Ambient Air Pollution and Respiratory Emergency Department Visits

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Background: A number of emergency department studies have corroborated findings from mortality and hospital admission studies regarding an association of ambient air pollution and respiratory outcomes. More refined assessment has been limited by study size and available air quality data.

Methods: Measurements of 5 pollutants (particulate matter [PM_{10}], ozone, nitrogen dioxide [NO_2], carbon monoxide [CO], and sulfur dioxide [SO_2]) were available for the entire study period (1 January 1993 to 31 August 2000); detailed measurements of particulate matter were available for 25 months. We obtained data on 4 million emergency department visits from 31 hospitals in Atlanta. Visits for asthma, chronic obstructive pulmonary disease, upper respiratory infection, and pneumonia were assessed in relation to air pollutants using Poisson generalized estimating equations.

Results: In single-pollutant models examining 3-day moving averages of pollutants (lags 0, 1, and 2): standard deviation increases of ozone, NO_2, CO, and PM_{10} were associated with 1–3% increases in URI visits; a 2 $\mu g/m^3$ increase of $PM_{2.5}$ organic carbon was associated with a 3% increase in pneumonia visits; and standard deviation increases of NO_2 and CO were associated with 2–3% increases in chronic obstructive pulmonary disease visits. Positive associations persisted beyond 3 days for several of the outcomes, and over a week for asthma.

Conclusions: The results of this study contribute to the evidence of an association of several correlated gaseous and particulate pollutants, including ozone, NO_2, CO, PM, and organic carbon, with specific respiratory conditions.

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A number of studies of emergency department visits, a relatively sensitive outcome for respiratory conditions, have corroborated findings from mortality and hospital admission studies regarding an association of ambient air pollution levels and respiratory health effects.[1–4] More refined assessment, including analysis of subgroups defined by specific illness or ages, or of air pollutants not routinely monitored, has been limited by study size and available air quality and health outcome data. Many of the single-city time-series studies have covered a relatively short time-span or involved a moderately low number of daily outcome events, resulting in imprecise effect estimates and often restricting analyses to broad outcome and age groups. Recent multicity time-series studies, although having a relatively large number of daily outcome counts, were limited to routinely available outcome and air-quality datasets.[5–7]

The present study is part of the Study of Particles and Health in Atlanta (SOPHIA). This collection of studies uses extensive air quality data, including detailed particulate matter (PM) component and size fraction information, from a monitoring station in Atlanta operated by the Aerosol Research and Inhalation Epidemiology Study (ARIES). Emergency department visits for respiratory illness were analyzed in relation to routinely collected criteria pollutant levels for the period 1 January 1993 through 31 August 2000, and in relation to additional air pollutants measured at the ARIES monitoring station for the period 1 August 1998 through 31 August 2000. The results for the cardiovascular visits are presented elsewhere.[8] In this work, we took advantage of the large number of respiratory emergency department visits and extensive air quality data to examine multiple pollutants in relation to specific respiratory outcomes.

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METHODS

Ambient Air Quality Data

We selected the pollutants and metrics for this analysis a priori on the basis of current hypotheses regarding potentially causal pollutants and components.[9,10] We also included pollutants in the a priori list that may be useful markers for sources or for groups of related pollutants (eg, carbon monoxide as a potential marker for primary traffic-related pollutants).

For the period 1 January 1993 through 31 August 2000, we obtained ambient air quality data for 24-hour average PM_{10} mass (PM with an average aerodynamic diameter less than 10 micrometers), 8-hour maximum ozone, and 1-hour maximum nitrogen dioxide (NO_2), sulfur dioxide (SO_2), and carbon monoxide (CO) from several existing monitoring networks, including the Air Quality System (AQS, formerly the Aeorometric Information Retrieval System or AIRS), the Georgia Department of Natural Resources, and Metro Atlanta Index. (See map, with the electronic version of this article.) Ozone levels were not monitored during the winter months when ozone levels in Atlanta are low; the remaining pollutants were measured year-round. The AQS air quality data have been described elsewhere.[8]

For the final 25 months of the study period (1 August 1998 through 31 August 2000), an extensive suite of pollutants, including PM size fractions and components, was measured at the ARIES monitoring station. We selected the following pollutants and metrics for this analysis a priori: oxygenated hydrocarbons (OHC), $PM_{2.5}$ mass (PM with an average aerodynamic diameter less than 2.5 micrometers), coarse PM (PM with an average aerodynamic diameter between 2.5 and 10 micrometers), ultrafine PM count (PM with an average aerodynamic diameter between 10 and 100 nanometers [nm]), and the $PM_{2.5}$ components sulfate, acidity, elemental carbon (EC), organic carbon (OC), and an index of water-soluble transition metals. The metrics for PM size fractions and components and for OHC were 24-hour averages, 8-hour maximum for ozone, and 1-hour maximum for NO_2, SO_2, and CO. The measurement methods for the ARIES monitoring station have previously been described.[8,11]

Average temperature and dew point temperature (average of the daily minimum and maximum), as well as additional meteorological data measured at Hartsfield-Atlanta International Airport, were obtained from the National Climatic Data Center network. Speciated pollen counts were obtained from the Atlanta Allergy Clinic.

Emergency Department Data

Of the 41 hospitals in the 20-county Atlanta metropolitan statistical area, 37 agreed to participate and 31 provided usable computerized billing records for at least part of the study period. (The map available with the electronic version of this article shows hospital locations.)

Computerized billing records for all emergency department visits between 1 January 1993 and 31 August 2000 were collected, including primary International Classification of Diseases 9th Revision (ICD-9) diagnostic code, secondary ICD-9 diagnosis codes, age, date of birth, sex, race, and residential zip code. Residents of the Atlanta metropolitan statistical area, determined by residential zip code at the time of the visit, were included in the analyses. Repeat visits within a single day were counted as a single visit.

Respiratory case groups of interest were defined using the primary ICD-9 diagnostic codes (all 2-digit extensions were used unless otherwise specified): asthma (493, 786.09), COPD (491, 492, 496), URI (460–466, 477), pneumonia (480–486), and an all-respiratory-disease group that combines the above 4 groups. We assessed the adequacy of the modeling approach using visits for finger wounds (883.0), an outcome group that has comparable temporal variations to the respiratory outcomes of interest and is expected to be unrelated to air pollution.

Analytic Methods

All analyses were performed using SAS statistical software, version 8.2 (SAS Institute, Inc., Cary, NC) unless otherwise indicated. We defined a priori single-pollutant models to control for long-term temporal trends and meteorological conditions. For the a priori analyses we used Poisson generalized estimating equations,[12] with a stationary 4-dependent correlation structure to account for possible autocorrelation in the outcome data (URI, asthma, all respiratory disease) and Poisson generalized linear models[13] for outcomes with minimal autocorrelation (pneumonia, COPD). Risk ratios and 95% confidence intervals were calculated for an increase of approximately a standard deviation of pollutant levels. The basic model had the following form:

$$\begin{aligned}\log(E(Y)) = {} & \alpha + \beta\, pollutant + \sum_k \lambda_k\, DOW_k \\ & + \sum_m \xi_m season_m + \sum_n \nu_n\, hospital_n \\ & + \sum_p \zeta_p\, holiday_p + g(\gamma_1,\ldots,\gamma_N;\, time) \\ & + g(\delta_1,\ldots,\delta_N;\, temp) + g(\eta_1,\ldots,\eta_N;\, dew\ point),\end{aligned}$$

where Y indicated the count of emergency department visits for a given day for the outcome of interest. The a priori models contained a 3-day moving average of pollution levels lagged 0, 1, and 2 days relative to the visits (levels on the same day as the visit, 1 day previous, and 2 days previous, respectively) (*pollutant*). Long-term temporal trends were accounted for using cubic splines with monthly knots [$g(\gamma_1,\ldots,\gamma_N;\, time)$]. Because ozone data were not available from November through March, ozone models used separate

time splines for each year. Additional season indicator variables (the 21st day of March, June, September, and December) were added to further control for seasonal trends (*season*). Cubic splines also were used to control for daily average temperature [$g(\delta_1,\ldots,\delta_N$; *temp)*] and dew point [$g(\eta_1,\ldots,\eta_N$; *dew point*)] with knots at the 25th and 75th percentiles (moving average of lags 0, 1, and 2). Indicator variables for day of week (*DOW*), federal holidays (*holiday*), and hospital entry and exit (*hospital*) also were included in the a priori model (as the hospitals provided data for varying amounts of time). The cubic splines, *g(x)*, were defined as follows:

$$g(\gamma_1,\gamma_2,\ldots\gamma_N;x) = \gamma_1 x + \gamma_2 x^2 + \gamma_3 x^3 + \sum_{j=4}^{N} \gamma_j w_j(x),$$

where $w_j(x) = (x\text{-}\tau_j)^3$ if $x \geq \tau_j$, and $w_j(x) = 0$ otherwise. The cubic splines were defined so that the first and second derivatives were continuous. We evaluated multipollutant models using the same covariates as the single-pollutant models.

We performed several secondary analyses. To assess the lag structure between pollutant levels and emergency department visits, we initially examined separate models for each lag from 0 to 7 days before the visit (up to 2 weeks prior to the visit for asthma). To estimate the overall effect of a unit increase in pollution during the previous 2 weeks, and to investigate whether associations persisted longer than 3 days, we ran unconstrained distributed lag models, including pollution levels from 0 to 13 days before the visit, with additional cubic terms for lags 3–13 for temperature and dew point (in addition to the cubic splines for lags 0–2). For the distributed lag models we presented results only for the pollutants available for the entire study period as the models became unstable for the pollutants available only 25 months.

We examined age-specific case groups (ages 0–1 year, 2–18, 19 years and older, and 65 years and older) as well as season-specific models for warm (April 15 to October 14) and cool (October 15 to April 14) periods. Daily pollen counts (grass, oak, and ragweed) and daily counts of influenza emergency department visits were assessed as confounders. We also assessed general additive models using S-Plus 2000 software (Insightful Corporation, Seattle, WA) with nonparametric LOESS smoothers and nonparametric smoothing splines (10^{-14} convergence criterion).[14,15]

In addition to examining the alternate outcome group believed unrelated to air pollution (finger wounds), we performed other analyses to evaluate the adequacy of the modeling approach. We explored negative lags for pollution (pollution levels on days after the visit) as exposure variables, controlling for positive lags, to evaluate the possibility that the modeling choices induced positive associations. We altered the placement (day of the month) and number of knots (degrees of freedom) in the cubic splines for time.

RESULTS

Descriptive statistics for the air quality variables are presented in Table 1; Spearman rank correlation statistics between the daily measures were previously published.[8] (Appendix Table 1, available with the electronic version of this article, presents the correlation statistics.) The extent of correlation among the pollutants followed expected patterns. Ultrafine PM count levels were negatively correlated with several pollutants, including ozone, PM, and PM components (sulfate, acidity, and metals). CO, NO_2, $PM_{2.5}$ organic carbon, and $PM_{2.5}$ elemental carbon were moderately correlated ($r = 0.55–0.68$). PM_{10} and $PM_{2.5}$ mass were moderately correlated with the $PM_{2.5}$ components ($r = 0.56–0.77$). Acidity and sulfate were highly correlated with each other ($r = 0.85$) and moderately correlated with ozone ($r = 0.64$ and 0.63, respectively) and temperature ($r = 0.84$ and $r = 0.64$, respectively). The diurnal patterns of CO and NO_2 indicate that mobile source emissions contributed substantially to these pollutant levels. SO_2 levels peaked in both summer and winter, corresponding to peak energy demands. SO_2 levels exhibited marked temporal and spatial variability, with occasional mid-afternoon peaks resulting from power plant plume fumigation events. Compared with other U.S. cities, ozone and $PM_{2.5}$ are relatively high (with sulfate and organic carbon comprising relatively high proportions of $PM_{2.5}$ mass), and acidity is relatively low.[16]

The 31 hospitals providing usable data for these analyses receive 80% of the annual emergency department visits in the Atlanta area, and contributed information on 4,407,535 total emergency department visits. Respiratory problems accounted for 11% of all emergency department visits. For the entire study period, average daily outcome counts of the subgroups ranged from 7 for COPD to 103 for URI, and the combined respiratory disease group had an average daily count of 172 (Table 2). For the final 25 months of the study, the 31 hospitals contributed 1,888,973 visits.

Results from the a priori single-pollutant models examining 3-day moving averages (lags 0, 1, and 2) of pollutant levels are shown in Table 3. PM_{10}, ozone, NO_2, and CO were individually associated with 1–3% increases of URI visits per standard deviation increase of pollutant; similar results were observed for the combined respiratory disease group (60% of all respiratory visits were for URI). Weak and less stable associations were observed for URI in relation to SO_2, $PM_{2.5}$, and organic carbon. A 20 pbb increase of NO_2 and a 1 ppm increase in CO were associated with 3.5% and 2.9% increases of COPD visits, respectively. Additional estimates for COPD were elevated, but COPD was the smallest outcome group and therefore had the widest confidence intervals. A 2.8%

TABLE 1. Mean, Standard Deviation, and Selected Percentiles of Daily Ambient Air Quality Measurements for 5 Criteria Pollutants From the AQS and for Pollutants From the ARIES Monitoring Station

	% Missing	Mean ± SD	10%	90%
24-h PM_{10} ($\mu g/m^3$)*‡	3	27.9 ± 12.3	13.2	44.7
8-h Ozone (ppb)*‡§	32	55.6 ± 23.8	26.8	87.6
1-h NO_2 (ppb)*‡	1	45.9 ± 17.3	25.0	68.0
1-h CO (ppm)*‡	2	1.8 ± 1.2	0.5	3.4
1-h SO_2 (ppb)*‡	1	16.5 ± 17.1	2.0	39.0
24-h $PM_{2.5}$ ($\mu g/m^3$)†	2	19.2 ± 8.9	8.9	32.3
24-h coarse PM ($\mu g/m^3$)†	11	9.7 ± 4.7	4.4	16.2
24-h 10–100 nm particle count ($^{\#}/cm^3$)†	44	38000 ± 40700	11500	74600
24-h $PM_{2.5}$ water-soluble metals ($\mu g/m^3$)†	9	0.028 ± 0.025	0.006	0.061
24-h $PM_{2.5}$ sulfate ($\mu g/m^3$)†	10	5.5 ± 3.7	1.9	10.7
24-h $PM_{2.5}$ acidity ($\mu \cdot equ/m^3$)†‖	15	0.018 ± 0.023	−0.001	0.045
24-h $PM_{2.5}$ organic carbon ($\mu g/m^3$)†	6	4.5 ± 2.2	2.2	7.1
24-h $PM_{2.5}$ elemental carbon ($\mu g/m^3$)†	6	2.0 ± 1.4	0.8	3.7
24-h oxygenated hydrocarbons (ppb)†	22	32.1 ± 15.3	15.0	53.1
Average temperature (°C)	0	17.5 ± 8.3	6.1	27.2
Average dew point (°C)	0	10.5 ± 8.9	−2.2	20.8

*Measurements available from AQS from 1 January 1993 to 31 August 2000.

†Measurements available from the ARIES monitoring station from 1 August 1998 to 31 August 2000.

‡Data were imputed for 17% (458 of 2703) of PM_{10} values, 2% (46 of 1892) of ozone values, 14% (398 of 2775) of NO_2 values, 6% (161 of 2758) of CO values, and 9% (237 of 2775) of SO_2 values.

§Ozone was measured for 1896 days: 1 March 1993 to 30 November 1993, 1 March 1994 to 30 November 1994, 1 March 1995 to 30 November 1995, 1 March 1996 to 31 October 1996, 1 April 1997 to 31 October 1997, 1 April 1998 to 31 October 1998, 1 April 1999 to 31 October 1999, 1 March 2000 to 31 August 2000.

‖Acidity reported in units of $\mu \cdot equ/m^3$, a measure of pH level, accounting for the negative values. If converted into units of nmol/ m^3, the mean is 18 and standard deviation is 23.

PPB, parts per billion; PPM, parts per million

TABLE 2. Mean, Standard Deviation, and Selected Percentiles of Daily Counts of Emergency Department Visits at 31 Participating Hospitals for the 2 Time Periods

		1 January 1993 to 31 August 2000			1 August 1998 to 31 August 2000		
	ICD-9 Codes	Mean ± SD	10%	90%	Mean ± SD	10%	90%
All emergency department visits		1574 ± 804	442	2572	2479 ± 252	2163	2814
All respiratory disease	460–465, 466.0, 480–486, 491–493, 496, 786.09	172 ± 93.7	61	286	241 ± 85.9	146	356
Upper respiratory infections	460–465, 466.0	103 ± 59.8	37	174	144 ± 59.9	84	225
Asthma	493, 786.09	39.0 ± 20.5	13	66	53.2 ± 15.2	34	73
Pneumonia	480–486	20.8 ± 14.4	6	39	30.7 ± 15.0	15	51
Chronic obstructive pulmonary disease	491, 492, 496	7.42 ± 5.86	1	15	12.2 ± 4.87	7	17
Finger wounds	883.0	21.4 ± 12.3	5	38	31.3 ± 6.94	23	40

ICD-9, *International Classification of Diseases*, 9th Revision.

increase in pneumonia visits was associated with a 2 $\mu g/m^3$ increase of organic carbon. Small increases of asthma visits were observed in relation to standard deviation increases of PM_{10}, ozone, NO_2, and CO; however, the confidence intervals were too wide to exclude a null association. Weak or no associations were observed for the finger wound group. Including daily pollen counts or daily influenza emergency department visits in the models did

TABLE 3. Results of a priori Models* for the Association of Daily Ambient Air Quality Measurements With Emergency Department Visits for Respiratory Disease

Pollutant	Unit†	All Respiratory Disease		URI		Asthma		Pneumonia		COPD	
		RR	(95% CI)	RR	(95% CI)	RR	(95% CI)	RR	(95% CI)	RR	(95% CI)
1 January 1993 to 31 August 2000											
24-h PM_{10}	10 $\mu g/m^3$	1.013	(1.004–1.021)	1.014	(1.004–1.025)	1.009	(0.996–1.022)	1.011	(0.996–1.027)	1.018	(0.994–1.043)
8-h O_3	25 ppb	1.024	(1.008–1.039)	1.027	(1.009–1.045)	1.022	(0.996–1.049)	1.015	(0.981–1.050)	1.029	(0.977–1.084)
1-h NO_2	20 ppb	1.016	(1.006–1.027)	1.019	(1.006–1.031)	1.014	(0.997–1.030)	1.000	(0.983–1.019)	1.035	(1.006–1.065)
1-h CO	1 ppm	1.011	(1.004–1.019)	1.012	(1.003–1.021)	1.010	(0.999–1.022)	1.009	(0.996–1.021)	1.026	(1.004–1.048)
1-h SO_2	20 ppb	1.008	(0.997–1.019)	1.010	(0.998–1.024)	1.001	(0.984–1.017)	1.003	(0.984–1.023)	1.016	(0.985–1.049)
1 August 1998 to 31 August 2000											
24-h $PM_{2.5}$	10 $\mu g/m^3$	1.016	(0.997–1.035)	1.018	(0.995–1.041)	1.005	(0.977–1.033)	1.011	(0.981–1.042)	1.015	(0.969–1.063)
24-h coarse PM	5 $\mu g/m^3$	1.003	(0.982–1.025)	1.013	(0.987–1.039)	0.998	(0.987–1.039)	0.975	(0.940–1.011)	0.948	(0.897–1.003)
24-h 10–100 nm particle count	30,000 #/cm^3	0.984	(0.968–1.000)	0.986	(0.966–1.006)	0.999	(0.977–1.021)	0.977	(0.953–1.002)	0.982	(0.942–1.022)
24-h $PM_{2.5}$ water-soluble metals	0.03 $\mu g/m^3$	1.005	(0.981–1.031)	1.010	(0.980–1.040)	1.007	(0.973–1.043)	0.997	(0.958–1.039)	0.971	(0.913–1.032)
24-h $PM_{2.5}$ sulfate	5 $\mu g/m^3$	0.998	(0.968–1.028)	1.001	(0.965–1.039)	0.991	(0.949–1.035)	1.013	(0.959–1.069)	1.004	(0.929–1.085)
24-h $PM_{2.5}$ acidity	0.02 $\mu equ/m^3$	1.005	(0.977–1.033)	1.012	(0.979–1.045)	0.986	(0.948–1.025)	1.010	(0.964–1.059)	0.997	(0.936–1.061)
24-h $PM_{2.5}$ organic carbon	2 $\mu g/m^3$	1.011	(0.997–1.025)	1.011	(0.995–1.028)	1.000	(0.978–1.023)	1.028	(1.004–1.053)	0.996	(0.959–1.035)
24-h $PM_{2.5}$ elemental carbon	1 $\mu g/m^3$	0.999	(0.987–1.011)	0.999	(0.985–1.013)	0.993	(0.976–1.011)	1.006	(0.987–1.026)	0.981	(0.952–1.012)
24-h oxygenated hydrocarbons	15 ppb	0.986	(0.969–1.004)	0.983	(0.961–1.006)	0.973	(0.943–1.004)	1.020	(0.984–1.057)	0.983	(0.929–1.041)

*Single pollutant GEE (asthma, URI, all respiratory illness) with a stationary 4-dependent correlation structure or GLM (pneumonia, COPD) including a 3-day moving average of lags 0, 1, and 2 for pollutant; indicators for day-of-week, hospital entry/exit, and holidays; cubic splines for time with monthly knots; season indicator variables; cubic splines for temperature and dew point temperature with knots at the 25th and 75th percentile

†Approximately 1 standard deviation.

not affect the observed estimates. General additive models provided similar estimates to those from the a priori models.

In the exploratory models assessing the lag structure between pollutant levels and emergency visits (separate models for each lag), the risk ratios for asthma visits were generally positive and strongest with a lag of 5 to 8 days (Fig. 1). The association with ozone appeared to have a shorter lag structure, with the strongest positive associations at lags of 1 and 2 days. The estimates for ultrafine PM count were negative for lags of 0 and 1 day, and positive for lags of 2 through 4 days. The estimates for URI visits were generally highest for the shorter lags (Fig. 2). The gaseous pollutants tended to have stronger positive associations with URI at a lag of 1 day, while the same-day associations were typically stronger for several particle measures (PM_{10}, $PM_{2.5}$, coarse PM, $PM_{2.5}$ components). Sulfate and acidity exhibited a similar trend in relation to URI visits, with positive same-day estimates and negative estimates for a lag of 2 days. Associations for pneumonia and COPD visits were generally positive and strongest for same-day pollutant levels and for levels lagged by 1 day.

Results from unconstrained distributed lags models (lags of 0–13 days) are presented in Table 4. The risk ratios from models using 3-day moving averages can be interpreted as the risk ratio per unit increase of a uniform 3-day moving average, while risk ratios from the distributed lag models can be interpreted as the risk ratio per unit increase of a weighted 14-day moving average. Estimates from distributed lag models (lags of 0–13 days) tended to be substantially higher than those from models using the 3-day moving average (lags of 0–2 days) for PM_{10}, NO_2, CO, and SO_2, reflecting an additional contribution of days 3–13 in the distributed lag model.

In age-specific analyses, associations for pediatric asthma visits (ages 2–18) in relation to PM_{10} (RR = 1.016 per 10 $\mu g/m^3$; 95% CI = 0.998–1.034), NO_2 (1.027 per 20 ppb; 1.005–1.050), and CO (1.019 per ppm; 1.004–1.035) were stronger than those for adult asthma visits. Associations for infant (ages 0–1) and pediatric URI visits were substantially stronger than those for adults. Infant URI visits were associated with PM_{10}, ozone, $PM_{2.5}$ mass, and $PM_{2.5}$ organic carbon (RRs s per standard deviation increase = 1.026–1.042), and pediatric URI visits were associated with these pollutants as well as NO_2 and CO (RRs per standard deviation increase = 1.025–1.047).

The associations for asthma tended to be stronger for several pollutants in the warm months (15 April to 14 October), especially for ozone and $PM_{2.5}$ organic carbon. The estimates for pneumonia and COPD tended to be higher in the cold months.

In sensitivity analyses that varied the numbers of knots in the time splines, there was a tendency toward lower point estimates and larger standard errors as the number of knots

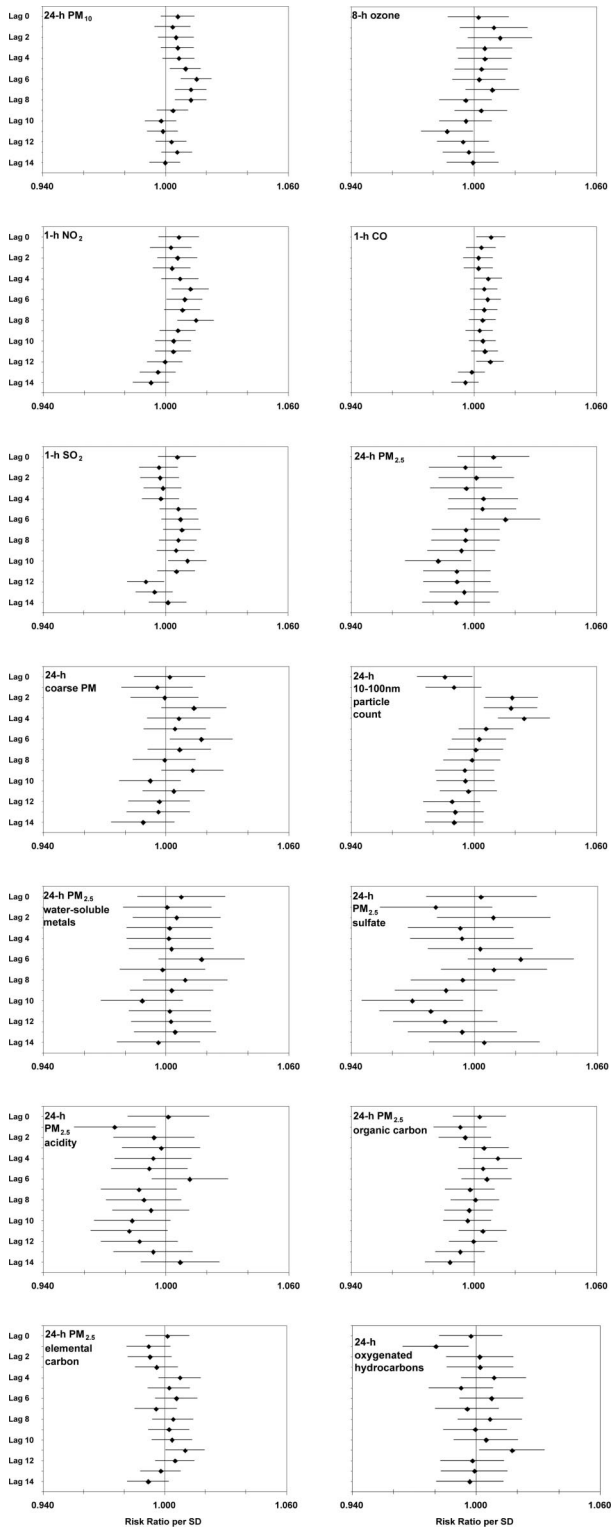

FIGURE 1. Risk ratios (diamonds) and 95% CIs (horizontal lines) per standard deviation increase from single-day lag models for the association of emergency department visits for asthma with daily ambient air quality measurements from AQS and the ARIES monitoring station.

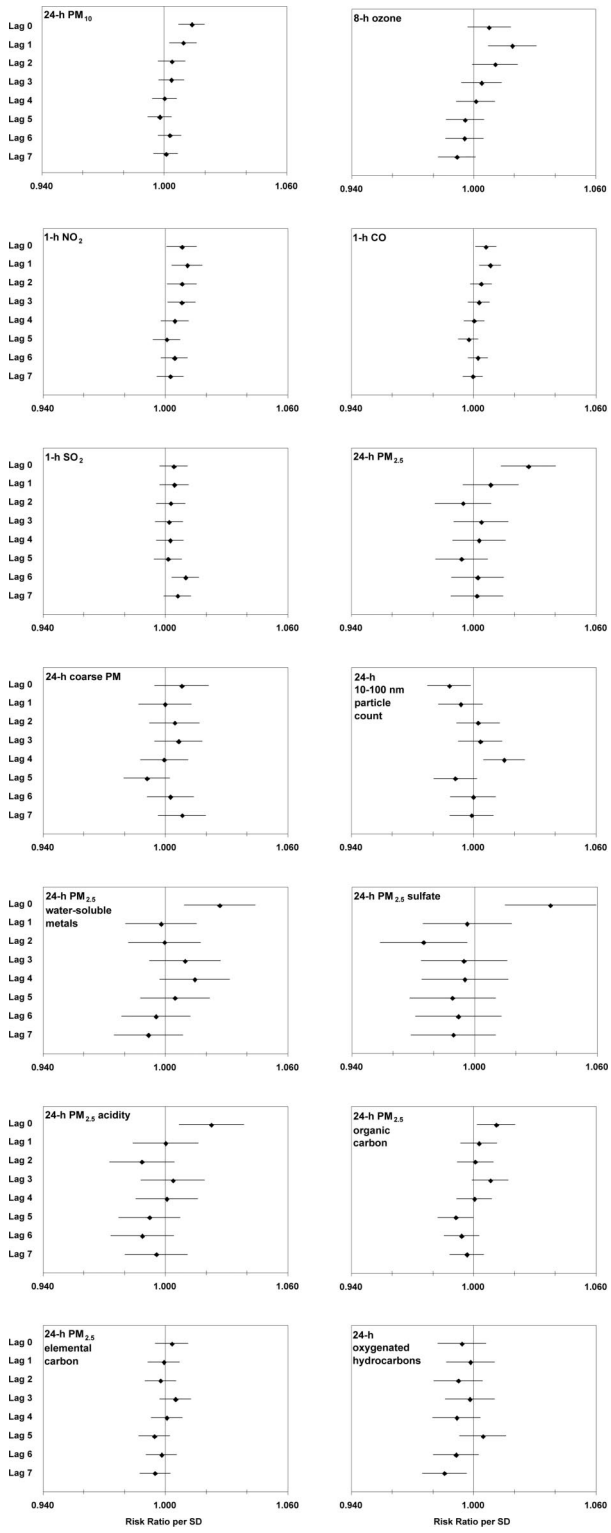

FIGURE 2. Risk ratios (diamonds) and 95% CIs (horizontal lines) per standard deviation increase from single-day lag models for the association of emergency department visits for upper respiratory illness with daily ambient air quality measurements from AQS and the ARIES monitoring station.

increased. (Appendix Table 2 presenting these results is available with the electronic version of this article.) Changing the placement of the knots in the cubic splines for time did not substantially alter the results. Estimates from models using negative lags for pollution, controlling for positive lags, were predominantly null. Results from models for the period 1 August 1998 through 31 August 2000 using the 2 sources of air quality data were not substantially different (Table 5).

Selected multipollutant analyses were performed. For URI visits, risk ratios for ozone were not substantially attenuated when PM_{10}, NO_2, and CO were included in the model (Fig. 3). For COPD, a much smaller outcome group, the risk ratios for both NO_2 and CO were attenuated in a 2-pollutant model (data not shown). As the estimates for asthma visits were somewhat elevated for several pollutants in the a priori models, we examined multipollutant models for asthma including all combinations of PM_{10}, ozone, NO_2, and CO. The estimates for NO_2 were generally not attenuated in multipollutant models, while the estimates for the other pollutants suggested weaker or no associations in the multipollutant models (data not shown).

DISCUSSION

This time-series study of respiratory emergency department visits provided a rare opportunity to examine associations of an extensive suite of ambient pollutant measures with specific respiratory conditions. In the a priori single-pollutant models (3-day moving average of lags of 0, 1, and 2 days for pollutant levels), URI visits were positively associated with PM_{10}, ozone, NO_2, and CO. The association with ozone persisted in multipollutant models. The associations observed for URI appeared to be specific to infants and children. COPD was positively associated with NO_2, and CO, while pneumonia was positively associated with $PM_{2.5}$ organic carbon. These results were generally robust to analytic method and model specification. We would expect several positive and negative associations by chance based on the number of tests performed. Overall, the a priori analyses yielded an abundance of positive associations and only a few negative associations.

Though few reasonably strong associations were observed with the PM finer size fraction and PM component measures, these data were available for a shorter time period and thus the estimates were less stable. The ultrafine particle count data, in particular, were missing for 44% of the days, often in blocks of time, which resulted in additional instability of the ultrafine particle models. Ultrafine particle levels also likely have considerable spatial and compositional heterogeneity. Additionally, high concentration days are potentially associated with different types of ultrafine nucleation events.[17,18] Further discussion of the ultrafine PM measurements can be found elsewhere.[17,18]

TABLE 4. Comparison of Results of a priori 3-Day Moving Average (Lags of 0, 1, and 2 Days) and Unconstrained Distributed Lag (Lags of 0 to 13 Days) Models for the Association of Daily Ambient Air Quality Levels With Respiratory Emergency Department Visits

			URI		Asthma		Pneumonia		COPD	
Pollutant	**Unit‡**	**Model**	**RR §**	**(95% CI)**	**RR §**	**(95% CI)**	**RR §**	**(95% CI)**	**RR §**	**(95% CI)**
PM_{10}	10 $\mu g/m^3$	3-day moving average*	1.014	(1.004–1.025)	1.009	(0.996–1.022)	1.011	(0.996–1.027)	1.018	(0.994–1.043)
		Unconstrained distributed lag†	1.073	(1.048–1.099)	1.099	(1.065–1.135)	1.087	(1.044–1.132)	1.092	(1.023–1.165)
Ozone	25 ppb	3-day moving average*	1.027	(1.009–1.045)	1.022	(0.996–1.049)	1.015	(0.981–1.050)	1.029	(0.977–1.084)
		Unconstrained distributed lag†	0.979	(0.942–1.017)	1.011	(0.957–1.067)	0.971	(0.900–1.047)	0.987	(0.880–1.109)
NO_2	20 ppb	3-day moving average*	1.019	(1.006–1.031)	1.014	(0.997–1.030)	1.000	(0.983–1.019)	1.035	(1.006–1.065)
		Unconstrained distributed lag†	1.057	(1.029–1.085)	1.047	(1.011–1.085)	1.024	(0.979–1.071)	1.018	(0.948–1.093)
CO	1 ppm	3-day moving average*	1.012	(1.003–1.021)	1.010	(0.999–1.022)	1.009	(0.996–1.021)	1.026	(1.004–1.048)
		Unconstrained distributed lag†	1.066	(1.045–1.087)	1.076	(1.047–1.105)	1.045	(1.011–1.080)	1.032	(0.975–1.092)
SO_2	20 ppb	3-day moving average*	1.010	(0.998–1.024)	1.001	(0.984–1.017)	1.003	(0.984–1.023)	1.016	(0.985–1.049)
		Unconstrained distributed lag†	1.062	(1.031–1.095)	1.015	(0.975–1.057)	1.022	(0.972–1.075)	1.116	(1.024–1.217)

*Single-pollutant models include cubic splines for temporal trends, temperature (lags 0, 1, and 2), and dew point temperature (lags 0, 1, and 2); indicators for day of the week, hospital entry/exit, and holidays; and a 3-day moving average of lags 0, 1, and 2 for pollutant

†Single-pollutant models include cubic splines for temporal trends, temperature (lags 0, 1, and 2), and dew point temperature (lags 0, 1, and 2); cubic terms of lags 3–13 for temperature and dew point temperature; indicators for day of the week, hospital entry/exit, and holidays; unconstrained distributed lag for pollutant lags 0–13.

‡Approximately 1 standard deviation.

§RR for 3-day moving average is per unit increase of the uniform 3-day moving average; RR for distributed lag is per unit increase of the weighted 14-day moving average.

TABLE 5. Comparison of Results of a priori Model* for the Association of Daily Ambient Air Quality Measures With Emergency Department Visits for All Respiratory Disease

		AQS 1 January 1993 to 31 August 2000		AQS 1 August 1998 to 31 August 2000		ARIES 1 August 1998 to 31 August 2000	
Pollutant	**Unit†**	**RR**	**(95% CI)**	**RR**	**(95% CI)**	**RR**	**(95% CI)**
24-h PM_{10}‡	10 $\mu g/m^3$	1.013	(1.004–1.021)	1.015	(1.003–1.029)	1.015	(0.999–1.032)
8-h O_3‡	25 ppb	1.024	(1.008–1.039)	1.027	(1.002–1.052)	1.025	(0.992–1.059)
1-h NO_2‡	20 ppb	1.016	(1.006–1.027)	1.028	(1.014–1.042)	1.024	(1.003–1.045)
1-h CO‡	1 ppm	1.011	(1.004–1.019)	1.010	(1.000–1.021)	1.018	(1.003–1.033)
1-h SO_2‡	20 ppb	1.008	(0.997–1.019)	1.010	(0.995–1.045)	1.020	(1.001–1.038)

*Single pollutant models include a 3-day moving average of lags 0, 1, and 2 for pollutant; cubic splines for temporal trends, temperature, and dew point temperature; indicators for day of the week, hospital entry/exit, and holidays.

†Approximately 1 standard deviation.

‡Spearman correlation coefficients for data on the same pollutant from AQS and ARIES monitoring stations for PM_{10}: r = 0.88; O_3: r = 0.98; NO_2: r = 0.78; CO: r = 0.70; and SO_2: r = 0.80.

In single-day lag models, estimates for URI, pneumonia and COPD were stronger for shorter pollutant lag structures (0–2 days), whereas associations for asthma were generally stronger at longer pollutant lags (5–8 days) and persisted for more than a week in distributed lag models. Results from the distributed lag models (lags of 0–13 days) suggest that associations for several of the outcomes persist for longer than the a priori 3-day moving average of lags 0, 1, and 2 days. A longer lag structure is plausible for emergency department visits for less severe respiratory conditions for biologic reasons (an underlying distribution of sensitivity or illness severity in the popu-

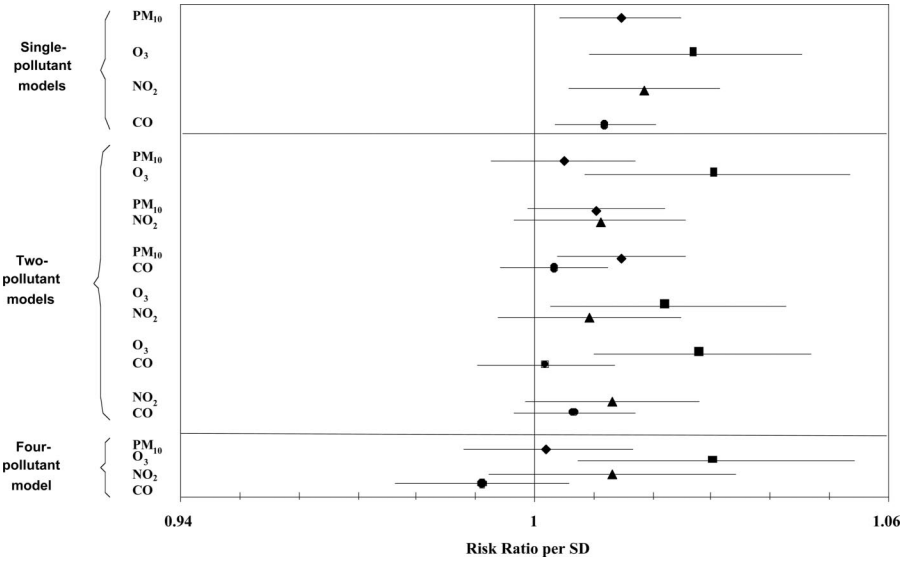

FIGURE 3. Risk ratios (diamonds) and 95% CIs (horizontal lines) per standard deviation increase from multipollutant models for the association of daily ambient air quality measurements with emergency department visits for upper respiratory illness.

lation) and for behavioral reasons (the time it takes for an exacerbation to become serious enough to necessitate a visit), especially compared with outcomes such as an acute cardiac event.

The results from this study are generally consistent with previously reported associations of ambient air pollution and respiratory morbidity.[1–4] (A brief description and supplemental references are provided in the electronic version of this article.) ED visits for respiratory outcomes have been relatively consistently associated with ozone and PM_{10}, and to a lesser extent with NO_2, SO_2, and CO.

In previous studies in Atlanta, which examined only asthma exacerbations, investigators reported associations of PM_{10} and ozone levels with pediatric asthma emergency department visits and hospital admissions in the summer.[19–21] In the present study, a 25 ppb increase in ozone was associated with a 2.6% increase in asthma visits in the warm months. Associations for pediatric asthma visits were somewhat stronger than those for adults for PM_{10}, NO_2 and CO.

Most previous studies that included PM component data (primarily $PM_{2.5}$ sulfate and acidity) have been in the northeastern United States and southeastern Canada.[22–29] Delfino et al[22] observed associations of $PM_{2.5}$ mass and sulfate, as well as of PM_{10} and ozone, with respiratory emergency department visits. Stieb et al[23] also reported positive associations for $PM_{2.5}$ mass and sulfate, as well as for ozone, SO_2, and PM_{10}, with asthma emergency department visits. Associations of acidity and sulfate with respiratory hospital admissions have been observed by several investigators.[24–29] We did not observe any associations for sulfate or acidity in the a priori analyses; however, given the width of the estimated confidence intervals, the study results are not inconsistent with even reasonably strong positive associations of respiratory outcomes with these and other pollutants. Additionally, acidity levels in the previous studies reporting associations with acidity were generally higher than the levels observed in Atlanta for this study.

Our understanding of the biologic mechanisms underlying associations between ambient air pollution and respiratory morbidity is evolving. Inhaled air pollutants may exacerbate existing respiratory disease, resulting in increased reactivity, decreased lung function, and increased respiratory symptoms.[30,31] In addition, inhaled pollutants may enhance the allergic response to an allergen.[32,33]

Many of the pollutant measurements at the ARIES monitoring site appeared to be spatially representative of Atlanta area. Measurements of criteria pollutants were available from both the ARIES and AQS monitoring sites; concentrations measured at the 2 sites were highly correlated and not substantially different in magnitude. Analyses of the ARIES criteria pollutant measurements yielded results comparable to those from analyses of the AQS measurement for the same pollutants. The spatial distribution of ambient $PM_{2.5}$ mass and several of its constituents, including sulfate, organic carbon, and elemental carbon, appeared to be relatively uniform across available monitoring stations; measurements from the ARIES monitoring site were similar to those from other monitoring sites in Atlanta. No information was available to assess the spatial variability for ultrafine particle count or oxygenated hydrocarbons.

Several issues need to be considered in interpreting the single- and multipollutant results. The single-pollutant results are likely confounded, at least in part, by correlated pollutants. Multipollutant models are typically used to address confounding by correlated pollutants, but results from multipollutant models may also be misleading. Pollutants are

measured with differing levels of error (including instrument error as well as other sources of error), whereas some potentially important pollutants may not be measured. A pollutant that exhibits a relatively strong association in a multipollutant model may be acting as a surrogate for an unmeasured or poorly measured pollutant.

The goal of this study was to assess the association between ambient pollution levels and respiratory morbidity. Ambient pollution levels are of interest for the assessment of population-level health effects of air pollution as well as for regulatory purposes. The measurement error that results from using centrally located monitors is likely to attenuate associations, but would not likely induce spurious associations. Additionally, personal behavior such as air conditioning use or time spent outdoors may affect personal exposure levels. This could affect the magnitude of the observed associations when compared with other locations with different behavior profiles. Eighty-three percent of households in Atlanta have central air conditioning,[34] which could weaken associations observed in Atlanta during the warm season relative to those observed in other areas.[35] However, in season-specific analyses, associations were often stronger or of similar magnitude in the warm season compared with the cool season or to the year-round analyses, especially for ozone.

We used an a priori approach to reduce possible biases associated with multiple testing and selective reporting of effect estimates. The pollutant metrics, outcome groups of interest, temporal relationship of the pollutant and outcome, and control for temporal trend were chosen prior to examining the data. We then performed secondary analyses to explore the associations further. Although there was some variability when we changed the number of knots to control for time, the overall conclusions would not have been substantially altered had we chosen a model with different knot frequency as the a priori model. We considered over-controlling for time a more conservative alternative to undercontrolling.

In this study, a large sample size and extensive air quality measurements allowed us to examine specific respiratory outcome groups in relation to air pollutants not routinely available for epidemiologic studies. The results contribute to the evidence of an association of several correlated gaseous and particulate pollutants (including ozone, NO_2, CO, PM, and organic carbon) with specific respiratory conditions.

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On Rounding

"It is the mark of an educated person to look for precision in each class of things only so far as the nature of the subject permits."

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