Relationship between Summertime Ambient Ozone Levels and Emergency Department Visits for Asthma in Central New Jersey

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The 5-year retrospective study of the association between temperature and emergency department (ED) visits for asthma with mean ambient ozone levels between 10:00 and 15:00 was conducted in central New Jersey during the summer months. An association was identified in each of the years (1986–1990). Between 8 and 34% of the total variance in ED visits for asthma was explained by the two environmental variables in the step-wise multiple regression analysis. ED visits occurred 28% more frequently when the mean ozone levels were >0.06 ppm than when they were <0.06 ppm. This result was statistically significant in a covariance analysis. An evaluation of the effects of ozone on asthmatics reported in the literature was completed to determine if, as proposed by Bates, the results from different types of studies were coherent among the health metrics. A consistency in the magnitude of reported effects and the time lag between exposure and response for four different health indices (symptom reports, decrements in expiratory flow, ED visits, and hospital admissions) was identified and indicates a coherence between ozone and respiratory response to ozone exposure. This supports a proposition that ozone adversely affects asthmatics at levels below the current U.S. standard. — Environ Health Perspect 103(Suppl 2):97–102 (1995)

Key words: ozone, temperature, asthma, emergency department, regression analysis, covariant analysis

Introduction

Measured associations between air pollutants and respiratory ailments have led to the establishment of National Ambient Air Quality Standards (NAAQS) for specific pollutants to protect public health. Ozone, an air pollutant found in photochemical smog, still exceeds the current U.S. NAAQS (0.120 ppm for 1 hr not to be exceeded more than once per year) during the summer in large regions of the United States, but the level and form of this standard is under review. Ozone is a lung irritant that affects the respiratory tract and can adversely affect sensitive subpopulations (*1,2*). In addition, the way that ozone initiates or promotes asthmatic attacks has been the subject of recent research and several review papers (*1,3,4*). Organizations have set different 1-hr ozone air quality standards or guidelines, e.g., United States, 0.12 ppm; Canada, 0.082; World Health Organization (WHO) for Europe, 0.075–0.10 ppm; and Japan, 0.060 ppm. Additionally, the respiratory responses to short-term peak exposures at high concentrations and extended exposures have resulted in recommendations for eight average standards in the United States (*5*). This is currently employed by WHO for Europe which has an 8-hr guideline with a range of 0.050 to 0.060 ppm.

Controlled clinical studies have demonstrated that decrements in forced expiratory volume in 1 sec (FEV_1), occurred in healthy, exercising adults exposed to ozone concentrations of 0.080 to 0.12 ppm for up to 6.6 hr (*6–8*). When asthmatics and healthy individuals were exposed solely to ozone, changes in respiratory function were similar (*9*), but asthmatics who were exposed to ozone and then challenged with an allergen had a greater response than a control group (*10*).

Studies have established a relationship between increases of ozone and respiratory function and a variety of asthmatic symptoms. Decrements in respiratory function with increasing ambient ozone levels have been measured in cohorts of active healthy adults (*11,12*), healthy children (*13–15*), and asthmatics (*16–19*). The number of respiratory symptoms reported by asthmatics increased on days when the ozone concentration was high (*17,20–23*). Further, an association has been identified between ambient ozone levels alone or in combination with acid aerosols and the number of visits to emergency departments (ED) for asthma attacks (*24–26*) and the number of hospital admissions for asthma (*27,28*). A few epidemiologic studies have not identified associations between increases in ozone or oxidant concentration and either the reporting of symptoms or visits to treatment centers by asthmatics (*29–32*).

Our previous 2-year study (*26*) was sufficient to observe a relationship between ambient ozone levels and ED visits by asthmatics. To determine whether this observation was part of a consistent trend, independent of interyear variations in ozone levels and meteorological conditions within a single locale, the present study was undertaken. The present study examined the relationship between ozone concentration and ED visits for asthma attacks in central New Jersey for 5 consecutive years, 1986 to 1990. The results obtained have been used to estimate the relative increase in the number of ED visits by asthmatics associated with increases in the ambient ozone concentrations. The observed consistency in results of this study suggest that an association between ozone levels and various health indices reported in the literature will lead to coherence, as suggested by Bates (*33*). This was also examined to identify the plausibility of a cause and effect relationship.

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Health Indices and Adverse Health Effects

Bates (*33*) has put forward the proposition that different health indices can be compared to differing levels of air pollutant exposure and used to determine if a coherence exists among available indices, which would support a conclusion that a particular air pollutant results in an adverse health effect. A number of the different health indices listed by Bates (*33*) have been examined for asthmatics in various field studies and can be compared to ambient ozone concentrations as a test of his proposition. The health indices that have been reported are: self reports of symptoms, peak flow measurements, emergency department visits, and hospital admissions. If ozone affects the respiratory system of asthmatics, then increases in the number of individuals expressing these health metrics would be expected at higher ozone concentrations. Bates (*33*) proposed the examination of a single locale to establish the relationship among indices; the coherence among health indices should also be present across studies at different locations, but at a potentially weakened level. The overall hierarchial relationship expected is as follows: *a*) the greatest change in an index occurs for self-reporting of symptoms and peak flow decrements; *b*) intermediate increase occurs for the number of visits to ED; and *c*) the smallest change occurs for the number of hospital admissions. A second parameter that may exhibit a relationship among the health indices as a function of the ozone concentration is the time lag between exposure and the change in health status. Self reports of symptoms and decrements in peak flow are the most transient and sensitive measures and are expected to occur during or shortly following the exposure. ED visits for asthmatics are associated with more severe responses and are expected to require a longer time after the exposure to be observed. Hospital admissions result from the most severe responses; the time period from the onset of symptoms until the admission is recorded is expected to have the longest time lag. In addition, hospital admissions are expected to occur after the other health indices have been manifested in particular individuals. Bates (*33*) indicated that lags of 24 to 48 hr might be expected for the latter two health indices.

Materials and Methods

Data on visits to ED in nine central and northern New Jersey hospitals were provided by the Emergency Medical Association for 1986 through 1990. These were sorted and quality assured as described by Cody et al. (*26*). Respiratory admissions with *International Classification of Diseases* (ICD-9) codes 493.9, 493.90, and 493.91 were classified as asthma cases. Air pollution data for 1986 through 1990 were obtained for criteria air pollutants measured by the New Jersey Department of Environmental Protection. These were compiled in the U.S. Environmental Protection Agency's AIRS database. Meteorological data collected at the Airways Surface Measurement Station in Newark, New Jersey, were obtained from the National Climatic Data Center of NOAA.

Data collected were analyzed from May through August, which is the time of the year when ozone concentrations and photochemical smog are the greatest in central/northern New Jersey. The ozone concentration typically peaks in the early afternoon. Thus, to minimize possible misclassification of ED visits for asthma associated with ambient ozone concentration, only those ED visits for asthma that occurred between 15:00 and 09:00 of the following day were compared to the mean ozone measured between 10:00 and 15:00 on that day or previous days (e.g., lag 24 or 48 hr).

Statistical Methods

The statistical software package SAS (version 6.07; SAS Institute, Cary, NC) was used to assemble and merge databases and to conduct statistical analyses. To investigate the effects of autocorrelation, all models were run using time series regression (PROC AUTOREG). For each of the years analyzed, the Durbin-Watson statistic was close to 2 and the r^2 and p values agreed closely, implying that the relationships are not influenced by autocorrelation. The r^2 and p values also agreed closely, with the nonautoregressive models. The distribution of ED visit frequencies for all years were similar to those reported in Cody et al. (*26*) where normal distribution was found to be a suitable model by goodness of fit chi square. Multiple regression analyses were conducted using forward stepwise regression analysis. The number of asthma visits to the ED was used as the dependent variable. Ozone concentrations during the day of the visit; ozone concentrations for the day previous to the visit (lag 24) and for two days previous to the visit (lag 48); the mean, minimum, and maximum ambient air temperature between 9:00 and 15:00; the mean, minimum, and maximum relative humidity between 9:00 and 15:00; and the rate change in the temperature and relative humidity between 9:00 and 15:00 were used as independent variables. The multiple regression analysis was done independently for each year. Analysis of covariance was also conducted using an ozone cutoff of 0.06 ppm and adjusted for temperature to determine if days with higher ozone levels had statistically more ED visits for asthma than days with lower concentrations. The need to adjust for temperature in statistical analyses of the association between ozone and asthma has been previously justified (*21,26,28*).

Results

General Descriptors

The annual mean, standard deviation, and median of the number of ED visits for asthma, mean A.M. temperature, and mean 1-hr ozone concentrations (between 10:00 and 15:00) are given in Table 1. The mean and median number of daily ED visits for asthma varied from 4.49 to 6.05 and from 4 to 6, respectively. The temperature variation was small, with 1988 being approximately 0.5 °C warmer than the other years. The ozone concentrations were also similar across the five years, with an ANOVA test showing only 1989 to be statistically lower than the other years, $p = 0.0016$.

Multiple Regression Analysis

Regression analyses were conducted using forward stepwise regression for each year (Table 2). Regression analyses were also performed for daily ED visits based on several different time intervals including 15:00

Table 1. Descriptive statistics of the variables retained in the regression analysis.

Year	Variable	Mean	SD	Median
1986	Asthma visits	4.49	2.77	4
	Mean A.M. temp.,°C	25.1	4.66	26.1
	Ozone, ppm	0.055	0.020	0.054
1987	Asthma visits	5.96	3.16	6
	Mean A.M. temp.,°C	25.5	5.67	25.9
	Ozone, ppm	0.053	0.023	0.050
1988	Asthma visits	4.74	2.85	4
	Mean A.M. temp.,°C	25.8	6.06	26.6
	Ozone, ppm	0.057	0.028	0.051
1989	Asthma visits	5.58	3.11	5
	Mean A.M. temp.,°C	25.0	5.39	25.9
	Ozone, ppm	0.046	0.020	0.047
1990	Asthma visits	6.05	3.32	5
	Mean A.M. temp.,°C	24.5	5.11	25.6
	Ozone, ppm	0.053	0.023	0.053

Table 2. Forward stepwise regression results for the years 1986 to 1990.

Year	Parameter	Estimate	SE	r^2	p-value
1986	Mean A.M. temperature	−0.112	0.039	0.055	0.009
	Ozone	31.69	15.99	0.028	0.06
1987	Mean A.M. temperature	−0.183	0.042	0.106	0.0002
	Ozone	45.66	18.02	0.045	0.0126
1988	Mean A.M. temperature	−0.121	0.037	0.018	0.14
	Ozone	33.45	14.30	0.058	0.007
1989	Mean A.M. temperature	−0.177	0.036	0.033	0.047
	Ozone	79.04	17.98	0.156	0.0001
1990	Mean A.M. temperature	−0.293	0.040	0.287	0.0001
	Ozone	42.09	15.49	0.054	0.0024

to 15:00 the next day; 21:00 to 21:00 the next day; and 15:00 to 21:00. These time intervals were included to confirm that no selection basis was apparent for the chosen time interval. The regression parameters for each time interval were similar but had a slightly smaller r^2 value than the selected time period, 15:00 to 09:00 of the following day. A smaller r^2 value indicates a weaker correlation. This weaker correlation is thought to be due to inclusion of ED visits not associated with temperature or ozone from the day of interest, but rather the next day when the time interval is extended past 09:00, or due to the exclusion of ED visits associated with ozone when the time cutoff was 21:00; since ozone typically remained high into the nighttime, a lag of hours might occur between the exposure, the onset of symptoms, and a visit to an ED. Other dependent variables, such as rate of temperature change, relative humidity, 24-hr average sulfate concentrations made every sixth day, nitrogen dioxide, sulfur dioxide, and visibility were not correlated with ED visits for asthma. They also did not explain additional variance during the initial regression analyses. Therefore, these variables were not included in the final analyses. Temperature and ozone concentration were the only two independent variables that were selected by the forward stepwise regression analyses, using an entry criteria of $p < 0.15$. The stepwise regression analyses of any year did not include the O_3 concentration with a lag of 24 or 48 hr. Based on the sum of their partial r^2 values, temperature and ozone explained between 7.6% (1988) and 34% (1990) of the total variance. All of the ozone coefficients are positive indicating that the increased number of visits to the ED classified as asthmatic was related to increases in ozone. The largest regression coefficient for ozone was found in 1989, the year with the lowest overall mean ozone concentration. An inverse relationship between temperature and visits to the ED was found for all years.

To investigate the effect of long-term temperature trends in our data, we ran two additional regression models, one restricted to the months of July and August, another to the months of June, July, and August. Other researchers (GD Thurston, personal communication) have indicated that long-wave temperature trends would not be a factor in these intervals. We ran the model for each of the years 1986 to 1990 and then with the years combined, to provide a more stable estimate of the regression coefficients. Using the combined 5 years of data, the ozone coefficient for the 2- and 3-month models was 15.99 ($p = 0.65$) and 19.3 ($p = 0.02$), respectively. Although these coefficients are smaller than those obtained in the larger model, they seem to indicate that long-term temperature changes are not seriously affecting the model.

Covariance Analysis

To test whether the mean number of ED visits above and below a cutoff ozone concentration differed, covariant analysis was performed for the number of ED visits for asthma and adjusted for temperature. The results of the analyses for each year using a cutoff of 0.06 ppm are given in Table 3. A cutoff of 0.06 ppm was chosen since: *a*) it is the concentration where changes in peak expiratory flow rates were observed (*20*); *b*) it is the lowest national standard currently enforced; and *c*) it provides a sufficient number of ED visits in both the above and below cutoff groups to detect statistical differences between two groups. The difference between the mean number of ED visits during the days when ozone exceeded 0.06 ppm was greater than on days when ozone was less than 0.06 ppm for all 5 years (Table 3). When all years were combined, a difference of 1.07 visits (5.27–4.20) was calculated which was statistically significant at a p-value of < 0.0001. The significant ozone-year interaction ($p = 0.0029$) indicates that the effect of the ozone level on asthma visits was not homogenous across years, although the direction was for increased visits at higher ozone levels for all five years (Figure 1).

Discussion

The number of visits to ED for asthmatics on days when summertime smog is higher was elevated in central New Jersey for all 5

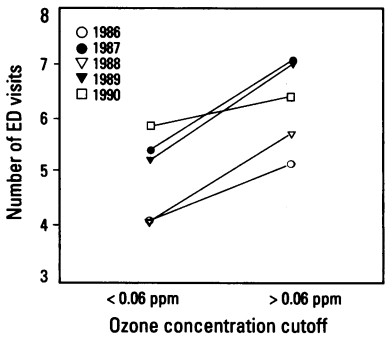

Figure 1. Mean concentration of ED visits for days with low and high ozone concentrations using a cutoff of 0.06 ppm for 1986 to 1990.

Table 3. Analysis of covariance results of ED visits using a cutoff ozone concentration of 0.06 ppm.

Year	Parameter	p-Value	Adjusted mean ED visit for low O_3 (<0.06 ppm)	Adjusted mean ED visit for high O_3 (≥0.06 ppm)	Difference in ED visits (high − low)
1986	Mean A.M. temperature	0.0016			
	Ozone level	0.0697	4.08	5.14	1.06
1987[a]	Mean A.M. temperature	0.0001			
	Ozone level	0.0243	5.40	7.07	1.67
1988	Mean A.M. temperature	0.0081			
	Ozone level	0.0216	4.07	5.69	1.62
1989	Mean A.M. temperature	0.0040			
	Ozone level	0.0226	5.20	6.99	1.79
1990	Mean A.M. temperature	0.0001			
	Ozone level	0.4082	5.83	6.40	0.57
All years two-way model	Mean A.M. temperature	0.0001			
	Ozone level interaction	0.0001			
	(Ozone*year)	0.0001	4.20	5.27	1.07

[a]Failed test for homogeneity of slope of the covariate by ozone level ($p = 0.0179$).

years. The regression coefficient of daily ED visits with ozone concentration (ppm) was positive in all years and varied between 32 and 79 indicating that an increase of between 0.3 and 0.8 visits/day is associated with each 0.01 ppm increase in ozone. The partial r^2 terms varied from 0.028 to 0.16 indicating that between 3 and 16% of the variance in number of ED visits was explained by the variations in ambient ozone concentrations. The mean A.M. temperature was inversely related to ED visits, consistent with studies by Whittemore (*21*) and Holguin (*18*) on changes in number of symptoms and decrements in forced expiratory volume (FEV) of asthmatics, but opposite the temperature and respiratory relationship observed by Thurston et al. (*28*). Since the temperature and ozone are highly correlated, the interrelationship between the two is important to decouple when analyzing pollutant health effect associations. Further, some of the variance that our regression analysis attributed to temperature could also be related to the ozone concentration since temperature entered the regression equation first.

The analysis of covariance showed a statistically significant difference in the mean number of visits to the ED for asthma on days when the ambient ozone concentration was > 0.06 ppm, when compared to days when the ambient ozone concentration was < 0.06 ppm. Overall, an additional 1.07 visits occurred on days when the ozone was higher than 0.06 ppm, which represents a 26% increase in daily visits.

The observed 26% increase in ED visits for asthmatics on higher ozone concentration days suggests that it may be feasible to examine the relationship of ozone with various health indices of asthma as outlined by Bates (*33*). A comparison of reported associations between health indices and ozone are given in Table 4. The overall relationship observed among the health indices and ozone is consistent with the coherence proposed by Bates (*33*), since the number of asthmatics affected was highest for the number of reported symptoms and decrements in measurements of peak flow, intermediate for ED visits, and smallest for hospital admissions. Similarly, the reported time lag between exposure and response increases in the same order. This trend is true even though the studies reported here are for different locations and populations, which would increase the number of confounders. The coherence of the reported exposure/response indices support the hypothesis that ambient ozone levels adversely affect the respiratory tract of asthmatics.

A detailed examination of the studies reported in Table 4 suggests why some studies attain coherence and others deviate from the expected coherence. The studies reporting symptoms and peak flow measurements of asthmatics have largely been done as panel studies. Kurata et al. (*29*) did not find an association between asthma symptoms and ozone concentration, but no control for temperature was done, which other studies have shown to be an important confounder. Holguin (*18*) followed 51 asthmatics in Houston and performed a multiple linear regression analysis using asthma attack status as a dependent variable and ozone, nitrogen dioxide, temperature, humidity, pollen, and asthma attack during previous 12-hr period as the independent variables. The risk of attack was found to be positively associated with ozone and inversely associated with temperature. He calculated that a 0.04 ppm increase in ozone levels resulted in an increase in the relative risk of attack by an individual of 1.2 to 2.2, dependent upon that individual's baseline probability of having an attack. Khan (*20*) followed 80 asthmatic children and observed that number, duration, and severity of asthmatic attacks were correlated with high ozone levels, with the environmental and meteorological factors accounting for between 5 to 15% of the total variance, but did not calculate a percent increase in incidents. A study in Los Angeles County, California, which examined medication use and peak expiratory flow rates (PEFR) in 83 asthmatics during 230 days, determined a consistent, statistical relationship between ozone concentration and respiratory status in 76% of the subjects and clinically significant response in symptom scores and PEFR predicted by the ozone concentration in 10% of the subjects (*16*). This differential

Table 4. Percent increase in morbidity with increased ozone concentration.

Health metric examined	Study	Location	Temperature covariance controlled	Mean (max) ozone conc., ppm	Ozone cutoff, ppm	Percent increase in incidents	Time lag to response
Self-report	Kurata et al., 1976 (*29*)	Los Angeles, CA	No	0.05 to 0.12 (0.37)	0.2	None	
of symptoms	Khan, 1977 (*20*)	Chicago, IL	Yes	Not provided	—	Not reported	
and	Holguin et al., 1987 (*18*)	Houston, TX	Yes	0.07	0.08–0.12	20–120%	Same day
peak flow	Krzyzanowski, 1992 (*19*)	Tucson, AZ	Yes	0.055 (0.092)	0.06	Not reported	Within hours
	Gong, 1987 (*16*)	Los Angeles, CA	Yes	0.1 (0.4)	>0.12	76% population for symptoms 9.6% for clinical response	Same day
	Whittemore, 1980 (*21*)	Los Angeles, CA	Yes	0.04 to 0.15		66%	Not reported
	Abbey et al., 1993 (*12*)	Southern California	No	0.3 (9.6)		40% new asthma cases	
Emergency	Bates et al., 1990 (*31*)	Vancouver, Canada	Yes	0.04 (0.08)		None	
department	White et al., 1991 (*24*)	Atlanta, GA	Yes	Not provided	0.11	38%	Later same day
	Ponka, 1991 (*25*)	Helsinki, Finland	Yes	0.01 (0.05)	0.02	6% (NS)	Same day, lag 1
	Rennick et al., 1992 (*32*)	Melbourne, Australia	No	Not provided	0.09	None	and 2 days
	This study	Central New Jersey	Yes	0.05 (0.13) 0.08–0.12 (regress.)	0.06 (Covar.)	28% 35%	Later same day
Hospital	Richards et al., 1981 (*30*)	Los Angeles, CA	No	0.03–0.14 (0.07–0.4)	—	None	
admissions	Bates and Sitzo, 1987 (*34*)	Ontario, Canada	Yes	0.04–0.06 (0.16)	0.082	7%	Lag 1 and 2 days
	Ponka, 1991 (*25*)	Helsinki, Finland	Yes	0.01 (0.05)	0.02	7% (NS)	Lag 1 and 2 days
	Thurston et al., 1992 (*28*)	Buffalo, NY	Yes	0.07 (0.16)	0.07–0.15	29%	Lag 3 days
		New York, NY		0.06 (0.21)	0.07–0.15	23%	Lag 3 days

NS, difference was not statistically significant; all other percent increase reported as statistically significant by authors at $p < 0.05$.

response among subjects indicates that even in a population thought to be sensitive to respiratory irritants (asthmatics), different degrees of response exists. Whittemore and Korn (*21*) identified a positive association between the number of attacks experienced by individuals in 16 panels of asthmatics in the Los Angeles area and the oxidant levels in southern California when controlling for temperature. They calculated an odds ratio of 1.66 for an asthmatic attack for ozone when other factors were held fixed. Studies of Seventh Day Adventists, whose lifestyle prohibits the use of smoking, a confounder in respiratory studies, found a statistically significant relationship between mean ozone concentration and the incidence of asthma and calculated a 40% higher incident of asthma for areas with higher ozone concentrations (*12,23*). Measurements of FEV_1 PEFR have been shown to decline by several percent in asthmatics during days of high ozone levels using a cutoff of 0.06 ppm (*19*).

Studies of more severe responses of asthmatics, requiring medical treatment at emergency departments or hospital admission as a function of ozone concentration, have been done. No increase in the number of visits to physician or ED or hospital admissions were identified in asthmatic children by Richards et al. (*30*), Bates et al. (*31*), or Rennick and Jarman (*32*). In the studies by Richards et al. (*30*) and Rennick and Jarman (*32*), the effect of temperature on asthmatics was not taken into account. Bates et al. (*31*) chose to study a region that had ambient ozone levels typically below 0.06 ppm in order to study effects of acidic aerosols on asthmatics in the absence of high oxidant levels. Comparison of the number of visits to ED for asthma attacks within inner cities on high ozone days with low ozone days has shown statistically significant elevations in Atlanta with a 38% increase on high ozone days (*24*). The current study identified a 30% increase in ED visits during a 5-year period. Neither study found a time lag between the day of exposure and the visit to the ED, as suggested by Bates (*33*). Ponkä (*25*) found a small increase in ED visits, with an indication of a lag of 24 to 48 hr between exposure and ED visits, but this study was conducted when the ozone concentrations were much lower than observed in other studies. He also identified a similar small increase, with a longer time lag, for hospital admissions.

Hospitalization of patients for asthma has been found to be elevated by 7% on days following high ozone with lags of 24 to 48 hr in Ontario, Canada (*34*). Larger increases in hospitalization, 29 and 23%, were identified in data collected in Buffalo, New York, and New York, New York, respectively, which have higher ozone levels than Canada (*28*). In these two studies, temperature, auto correlations, and day of week effects were removed via regression analysis. Ponkä (*25*) identified a correlation between ozone concentration and ED visit and hospital admission, but the increase between two stratified groups was not statistically significant, probably due to the relatively low ozone levels present, < 0.05 ppm.

The studies outlined in Table 4 that have been careful to control for temperature effects have generally established a statistically significant association between ambient ozone levels and adverse respiratory responses by asthmatics, when the levels of ozone have been sufficiently high. The common lower end cutoff is 0.06 ppm although one study observed effects at lower concentration (*25*). This consistent trend is across diverse cities, often without control of numerous confounders that occur in epidemiological studies including: the lack of control or knowledge of medications used, possible adaptation by subjects to ozone's effect, the maximum ozone concentrations, and the number of days that ozone exceeded a level that effects occur, such as 0.06 ppm.

Conclusions

An increase in the number of the ED visits for asthma with increasing ambient ozone concentration has been identified in central New Jersey for 5 consecutive years, 1986–1990. An estimated increase of 26% in the number of daily ED visits for asthma occurred for days when the ozone level was > 0.06 ppm, compared to days when it was < 0.06 ppm. An evaluation of the changes in percent morbidity reported for asthmatics for different health indices, as proposed by Bates (*33*), presents a coherent picture that ozone does adversely affect asthmatics at levels that are below the current U.S. standard.

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