Repeated hospital encounters for asthma in children and exposure to traffic-related air pollution near the home

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Abstract

Background: Aggregate hospital encounters for asthma (admissions or emergency department visits) have been associated with daily regional air pollution. There are fewer data on relationships between repeated hospital encounters and traffic-related air pollution near the home.

Objective: To estimate the association of local traffic–generated air pollution with repeated hospital encounters for asthma in children.

Methods: Hospital records for 2,768 children aged 0 to 18 years (697 of whom had ≥2 encounters) were obtained for a catchment area of 2 hospitals in northern Orange County, California. Residential addresses were geocoded. A line source dispersion model was used to estimate individual seasonal exposures to local traffic–generated pollutants (nitrogen oxides and carbon monoxide) longitudinally beginning with the first hospital encounter. Recurrent proportional hazards analysis was used to estimate risk of exposure to air pollution adjusting for sex, age, health insurance, census-derived poverty, race/ethnicity, residence distance to hospital, and season. The adjustment variables and census-derived median household income were tested for effect modification.

Results: Adjusted hazard ratios for interquartile range increases in nitrogen oxides (4.00 ppb) and carbon monoxide (0.056 ppm) were 1.10 (95% confidence interval, 1.03–1.16) and 1.07 (1.01–1.14), respectively. Associations were strongest for girls and infants but were not significantly different from other groups. Stronger associations in children from higher-income block groups ($P < .09$ for trend) may have been due to more accurate data.

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Conclusions: Associations for repeated hospital encounters suggest that locally generated air pollution near the home affects asthma severity in children. Risk may begin during infancy and continue in later childhood, when asthma diagnoses are clearer.

INTRODUCTION

Many studies[1] show that children with asthma are susceptible to acute adverse changes in asthma outcomes from short-term increased exposure to ambient air pollutants measured at central regional sites at some distance from their residence. A few studies[2–5] have shown associations of pediatric asthma outcomes with personal exposure measurements of air pollutants. There are fewer data on whether certain air pollution sources cause these asthma associations largely because analyses have focused on temporal rather than spatial differences in exposure. Spatial heterogeneity of potentially toxic pollutant components are not well represented by data from ambient air monitoring sites, which have provided the bulk of exposure data in previous studies.[6,7]

A major contributor to air pollution exposure in urban areas is from mobile transportation sources. In southern California, for example, on-road emission sources alone contribute approximately 45% of volatile organic compounds, 63% of nitrogen oxide (NO_x), and 76% of carbon monoxide (CO) in the air.[8] There is a growing view that to accurately measure the magnitude of pediatric respiratory associations, air pollutant exposures are best evaluated closer to where children reside.[9] High home or school traffic density has been associated with prevalence of diagnosed asthma in epidemiologic studies.[10,11] Cohort studies[12–18] have shown associations between asthma incidence or early wheeze or cough without a cold and traffic-related air pollution near the homes of preschool children using geographic information system (GIS)–based exposure models.

Numerous experimental studies have provided evidence that exposure to chemicals capable of inducing airway oxidative stress, such as polycyclic aromatic hydrocarbons from diesel and auto exhaust, may play a role in the onset of allergic sensitization that could lead to asthma and in the acute exacerbation of respiratory allergic diseases, including asthma.[19] However, the impact of exposure to traffic on repeated episodes of asthma requiring hospital care is unclear.

Time-series studies have generally evaluated the relationship between central site air pollution measurements and aggregate (nonindividual) daily data for asthma emergency department (ED) visits and hospital admissions.[1] We conducted the first longitudinal study[20] of the relationship between repeated hospital encounters for individual children admitted with an asthma diagnosis and traffic-related air pollution in the outdoor home environment of these children. We found increased risk of repeated ED visits and hospitalizations for children 18 years and younger with a primary or secondary diagnosis of asthma in those living within 300 m of arterial roads or freeways. At highest risk were children in the top quintile of traffic density and those who had 750 m or more of arterial road and freeway length within 300 m of their residence. The present study advances this analysis of home traffic indices by using an improved GIS-based exposure evaluation method and air dispersion models. We aimed to estimate the risk of repeated hospital encounters in a cohort

of children with a primary diagnosis of asthma in relation to individual exposures to local traffic–generated air pollution. Furthermore, given previous evidence,[21] we tested whether children of lower socioeconomic status are at increased risk from air pollution exposures.

METHODS

Population and Outcomes

We passively followed up patients aged 0 to 18 years admitted to the hospital or seen in the ED with a primary diagnosis of asthma. Hospital data were extracted from billing records at 2 hospitals primarily serving the urban core of Northern Orange County. The study region consists of census block areas located within 13 km of the Children's Hospital of Orange County (CHOC) or the University of California Irvine Medical Center (UCIMC) (within 2.5 km of each other). This region was determined by mapping all records and finding a high density of patients visiting the CHOC and the UCIMC from this catchment area. This provided a reasonable evaluation of repeated hospital utilization for individual patients.

We identified 2,768 patients seen at the CHOC or the UCIMC between January 1, 2000, and December 31, 2003. Hospital data included health insurance, sex, age, race/ethnicity, and home address. For the 2,768 identified patients there were 4,020 unique hospital encounters (ED visits or hospitalizations by a particular patient 8 days apart). We geocoded the home addresses of all the patients and linked this to US Census 2000 block group socioeconomic data and traffic data using the GIS. The institutional review boards of the UCIMC and the CHOC approved the study protocol and establishment of the hospital records surveillance system for respiratory illnesses.

Exposure Evaluation

Residential addresses at the first hospital encounter were successfully geocoded for 93% of the patients (Tele Atlas North America Inc, Boston, Massachusetts). California Department of Transportation traffic data for major roads and highways were linked to the home locations.

We applied CALINE4 dispersion models to estimate nitrogen dioxide (NO_2), NO_x (nitric oxide + NO_2), and CO concentrations at each residence from local traffic emissions of gasoline vehicles and diesel trucks within a 5-km radius of each residence. The 5-km radius was used previously, and reasonable agreement was observed between CALINE4-modeled and measured 2-week average NO_2 concentrations at 260 residences in southern California ($R^2 = 0.3$–0.9).[22] The CALINE4 model is a gaussian line source dispersion model designed to estimate local pollutant concentrations from motor vehicle emissions based on traffic volumes, roadway geometry, vehicle emission rates, and meteorologic conditions (wind speed and direction, atmospheric stability, and mixing heights).[23] Wind patterns affect the general direction and dispersion of pollutants, leading to different exposures for individuals on the upwind vs downwind side of traffic sources.[6] Average diurnal and day-of-week freeway and non-freeway traffic variations were included. Emission factors were obtained from the California Air Resources Board's EMFAC2007 (v2.3) vehicle emissions model. Meteorologic data were obtained from the National Weather Service.

Exposures were updated for each participant every 6-month season from the time of entry into the study at first admission or ED visit (event) to the end of follow-up. Seasons were divided into 2 periods of southern California weather for CALINE4 estimates (warm season: May-October; cool season: November-April). Therefore, exposures during follow-up were estimated seasonally across the 4-year study.

Statistical Analysis

The relationship between hospital encounters and traffic-related air pollution (dispersion-modeled CO, NO_2, and NO_x) was tested using recurrent event proportional hazards models in SAS version 9.2 (SAS Institute Inc, Cary, North Carolina). We estimated the baseline hazard for admission separately for each recurrence time and assumed a common log–hazard ratio for the exposure association across all recurrence times.[24] We accounted for within-patient correlation of recurrent events by using robust variance estimates.[25] Only 3 patients had 11 to 12 readmissions, so we considered only 10 or fewer readmissions per patient in the analysis. Patients were considered to be at risk for recurrence from the time of first hospital encounter until the end of the observation period (December 31, 2003) or their 19th birthday. Time at risk started at the first or a subsequent event and ended with each season (when time at risk begins with the next seasonal exposure) or at the next event (when time at risk begins again with the current seasonal exposure).

We adjusted for a priori–identified potential confounders available from hospital records: age group (0, 1–5, 6–18 years), sex, race/ethnicity, insurance status, and residence distance to hospital. We also controlled for neighborhood socioeconomic status using US Census 2000 percent of households below the poverty level, for which there was no clear evidence of effect modification. We tested covariates for effect modification in separate models including product terms (interactions) of the air pollutant with the potential effect modifier, including age group, sex, race/ethnicity, insurance status, season, residence distance to hospital, and another indicator of neighborhood socioeconomic status (median household income). Significance tests for product terms were evaluated using the Wald χ^2 test for each contrast. We assume that product term $P < .10$ indicates significant interaction, ie, that the association with air pollution differs in one group compared with a reference group (eg, boys vs girls). Results stratified by group were obtained from product term models. Hazard ratios and 95% confidence intervals were calculated for interquartile range increases in air pollutants to standardize and compare associations regardless of pollutant concentration ranges or units of measurement.

RESULTS

There were 2,071 children (74.8%) with 1 hospital encounter during follow-up and 697 (25.2%) with 2 or more (Table 1). There was an expected predominance of boys, and 1,666 children (60.2%) were 0 to 5 years old at their first hospital encounter and had 66.7% of readmissions. Seasonal air pollutant exposures are given in Table 2. Pollutants were strongly correlated ($R > 0.9$). The modeled concentrations of fresh traffic emissions equaled approximately 20% of ambient NO_x concentrations at the regional station (38 ppb). Other

pollutants (CO and NO_2) shared a similar pattern. Nevertheless, fresh traffic-generated air pollution contributes greatly to the spatial heterogeneity of ambient pollution.[6]

Table 3 indicates the significant increased risks of repeated hospital encounters of 7% to 10% per interquartile range increase in traffic-related NO_x and CO exposures. Associations for NO_2 are approximately half that for NO_x and do not reach significance at $P < .05$. There is little difference in coefficients between adjusted and unadjusted models and between NO_x and CO. The remaining models include NO_x but not NO_2.

Table 4 gives the models stratified by sex and age group. Although the product terms (interactions) are not significant, the point estimates for CO and NO_x are stronger in girls than in boys and in infants than in older children. Hazard ratios for children aged 6 to 18 years are more positive than for those aged 1 to 5 years, but the lower 95% confidence limits dip below 1.0 in both groups.

Table 5 provides the models stratified by census block group poverty and median household income above vs below the median population distribution. Although hazard ratios were larger for those in block groups with more families below the poverty level, product terms were nonsignificant. Models stratified by median household income showed stronger and significant associations for both pollutants in those in the upper half of income distribution. We did not find significant differences by health insurance status, although coefficients were larger for those with private insurance (Table 5). Results by race/ethnicity showed a lower risk estimate for nonwhite patients attributable to black, Asian, and other patients (data not shown). Therefore, we combined these non-Hispanic nonwhite groups because of low sample sizes in each (Table 1) and compared regression estimates for them and for Hispanic patients with those of white patients (Table 5). There were no significant differences in associations between white and Hispanic patients for NO_x or CO. However, there were significantly smaller associations for non-Hispanic nonwhite patients than for white patients. There was no significant interaction for residence distance to hospital or for season (data not shown).

DISCUSSION

Overview of Findings and Implications

We found that residential exposure to traffic-related air pollution is associated with increased risk of hospital encounters for asthma in children. There was some evidence that infants and girls were at highest risk. These results are consistent with those of a cross-sectional study[26] and 2 case-control studies[27,28] showing increased risk of asthma hospitalizations or other medical care visits with increasing home traffic density indices. These results are also consistent with those of a longitudinal analysis of recurrent respiratory hospital encounters using traffic indices,[20] but associations in that study were not significant for patients with a primary diagnosis of asthma. The present results support the use of dispersion modeling for evaluating traffic-related exposures, but other methods involving direct home or neighborhood air pollutant measurements have been proposed to further limit exposure error.[6]

We did not include background pollutant concentrations because variability is likely low within the 13-km study radius. Dispersion-modeled gases are considered surrogates of other more toxic gases and particles emitted from nearby diesel trucks and automobiles, including ultrafine particles that have been found at notably higher concentrations near roadways along with black carbon, particle number, and CO.[29] The stronger associations for NO_x compared with NO_2 support this because NO_2 is strongly affected by photochemical reactions that can occur across time away from roadways, whereas NO_x is expected to capture traffic emissions more generally.[30] Key pollutants likely represented by NO_x and CO are those carried by ultrafine particles. Ultrafine particles carry more redox-active components than larger particles, which are more spatially homogenous.[7,31,32] Based in large part on experimental evidence, it has been hypothesized that particles from vehicular exhaust, especially in the ultrafine range, can trigger oxidative stress. When antioxidant responses are then overwhelmed, airway inflammation may follow, leading to increasing asthma symptoms in susceptible children.[19,33]

Potentially Susceptible Subgroups

There was no significant difference in association by sex or age group, and widened confidence intervals in stratified results suggest that subsample sizes may have limited the ability to compare these groups. Nevertheless, the largest associations were for infants, followed by children aged 6 to 18 years, in whom the diagnosis of asthma is clearest. There was limited evidence of stronger associations in girls, consistent with other studies of traffic-related air pollution and respiratory outcomes.[10,20,27,34] The underlying reasons for sex differences are unknown.

Significant associations for NO_x and CO in infants are intriguing. Approximately half of the repeated encounters in this group occurred between ages 1 and 3 years. These findings suggest that early-life exposures to traffic pollutants may affect asthma severity and development. This view is supported by studies[12–18] of preschool children that have found increased risk of incident asthma or wheeze or cough without a cold from long-term exposures to local traffic–related air pollutants using GIS-based methods. A recent study[35] showed acute increases in wheeze occurrence with elevations in daily regional NO_2 and NO_x levels in infants and children followed up during their first 3 years of life. These findings are consistent with emerging views that gene-environment interactions during early life are important in the prognosis of early-onset wheeze and in the development of lung function deficits and asthma in later life.[36,37]

In contrast to previous findings,[20] we did not find stronger associations in children without insurance or with government-sponsored insurance than in children with private insurance. Instead, there were significantly stronger associations in patients living in census block groups in the upper half of the distribution of median household income. However, there was no significant difference in associations between white and Hispanic patients (the predominant minority group). Both groups showed significant or nearly significant associations for NO_x and CO. The remaining minorities (blacks, Asians, and others) showed significantly smaller null associations compared with white patients. The distribution of median household income by race/ethnicity did not explain these findings because only

Hispanic patients showed significantly more families below the median income distribution (61%) compared with white patients (40%) and the remaining minorities (44%) ($P < .001$ by χ^2 test).

Evaluating community-level contextual factors may be important in understanding the heterogeneity in asthma expression and risk.[38] Environmental disparities, such as exposure to traffic and indoor allergens, have been proposed to explain the increased asthma burden in minority and lower socioeconomic groups.[39] Although the present findings do not support this hypothesis, the number of potentially important environmental factors that differ by communities is large[39] and mostly unmeasured in the present study. Furthermore, we speculate that these findings of smaller associations in children living in lower-income census block groups may have been attributable to 2 factors: (1) less consistent exposure as evaluated at study entry due to less stable residence and (2) less consistent outcome data due to more variable use of hospitals, including those not evaluated in this study. To test this possibility, we successfully contacted parents and administered a short survey for 250 randomly selected patients aged 0 to 8 years seen at the participating EDs or admitted to the hospitals with lower respiratory tract illnesses. Of 103 respondents with nonmissing data, those with survey-reported household annual incomes less than $30,000 (N = 58) were significantly more likely to have lived in the same residence for 12 or fewer months (29%) than were the 45 families making $30,000 or more (9%) ($P < .02$ by χ^2 test). In addition, those with annual household incomes less than $30,000 were more likely to have gone to a hospital not captured in the surveillance data (26%) than were families making at least $30,000 (13%) ($P < .12$ by χ^2 test).

There are several limitations to the present study design. First, some children used other hospitals, and, therefore, the outcome ascertainment is incomplete and the censoring assumption underlying the analysis is subject to some error. We also did not directly contact parents. Therefore, we could not ascertain any change in residence after the first event, leading to potential exposure error. Additional exposure error comes from unmeasured exposures occurring when children are away from their residence. We also did not have data on other known or suspected risk factors that may have confounded associations, including family history of asthma and environmental exposures (eg, second-hand smoke, aeroallergens, endotoxin, family size).[38,39] This may have led to misclassification of risk. For example, differences in the distribution of indoor and outdoor allergen triggers may have biased associations. Because the study used a retrospective cohort design, it was not possible to obtain allergen measurements during times at risk. Such measures could be used in future prospective cohort studies.

Finally, we could not confirm asthma diagnoses independently, which is especially important in younger patients, in whom lower respiratory tract illnesses can often induce asthma-like symptoms that resolve at later ages. Asthma diagnosis can be made using objective methods, such as spirometry, at school ages. Nevertheless, given other evidence,[12,40] it is conceivable that air pollutants also enhanced the propensity toward lower respiratory tract illness–related wheeze in the present population.

Conclusions

Traffic-related NO$_x$ and CO were associated with repeated hospital encounters for asthma in children, suggesting that traffic-generated air pollution near the home affects asthma symptom severity. These findings suggest that this potential risk may begin during infancy. Early lower respiratory tract illness with recurrent wheeze symptoms can increase asthma risk in later childhood, when the diagnosis of asthma is clearer.[41,42] Evidence from the present study supports a possible role of pollutants from traffic emissions in this progression.

Cohort studies of asthma risk in children to date have focused on general populations or on children with family histories of atopy. Prospective environmental data are sparse for high-risk populations who present to the hospital with asthma exacerbations. Additional work with improved assessments of air pollutant exposures and asthma outcomes in such high-risk populations is likely to be fruitful given the present results.

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Table 1.

Demographic Characteristics of the 2,768 Study Participants

Characteristic	Readmission for asthma, No. (%)	
	No (n = 2,071)	Yes (n = 697)
Sex		
Female	892 (43.1)	277 (39.7)
Male	1,179 (56.9)	420 (60.3)
Age group at study entry, y		
0	311 (15.0)	197 (28.3)
1–5	890 (43.0)	268 (38.4)
6–18	870 (42.0)	232 (33.3)
Race		
White non-Hispanic	910 (43.9)	327 (46.9)
White Hispanic	904 (43.7)	317 (45.5)
Black	56 (2.7)	15 (2.2)
Asian	52 (2.5)	14 (2.0)
Other	94 (4.5)	17 (2.4)
Unknown	55 (2.7)	7 (1.0)
Insurance status at study entry		
Private	799 (38.6)	295 (42.3)
Government sponsored or self-pay	1,042 (50.3)	371 (53.2)
Unknown	230 (11.1)	31 (4.5)
Census block median household income (quartiles), $		
36,672	516 (24.9)	176 (25.2)
>36,672–45,000	517 (25.0)	176 (25.2)
>45,000–59,375	507 (24.5)	190 (27.3)
>59,375	531 (25.6)	155 (22.2)
Census block percentage below poverty (quartiles)		
6.3	527 (25.4)	164 (23.5)
>6.3–14.0	515 (24.9)	178 (25.5)
>14.0–23.3	509 (24.6)	173 (24.8)
>23.3	520 (25.1)	182 (26.1)
Residence distance to treating hospital, median, km		
6.36	1,022 (49.3)	362 (51.9)
>6.36	1,049 (50.7)	335 (48.1)

Table 2.

Distribution of Traffic-Related Air Pollution Exposures by Season[a]

Exposure and season	Mean (SD)	Minimum	25th percentile	Median	75th percentile	Maximum	Interquartile range
NO2, ppb							
Cool	5.24 (2.39)	0.66	3.72	4.86	6.42	17.9	2.70
Warm	5.66 (2.61)	0.71	4.00	5.15	6.72	26.0	2.72
NOx, ppb							
Cool	8.10 (3.75)	1.00	5.70	7.52	9.98	27.0	4.29
Warm	6.35 (2.99)	0.76	4.45	5.75	7.59	29.7	3.14
CO, ppm							
Cool	0.114 (0.052)	0.014	0.081	0.106	0.140	0.378	0.060
Warm	0.103 (0.048)	0.013	0.072	0.093	0.123	0.482	0.051

Abbreviations: CO, carbon monoxide; NO2, nitrogen dioxide; NOx, nitrogen oxide.

[a]The cool season is November through April, and the warm season is May through October. Exposures are estimated from all person-times of observation during follow-up.

Table 3.

Traffic-Related Air Pollution and Repeated Hospital Encounters for Asthma in 2,768 Children Aged 0 to 18 Years

Exposure	Unadjusted HR (95% CI)[a]	P value	Adjusted HR (95% CI)[b]	P value
NO_2	1.044 (0.992–1.098)	.10	1.042 (0.987–1.101)	.14
NO_x	1.094 (1.035–1.156)	.002	1.097 (1.034–1.164)	.002
CO	1.072 (1.016–1.131)	.01	1.073 (1.013–1.137)	.02

Abbreviations: CI, confidence interval; CO, carbon monoxide; HR, hazard ratio; NO_2, nitrogen dioxide; NO_x, nitrogen oxide.

[a] Values are for an interquartile range increase in the air pollutant (NO_2, 2.68 ppb; NO_x, 4.00 ppb; and CO, 0.056 ppm).

[b] Adjusted for sex, age group, race, health insurance status, residence distance to hospital, and poverty.

Table 4.

Traffic-Related Air Pollution and Repeated Hospital Encounters for Children With Asthma by Sex and Age

Model	Patients, No.	Exposure	HR (95% CI)[a]	P value	Product term P value
Sex					
Male	1,599	NO_x	1.071 (0.991–1.158)	.08	.30
		CO	1.054 (0.978–1.137)	.17	.45
Female	1,169	NO_x	1.136 (1.043–1.238)	.003	Reference
		CO	1.100 (1.011–1.197)	.02	Reference
Age group, y					
0	508	NO_x	1.197 (1.075–1.333)	.02	.22
		CO	1.158 (1.041–1.289)	.007	.32
1–5	1,158	NO_x	1.042 (0.952–1.140)	.18	.52
		CO	1.021 (0.933–1.117)	.65	.44
6–18	1,102	NO_x	1.090 (0.979–1.212)	.12	Reference
		CO	1.076 (0.972–1.191)	.16	Reference

Abbreviations: CI, confidence interval; CO, carbon monoxide; HR, hazard ratio; NO_x, nitrogen oxide.

[a]Values are for an interquartile range increase in the air pollutant (NO_x, 4.00 ppb; CO, 0.056 ppm) adjusted for sex, age group, race, health insurance status, residence distance to hospital, and poverty. Stratified results are from the product term models.

Table 5.

Traffic-Related Air Pollution and Repeated Hospital Encounters for Children With Asthma by Socioeconomic Status and Race/Ethnicity

Model	Patients, No.	Exposure	HR (95% CI)[a]	P value	Product term P value[b]
Poverty[c]					
Median or less	1,384	NO$_x$	1.078 (0.999–1.163)	.05	Reference
		CO	1.054 (0.979–1.134)	.16	Reference
Greater than the median	1,384	NO$_x$	1.116 (1.026–1.214)	.01	.39 (.70)
		CO	1.094 (1.006–1.190)	.04	.49 (.71)
Median household income[d]					
Greater than the median	1,383	NO$_x$	1.145 (1.054–1.244)	.001	Reference
		CO	1.120 (1.034–1.213)	.005	Reference
Median or less	1,385	NO$_x$	1.068 (0.983–1.160)	.12	.23 (.09)
		CO	1.041 (0.959–1.129)	.34	.20 (.07)
Insurance status					
Private	1,094	NO$_x$	1.136 (1.036–1.247)	.007	Reference
		CO	1.102 (1.006–1.206)	.04	Reference
Government sponsored or self-pay	1,413	NO$_x$	1.080 (1.005–1.160)	.04	.38
		CO	1.061 (0.989–1.138)	.10	.51
Unknown	261	NO$_x$	0.886 (0.569–1.379)	.59	.28
		CO	0.913 (0.591–1.412)	.68	.41
Race/ethnicity					
White	1,237	NO$_x$	1.145 (1.055–1.243)	.001	Reference
		CO	1.113 (1.027–1.205)	.009	Reference
Hispanic	1,221	NO$_x$	1.097 (1.008–1.193)	.03	.46
		CO	1.081 (0.996–1.173)	.06	.62
Non-Hispanic nonwhite	310	NO$_x$	0.829 (0.624–1.102)	.20	.03
		CO	0.804 (0.601–1.074)	.14	.03

Abbreviations: CI, confidence interval; CO, carbon monoxide; HR, hazard ratio; NO$_X$, nitrogen oxide.

[a] Values are for an interquartile range increase in the air pollutant (NO_X, 4.00 ppb; CO, 0.056 ppm) adjusted for sex, age group, race, health insurance status, residence distance to hospital, and poverty. Stratified results are from product term models.

[b] The P value for interaction of air pollution with socioeconomic variables and race/ethnicity. The P value for trend from continuous poverty and median household income is given in parentheses.

[c] The median of Census 2000 block group percentage below the federal poverty level was 14%.

[d] The median of Census 2000 block group of median household income was $45,000.