Air Quality and Pediatric Emergency Room Visits for Asthma in Atlanta, Georgia

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Pediatric emergency room visits for asthma were studied in relation to air quality indices in a spatio-temporal investigation of approximately 130,000 visits (~6,000 for asthma) to the major emergency care centers in Atlanta, Georgia, during the summers of 1993–1995. Generalized estimating equations, logistic regression, and Bayesian models were fitted to the data. In logistic regression models comparing estimated exposures of asthma cases with those of the nonasthma patients, controlling for temporal and demographic covariates and using residential zip code to link patients to spatially resolved ozone levels, the estimated relative risk per 20 parts per billion (ppb) increase in the maximum 8-hour ozone level was 1.04 (p < 0.05). The estimated relative risk for particulate matter less than or equal to 10 μm in aerodynamic diameter (PM10) was 1.04 per 15 μg/m3 (p < 0.05).

Exposure-response trends (p < 0.01) were observed for ozone (>100 ppb vs. <50 ppb: odds ratio = 1.23, p = 0.003) and PM10 (>60 μg/m3 vs. <20 μg/m3: odds ratio = 1.26, p = 0.004). In models with ozone and PM10, both terms became nonsignificant because of collinearity of the variables (r = 0.75). The other analytical approaches yielded consistent findings. This study supports accumulating evidence regarding the relation of air pollution to childhood asthma exacerbation. Am J Epidemiol 2000; 151:798-810.

It has been estimated that 2.3 million asthmatic children live in areas of the United States that have not met one or more of the National Ambient Air Quality Standards (1). Recently, the US Environmental Protection Agency promulgated revised standards for both ozone and particulate matter because of concerns about health risks at existing levels. While a substantial body of evidence has accumulated supporting the hypothesis that air pollution can exacerbate asthma symptoms (2), much remains to be understood regarding the quantitative relation between specific ambient air quality indices and asthma exacerbation. Furthermore, studies of the relation of air pollution to asthma exacerbation have generally not focused on African-American populations, who are at particularly high risk of asthma morbidity and mortality. At least one study, however, has suggested effect modification of the association of air pollution with asthma by socioeconomic status, with a stronger effect in persons of lower socioeconomic status (3).

We undertook a study to further characterize the relation of air quality with pediatric asthma exacerbation, using ambient monitoring data and billing records on visits to emergency rooms in Atlanta, Georgia, for the summers of 1993–1995.

MATERIALS AND METHODS

Air quality data

Air quality indices used in this study include ozone, particulate matter less than or equal to 10 μm in aerodynamic diameter (PM10), total oxides of nitrogen (NOx), pollen, mold, and various meteorologic data, including temperature and humidity. Sulfur dioxide data were also obtained but were not used in the present analyses because they appeared to be representative of only a small portion of the study area; moreover, sulfur dioxide levels in Atlanta are very low. No PM2.5 measurements were available. Air quality data were

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Abbreviations: GEE, generalized estimating equations; NOx, total oxides of nitrogen; PM10, particulate matter ≤10 μm in aerodynamic diameter.

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acquired for June 1 through August 31 of the years 1993–1995 from 13 monitoring stations located within or near the border of the Atlanta Metropolitan Statistical Area. The Atlanta Metropolitan Statistical Area consists of 20 counties, which, in 1990, contained 162 zip code areas in full or in part. The study area extends a radius of approximately 45 miles (72.4 km) from the center of Atlanta, encompassing roughly 6,000 square miles (15,540 km²) and over 3 million people. Monitoring station locations are shown in figure 1. Data were obtained from the following data sources: the Environmental Protection Agency’s Aerometric Information Retrieval System, the Clean Air Status and Trends Network, the Georgia Department of Natural Resources, the Fulton County Health Department, the National Climatic Data Center, the US Geological Survey, the Southeastern Consortium for Intensive Oxidant and Nitrogen Measurements, and the Atlanta Allergy Clinic.

Raw station data include hourly averages of continuous ozone and NOₓ measurements and 24-hour mea-

FIGURE 1. Locations of monitoring stations and participating medical facilities in a study of pediatric asthma emergency room visits, Atlanta, Georgia, 1993–1995. (1 mile = 1.61 km.) NOₓ, total oxides of nitrogen.

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measurements of PM$_{10}$, pollen, and mold. All data were obtained 7 days per week with the exception of the PM$_{10}$ (5–6 days/week) and pollen and mold data (5 days/week).

Spatial resolution of the ozone data was carried out by the geostatistical interpolation procedure known as “universal kriging,” using the Geographic Information System ARC/INFO® facility. The kriging procedure involves estimating a smooth surface from irregularly spaced data points based on the assumption that the spatial variation is homogeneous over the domain and depends only on the distance between sites. In addition to providing a predicted value, kriging provides an estimate of variance associated with the uncertainty in the spatial resolution of data. The universal kriging procedure uses a linear model of the semivariogram and, unlike ordinary kriging, incorporates a drift function to account for a structural component in the spatial variation. A linear drift function was selected because this method resulted in lower kriging variances in ozone estimates than did a quadratic drift function. A 3 km x 3 km grid was used with ozone levels at zip code centroids estimated by bilinear interpolation.

Insufficient data were available with which to spatially resolve the other pollutants. Twenty-four-hour PM$_{10}$ data from the Fulton County Health Department, located in downtown Atlanta, were used as the primary particulate matter variable. For the NO$_x$ variable, data from two stations near the center of the Atlanta Metropolitan Statistical Area were used, because this is the area of highest population; average values from the Georgia Institute of Technology and South Dekalb College stations were used. Pollen and mold data, which are 24-hour measurements, were available from only one location. Daily meteorologic data were obtained from the Hartsfield International Airport, the meteorologic station nearest the center of the study area.

For ozone and NO$_x$, hourly values were interpolated within a day if 1–2 hours of data were missing, to allow computation of a daily value. If more than 2 hours of data were missing, the daily value assigned to the day was treated as missing.

Outcome assessment and collection of demographic data

Participation was obtained from all but one Atlanta-area emergency departments that reportedly handle at least 10,000 pediatric visits annually. Participating hospitals were the Grady Hospital/Hughes Spalding Pediatric Center, Egleston Children’s Hospital, Scottish Rite Children’s Hospital, South Fulton Hospital, Southern Regional Hospital, Gwinnett Hospital, and Henry Hospital (figure 1). The participating facilities handle approximately 80 percent of pediatric emergency care visits in Atlanta, according to data provided by the Georgia State Health Planning Agency, and the clienteles served encompass a wide range of socioeconomic levels.

For all visits to the participating emergency departments during June through August, 1993–1995, the following information was requested from computerized billing records maintained by each facility: medical record number, date and time of admission, International Classification of Diseases, Ninth Revision (4), diagnostic code, procedure code, age, sex, race, zip code of residence, and payment method (payment by Medicaid was used as a crude proxy for socioeconomic status). The age range included in this study was 0–16 years, and zip code of residence was required to be one of 162 zip codes that fall at least partially within the Atlanta Metropolitan Statistical Area. Repeat visits within a single day were counted only once. For the purposes of this study, a patient was categorized as an asthma case if any of the International Classification of Diseases, Ninth Revision, diagnostic codes for the visit were 493 (any fourth and fifth digit extension, “asthma”), 786.09 (“wheezing”), or 519.1 (includes “reactive airway disease,” applicable only in 1993). All other patients were considered controls.

Statistical analysis

A priori specification of terms. Based on results of similar studies, a 1-day lag was selected to be the lag-time applied for each of the pollutants and meteorologic variables other than temperature (5–14). For the temperature covariate, the minimum temperature in the 24-hour period preceding the emergency room visit was selected. The 8-hour ozone metric was used in most of the analyses, because this is the averaging time of the new ozone standard and our findings for the 1-hour and 8-hour metrics are indistinguishable because of their high correlation in our data ($r = 0.99$). Averaging times used for the other variables were the following: PM$_{10}$, 24-hour average; NO$_x$, 1-hour maximum; pollen, 24-hour average; mold, 24-hour average; and temperature, daily minimum. In all analyses, missing pollutant data led to exclusion of the day (generalized estimating equations (GEE) analysis) or patient (logistic regression analysis) from the analysis, while missing covariate data were handled by the use of dummy variables for the unknown values.

GEE analyses. The data were initially treated ecologically, modeling the daily count of asthma presentations throughout Atlanta as a function of average Atlanta-wide air quality indices, as well as time.
covariates. A Poisson model with a log link function was fitted using GEE analysis to allow for autocorrelation in the daily counts (15). Models were implemented using the GENMOD procedure in SAS (16), with AR-1 to account for correlation in asthma visits on a given day with the previous day’s visits (this autocorrelation was actually quite minimal: Durbin-Watson statistic = 1.98 (not significant)). Let \( Y_t \) be the total number of observed pediatric emergency room asthma visits in the study area on day \( t \). The expected value of \( Y_t \) was modeled as \( n_t \lambda_t \), where \( n_t \) is the observed number of pediatric emergency room visits and \( \lambda_t \) is the rate of asthma visits on day \( t \). The natural log of \( \lambda_t \) was, in turn, modeled as follows:

\[
\ln \lambda_t = \alpha_0 + (\beta_1 \text{Exp}1_{t-1} + \beta_2 \text{Exp}2_{t-1} + ...) +
\gamma_1 \text{Tues} + \gamma_2 \text{Wed} + \gamma_3 \text{Thurs} + \gamma_4 \text{Fri} + \gamma_5 \text{Sat} +
\gamma_6 \text{Sun} + \gamma_7 \text{Day} + \gamma_8 \text{Day}^2 + \gamma_9 \text{Year}94 +
\gamma_{10} \text{Year}95 + \text{interaction terms},
\]

where \( \alpha_0 \) was the intercept term, \( \text{Exp}1_{t-1} \) was the daily value of the first air quality measure on day \( t - 1 \), and \( \text{Exp}2_{t-1} \) was the daily value of the second air quality measure on day \( t - 1 \). \text{Day} was the centered index for the day of summer. Because pediatric asthma emergency room visits followed a rough U-shape over the course of each summer, we included both a linear term and a quadratic term for day of summer. \text{Year}94 and \text{Year}95 were indicator variables for year, with 1993 being the reference year. \text{Tues, Wed, Thurs, Fri, Sat,} and \text{Sun} were indicator variables for day of the week, with Monday used as the referent. Interaction terms for \text{Day} and \text{Year}94 and for \text{Day} and \text{Year}95 were included. Because this parametric model fitted the data well and the study period was limited to summers, we did not use semiparametric or nonparametric methods commonly used in these types of studies; sensitivity analyses using smoothing splines for time yielded similar results.

Air pollutants (\( \text{Exp1}, \text{Exp2}, \) etc.) were treated as continuous variables in most analyses, and residuals were studied to assess the appropriateness of the implicit exposure-response form. The daily average of the ozone values obtained from all of the ozone monitoring stations was used in these analyses. Each air quality variable was scaled by its estimated standard deviation, facilitating comparisons of rate ratios for different air quality variables.

Logistic regression analyses. Secondly, in a case-control treatment of the data, asthma cases were compared with the emergency room patients who presented with nonasthma diagnoses with respect to air quality indices of interest. Unconditional logistic regression (using the LOGISTIC procedure in SAS (16)) was performed to model the probability of a pediatric asthma emergency room visit as a function of the estimated ozone level, other air quality indices, temporal covariates, and demographic covariates. Let \( P \) be the probability that a given emergency room visit is due to asthma. The general form of the logistic regression model was as follows:

\[
\logit(P) = \alpha_0 + (\beta_1 \text{ozone} + \beta_2 \text{Exp2} + ...) +
\gamma_1 \text{Tues} + \gamma_2 \text{Wed} + \gamma_3 \text{Thurs} + \gamma_4 \text{Fri} + \gamma_5 \text{Sat} +
\gamma_6 \text{Sun} + \gamma_7 \text{Day} + \gamma_8 \text{Day}^2 + \gamma_9 \text{Year}94 + \gamma_{10} \text{Year}95 + 
\gamma_{11} \text{Age}_{2-5} + \gamma_{12} \text{Age}_{6-10} + \gamma_{13} \text{Age}_{11-16} + 
\gamma_{14} \text{Race}_{\text{African American}} + \gamma_{15} \text{Race}_{\text{Unknown}} + 
\gamma_{16} \text{Race}_{\text{Other}} + \gamma_{17} \text{Male} + \gamma_{18} \text{Medicaid} + 
\text{interaction terms},
\]

where \( \alpha_0 \) was the intercept term, \( \text{ozone} \) was the daily value for ozone on the previous day for the residential zip code of the patient, and \( \text{Exp2} \) was the average value of the second air quality measure on the previous day. In these analyses, spatially resolved estimates of each patient’s ozone level were used, by linking zip code of residence to the kriged ozone values. The temporal control variables were analogous to those used in the GEE models above. \( \text{Age}_{2-5}, \text{Age}_{6-10}, \) and \( \text{Age}_{11-16} \) were indicator variables for age groups 2–5, 6–10, and 11–16 years, respectively, with age group 0–1.9 years being the reference group. \( \text{Race}_{\text{African American}}, \text{Race}_{\text{Unknown}}, \) and \( \text{Race}_{\text{Other}} \) were indicator variables for racial groups, with Whites used as the reference group. \( \text{Male} \) was an indicator variable for males. \( \text{Medicaid} \) was the indicator variable for payment by Medicaid, with payment by other methods used as the reference group. To assess the impact of nonindependence of repeat visitors, we conducted a sensitivity analysis using GEE analysis with a logit link and exchangeable correlation structure; the impact was negligible.

Bayesian analyses. In order to evaluate the impact of spatial correlation in the asthma presentation rates and the prediction error associated with the kriged ozone values, we fitted an additional model to these data. The augmented model was based on a Bayesian approach in which a conditional autoregressive correlation structure (17) was used to impart spatial and temporal dependence among the asthma rates. In addition,
the model was adjusted to reflect the fact that predicted (i.e., kriged) measurements for ozone, as opposed to actual measurements, were used in the spatially resolved analysis. Estimates of the magnitude of the uncertainty in the predicted ozone estimates were based on the prediction errors produced by the kriging approach (18). A model analogous to that used in the GEE analyses (but using zip code-specific data on daily ozone levels, percentage of emergency room visits by African-American children, and percentage of emergency room visits by children using Medicaid), adjusted to reflect spatial/temporal dependence in the asthma rates and uncertainty in the predicted ozone values, was fitted to the data with estimates of the posterior distributions of the coefficients derived using a Markov chain Monte Carlo approach (19). Details on the methods used in this analysis are given elsewhere (20).

RESULTS

Descriptive analyses of air quality data

Descriptive analyses of the air quality data are presented in table 1, and time plots are presented in figure 2. There were 34 ozone exceedance days in Atlanta during the three summers of the study, most of which occurred during two episodes in each of the summers of 1993 and 1995. The summer of 1994 was cool and wet, and both ozone and \( \text{PM}_{10} \) levels were lower than in the summers of 1993 and 1995. Correlation among concentrations of the various air pollutants was expected because of their dependence on atmospheric mixing. Positive correlations were observed among ozone, \( \text{PM}_{10} \), and NO\(_x\) levels. Ozone was most correlated with \( \text{PM}_{10} \). While, on average, ozone peak values were highest near downtown Atlanta, the spatial distribution of peak ozone levels varied, depending on wind speed and direction (21).

### TABLE 1. Means values, ranges, and Spearman's rank correlation coefficients for air quality variables in a study of pediatric asthma emergency room visits, Atlanta, Georgia, June through August, 1993-1995

<table>
<thead>
<tr>
<th>Mean</th>
<th>Range</th>
<th>Spearman's rank correlation coefficient</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>8-hour ozone</td>
</tr>
<tr>
<td>8-hour ozone (ppb)</td>
<td>59.3 (19.1)‡</td>
<td>18.2-113</td>
</tr>
<tr>
<td>1-hour ozone (ppb)</td>
<td>68.6 (21.1)</td>
<td>22.8-132</td>
</tr>
<tr>
<td>24-hour PM(_{10}) (ug/m(^3))</td>
<td>38.9 (15.5)</td>
<td>9-105</td>
</tr>
<tr>
<td>1-hour NO(_x) (ppb)</td>
<td>81.7 (53.8)</td>
<td>5.35-306</td>
</tr>
<tr>
<td>24-hour pollen (grains/m(^3))</td>
<td>3.8 (4.5)</td>
<td>0-29.8</td>
</tr>
<tr>
<td>24 hour mold (grains/m(^3))</td>
<td>474 (342)</td>
<td>91-2,710</td>
</tr>
<tr>
<td>Minimum temperature (°F)</td>
<td>71.4 (3.4)</td>
<td>57-78</td>
</tr>
<tr>
<td>Wind speed (m/s)</td>
<td>8.28 (2.37)</td>
<td>4.1-19.3</td>
</tr>
</tbody>
</table>

* \( p < 0.05 \)

‡ Numbers in parentheses, standard deviation.

Descriptive analyses of outcome data

A total of 128,969 pediatric emergency room visits occurred at the participating medical facilities between June 1 and August 31 of 1993–1995. Of these, 5,934 (5 percent) patients presented with asthma, as defined above. The average number of daily emergency room visits was 467 (standard deviation, 84). The average number of daily emergency room visits for asthma was 22 (standard deviation, 9; range, 5–52).

The distributions of various demographic characteristics by case status are presented in table 2. Demographic distributions exhibited expected patterns: Relative to the nonasthma cases, the asthma cases had higher proportions of males, higher proportions of African Americans, and higher proportions of patients using Medicaid. The total number of emergency room visits showed variation across weekdays, with Saturday and Sunday being notably higher usage days than weekdays, particularly Friday. The proportion of all emergency room visits that was due to asthma was highest on Sunday and Tuesday and lowest on Friday. While total emergency room visits exhibited a slight U-shaped distribution through the summer months (with June and August being higher than July), the asthma cases showed a more pronounced U shape, leading to a U-shaped distribution in the proportion of all emergency room visits that were asthma cases. The total number of emergency room visits rose each year from 1993 to 1995, with asthma cases comprising an increasing proportion each year.

Time plots of daily air quality levels, asthma counts, and total emergency room visits are presented in figure 2. The asthma counts are crude (i.e., unadjusted for emergency room usage patterns, etc.), and the expected air quality effects are subtle relative to the amount of noise expected in the data, and thus model-
FIGURE 2. Time plots of crude data on air quality, asthma visits, and total emergency room (ER) visits in a study of pediatric asthma ER visits, Atlanta, Georgia, 1993–1995. PM_{10}, particulate matter ≤ 10 μm in aerodynamic diameter; NO_{x}, total oxides of nitrogen.
ing is a critical tool for the assessment of the temporal relation between these variables.

Maps of the geographic distribution of several variables by zip code are presented in figure 3. Visual inspection of these maps in comparison with the map of kriged ozone levels by zip code suggests that ozone level, race, Medicaid payment status, and asthma presentation rate may be correlated in space.

**GEE analyses**

Table 3 presents the results for six models modeling the air quality variables one at a time, adjusting for the temporal variables and incorporating the offset term. The six air quality indices are average maximum 8-hour ozone, 24-hour PM$_{10}$, NO$_x$, pollen, mold, and minimum temperature. Statistically significant positive associations were observed for maximum 8-hour ozone and 24-hour PM$_{10}$. For each of these indices, there was an increase in asthma rates of approximately 4 percent per standard deviation increase in the pollutant (20 parts per billion (ppb) for ozone and 15 μg/m$^3$ for PM$_{10}$). As expected based on their high correlation ($r = 0.99$), results for 1-hour ozone were similar to those obtained using the 8-hour metric (relative risk = 1.04, $p = 0.01$). Because the new Environmental Protection Agency standard makes use of the maximum 8-hour ozone concentration, all further analyses were based on the 8-hour metric. For NO$_x$, pollen, mold, and temperature, the rate ratios were near unity and were not statistically significant. Quadratic terms for ozone, PM$_{10}$, temperature, pollen, and mold were assessed in models with a linear term for the air quality indicator, and none of the quadratic terms was significant. In sensitivity analyses of alternative lag times, the 1-day lag was found to be the only significant predictor of asthma visits in the PM$_{10}$ analyses, and in the ozone analyses the 2-day lag was found to be significant and slightly stronger than the 1-day lag (relative risk = 1.044, $p = 0.007$).

**Logistic regression analyses**

Results of logistic regression modeling of the data are presented in tables 4–6. Table 4 shows the estimated odds ratios for a model fitting the kriged 8-hour ozone and the temporal and demographic covariates. The odds ratio for ozone was significantly positive, with a magnitude similar to that observed in the GEE analysis of the 8-hour ozone variable. Each of the odds ratios for the covariates had the expected direction; that is, males, African Americans, and persons who paid via Medicaid all had an increased risk of being an asthma case (table 4).

A comparison of models for the kriged and unkriged (average across monitoring stations) 8-hour ozone metrics is presented elsewhere (21). In summary, when models controlling only for temporal variables were run, the magnitude of the odds ratio for the kriged ozone metric was substantially larger than that for the unkriged metric. This is apparently a reflection of the spatial correlation of ozone with race and socioeconomic status, because after adjustment for the demographic variables, the magnitude of the odds ratios for the kriged and unkriged metrics was the same (both odds ratios were 1.04), with only slightly smaller $p$ values in the kriged analysis. Nonetheless, this suggests that there was spatial variation in ozone that was captured by the kriging process. The remainder of the analyses made use of the kriged ozone data, adjusting for temporal and demographic covariates.

To evaluate the exposure-response relation of ozone and asthma exacerbation in our data, we ran a logistic regression model with multiple ozone levels using dummy variables (table 5). The odds ratios for
each level of ozone relative to a level of <50 ppb showed a generally increasing pattern (p for trend = 0.005). Starting at 70 ppb, the odds ratios were consistently elevated relative to <50 ppb, and for the highest category (>100 ppb) the odds ratio was 1.23 (p = 0.003).

A logistic regression model assessing PM$_{10}$ controlling for demographic and temporal covariates yielded an odds ratio of 1.042 per 15-µg/m$^3$ increase in 24-hour PM$_{10}$ (95 percent confidence interval: 1.008, 1.077; p = 0.014). When multiple levels of PM$_{10}$ were assessed, an exposure-response relation was apparent, with a p value for the trend test of 0.01 (table 5). All odds ratios for categories above 20 µg/m$^3$ were significantly elevated relative to <20 µg/m$^3$, with the highest category, >60 µg/m$^3$, having an odds ratio of 1.26 (p = 0.004).
TABLE 3. Rate ratios from separate generalized estimating equation models for each air quality indicator (lagged 1 day), adjusted for temporal covariates* and including an offset term for total emergency room visits, Atlanta, Georgia, June through August, 1993–1995

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Rate ratio</th>
<th>95% Cl†</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Average maximum 8-hour ozone (per 20 ppb)‡</td>
<td>1.040</td>
<td>1.008, 1.074</td>
<td>0.013</td>
</tr>
<tr>
<td>PM₁₀ (per 15 μg/m³)†</td>
<td>1.038</td>
<td>1.004, 1.074</td>
<td>0.029</td>
</tr>
<tr>
<td>NO₂ (per 50 ppb)‡</td>
<td>1.012</td>
<td>0.987, 1.039</td>
<td>0.348</td>
</tr>
<tr>
<td>Pollen (per 5 grains/m³)‡</td>
<td>0.980</td>
<td>0.937, 1.025</td>
<td>0.375</td>
</tr>
<tr>
<td>Mold (per 300 grains/m³)‡</td>
<td>0.985</td>
<td>0.954, 1.016</td>
<td>0.341</td>
</tr>
<tr>
<td>Minimum temperature (per 5°F)‡</td>
<td>1.000</td>
<td>0.991, 1.001</td>
<td>0.955</td>
</tr>
</tbody>
</table>

* Temporal covariates: day of week (dummy variables), linear and quadratic terms for day of summer (centered), year (dummy variables, 1994 and 1995), and interaction terms for day of summer x year.
† CI, confidence interval; PM₁₀, particulate matter ≤ 10 μm in aerodynamic diameter; NO₂, total oxides of nitrogen.
‡ Exposure increment is approximately equal to 1 standard deviation of the observed exposure variables (see table 1), for roughly comparable scales across continuous exposure indices.

Results from a series of models are presented in table 6, fitting ozone, PM₁₀, and minimum temperature. Temperature adjustment did not alter the observed association with either ozone or PM₁₀. When PM₁₀ and ozone were fitted simultaneously, the magnitudes of both odds ratios remained greater than the null value but became statistically nonsignificant. When we added an interaction term for ozone and PM₁₀ (model not shown), the interaction term was nonsignificant. It is possible that the ozone term became nonsignificant when PM₁₀ was added to the model, because the power of this model was reduced as a consequence of PM₁₀ measurements not being available on all days. To check this, we ran a model with ozone as the only air quality variable, deleting the days on which PM₁₀ was missing. In this model, ozone remained significant, which suggests that its nonsignificance in the model with PM₁₀ was a result of collinearity of ozone and PM₁₀ rather than loss of power due to missing data. The multivariate models are highly complex in terms of the interrelations among the air quality parameters. In particular, some of the air quality variables may be intervening variables (e.g., while both temperature and ozone may be independently associated with asthma exacerbation, elevated temperature also leads to ozone formation).

We assessed the interaction of ozone with either race or Medicaid payment status in the prediction of asthma risk; the interaction of PM₁₀ with race or Medicaid payment status was also assessed. None of these interaction terms was statistically significant. The interaction of ozone or PM₁₀ with age was nonsignificant.

Bayesian analyses

The Bayesian analyses yielded results that were consistent with the above analyses. A positive association of ozone level and asthma emergency room visits was observed, with a fitted relative risk of 1.026 per 20 ppb of ozone, and the 95 percent posterior credible set for the ozone parameter did not include the null value. The impact of adjusting for kriging error and for spatial correlation was considered separately. Neither adjust-

TABLE 4. Odds ratios for ozone (maximum 8-hour, lagged 1 day) and demographic variables from logistic regression models predicting the probability of an emergency room patient's being an asthma case, adjusted for temporal covariates,* Atlanta, Georgia, June through August, 1993–1995

<table>
<thead>
<tr>
<th>Variable</th>
<th>Odds ratio</th>
<th>95% Cl†</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ozone (kriged, 8-hour average, 1-day lag)</td>
<td>1.04</td>
<td>1.02, 1.07</td>
<td>0.0010</td>
</tr>
<tr>
<td>Age (years)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2–5 vs. 0–1.9</td>
<td>1.37</td>
<td>1.26, 1.49</td>
<td>0.0001</td>
</tr>
<tr>
<td>6–10 vs. 0–1.9</td>
<td>1.36</td>
<td>1.25, 1.49</td>
<td>0.0001</td>
</tr>
<tr>
<td>11–16 vs. 0–1.9</td>
<td>1.34</td>
<td>1.22, 1.48</td>
<td>0.0001</td>
</tr>
<tr>
<td>Race</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Black vs. White</td>
<td>2.17</td>
<td>2.03, 2.31</td>
<td>0.0001</td>
</tr>
<tr>
<td>Unknown vs. White</td>
<td>1.06</td>
<td>0.86, 1.31</td>
<td>0.59</td>
</tr>
<tr>
<td>Other vs. White</td>
<td>1.04</td>
<td>0.91, 1.20</td>
<td>0.57</td>
</tr>
<tr>
<td>Sex: male vs. female</td>
<td>1.40</td>
<td>1.33, 1.48</td>
<td>0.0001</td>
</tr>
<tr>
<td>SES†: Medicaid vs. non-Medicaid</td>
<td>1.25</td>
<td>1.18, 1.33</td>
<td>0.0001</td>
</tr>
</tbody>
</table>

* Temporal covariates: day of week (dummy variables), linear and quadratic terms for day of summer (centered), year (dummy variables, 1994 and 1995), and interaction terms for day of summer x year.
† CI, confidence interval; SES, socioeconomic status.
TABLE 5. Odds ratios for multiple levels of ozone (maximum 8-hour, kriged, lagged 1 day) and PM₁₀* from separate logistic regression models for the two pollutants, adjusted for temporal and demographic covariates,† Atlanta, Georgia, June through August, 1993–1995

<table>
<thead>
<tr>
<th>Air quality variable</th>
<th>No. of asthma cases</th>
<th>Odds ratio</th>
<th>95% CI</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>8-hour ozone level (ppb)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;50</td>
<td>1,875</td>
<td>1.0 §</td>
<td></td>
<td></td>
</tr>
<tr>
<td>50–59 vs. &lt;50</td>
<td>303</td>
<td>1.07</td>
<td>0.98, 1.16</td>
<td>0.14</td>
</tr>
<tr>
<td>60–69 vs. &lt;50</td>
<td>963</td>
<td>1.00</td>
<td>0.92, 1.09</td>
<td>0.30</td>
</tr>
<tr>
<td>70–79 vs. &lt;50</td>
<td>954</td>
<td>1.07</td>
<td>0.99, 1.17</td>
<td>0.10</td>
</tr>
<tr>
<td>80–89 vs. &lt;50</td>
<td>566</td>
<td>1.06</td>
<td>0.96, 1.17</td>
<td>0.26</td>
</tr>
<tr>
<td>90–99 vs. &lt;50</td>
<td>308</td>
<td>1.11</td>
<td>0.98, 1.26</td>
<td>0.11</td>
</tr>
<tr>
<td>≥100 vs. &lt;50</td>
<td>287</td>
<td>1.23</td>
<td>1.07, 1.40</td>
<td>0.003</td>
</tr>
<tr>
<td>PM₁₀ level (µg/m³)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;20</td>
<td>427</td>
<td>1.0 §</td>
<td></td>
<td></td>
</tr>
<tr>
<td>20–29 vs. &lt;20</td>
<td>752</td>
<td>1.16</td>
<td>1.02, 1.33</td>
<td>0.03</td>
</tr>
<tr>
<td>30–39 vs. &lt;20</td>
<td>1,079</td>
<td>1.14</td>
<td>1.00, 1.30</td>
<td>0.04</td>
</tr>
<tr>
<td>40–49 vs. &lt;20</td>
<td>1,904</td>
<td>1.17</td>
<td>1.03, 1.33</td>
<td>0.02</td>
</tr>
<tr>
<td>50–59 vs. &lt;20</td>
<td>623</td>
<td>1.19</td>
<td>1.03, 1.37</td>
<td>0.02</td>
</tr>
<tr>
<td>≥60 vs. &lt;20</td>
<td>331</td>
<td>1.26</td>
<td>1.07, 1.47</td>
<td>0.004</td>
</tr>
</tbody>
</table>

* PM₁₀, particulate matter ≤ 10 µm in aerodynamic diameter; CI, confidence interval.
† Day of week (dummy variables), linear and quadratic terms for day of summer (centered), year (dummy variables, 1994 and 1995), interaction terms for day of summer x year, race (dummy variables), Medicaid payment status, sex, and age group (dummy variables).
‡ p value for trend test: 0.005 (ozone), 0.01 (PM₁₀).
§ Reference category.

TABLE 6. Odds ratios from logistic regression models fitting combinations of air quality variables,* adjusted for temporal and demographic covariates, Atlanta, Georgia, June through August, 1993–1995

<table>
<thead>
<tr>
<th>Model</th>
<th>Variable†</th>
<th>Odds ratio</th>
<th>95% CI‡</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Ozone (per 20 ppb, kriged, 8-hour)</td>
<td>1.042</td>
<td>1.017, 1.068</td>
<td>0.001</td>
</tr>
<tr>
<td>2</td>
<td>PM₁₀‡ (per 15 µg/m³)</td>
<td>1.042</td>
<td>1.008, 1.077</td>
<td>0.014</td>
</tr>
<tr>
<td>3</td>
<td>Ozone (per 20 ppb, kriged, 8-hour) Temperature (per 5°F)</td>
<td>1.046</td>
<td>1.019, 1.073</td>
<td>0.0007</td>
</tr>
<tr>
<td>4</td>
<td>PM₁₀ (per 15 µg/m³) Temperature (per 5°F)</td>
<td>1.048</td>
<td>1.012, 1.086</td>
<td>0.010</td>
</tr>
<tr>
<td>5</td>
<td>Ozone (per 20 ppb, kriged, 8-hour) deleting PM₁₀ &quot;missingness&quot;</td>
<td>1.040</td>
<td>1.008, 1.072</td>
<td>0.012</td>
</tr>
<tr>
<td>6</td>
<td>Ozone (per 20 ppb, kriged, 8-hour) PM₁₀ (per 15 µg/m³)</td>
<td>1.024</td>
<td>0.982, 1.069</td>
<td>0.270</td>
</tr>
</tbody>
</table>

* Day of week (dummy variables), linear and quadratic terms for day of summer (centered), year (dummy variables, 1994 and 1995), interaction terms for day of summer x year, race (dummy variables), Medicaid payment status, sex, and age group (dummy variables).
† Exposure increment is approximately equal to 1 standard deviation of the observed exposure values (see table 1), in order to provide roughly comparable scale across continuous exposure indices.
‡ CI, confidence interval; PM₁₀, particulate matter ≤ 10 µm in aerodynamic diameter.

DISCUSSION

These analyses suggested a positive association between pediatric asthma presentation rates and the pollutant of primary interest in this study, ozone, when modeled either singly (with adjustment only for tem-
polar and demographic covariates) or with temperature adjustment. In both the GEE and logistic regression analyses lagging ozone values 1 day, the estimated relative risk per 20-ppb increase (an increment roughly equal to 1 standard deviation of the observed ozone values) in the maximum 8-hour ozone level is 1.04. An exposure-response trend is suggested in our data, with odds ratios being consistently elevated for categories of 70–79 ppb and above (relative to <50 ppb), increasing to a highly significant odds ratio of 1.23 for the highest ozone category (>100 ppb). While the odds ratios showed a generally increasing pattern, the data are consistent with a variety of exposure-response shapes ranging from a monotonically increasing trend to a threshold form.

With respect to particulate matter, our analyses also indicated a positive association with PM$_{10}$, modeled singly or with temperature adjustment, with an estimated relative risk of 1.04 per 15-μg/m$^3$ increase (1 standard deviation) in PM$_{10}$. An exposure-response relation was again evident. The odds ratios for all categories above 20 μg/m$^3$ were greater than unity relative to <20 μg/m$^3$, with the highest category having a highly significant odds ratio of 1.26, the confidence intervals again accommodating a variety of exposure-response shapes.

Finally, in a model including both ozone and PM$_{10}$, neither association was significant, reflecting a collinearity problem ($r = 0.75$). Because these air quality parameters covary, this study could not distinguish between the effects of the two pollutants.

Whereas race and Medicaid payment status were strong independent predictors of asthma presentation rates, they did not appear to modify either the ozone-asthma or the PM$_{10}$-asthma relation. Thus, our results do not lend support to the hypothesis that the association of pollution with asthma is stronger in lower socioeconomic status groups (African Americans, Medicaid recipients) because of factors such as a lower prevalence of air conditioning. However, it should be noted that the power to detect interaction is always lower than the power to observe a main effect, the expected amount of effect modification was small, and both race and Medicaid payment status were crude, dichotomized socioeconomic status indicators.

This study was limited at the outset by several design features. Because it was a retrospective study, it was not possible to obtain information not present in historical records. This necessitated reliance on a number of assumptions, and the degree to which these assumptions were met will have affected the precision and validity of the results. Regarding exposure issues, an important assumption was that ambient levels of pollutants are meaningfully related to personal exposures. Our study did not account for many factors that might affect personal exposures, such as air conditioning use and time spent outdoors in the relevant time period. Data on personal exposure to potential confounders such as cigarette smoke, cockroaches, and indoor mold also were not available. Furthermore, the spatially resolved analyses assumed that the patient was in the zip code of residence during the day before he or she went to the emergency room; information on where the patient actually was would have been useful. Regarding ambient air quality data, it would have been useful to have PM$_{10}$ data and pollen counts for all weekend days, information on fine particulate matter and particulate matter composition for all days, and spatially resolved data for the air quality indicators other than ozone.

Regarding the study population, we were limited to the emergency rooms that agreed to participate in the study, estimated to comprise 80 percent of the total emergency room visits in the Atlanta Metropolitan Statistical Area. In addition to the 20 percent of emergency room visits that were not included, the study population lacked individuals who presented at facilities other than emergency rooms. These individuals are likely to have differed with respect to socioeconomic status and other factors. Furthermore, there may be changes through time in emergency room usage resulting from such factors as changes in Medicaid reimbursement policies. Because of these considerations, we chose to use as our "denominator" the total number of visits made each day to the study emergency rooms, rather than the census data on the population of each zip code. The assumption here was that the patients who went to the study emergency rooms for nonasthma reasons were representative of the population from which the asthma cases appearing at those emergency rooms arose.

Our results add to the body of evidence that supports an association of air pollution with exacerbation of asthma. Comparison of our quantitative findings with those of similar studies (studies that assessed the relation of ambient air pollution to asthma emergency room visits in patient populations that included children (5–14)) is not entirely straightforward because of differences in analytical approaches, control variables, populations studied, and exposure variable averaging times and cutpoints. For instance, Cody et al. (11) reported results using multiple regression analysis leading to a risk estimate expressed as number of visits per ppb. For studies that reported statistically significant positive results in terms of rate ratios, we have recomputed GEE rate ratios using the same averaging time and cutpoint or increment, to facilitate...
comparison (although we did not change our control variables). To compare our results with those of Romieu et al. (9), who reported a rate ratio of 1.43 per 50-ppb change in maximum 1-hour ozone lagged 1 day, we obtained a rate ratio of 1.10 per 50-ppb change in maximum 1-hour ozone lagged 1 day. White et al. (8) reported a rate ratio of 1.37 in a comparison of visits following days on which the maximum 1-hour ozone level was at least 110 ppb with visits following days on which the ozone level was less than 110 ppb. When we categorized our data in this way, we obtained a rate ratio of 1.17 (p = 0.057). Steib et al. (13) used a cut-point of 75 ppb and observed an increase in asthma visits of 33 percent 2 days after days on which the maximum 1-hour ozone level was greater than this value; when we applied this cutpoint and lag period, we obtained a rate ratio of 1.07 (p = 0.035). For PM_{10}, Schwartz et al. (7) presented an overall rate ratio of 1.04 for a 4-day average increment of 10 μg/m³, and for ages 0–5 and 5–20 years the rate ratios were 1.03 and 1.02. For this increment in PM_{10}, we found a rate ratio of 1.02 (p = 0.44). Lipsett et al. (14) reported rate ratios ranging from 1.11 to 1.43 per 60 μg/m³ PM_{10}, lagged 2 days for three temperature groups. For this increment and lag period, our rate ratio was 1.02 (p = 0.73).

While our results appear to be generally consistent with those of these prior studies, there are a number of qualitative differences among the studies that should be kept in mind. For instance, whereas we studied urban air pollution in the summer months, Lipsett et al. (14) studied particulate pollution primarily from the use of wood-burning stoves in winter. More generally, given the broad distribution of geographic location and degree of urbanization of the study sites, the pollutant mix is likely to differ across the studies. Several of the above studies that included all ages did not report results for children separately. Furthermore, our study included a broader range of race and socioeconomic status than did others.

In most of the previous ozone studies, PM_{10} data were generally lacking, so the issue of collinearity could not be assessed. However, White et al. (8) controlled for PM_{10} in the analyses of ozone, and the ozone effect remained statistically significant. In the study by Schwartz et al. (7), no association was observed with ozone, so lack of control of the PM_{10} analyses for ozone was not an issue. The Lipsett et al. (14) study findings for PM_{10} are also unlikely to have been confounded by ozone, since the study period was winter, when ozone levels are low. Thus, several previous studies provide evidence of an effect of either ozone or PM_{10} independent of the other factor. In addition, chamber studies provide positive evidence for ozone alone (22, 23), and panel studies provide positive evidence for each type of pollutant (24–26).

Therefore, while the most cautious interpretation of the positive findings in our study is to consider ozone and PM_{10} indicators of general air pollution due to their collinearity, evidence to date suggests that both ozone and PM_{10} are associated independently with asthma exacerbation.

The current study had several strengths. It contained perhaps the largest number of pediatric asthma visits of the studies performed to date, and it included a broad range of exposure levels, information on a variety of pollutants, including spatial resolution of ozone information, and a balanced distribution of socioeconomic status in the study population. In light of these advantages, this study makes an important contribution to the body of knowledge regarding associations of air pollution with pediatric asthma exacerbation. The associations observed in this study were robust to analytical method and model specifications, are consistent with previous reports, and suggest continuing health risks at pollution levels that commonly occur in many US cities.

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