.01–1.12) at 2 days' lag and 1.08 (95% CI, 1.02–1.14) at 4 days' lag.

Conclusion: Ozone exposure exacerbates asthma in people in the Seattle area, especially in children.

Ann Allergy Asthma Immunol. 2009;103:474–479.

INTRODUCTION

Children with asthma are especially susceptible to adverse respiratory effects from air pollution exposure because underlying airway inflammation is a hallmark of asthma. Much of the research documenting this association has been conducted with particulate matter (PM) air pollution.^{1–4} Outdoor exposure of children to ozone in the summer months is common. Children constitute an especially vulnerable group because they tend to be outside during the peak ozone periods (10 AM to 6 PM). Children often are engaged in sports programs during these months and, thus, have increased exposure due to increased respiratory rates. Because ozone is known to cause increases in oxidative stress and immunologic disease,⁵ there is concern that ozone exposure may be associated with aggravation of asthma in children. Recently, the Clean Air Science Advisory Committee of the Environmental Protection Agency recommended tightening the National Ambient Air Quality Standard for ozone owing to its known toxic effects. Bates⁶ suggested that ozone exposure may be associated with small airway disease, resulting in chronic bronchiolitis.

Few studies document an association between ozone exposure and asthma exacerbation in children. Controlled hu-

man studies found associations between individuals with asthma and changes in pulmonary function.⁷ An early epidemiologic study by Weisel et al⁸ found an association between ozone exposure and emergency department (ED) visits for asthma in children in New Jersey. In a study conducted in Atlanta, Georgia, during the 1996 Olympic games, Friedman et al⁹ investigated the effects of traffic changes brought about by the games on childhood asthma events. They reported that prolonged reductions in ozone concentrations were associated with significantly fewer unscheduled hospital visits by children with asthma. In Paris, France, Fauroux et al¹⁰ conducted a time-series study on urban air pollution and daily ED visits for asthma. A positive association was found between daily asthma visits and daily variations in ozone levels. Tolbert et al¹¹ reported a significant association between ED visits for pediatric asthma and ozone levels in Atlanta. Gent and co-workers¹² reported an association between low levels of ozone (mean 1-hour average of 51 ppb) and respiratory symptoms in children with asthma in southern New England. The outcomes reported were daily respiratory symptoms and use of rescue medications. They also found an association with fine particles. In Seattle, several studies have found associations between asthma aggravation and air pollution measured by ED visits,^{13,14} asthma symptoms,^{15,16} and increases in exhaled nitric oxide levels.¹⁷ These health outcomes were associated with fine particles and other primary traffic-related pollutants. Ozone exposures were not considered in these studies because ozone is monitored only from May through September in the Seattle area. We decided to evaluate the

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Disclosures: Authors have nothing to disclose.

Received for publication March 25, 2009; Received in revised form June 23, 2009; Accepted for publication July 12, 2009.

relationship between outdoor ozone in the summer and asthma aggravation in the Seattle area. We hypothesized that peak daily ozone concentrations and the maximum daily 8-hour average ozone concentration would be associated with ED visits for asthma. To our knowledge, this is the first time-series analysis of associations between ED visits for asthma and ozone exposure in the greater Seattle region.

METHODS

Health Data

Daily hospital counts of ED visits for children were obtained from 3 Seattle-area hospitals located east of the city in the geographic area where ozone levels rise due to air chemistry and wind patterns. Only ED visits where the primary diagnosis was asthma were included in the study. Asthma ED visits were identified by *International Classification of Diseases, Ninth Revision*, codes 493 to 493.9. In the Seattle area, the highest ozone concentrations are located southeast of the city. This research was approved by the University of Washington Human Subjects Division.

Air Pollution and Meteorologic Data

Ozone precursors are emitted from mobile sources on freeways I-5 and I-90 and on other major highways in urban areas and are transformed photochemically into ozone over several hours. Ozone data from May to October (the only months that ozone data are available in the Puget Sound area) from 2 monitoring sites (Enumclaw and Lake Sammamish Park) were obtained from the Washington Department of Ecology. Yearly data for PM 2.5 μm in diameter or smaller (PM_{2.5}) from 3 monitoring sites (Kent, North Bend, and Bellevue) were obtained from the Puget Sound Clean Air Agency. Daily average temperatures were obtained from SeaTac Airport. The map in Figure 1 shows the locations of the ozone monitors and the cities where the 3 hospitals are located.

Statistical Analyses

Poisson regression models were used to evaluate the association between asthma ED visits and ozone or PM_{2.5} concentrations. Generalized additive models with stringent convergence criteria (maximum iterations used was 1,000, with the convergence criterion set to 10e-8) and asymptotically exact standard errors were used for the analysis.¹⁸ The model controlled for seasonal trends, daily temperature, and day of the week. Because we had 20 months of ozone data from 1999 to 2002, we used 7 *df* for the smoothing of time trend. We used 3 *df* for the smooth of temperature. The temperature term was averaged over the same period as the ozone concentration; that is, when we analyzed the maximum 8-hour average ozone effect, temperature was averaged over the same period. In addition, the temperature term was included in the model with the same lag time as the ozone term. For example, for a 1-day lag in ozone, the temperature term was also lagged 1 day. An indicator variable was used for day of the week. We analyzed the relationship between daily ED visits for asthma and 0- to 5-day lags of ozone and PM_{2.5} concentrations. We



Figure 1. Map of the ozone monitors and the area hospitals.

evaluated the association for the maximum daily 1-hour average ozone concentration, the maximum daily 8-hour average ozone concentration, and the daily average PM_{2.5} concentration. Separate analyses were conducted for children (<18 years) and adults (≥18 years). Statistical analyses were conducted using SPLUS 2000 (Insightful Inc, Seattle). Results of the analyses are reported as the relative risk (RR) of ED visits for asthma per 10-ppb increase in ozone concentration and 10-μg/m³ increase in PM_{2.5} concentration.

RESULTS

Air Pollution and Meteorologic Data

Summary statistics for pollution and temperature data in Enumclaw and Lake Sammamish Park are given in Tables 1 and 2, respectively. From 1998 to 2002, the maximum 1-hour average ozone value was 39 ppb averaged for the 2 monitoring sites. The maximum 8-hour average ozone value was 32 ppb. The 24-hour daily average PM_{2.5} values ranged from 8 to 11 $\mu\text{g}/\text{m}^3$. The mean (SD) daily temperature was 51°F (8.8°F), and the temperature on days with ozone was 59.5°F (5.7°F).

Asthma ED Data

We obtained ED data from 3 area hospitals between November 1, 1998, and November 30, 2002, totaling 9,028 cases, with 3,042 of these cases occurring in children. For only those days with ozone data (May 1 to September 30), we had a total of 2,080 adults and 1,137 children. Most of the children with asthma were boys, and most of the adults with asthma were women. Characteristics of the study population are given in Table 3. During the months with available ozone data, there was an average of 5.5 cases per day (Table 4).

Table 1. Summary of Air Pollution Data^a

	Enumclaw	Lake Sammamish Park	Average
Ozone, maximum 1-h average, ppb			
Mean	41.6	35.7	38.6
75th percentile	49.0	43.0	45.5
25th percentile	31.0	27.0	29.0
IQR	18.0	16.0	16.5
Ozone, maximum 8-h average, ppb	35.8	28.6	32.2
Mean	43.0	36.3	39.2
75th percentile	27.1	20.5	23.7
25th percentile	15.9	15.8	15.5
IQR	11.1	4.7	8.2
24-h average, ppb			
Mean	24.4	16.0	20.2
75th percentile	30.0	20.0	25.0
25th percentile	18.0	11.0	15.0
IQR	12.0	9.0	10.0
PM _{2.5} , µg/m ³	Kent	Bellevue	North Bend
	11.3	8.8	8.0

Abbreviations: IQR, interquartile range; PM_{2.5}, particulate matter 2.5 µm in diameter or smaller.

^a The National Ambient Air Quality Standard for ozone is 70 ppb for an 8-hour average and for PM_{2.5} is 35 µg/m³ for a 24-hour average.

Table 2. Summary of Temperature Data

	Temperature, °F			
	Daily mean (SD)	Range	Maximum 8-h mean (SD)	Maximum 1-h mean (SD)
Ozone months (May-Sep)	59.5 (5.7)	40–76	64.8 (7.1)	66.8 (7.5)
Annual	50.95 (8.8)	22–76	54.9 (10.3)	56.6 (10.7)

Table 3. Age and Sex Distributions of Patients With Asthma

	Males	Female	Total
Children (0–18 y)			
No.	727	410	1,137
Average age, y	5.9	8.4	
Adults (>18 y)			
No.	633	1,447	2,080
Average age, y	39.4	40.3	

Statistical Results

The RRs of an asthma ED visit associated with a 10-ppb increase in the maximum 1- and 8-hour average ozone concentrations are shown for children and adults in Figures 2 and 3, respectively. For children, the maximum 1- and 8-hour average ozone concentrations were associated with an increased risk of an asthma ED visit. The RR of an asthma ED visit associated with a 10-ppb increase in the maximum 1-hour ozone concentration on the same day was 1.10 (95% confidence interval [CI], 1.02–1.18). For a 10-ppb increase in the maximum 8-hour average ozone concentration on the same day, the RR was 1.10 (95% CI, 1.01–1.19). In children, there were also significant associations with ozone at 3 days' lag. Smaller effects were also observed at 4 and 5 days' lag

Table 4. Age Distribution of Patients With Asthma for Ozone and PM_{2.5} Analysis

	Patients, No.	Patients per day, mean No.
Ozone months (May-Sep)		
All patients	2,540	5.5
Adults	1,654	3.6
Children	886	1.9
All year (PM _{2.5})		
All patients	9,028	6.1
Adults	5,986	4.0
Children	3,042	2.0

Abbreviation: PM_{2.5}, particulate matter 2.5 µm in diameter or smaller.

with ozone measured at only 1 location but not with the average concentration from both locations (Table 5). For adults, associations were found with the maximum 8-hour average ozone concentration at 2, 4, and 5 days' lag and with the maximum 1-hour average ozone concentration at 4 and 5 days' lag (Table 6). For children and adults, there were no significant associations between asthma ED visits and PM_{2.5} concentration at any of the monitoring sites. To further investigate the effects of lag times, we also analyzed associa-

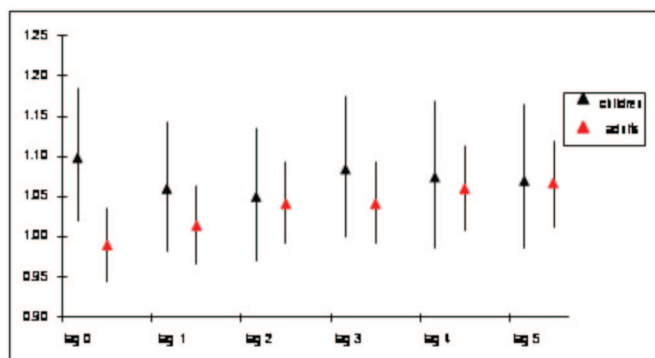


Figure 2. Relative risk of an asthma emergency department visit associated with a 10-ppb increase in the maximum 1-hour average ozone concentration.

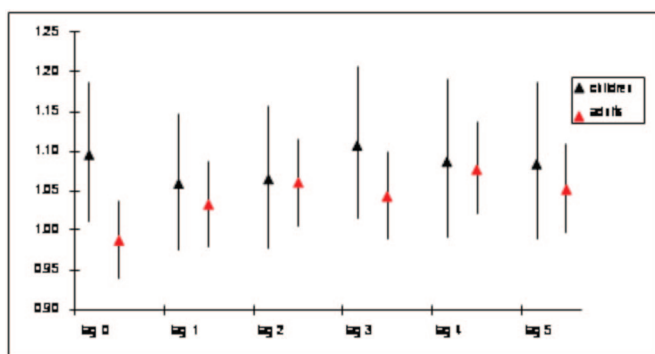


Figure 3. Relative risk of an asthma emergency department visit associated with a 10-ppb increase in the maximum 8-hour average ozone concentration.

tions between the running average levels (0–1 days' lag, 0–2 days' lag, 0–3 days' lag, 1–2 days' lag, and 1–3 days' lag) for the maximum 1- and 8-hour average ozone concentrations. No significant associations were found with the running average data.

DISCUSSION

This study found a robust relationship between visits to the hospital ED for pediatric asthma and ambient ozone levels at 3 days' lag during the summer months in the greater Seattle area. A 10-ppb increase in the maximum daily 1- and 8-hour average ozone concentrations was associated with an 8% and 11% increase in the risk of an asthma ED visit, respectively. Although there is considerable evidence that asthma exacerbation is associated with fine particulate air pollution in Seattle, this is the first study, to our knowledge, to implicate ozone as a trigger for childhood asthma aggravation in this location. This analysis also found an association between adult asthma ED visits and the maximum 1-hour average ozone level on lag days 4 and 5 and the maximum 8-hour average ozone level on lag days 2, 4, and 5. The difference between children and adults with respect to lag time between

Table 5. Relative Risk Associated With a 10-ppb Increase in Ozone in Children

Site and lag, d	Relative Risk (95% CI)
Maximum Daily 1-h Average Ozone Concentration	
Average of both sites	
0	1.10 (1.02–1.18)
1	1.06 (0.98–1.14)
2	1.05 (0.97–1.14)
3	1.08 (1.00–1.18)
4	1.07 (0.99–1.17)
5	1.07 (0.99–1.17)
Enumclaw	
0	1.08 (1.01–1.17)
1	1.03 (0.95–1.11)
2	1.04 (0.96–1.12)
3	1.07 (0.99–1.15)
4	1.06 (0.98–1.14)
5	1.06 (0.98–1.15)
Lake Sammamish Park	
0	1.09 (1.01–1.18)
1	1.08 (1.00–1.17)
2	1.06 (0.98–1.14)
3	1.08 (1.00–1.17)
4	1.07 (1.00–1.16)
5	1.07 (0.99–1.15)
Maximum Daily 8-h Average Ozone Concentration	
Average of both sites	
0	1.10 (1.01–1.19)
1	1.06 (0.98–1.15)
2	1.06 (0.98–1.16)
3	1.11 (1.02–1.21)
4	1.09 (0.99–1.19)
5	1.08 (0.99–1.19)
Enumclaw	
0	1.08 (1.00–1.17)
1	1.03 (0.95–1.11)
2	1.06 (0.98–1.15)
3	1.08 (1.00–1.18)
4	1.09 (1.00–1.18)
5	1.08 (0.99–1.18)
Lake Sammamish Park	
0	1.09 (1.01–1.18)
1	1.07 (0.99–1.16)
2	1.07 (0.99–1.16)
3	1.11 (1.02–1.20)
4	1.07 (0.98–1.16)
5	1.08 (0.99–1.17)

Abbreviation: CI, confidence interval.

ozone exposure and asthma ED visit suggests that children are more immediately responsive to adverse effects of ozone exposure. Children are known to be more susceptible to the effects of air pollution owing to their undeveloped immune systems and their increased outdoor activity patterns. The outdoor activity of children makes them highly vulnerable to the adverse effects of ozone because ozone levels are elevated during the summer months, when children are ordinarily out

Table 6. Relative Risk Associated With a 10-ppb Increase in Ozone in Adults

Site and lag, d	Relative Risk (95% CI)
Maximum Daily 1-h Average Ozone Concentration	
Average of both sites	
0	0.99 (0.94–1.04)
1	1.01 (0.97–1.06)
2	1.04 (0.99–1.09)
3	1.04 (0.99–1.09)
4	1.06 (1.01–1.11)
5	1.06 (1.01–1.12)
Enumclaw	
0	0.98 (0.94–1.03)
1	1.01 (0.97–1.06)
2	1.04 (0.99–1.09)
3	1.04 (1.00–1.09)
4	1.05 (1.00–1.10)
5	1.05 (1.01–1.10)
Lake Sammamish Park	
0	1.00 (0.96–1.05)
1	1.02 (0.97–1.07)
2	1.03 (0.99–1.08)
3	1.03 (0.99–1.08)
4	1.06 (1.01–1.11)
5	1.06 (1.01–1.11)
Maximum Daily 8-h Average Ozone Concentration	
Average of both sites	
0	0.99 (0.94–1.04)
1	1.03 (0.98–1.09)
2	1.06 (1.01–1.12)
3	1.04 (0.99–1.10)
4	1.08 (1.02–1.14)
5	1.05 (1.00–1.11)
Enumclaw	
0	0.99 (0.94–1.04)
1	1.04 (0.98–1.09)
2	1.06 (1.00–1.11)
3	1.06 (1.00–1.11)
4	1.08 (1.02–1.11)
5	1.05 (1.00–1.11)
Lake Sammamish Park	
0	0.99 (0.94–1.04)
1	1.03 (0.98–1.08)
2	1.05 (1.00–1.10)
3	1.03 (0.98–1.08)
4	1.06 (1.01–1.12)
5	1.04 (0.99–1.09)

Abbreviation: CI, confidence interval.

of school and are more likely to be outdoors. Ozone does not penetrate indoors with the same efficiency as fine particles, and, therefore, individuals who spend most of their time indoors experience lesser exposure. Although we found an association between ED visits and ozone concentrations on the same day (lag day 0), we believe that caution should be used in interpreting these results because for same-day exposures, the ED visit may have preceded the maximum 1- and

8-hour average ozone concentrations. The associations observed at 3 days' lag in children and the effect observed at 2 days' lag in adults may be explained by differences in the delayed inflammatory response due to ozone and the onset of symptoms or by differential abilities to manage asthma symptoms at home. Adults may be better able to manage their asthma symptoms at home. Other investigators¹⁹ have also observed that in adults with asthma, there is often a delay between onset of symptoms and ED visits.

Although this is the first report of an association between asthma and ozone in Seattle, several studies document such an association in other cities. The effects of ambient ozone on childhood asthma have been studied in summer camps, an environment where children are often very active, thus increasing their ventilation rates and subsequent exposure to air pollution. These studies, as reviewed by Kinney et al,²⁰ found decreased lung function in the camp children. Mortimer et al²¹ reported an association between an interquartile increase in ambient ozone levels and the incidence of morning symptoms in inner-city children with asthma. Babin et al²² reported an association between daily ozone exposure and ED visits for asthma in children, with the greatest effects in the 5- to 12-year-old age group. They saw a small effect using same-day ozone concentration and a stronger lagged effect (1.9% increase in visits per 0.01-ppm ozone increase).

As mentioned previously, Bates⁶ suggested that ozone may be responsible for recent increases in bronchiolitis in children. Burnett et al²³ studied children younger than 2 years and found a 35% (95% CI, 19%–52%) increase in the daily hospitalization rate for respiratory problems associated with a 5-day moving average of the maximum daily 1-hour ozone concentration (45 ppb in this study). Galizia and Kinney²⁴ and Kunzli et al²⁵ reported that long-term residence in areas with high ozone concentrations is associated with lower lung function measured in early adulthood.

It is surprising that this study did not find an association between fine particles and visits to the ED for asthma. As mentioned previously herein, similar studies in Seattle have found robust associations. For example, using a 15-month data set in 1989 to 1990, Schwartz et al¹³ found that daily counts of ED visits for persons younger than 65 years were significantly associated with PM₁₀ exposure on the previous day. The RR for a 30- $\mu\text{g}/\text{m}^3$ increase in PM₁₀ was 1.12. Nearly the same association was seen for finer particles measured using a light-scattering nephelometer. In an analysis using only individuals younger than 18 years, Norris et al¹⁴ reported a RR of 1.15 (95% CI, 1.08–1.23) for an association between PM_{2.5} and ED visits for asthma in Seattle. More recently, in Tacoma, Washington, a city that shares the Puget Sound airshed with Seattle, we observed an association between ED visits and PM_{2.5} in children. Therese F. Mar, PhD, Jane Q. Koenig, PhD, and Janet Primomo, PhD, unpublished data). The most likely explanation for the lack of an association between PM and asthma ED visits in the present analysis is location. The analyses by Schwartz et al¹³ and Norris et al¹⁴ looked at asthma cases in Seattle, whereas

the present analysis looked at asthma cases in areas to the southeast of Seattle, where ozone concentrations are highest (see Fig 1 for geographic information). Because PM_{2.5} is a traffic-related air pollutant, it is expected to be in higher concentrations near interstate freeways and major busy roadways. Ozone, on the other hand, due to its dependence on photochemistry, tends to have a higher concentration in areas 2 to 4 hours downwind of the traffic sources.

One limitation of this study is the use of population- or community-based air pollution data, which may not be representative of all individuals. Furthermore, the lag time between air pollution exposure and ED visit is unknown and may vary from child to child. Finally a limitation of time-series studies is the lack of individual data on health, behavior characteristics, and activity levels, which affect air pollution exposure levels. However, the advantage of such studies is that there is no need to control for modifying factors such as preexisting conditions (such as atopy) or concomitant illnesses, socioeconomic issues, and access to health care, which remained relatively the same during the study.

In conclusion, these data suggest that a relatively low concentration of outdoor ozone is associated with asthma exacerbation in children and adults. Thus, ozone is a potential public health threat that needs to be recognized.

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