Impact of Changes in Transportation and Commuting Behaviors During the 1996 Summer Olympic Games in Atlanta on Air Quality and Childhood Asthma

Michael S. Friedman, MD

Kenneth E. Powell, MD, MPH

Lori Hutwagner, MS

LeRoy M. Graham, MD

W. Gerald Teague, MD

DESPITE ADVANCES IN ASTHMA therapy, asthma remains a substantial public health problem. In the United States, asthma is a leading cause of childhood morbidity, with an estimated prevalence of 6.9% in children and youth younger than 18 years.[1] Numerous studies have documented a rise in the morbidity, mortality, and prevalence of asthma in different populations.[2-8] The cause or causes of this trend remain controversial.[9-11]

Experimental, laboratory, and epidemiologic studies in the last several years have linked high concentrations of known air pollutants to respiratory health problems, most notably exacerbations of asthma.[12-23] However, opportunities to study the health effects of anthropogenic improvements in air quality are rare. One study found a decrease in particulate pollution and respiratory hospital admissions associated with the closure of an industrial factory in that community.[24] To our knowledge, no study has examined the impact of improved ozone pollution for an extended period of time on asthma exacerbations or other markers of asthma morbidity. Also, the extent to which moderate concentrations of

ozone (ie, daily peak of 50-100 ppb) during various exposure lengths affects asthma morbidity remains controversial.[12-16]

Context Vehicle exhaust is a major source of ozone and other air pollutants. Although high ground-level ozone pollution is associated with transient increases in asthma morbidity, the impact of citywide transportation changes on air quality and childhood asthma has not been studied. The alternative transportation strategy implemented during the 1996 Summer Olympic Games in Atlanta, Ga, provided such an opportunity.

Objective To describe traffic changes in Atlanta, Ga, during the 1996 Summer Olympic Games and concomitant changes in air quality and childhood asthma events.

Design Ecological study comparing the 17 days of the Olympic Games (July 19–August 4, 1996) to a baseline period consisting of the 4 weeks before and 4 weeks after the Olympic Games.

Setting and Subjects Children aged 1 to 16 years who resided in the 5 central counties of metropolitan Atlanta and whose data were captured in 1 of 4 databases.

Main Outcome Measures Citywide acute care visits and hospitalizations for asthma (asthma events) and nonasthma events, concentrations of major air pollutants, meteorological variables, and traffic counts.

Results During the Olympic Games, the number of asthma acute care events decreased 41.6% (4.23 vs 2.47 daily events) in the Georgia Medicaid claims file, 44.1% (1.36 vs 0.76 daily events) in a health maintenance organization database, 11.1% (4.77 vs 4.24 daily events) in 2 pediatric emergency departments, and 19.1% (2.04 vs 1.65 daily hospitalizations) in the Georgia Hospital Discharge Database. The number of nonasthma acute care events in the 4 databases changed –3.1%, +1.3%, –2.1%, and +1.0%, respectively. In multivariate regression analysis, only the reduction in asthma events recorded in the Medicaid database was significant (relative risk, 0.48; 95% confidence interval, 0.44-0.86). Peak daily ozone concentrations decreased 27.9%, from 81.3 ppb during the baseline period to 58.6 ppb during the Olympic Games ($P<.001$). Peak weekday morning traffic counts dropped 22.5% ($P<.001$). Traffic counts were significantly correlated with that day's peak ozone concentration (average $r=0.36$ for all 4 roads examined). Meteorological conditions during the Olympic Games did not differ substantially from the baseline period.

Conclusions Efforts to reduce downtown traffic congestion in Atlanta during the Olympic Games resulted in decreased traffic density, especially during the critical morning period. This was associated with a prolonged reduction in ozone pollution and significantly lower rates of childhood asthma events. These data provide support for efforts to reduce air pollution and improve health via reductions in motor vehicle traffic.

JAMA. 2001;285:897-905   www.jama.com

Author Affiliations are listed at the end of this article.
Corresponding Author and Reprints: Michael S. Friedman, MD, Air Pollution and Respiratory Health Branch, National Center for Environmental Health, Centers for Disease Control and Prevention, Atlanta, GA 30333 (e-mail: mff7@cdc.gov).

The main sources of ambient air pollutants are vehicle exhaust, industry, power generation plants, and background contamination. Compared with emissions from nonvehicle sources, the relative amounts of nitrogen oxides, carbon monoxide, and small particulate matter emitted from vehicles have increased disproportionately due to the dramatic increase in worldwide automobile use in the past 30 years.[12,15] Many studies have found positive associations between traffic density on street of residence and either asthma events or asthma prevalence.[25-29] However, the impact of citywide automobile use and traffic flow on ambient air pollution and asthma morbidity has not been studied.

The 1996 Summer Olympic Games in Atlanta, Ga, provided a unique opportunity to study the relationship between automobile traffic, air quality, and asthma morbidity. Preparations for the Olympic Games required a strategy for minimizing road traffic congestion and ensuring that spectators could reach Olympic events in a reasonable amount of time. Additionally, the more than 1 million visitors to Atlanta (resulting in increased regional transportation demands) could have magnified the region's existing air quality violations for ozone pollution that occur each summer. Atlanta's strategy was similar to that used in Los Angeles, Calif, for the 1984 Summer Olympic Games.[30] It included the development and use of an integrated 24-hour-a-day public transportation system, the addition of 1000 buses for park-and-ride services, local business use of alternative work hours and telecommuting, closure of the downtown sector to private automobile travel, altered downtown delivery schedules, and public warnings of potential traffic and air quality problems.[31-33]

METHODS
Study Design

We used an ecological study design and compared the 17 days of the 1996 Summer Olympic Games (July 19–August 4) to a summertime baseline period defined as the 4-week periods before and after the Olympic Games (June 21–July 18 and August 5–September 1). We measured the number of asthma acute care events, number of nonasthma acute care events, air pollution, meteorological conditions, and amount of vehicular traffic. The specific dates for study were determined before any data were analyzed. Four-week baseline periods were used to avoid the spring and fall seasons, which can affect ozone levels[12,13] and asthma exacerbation rates.[34]

Medical Definitions and Data Collection

Our primary outcome measure was the number of hospitalizations, emergency department visits, and urgent care center visits for asthma. The study population included persons aged 1 through 16 years residing in the 5 central counties of metropolitan Atlanta (ie, Fulton, DeKalb, Cobb, Gwinnett, and Clayton). Because of their central location, these densely populated counties were more likely to experience dramatic changes in air quality in response to changes in driving and commuting behaviors (ie, greater access to public transportation as an alternative). Visitors to Atlanta were excluded from the study.

Data regarding asthma acute care events were collected from Georgia's Medicaid claims file, the patient database of a health maintenance organization (HMO), computerized emergency department records from 2 of the 3 pediatric hospitals in Atlanta (combined to create a single emergency department data source), and the Georgia Hospital Discharge Database, which includes hospitalization records from all metropolitan Atlanta hospitals. Medical records at the 1 publicly funded pediatric hospital in Atlanta were not available for review. However, 60% to 66% of children seeking emergency care at that hospital were receiving Medicaid in 1996 (Robert J. Geller, MD, written communication, January 17, 2001). Therefore, we believe that the Medicaid files captured clinical information on most of this population. For the Georgia Hospital Discharge Database, a child admitted to a metropolitan At-lanta hospital during the study period with a primary diagnosis of asthma (*International Classification of Diseases, Ninth Revision [ICD-9]*, code 493) met our study definition for an asthma acute care event. For the other 3 data sources, a child seen in an emergency department or urgent care center with a primary diagnosis of asthma met our study definition for an asthma acute care event, regardless of whether the child was hospitalized.

To determine if the study population was more, less, or as likely to seek emergency services in general during the Olympic period compared with the periods before and after the Olympic Games, or if the size of the study population significantly changed during the Olympic Games, we collected and analyzed data on the day-by-day total number of all nonasthma-related acute care events during the study period among Atlanta residents aged 1 through 16 years from the same 4 data sources.

Air Quality Data Collection

All data on primary pollutants (ie, particulate matter of 10 μm or smaller [PM_{10}], carbon monoxide, nitrogen dioxide, and sulfur dioxide) and secondary pollutants (ozone) were obtained jointly from the Environmental Protection Agency (EPA) and the Environmental Protection Division of Georgia's Department of Natural Resources. All air quality measurement sites used in the study were located in the 5 central counties of Atlanta and were state operated and EPA regulated (**FIGURE 1**).

The ozone concentration chosen to represent each day's exposure was the average of the peak 1-hour ozone concentrations from the 3 monitoring sites in the study area. The daily 1-hour peak ozone levels for the same time periods in 1997-1998 were used for comparison. To see if other areas in the region with similar weather patterns experienced similar ozone patterns during the 1996 summer, we obtained daily 1-hour peak ozone concentrations at 3 other Georgia sites (Fannin County, Augusta, and Columbus), all 60 to 150 miles from Atlanta.

We used the EPA's standard method of measuring the 4 major primary pollutants.[35] Data on PM_{10} were collected at the 1 site capable of continuous 24-hour monitoring and expressed as the cumulative total for each 24-hour period. The mean 8-hour running daily peak carbon monoxide level, the 24-hour daily mean sulfur dioxide level, and the 1-hour daily peak nitrogen dioxide level were collected at 1, 2, and 3 monitoring sites, respectively. The daily levels obtained at the 2 sulfur and 3 nitrogen dioxide sites were then averaged. Allergen exposure was determined by total daytime mold counts (the predominant summertime allergen in Atlanta) collected weekdays during the study period at the Atlanta Allergy and Asthma Clinic.

Meteorological Data

We obtained hourly data for 5 weather-related variables (temperature, wind speed, relative humidity, barometric pressure, and solar radiation) from a state-run weather monitoring site located east of downtown Atlanta. These 5 variables have a direct or indirect impact on the rate of ozone formation and clearance in the lower atmosphere and, to a lesser extent, can affect levels of the primary pollutants.[12] For each day and for each variable, we calculated the mean of the 12 readings from 6 AM to 6 PM.

Traffic Counts

We obtained hourly traffic data collected by the Georgia Department of Transportation from the 4 functioning sites (2 highways and 2 local roads) located within Atlanta's perimeter interstate highway. Total 24-hour and 1-hour morning peak bidirectional traffic counts were available for analysis for 92% of the study weekdays and 85% of the study weekend days.

Public Transportation Data

We examined the total number of passenger trips per day on Atlanta's public buses and rail lines during the study period. The Metro Atlanta Rapid Transit Authority provided data for average weekday and weekend daily rider-

Figure 1. Functioning Traffic Count and Air Quality Monitoring Sites in Atlanta, Ga, During the 1996 Summer Olympic Games
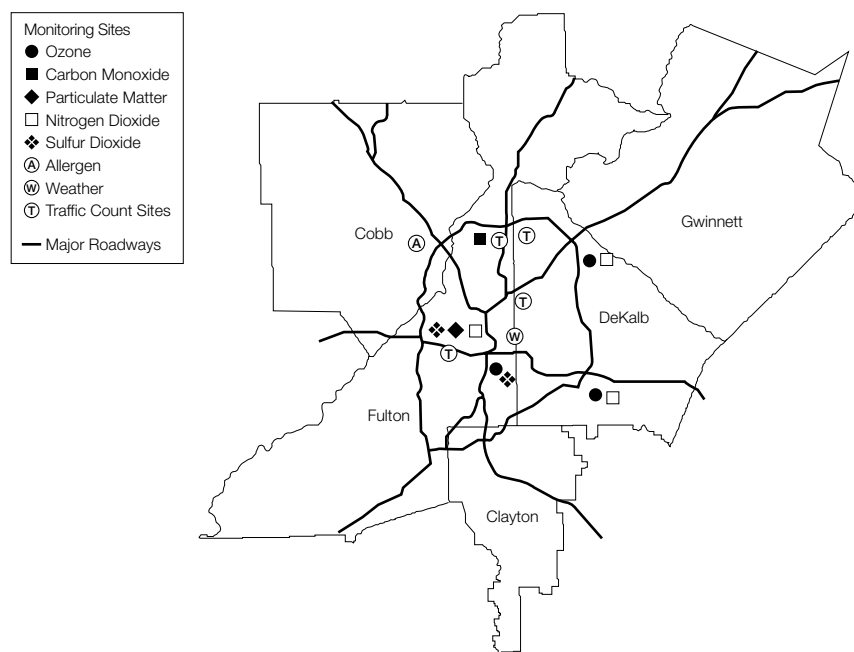
Borders indicate the 5 central counties of metropolitan Atlanta included in the study.

ship totals for the pre- and post-Olympic Games periods. For the Olympic period, totals for each of the 17 days were available for analysis.[33]

Statewide Gasoline Sales

We collected and compared total gallons of gasoline purchased in the state of Georgia in June, July, and August 1991-1997. The Georgia State Department of Revenue routinely calculates these gallon totals based on receipts of the state fuel tax. The month of purchase was determined by the month in which the fuel distributors delivered the gasoline to individual filling stations.

Statistical Analysis

We analyzed all collected data to determine the percentage change in mean values during the Olympic period compared with the 1996 summertime baseline period. One-way analysis of variance t testing was used to determine if the daily air pollutant, meteorological, and traffic count values differed significantly between the 2 study periods. Significance was defined as $P \le .05$.

Further analysis of the asthma event data was performed using a time-series Poisson regression model. Univariate and adjusted relative risk (RR) (with 95% confidence intervals [CIs]) of asthma acute care events during the Olympic period compared with the baseline period was calculated for each of the 4 sources of data. The univariate analysis was based on the fraction of total acute care events with a primary diagnosis of asthma. The multivariate time-series Poisson model was fitted using generalized estimating equations to address possible serial (auto) correlation in the number of asthma events.[36] Models were implemented using the GENMOD procedure in SAS with AR(1) (SAS Institute Inc, Cary, NC) to account for correlation in asthma events on a given day with the previous day's events. The Durbin-Watson statistic was 1.96-2.01, which indicates minimal residual serial correlation. This model was adjusted for day of the week (weekday vs weekend) and minimum temperature (lagged 1 day to improve the fit of the model). Total mold counts were only

collected on weekdays, and therefore, were not included in the multivariate model. Seasonality and time trends were not included in this model due to the very short study period covering only the summer season. The Olympic period was modeled here instead of ozone or PM_{10} levels due to the correlation between these variables and because we wanted to emphasize the changes directly associated with the Olympic Games.

A second autoregressive time-series model analyzed the change in ozone concentrations in Atlanta and the other 3 Georgia sites during the Olympic Games. The model was used to adjust for day of the week (weekday vs weekend) and trends in weather conditions (mean daytime wind speed, temperature, solar radiation, and barometric pressure). Relative humidity was not included because it was strongly correlated with solar radiation (inverse relationship). The model (using AR[1]) found minimal serial correlation in daily ozone levels (Durbin-Watson statistic, 1.94).

In a separate analysis, Pearson coefficients were used to measure the correlation between morning peak–total traffic counts and that day's peak ozone concentration. Significance was defined as $P \leq .05$.

In addition to our analyses comparing the Olympic period with the baseline period, data for all 73 study days were used to examine the relationship between ozone accumulation and daily asthma acute care events. The mean number of asthma events was determined for days in which the peak ozone concentrations for that day plus the previous 2 days averaged less than 60 ppb (low cumulative exposure), 60 to 89 ppb (moderate cumulative exposure), or 90 ppb or more (high cumulative exposure). Controlling for day of the week and minimum temperature (lagged 1 day) in a Poisson regression model, the RR of asthma events was determined at cumulative ozone concentrations of 60 to 89 ppb and 90 ppb or more compared with ozone concentrations less than 60 ppb.

To test our hypothesis that cumulative exposures to air pollutants for 2 to 3 days are more strongly associated with asthma acute care events than traditional single-day exposures, we compared the RR of asthma events per 50 ppb incremental change in ozone and 10-$\mu g/m^3$ incremental change in PM_{10} levels based on a same-day, 2-day, and 3-day cumulative exposure measure. We used time-series regression models similar to our primary asthma event model except that the dependent variable (Olympic vs baseline period) was replaced by a single pollutant exposure variable.

RESULTS

Among children aged 1 through 16 years in the Medicaid claims file database, the number of asthma emergency care visits and hospitalizations decreased from 4.23 events per day during the baseline period to 2.47 events per day during the Olympic period, a 41.6% overall decrease (TABLE 1). The number of asthma-related emergency department, urgent care visits, and hospitalizations among HMO enrollees decreased 44.1% during the same time period. Asthma-related visits to 2 large pediatric emergency departments in Atlanta decreased 11.1%, and citywide hospitalizations for asthma were reduced 19.1%. Using our 4 data sources, this equates to respectively 30, 10, 9, and 7 fewer emergency asthma events during the Olympic Games than would have been expected based on the pre- and post-Olympic period averages.

The total number of nonasthma-related acute care events per day decreased only 3.1% in the Medicaid database and 2.1% at the pediatric emergency departments; this number increased 1.3% among HMO enrollees. Nonelective nonasthma hospitalizations increased 1.0% during the Olympics.

As illustrated in TABLE 2, the univariate RR of asthma acute care events during the Olympics compared with the baseline period was significantly reduced in the Medicaid database (RR, 0.61; 95% CI, 0.44-0.85) and approached statistical significance in the HMO database (RR, 0.56; 95% CI, 0.31-1.02). Although less than 1, the RR at the 2 pediatric emergency departments and among those hospitalized was not significant (RR, 0.91 and 0.81, respectively). Adjusting the RR to con-

Table 1. Acute Asthma Events and Acute Nonasthma Events Among Children and Youth During the 1996 Summer Olympic Games Compared With the 1996 Summertime Baseline Period

Data Source	Type of Asthma Event	Acute Asthma Events			Acute Nonasthma Events		
		Mean (SD) No. of Events Per Day			Mean (SD) No. of Events Per Day		
		Baseline Period*	Olympic Period†	% Change	Baseline Period*	Olympic Period†	% Change
Georgia Medicaid claims file	Emergency care and hospitalizations	4.23 (2.81)	2.47 (1.46)	−41.6	100.5 (18.6)	97.4 (16.4)	−3.1
Health maintenance organization	Emergency care, urgent care, and hospitalizations	1.36 (1.63)	0.76 (0.83)	−44.1	37.6 (19.6)	38.1 (18.4)	+1.3
Pediatric emergency departments	Emergency care and hospitalizations	4.77 (2.52)	4.24 (2.49)	−11.1	118.4 (20.5)	115.9 (15.9)	−2.1
Georgia Hospital Discharge Database	Hospitalizations	2.04 (1.53)	1.65 (1.50)	−19.1	19.7 (5.1)	19.9 (3.5)	+1.0

*Defined as June 21–July 18 and August 5–September 1, 1996.
†Defined as July 19–August 4, 1996.

trol for minimum temperature and day of the week did not alter these findings. The data from Atlanta's Medicaid database remained statistically significant (adjusted RR, 0.48; 95% CI, 0.44-0.86).

FIGURE 2 shows the daily time series of each of the 5 measured pollutants and mold counts during the Olympic and baseline periods. The 1-hour peak ozone concentration in Atlanta decreased 27.9% from an average daily peak of 81.3 ppb during the baseline period to 58.6 ppb during the Olympic Games ($P<.001$). The daily ozone concentrations at the 3 monitoring sites in Atlanta were highly correlated ($r=0.91-0.97$). Combined ozone data for the 1997 and 1998 summer seasons did not show a similar decrease—the average peak ozone concentration was 77.2 ppb during July 19–August 4 and 78.8 ppb during the remainder of the study dates.

Ozone concentrations at Georgia sites outside of Atlanta decreased 11.1% in Augusta (58.8 ppb vs 66.2 ppb; $P=.11$), 17.5% in Fannin County (50.5 ppb vs 61.2 ppb; $P=.003$), and 18.5% in Co-

lumbus (52.2 ppb vs 64.1 ppb; $P=.01$) during the Olympic Games. These ozone reductions were, respectively, 60%, 37%, and 34% less than the ozone reductions experienced in Atlanta during the same period with similar weather conditions. After controlling for the 4 weather variables, serial correlation, and day of the week in a time-series regression model, the reduction in Atlanta's ozone concentrations during the Olympic Games was 13% ($P=.06$). Comparatively, the reduction in ozone was calculated at 2% in Augusta ($P=.71$), 7% in Fannin County ($P=.12$), and 6% in Columbus ($P=.30$).

During the Olympic period, Atlanta additionally experienced significant reductions in daily carbon monoxide levels (1.26 vs 1.54 ppm, 18.5% decrease; $P=.02$) and PM_{10} concentrations (30.8 vs 36.7 µg/m^3, 16.1% decrease; $P=.01$). Nitrogen dioxide levels decreased 6.8% (36.5 vs 39.2 ppb; $P=.49$), whereas sulfur dioxide levels increased 22.1% (4.29 vs. 3.52 ppb; $P=.65$). FIGURE 3 summarizes these findings relative to the EPA National Ambient Air Quality Standards for each of these pollutants.[35] Data for the baseline period are divided into pre- and post-Olympic time periods

Table 2. Univariate and Adjusted Relative Risk of Acute Asthma Events During the 1996 Summer Olympic Games Compared With the 1996 Summertime Baseline Period*

Data Source	Univariate RR† (95% CI)	P Value	Adjusted RR‡ (95% Confidence Interval)	P Value
Georgia Medicaid claims file	0.61 (0.44-0.85)	.003	0.48 (0.44-0.86)	.005
Health maintenance organization	0.56 (0.31-1.02)	.06	0.58 (0.32-1.06)	.10
Pediatric emergency departments	0.91 (0.85-1.42)	.48	0.93 (0.71-1.22)	.69
Georgia Hospital Discharge Database	0.81 (0.54-1.23)	.34	0.71 (0.46-1.11)	.22

*RR indicates relative risk; CI, confidence interval. For definition of baseline period, see "Study Design" subsection of "Methods" section.
†Relative risk based on Poisson model (fraction of total acute care events with a primary diagnosis of asthma).
‡Time-series regression analysis was adjusted for day of week (weekday vs weekend) and minimum daily temperature (lagged 1 day to minimize serial correlation).

Figure 2. Daily Time Series of Individual Air Pollutant Levels and Mold Counts During the 1996 Summer Olympic Games and Baseline Period
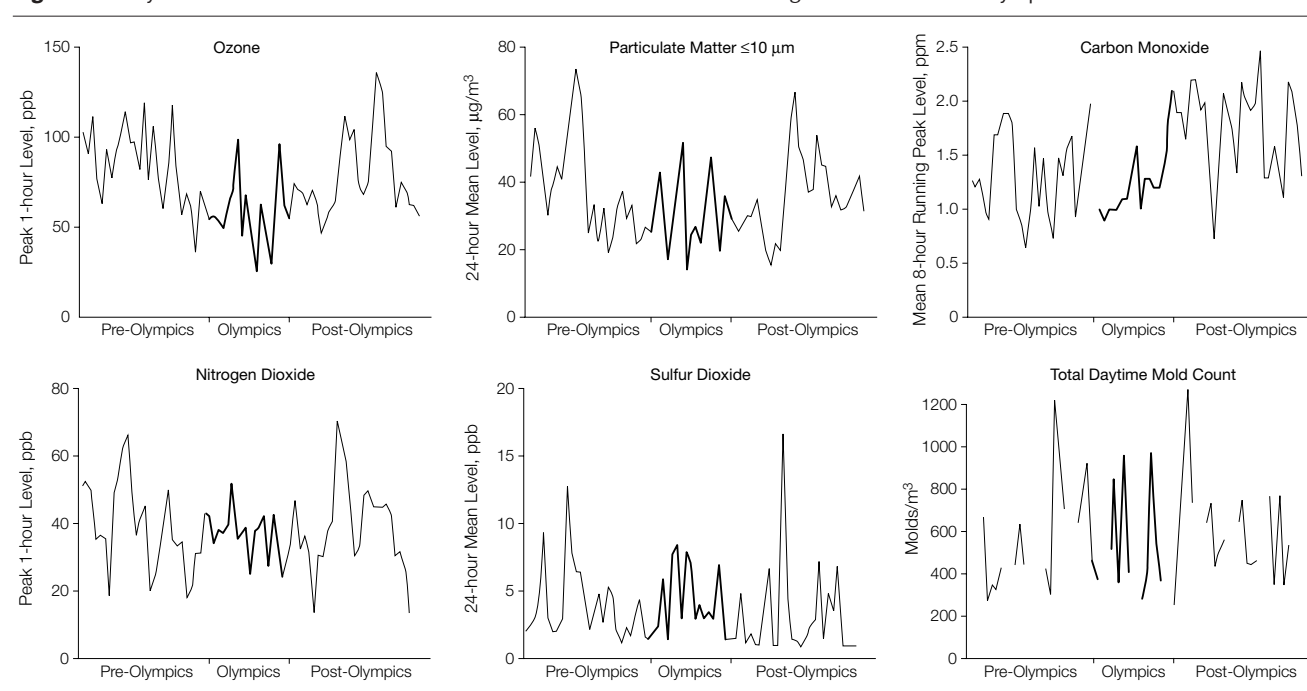
Broken line indicates incomplete data (eg, mold counts were available weekdays only).

demonstrating the uncharacteristic decrease in air pollution levels during the Olympic Games.

Mean daytime weather conditions in Atlanta were determined for both the Olympic and baseline periods. Temperature decreased 0.67°C, wind speed increased 0.19 m/sec, and solar radiation decreased 29.6 W/m² during the Olympic Games. These changes were not statistically significant. Barometric pressure did not change. Total mold counts did not differ significantly during the Olympic vs the baseline period (daily mean, 597 vs 551 molds/m³; $P = .58$; Figure 2). Moreover, mold

Figure 3. Mean Levels of Major Pollutants Before, During, and After the 1996 Summer Olympic Games as a Percentage of the National Ambient Air Quality Standard (NAAQS)

National Ambient Air Quality Standard at time of study: ozone 1-hour peak average, 120 ppb; particulate matter of 10 μm or smaller (PM$_{10}$) 24-hour average, 150 μg/m³; carbon monoxide (CO) 8-hour average, 9 ppm; nitrogen dioxide (NO$_2$) 1-hour peak average, 600 ppb; sulfur dioxide (SO$_2$) 24-hour average, 140 ppb.[35]

counts were not correlated with same-day asthma events (average $r = -0.15$).

Weekday 1-hour morning peak traffic counts decreased 22.5% overall during the Olympic Games (range, 17.5%-23.6%; $P<.001$ for all 4 sites). This amounted to a reduction of 4260 vehicle trips during the peak morning traffic hour on these 4 roads. Weekend morning peak traffic counts decreased 9.7% overall (range, 3.6%-12.3%), although only the change in traffic counts at the site closest to downtown was significant. Weekday total 24-hour traffic counts decreased 2.8% overall (range, 1.3%-3.6%), with the significant changes occurring at the 2 sites closest to downtown. Public transportation ridership increased 217% (190% on weekdays; 334% on weekends) during the Olympic Games. A total of 17.5 million more trips occurred on public transportation throughout the Olympic Games than would be expected based on the baseline period ridership.

Weekly total gallons of gasoline purchased in Atlanta were not available for analysis. The number of gallons of gasoline purchased statewide in July 1996 was 3.9% lower than June and August 1996. In contrast, July sales for 1995 and 1997 were 1.2% higher than the June and August sales for those 2 years.

To explore whether automobile traffic is a critical factor in urban ozone accumulation, we analyzed the relation-

ship between weekday traffic counts and peak ozone concentrations on that day. We found a significant correlation between 1-hour morning peak traffic counts and peak ozone concentrations at all 4 traffic-count sites (Pearson $r=0.29, 0.42, 0.34,$ and 0.39, respectively; average $r=0.36$). No difference in this correlation was seen between the Olympic and baseline periods. An equally strong and significant correlation was found between total 24-hour traffic counts and ozone concentrations (average $r=0.38$; range, 0.33-0.48).
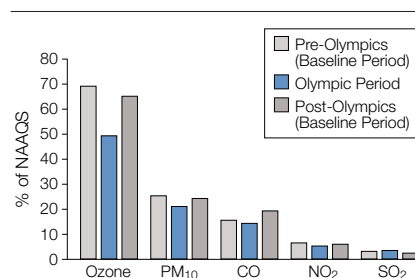
We used data from the entire 73-day study period to analyze the relationship between the number of asthma acute care events on a given day and the average daily peak ozone concentration during the preceding 48 to 72 hours. For data from the Medicaid, HMO, and emergency department databases, the RR of asthma events increased stepwise at cumulative ozone concentrations 60 to 89 ppb and 90 ppb or more compared with ozone concentrations of less than 60 ppb (TABLE 3). This trend was significant for the Medicaid and emergency department data.

A 3-day cumulative exposure measure was selected for the analysis shown in Table 3 instead of a single-day or 2-day exposure measure because it was found to be more consistently correlated with asthma events (TABLE 4). For data from the Medicaid and pediatric emergency department databases, the RR of asthma events per incremental changes in ozone and PM$_{10}$ levels increased as the number of cumulative exposure days included increased from 1 to 2. The RR per incremental change in PM$_{10}$ further increased when the number of cumulative exposure days increased from 2 to 3. However, increasing the cumulative exposure from 2 to 3 days did not further increase the RR per incremental change in ozone.

COMMENT

Evidence linking air quality to respiratory health has been accumulating in recent years. Many studies, including 2 conducted in Atlanta,[16,37] have demonstrated significant associations be-

Table 3. Relationship Between Acute Asthma Events and the 3-Day Cumulative Ozone Levels During the 73-day Summer Study Period*

Data Source	Ozone <60 ppb†		Ozone 60-89 ppb†		Ozone ≥90 ppb†	
	Mean Daily Asthma Events	RR	Mean Daily Asthma Events	RR (95% CI)	Mean Daily Asthma Events	RR (95% CI)
Georgia Medicaid claims file	2.20	1.00	3.85	1.61 (1.13-2.30)‡	5.11	1.88 (1.24-2.83)‡
Health maintenance organization	1.07	1.00	1.15	1.11 (0.63-1.96)	1.50	1.33 (0.68-2.61)
Pediatric emergency departments	3.00	1.00	4.65	1.33 (0.98-1.81)	6.00	1.46 (1.02-2.09)§
Georgia Hospital Discharge Database	1.67	1.00	2.15	1.19 (0.77-1.84)	1.72	1.03 (0.58-2.11)

*CI indicates confidence interval. Relative risk (RR) based on Poisson model (fraction of total acute care events with a primary diagnosis of asthma); time-series regression analysis was adjusted for day of the week (weekday vs weekend) and minimum daily temperature (lagged 1 day to minimize serial correlation).
†Average of the peak ozone concentrations for 3 days (day of event plus the preceding 2 days).
‡$P\leq.01$.
§$P = .04$.

tween days with high ozone levels and increased rates of asthma exacerbations.[12-15,20-23] Our results support these previous findings and also indicate that extended reductions in ozone and PM_{10} concentrations at levels considerably below the EPA's National Ambient Air Quality Standards can reduce asthma morbidity in children. Furthermore, our findings suggest that by decreasing automobile emissions through citywide changes in transportation and commuting practices, a substantial number of asthma exacerbations requiring medical attention can be prevented.

We found variation in the relative change in asthma acute care events during the Olympic Games among the 4 sources of medical data. All showed a decrease in asthma events, ranging from 11% to 44%. Only the data from the Georgia Medicaid claims file reached statistical significance, which may be related to the low power of this short-term intervention. Based on a power of 80% and the number of events in each database, the percent reduction in asthma events needed to detect a significant difference between the baseline and Olympic periods was 37% for the Georgia Medicaid claims file, 58% for the health maintenance organization database, 33% for the pediatric emergency room database, and 51% for the Georgia Hospital Discharge database (without adjustment for serial correlation). Other possible explanations include differences between the study

population in the 4 data sources in asthma severity, medication use,[38] exposure to other allergens, particularly those that could synergistically worsen the untoward effects of ozone and other air pollutants,[39,40] extent of indoor ozone exposure,[41] and of outdoor exposure.

The observed reductions in asthma events require us to address the following potentially confounding situation: did enough Atlanta children leave the city during the Olympics to significantly reduce the number of children with asthma who would seek medical attention for an acute asthma exacerbation? If true, this could account, in whole or in part, for the reduction in asthma events observed. However, for all 4 data sources, use of nonasthma-related urgent and emergency medical services by Atlanta children changed minimally. This suggests that neither the size of the study population nor use of emergency medical services by this population changed significantly during the Olympic games.

Of the factors potentially affecting asthma morbidity that we could readily assess, air quality remains the likely cause for the decline in asthma acute care events. Our analysis demonstrated a large and significant decrease in ozone concentrations, and to a lesser extent, PM_{10}, and carbon monoxide concentrations. Of all the pollutants, Atlanta's ozone concentrations in the summer most frequently violate the National Ambient Air Quality Standards.[42] These

standards attempt to set a maximum pollution level, which, if exceeded, may be hazardous to the general public's health. The standards are based, in part, on the available information regarding the health effects of the 5 major air pollutants. However, pollution levels below these standards may be harmful to certain, high-risk populations (such as individuals with asthma and the elderly). Therefore, the 28% drop in ozone concentrations during the Olympic Games represented a substantial decrease in a potential health hazard.

Our study design and findings make it difficult to determine to what degree the observed reductions in ozone, PM_{10}, carbon monoxide, and nitrogen dioxide pollution individually contributed to the observed changes in health. As controlled studies of human exposure to multiple pollutants have demonstrated,[12,13,19] the effects of reduced levels of ozone and these primary pollutants likely were additive. This is supported by the finding that both ozone and PM_{10} levels were similarly correlated with asthma events. The fact that ozone and PM_{10} levels were highly correlated with each other ($r = 0.58-0.69$) additionally limits our ability to determine which pollutant(s) accounted for the reduction in asthma events. This correlation between ozone and PM_{10} levels should have been expected, given that the environmental changes occurring during the study period (ie, decreased automobile emissions and

Table 4. Comparison of the Relative Risk of Asthma Events per 50-ppb Incremental Change in Ozone and 10-μg/m^3 Incremental Change in PM_{10} Levels Based on a Same-Day, 2-Day, and 3-Day Cumulative Exposure Measure*

| | Relative Risk (95% Confidence Interval) | | | | | |
| | Ozone | | | PM_{10} | | |
Data Source	Same Day	2 Day	3 Day	Same Day	2 Day	3 Day
Georgia Medicaid claims file	1.1 (0.88-1.47)	1.4 (1.05-1.93)†	1.4 (1.01-1.94)†	0.9 (0.57-1.49)	1.1 (0.62-1.78)	1.4 (0.80-2.48)
Health maintenance organization	1.4 (0.88-2.14)	1.2 (0.68-1.96)	1.2 (0.69-2.11)	2.1 (0.92-4.61)	1.8 (0.72-4.40)	1.8 (0.68-4.81)
Pediatric emergency departments	1.2 (0.99-1.56)	1.4 (1.04-1.79)†	1.4 (1.03-1.86)†	1.2 (0.81-1.89)	1.3 (0.81-2.08)	1.5 (0.91-2.47)
Georgia Hospital Discharge Database	0.9 (0.64-1.34)	1.0 (0.62-1.51)	1.0 (0.61-1.58)	0.7 (0.34-1.45)	0.6 (0.29-1.45)	0.8 (0.33-1.88)

*PM_{10} indicates particulate matter of 10 μm or smaller; same day, single-day measure of exposure (day of asthma event only); 2 day, 2-day measure of exposure (day of asthma event plus the preceding day); and 3 day, 3-day measure of exposure (day of asthma event plus the preceding 2 days). Relative risk based on Poisson time-series regression analysis was adjusted for day of the week (weekday vs weekend) and minimum daily temperature (lagged 1 day to minimize serial correlation).
†$P \leq .05$.

weather variability) would have had a strong influence on the daily levels of both these pollutants.

The more immediate question is what accounted for this change in air quality. We suggest that it was caused by changes in both meteorological conditions and automobile emissions, with decreases in peak morning rush hour traffic playing a major role. Weather conditions during the Olympic Games (increased wind speed and decreased temperature and solar radiation) favored less accumulation of ozone, but the degree of weather improvements was measurably small and not statistically significant. Even when controlling for these weather variables in a multivariate regression model, ozone levels in Atlanta during the Olympic Games were reduced 13% whereas the changes in ozone levels at the other 3 Georgia sites with the same prevailing weather patterns were reduced between 2% and 7%.

Other indirect evidence supports our conclusion. The concentration of carbon monoxide, which is primarily emitted directly from automobiles and is much less dependent on weather conditions for its accumulation in the lower atmosphere, decreased significantly during the Olympic Games. The small increase in sulfur dioxide levels (far below health hazard levels) during the Olympic Games is consistent with the increased use of diesel-powered buses,[31,32] and should not have increased if the prevailing weather conditions had indeed prevented the normal accumulation of air pollutants in Atlanta. The amount of emissions from stationary sources (eg, power plants and industry) did not change during the Olympic Games.[31,32] The additional electrical needs required during the Olympic Games came from power stations outside the immediate Atlanta area and, therefore, would not have caused the increase in sulfur dioxide observed.

Evidence of changes in automobile traffic and emissions include the marked decreases in weekday and weekend morning peak traffic counts at all 4 traffic-count sites, the statistically significant decreases in weekday total traffic counts at the 2 traffic-count sites closest to downtown Atlanta, the statistically significant correlation between weekday morning peak and 24-hour total traffic counts and that day's peak ozone concentration, the 3.9% decrease in statewide gasoline sales in July compared with June and August, and the 217% increase in overall public transportation use. These traffic data probably underestimate the impact of the alternative transportation strategies on local residents of Atlanta because they include automobile use by the estimated 1 million visitors during the Olympic intervention period. Using this same logic, however, the increase in public transportation use is probably an overestimation of the behaviors of local residents since it also includes use by visitors to Atlanta.

The science of ozone formation helps explain our findings. The moderate alterations in morning traffic levels (and probably traffic flow) experienced during the Olympic Games would have decreased the buildup of ozone precursors emitted into the atmosphere from 7 AM through 2 PM. Without sufficient atmospheric concentrations of these precursors being present during this time of maximum sunlight and heat, rapid ozone production and accumulation could not occur, thus leading to lower than anticipated peak ozone levels. During a period of 17 days, this appeared to have contributed to the improved respiratory health of children with asthma residing in Atlanta. What motivated businesses and individuals to change their transportation and commuting behaviors temporarily is a crucial question, which has not been properly addressed. Fear of traffic and lack of parking, and social pressures to conform certainly played a role. How this can be adapted to more routine conditions remains a major public health challenge. For example, Atlanta's Clean Air Campaign[43] (largely initiated after the Olympic Games) has been shown to increase use of alternative commuting methods within 3 companies that promoted this.[44] But the effects of this citywide campaign on air pollution to date appear to be small compared with what was observed during the Olympic Games.

The weight of evidence linking air quality to respiratory health continues to grow. Our findings suggest that efforts to decrease ozone and PM_{10} concentrations from moderate to low levels can decrease the burden of asthma. Consistent with our findings for the Olympic period, we found an increasing correlation between daily asthma events and ozone and PM_{10} levels as the period of exposure was increased from less than 24 hours preceding the asthma event to the 48 to 72 hours preceding the event. When using this 3-day cumulative exposure measure, the risk of asthma exacerbations increased substantially after only moderate levels of ozone exposure (60-89 ppb). These data suggest that the cumulative effect of moderate levels of ozone and other pollutants during a more extended period is as or more important to respiratory health than single-day levels exceeding the national standards (ie, 1-hour peak ozone >120 ppb or 8-hour peak >80 ppb). Recent research with PM_{10} supports the important effect of extended exposure to pollutants on respiratory health.[45,46]

Our study methods and findings have several limitations. The special nature of the Olympic Games, the relatively short intervention period and limited statistical power, the lack of an updated traffic counting system, and the limited number of air pollution monitoring sites (for PM_{10} and carbon monoxide) make firm conclusions difficult. Unmeasured medical or social factors may have influenced our findings. A citywide alternative transportation and commuting intervention, not associated with a special event, would provide a better study situation. However, the power of the Olympic Games to transform behavioral norms should not be underestimated and deserves close scrutiny for its lessons.

We conclude that the alternative transportation plan in Atlanta during the Olympic Games reduced ozone and other air pollutants and was associated

with a significant, albeit temporary, decrease in the burden of asthma among Atlanta's children.

Author Affiliations: Epidemic Intelligence Service (Dr Friedman) and Chronic Disease, Injury, and Environmental Epidemiology Section (Dr Powell), Epidemiology and Prevention Branch, Georgia Division of Public Health, and Epidemiology Program Office, Centers for Disease Control and Prevention (Dr Friedman and Ms Hutwagner), Department of Pediatrics, Morehouse School of Medicine, and Georgia Pediatric Pulmonary Associates (Dr Graham), Division of Pediatric Pulmonary and Critical Care Medicine, Egleston Children's Hospital and Emory University (Dr Teague), Atlanta, Ga.

REFERENCES

1. Centers for Disease Control and Prevention. Measuring childhood asthma prevalence before and after the 1997 redesign of the National Health Interview Survey—United States. *MMWR Morb Mortal Wkly Rep*. 2000;49:908-911.
2. Centers for Disease Control and Prevention. Surveillance for asthma—United States, 1960-1995: CDC surveillance summaries. *MMWR Morb Mortal Wkly Rep*. 1998;47:SS1-SS27.
3. Centers for Disease Control and Prevention. Asthma—United States, 1982-1992. *MMWR Morb Mortal Wkly Rep*. 1995;43:952-955.
4. Centers for Disease Control and Prevention. Asthma mortality and hospitalization among children and young adults—United States, 1980-1993. *MMWR Morb Mortal Wkly Rep*. 1996;45:350-353.
5. Abramson MJ, Kutin J, Czarny D, Walters EH. The prevalence of asthma and respiratory symptoms among young adults: is it increasing in Australia? *J Asthma*. 1996;33:189-196.
6. Anderson HR. Increase in hospital admissions for childhood asthma: trends in referral, severity and readmissions from 1970 to 1985 in a health region of the United Kingdom. *Thorax*. 1989;44:614-619.
7. Goren AI, Hellmann S. Has the prevalence of asthma increased in children? evidence from a long term study in Israel. *J Epidemiol Community Health*. 1997;51:227-232.
8. Mullaly DI, Howard WA, Hubbard TJ, et al. Increased hospitalizations for asthma among children in the Washington, DC area during 1961-1981. *Ann Allergy*. 1984;53:15-19.
9. Becklake MR, Ernst P. Environmental factors. *Lancet*. 1997;350(suppl 2):10-13.
10. Schenker M. Air pollution and mortality. *N Engl J Med*. 1993;329:1807-1808.
11. Abramson MJ, Marks GB, Pattemore PK. Are non-allergenic environmental factors important in asthma ? *Med J Aust*. 1995;163:542-545.
12. Holgate ST, Samet JM, Koren HS, Maynard RL. *Air Pollution and Health*. London, England: Academic Press; 1999.
13. Committee of the Environmental and Occupational Health Assembly, American Thoracic Society. Health effects of outdoor air pollution. *Am J Respir Crit Care Med*. 1996;153:3-50.
14. Brunekreef B, Dockery DW, Krzyzanowski M. Epidemiologic studies on short-term effects of low levels of major ambient air pollution components. *Environ Health Perspect*. 1995;103(suppl 2):3-13.
15. Tatterfield AE. Air pollution: brown skies research. *Thorax*. 1996;51:13-22.
16. White MC, Etzel RA, Wilcox WD, Lloyd C. Exacerbations of childhood asthma and ozone pollution in Atlanta. *Environ Res*. 1994;65:56-68.

17. Anderson HR, Ponce de Leon A, Bland JM, et al. Air pollution and daily mortality in London: 1987-92. *BMJ*. 1996;312:665-669.
18. Buchdahl R, Parker A, Stebbings T, Babiker A. Association between air pollution and acute childhood wheezy episodes: prospective observational study. *BMJ*. 1996;312:661-665.
19. Avol EL, Linn WS, Shamoo DA, et al. Respiratory effects of photochemical oxidant air pollution in exercising adolescents. *Am Rev Respir Dis*. 1985;132:619-622.
20. Cody RP, Weisel CP, Birnbaum G, Lioy PJ. The effect of ozone associated with summertime photochemical smog on the frequency of asthma visits to hospital emergency departments. *Environ Res*. 1992;58:184-194.
21. Romieu I, Meneses F, Sienra-Monge JJ, et al. Effects of urban air pollutants on emergency visits for childhood asthma in Mexico City. *Am J Epidemiol*. 1995;141:546-553.
22. Romieu I, Meneses F, Ruiz S, et al. Effects of air pollution on the respiratory health of asthmatic children living in Mexico City. *Am J Respir Crit Care Med*. 1996;154:300-307.
23. Thurston GD, Lippmann M, Scott MB, Fine JM. Summertime haze air pollution and children with asthma. *Am J Respir Crit Care Med*. 1997;155:654-660.
24. Pope CA. Respiratory disease associated with community air pollution and a steel mill, Utah Valley. *Am J Public Health*. 1989;79:623-628.
25. Livingstone AE, Shaddick G, Grundy C, Elliott P. Do people living near inner city main roads have more asthma needing treatment? case-control study. *BMJ*. 1996;312:676-677.
26. Edwards J, Walters S, Griffiths RC. Hospital admissions for asthma in pre-school children: relationship to major roads in Birmingham UK. *Arch Environ Health*. 1994;49:223-237.
27. Wjst M, Reitmeir P, Dold S, et al. Road traffic and adverse effects on respiratory health in children. *BMJ*. 1993;307:596-600.
28. Weiland SK, Mundt KA, Ruckmann A, Keil U. Self-reported wheezing and allergic rhinitis in children and traffic density on street of residence. *Ann Epidemiol*. 1994;4:243-247.
29. Murakami M, Ono M, Tamura K. Health problems of residents along heavy traffic roads. *J Hum Ergol (Tokyo)*. 1992;19:101-106.
30. *What You Can Do to Reduce Air Pollution*. Washington, DC: United States Environmental Protection Agency Office of Air and Radiation; 1992;12. EPA 450-K-92-002.
31. Balagas R. *Improved Air Quality in Atlanta During the 1996 Olympic Summer Games*. Atlanta: Georgia Environmental Protection Division, Georgia Department of Natural Resources; 1996.

32. Porter C. *Changes in Air Quality and Transportation Associated With the 1996 Atlanta Summer Olympics*. Cambridge, Mass: Cambridge Systematics Inc; 1997. Prepared for: National Cooperative Highway Research Program (NCHRP 8-33).
33. Metropolitan Atlanta Rapid Transit Authority. *The Way to the Games: A Report on Mass Transit During the 1996 Summer Olympic Games*. Atlanta, Ga: MARTA; 1996.
34. Busse WW, Holgate ST. *Asthma and Rhinitis*. Cambridge, Mass: Blackwell Science; 1995.
35. *Measuring Air Quality: The Pollutant Standards Index*. Washington, DC: Environmental Protection Agency Office of Air Quality Planning and Standards; 1994. EPA 451-K-94-001.
36. Zeger SL, Liang KY. Longitudinal data analysis for discrete and continuous outcomes. *Biometrics*. 1996;42:121-130.
37. Tolbert P, Mulholland JA, MacIntosh DL, et al. Air quality and pediatric emergency room visits for asthma in Atlanta, Georgia. *Am J Epidemiol*. 2000;151:798-810.
38. Hartert TV, Windom HH, Peebles RS, Friedhoff LR, Togias A. Inadequate outpatient medical therapy for patients with asthma admitted to two urban hospitals. *Am J Med*. 1996;100:386-394.
39. Jorres R, Nowak D, Magnussen H. The effect of ozone exposure on allergen responsiveness in subjects with asthma or rhinitis. *Am J Respir Crit Care Med*. 1996;153:56-64.
40. Molfino NA, Wright SC, Katz I, et al. Effect of low concentrations of ozone on inhaled allergen responses in asthmatic subjects. *Lancet*. 1991;338:199-203.
41. Hayes SR. Use of an indoor air quality model (IAQM) to estimate indoor ozone levels. *J Air Waste Manage Assoc*. 1991;41:171-181.
42. Environmental Protection Agency Green Book. Available at: http://www.epa.gov/oar/oaqps/greenbk/index.html. Accessed February 2, 2001.
43. Clean Air Campaign. Available at: http://www.cleanaircampaign.com. Accessed February 2. 2001.
44. Centers for Disease Control and Prevention. Corporate action to reduce air pollution—Atlanta, Georgia, 1998-1999. *MMWR Morb Mortal Wkly Rep*. 2000;49:153-156.
45. Delfino RJ, Zeiger RS, Seltzer JM, Street DH. Symptoms in pediatric asthmatics and air pollution: differences in effects by symptom severity, anti-inflammatory medication use and particulate averaging time. *Environ Health Perspect*. 1998;106:751-761.
46. Schwartz J. The distributed lag between air pollution and daily deaths. *Epidemiology*. 2000;11:320-326.