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Effect of Insurance Coverage on the Relationship between Asthma Hospitalizations and Exposure to Air Pollution

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SYNOPSIS

Objective. Based on the assumption that people without health insurance have limited access to the primary care services needed to prevent unnecessary hospitalizations for asthma, the authors hypothesized that insurance is a factor in the strength of the association between hospital admissions for asthma and exposure to air pollution. They tested this hypothesis with 1991–1994 data from central Los Angeles.

Methods. The authors analyzed the effect of insurance status on the association between asthma-related hospital admissions and exposure to atmospheric particulates (PM_{10}) and ozone (O_3) using hospital discharge and air quality data for 1991–1994 for central Los Angeles. They used regression techniques with weighted moving averages (simulating distributed lag structures) to measure the effects of exposure on overall hospital admissions, admissions of uninsured patients, admissions for which MediCal (California Medicaid) was the primary payer, and admissions for which the primary payer was another government or private health insurance program.

Results. No associations were found between asthma admissions and O_3 exposure. An estimated increase from 1991 to 1994 of 50 micrograms per cubic meter in PM_{10} concentrations averaged over eight days was associated with an increase of 21.0% in the number of asthma admissions. An even stronger increase—27.4%—was noted among MediCal asthma admissions.

Conclusions. The authors conclude that low family income, as indicated by MediCal coverage, is a better predictor of asthma exacerbations associated with air pollution than lack of insurance and, by implication, a better predictor of insufficient access to primary care.

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Days with high levels of atmospheric pollutants are associated with increases in asthma-related hospital admissions.[1-7] This association suggests a possible link between severe exacerbations of asthma and exposure to air pollution—in particular, to PM_{10} (small, airborne particles less than 10 microns in diameter) and O_3 (ozone). However, levels of these two pollutants have decreased in the United States over the last 20 years[8-10] as asthma morbidity and mortality have risen significantly.[10-13] Therefore, if exposure to air pollution is at least partly responsible for the reported increase in the severity of asthma, an interaction with another factor must be compensating for decreased exposures. A recent report suggests that increased indoor exposure to allergens, such as dust mites and cockroach eggs, might be partially responsible for the rise in asthma morbidity and mortality.[14] Another recent paper suggests that the sanitary conditions of developed societies reduce exposure to infectious agents, weakening immunologic resistance to allergens that may trigger asthma episodes.[15] However, these factors may function more as subtle cumulative causes of bronchial hyperreactivity than as sudden incremental boosts to the effects of exposure to pollution; if so, another factor must be augmenting the effect of this exposure.

In contrast, obstacles to obtaining primary care services may incrementally boost the severity of pollution-related exacerbations by allowing them to fester unarrested until hospitalization becomes necessary. It is known that difficulty in accessing good primary care is a risk factor for asthma-related hospitalizations.[18] A well-trained primary care clinician—a physician, nurse practitioner, or physician assistant—can help avoid hospitalizations by managing asthma with appropriate education and medications. Insufficient access is endemic among the population most at risk for asthma—the inner-city poor[16-18]—and there is strong evidence to suggest that the uninsured have difficulties in accessing the primary care services needed to prevent impending asthma attacks.[19,20] Thus, reduced access to primary care services interacting with exposure to air pollution may be at least partially responsible for the 21% increase (from 386,000 to 466,000) in annual asthma hospitalizations from 1979 to 1994.[21]

For the present study, we investigated whether the association between exposures to both PM_{10} and O_3 and the census of daily asthma hospital admissions in one urban area varied by type of insurance coverage.

Although cutbacks in primary care services have been documented in other countries where health insurance is universal,[22] it is difficult to measure the effects of changes in access on asthma morbidity because such changes normally occur over long time periods. Given that more than 16% of Americans are currently uninsured,[23] the United States is an ideal laboratory for conducting a cross-sectional study of the effects of exposure to pollution by level of access to primary care—as measured by a proxy, insurance status.

Many recent studies of pollution exposure have been conducted in Canada or Great Britain. These studies have not consistently reported a significant association between exposure to pollution and asthma admissions, at least in part because in these countries universal insurance coverage ensures access to primary care. Perhaps, though, there exists a threshold level of exposure below which effects are negligible or there may be confounding factors in certain geographic areas, such as high levels of airborne sulfates, which have been shown to exacerbate asthma.[1-3,24-27]

A study of the interaction between insurance status and air pollution exposure requires a location with relatively high baseline levels to ensure an exposure effect, low levels of sulfates to avoid confounding, and a large population of uninsured people. The site of the present study, central Los Angeles, California, meets all of these criteria. It has high levels of environmental PM_{10} and O_3, lower levels of sulfates than most other US cities, and a high percentage of residents without health insurance coverage.

METHODS

Sample. The sample consisted of all emergency and urgent hospital admissions for asthma during 1991–1994 of people residing in central Los Angeles (zip code of residence 900XX).

We obtained hospital discharge records for 1991–1994 from the California Office of Statewide Health Planning and Development (OSHPD). To focus on unscheduled admissions, which were most likely to be precipitated by high pollution levels, we analyzed only emergency and urgent asthma-related admissions, excluding scheduled admissions and transfers from other facilities. The final sample consisted of 11,240 admissions for asthma in the 900XX zip code region during 1991–1994.

We selected the central Los Angeles (900XX) zip code region because we found—using OSHPD hospital discharge data, air quality data from the California Air

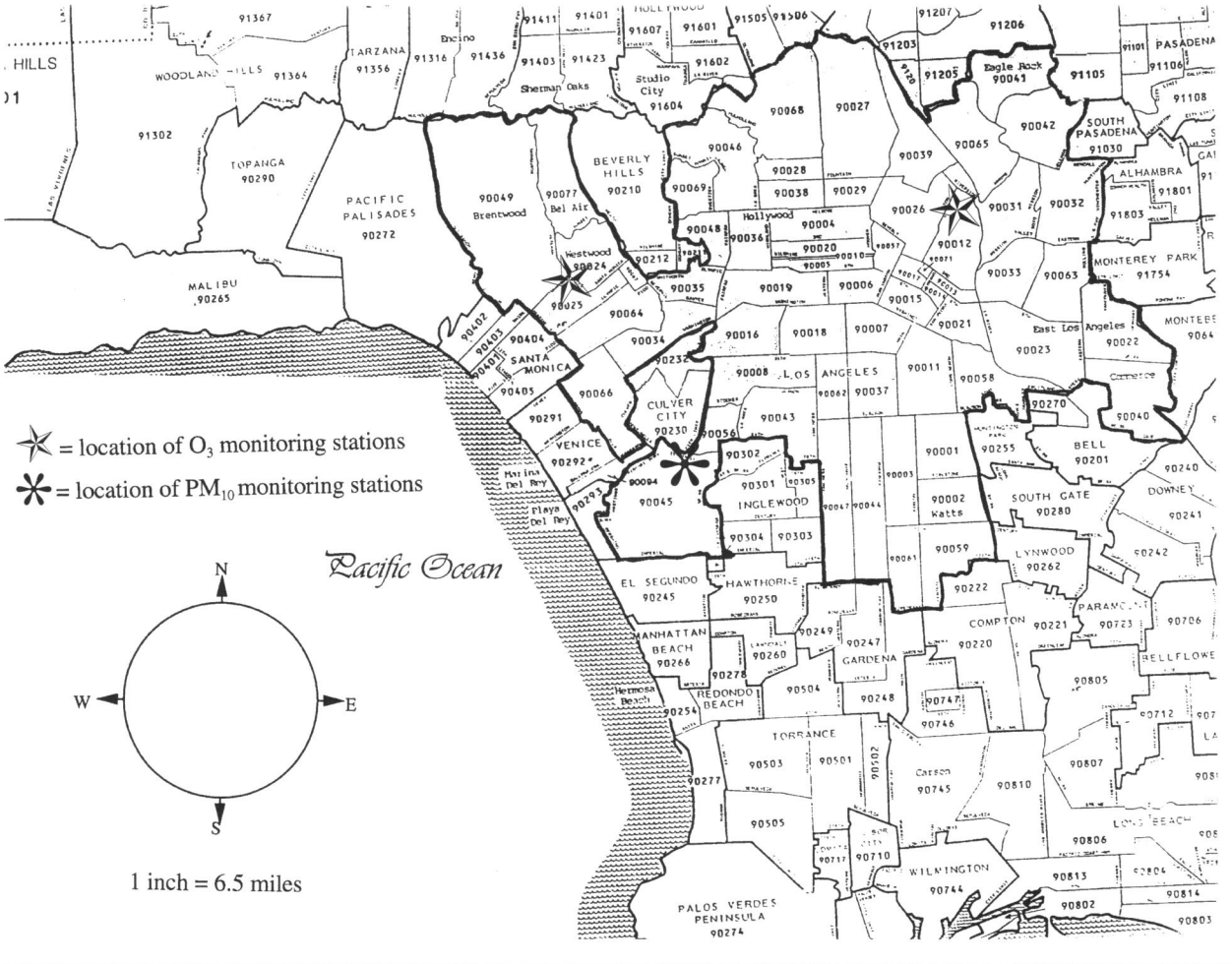
Resources Board, and population data from the 1990 US Census—that it has the largest population of any zip code region in California, the greatest variation in daily hospital admissions, and the greatest variation in O_3 levels. Central Los Angeles is surrounded by hills and mountains, which tend to confine pollutants to the area. With a population of 2.36 million people, the 900XX three-digit zip code region includes a large portion of Los Angeles county and a range of socioeconomic strata from affluent Brentwood and Bel Air in the north to impoverished Watts in the south (see Figure 1).

In Los Angeles, studying an area defined by a three-digit zip code is appropriate for several reasons. First, air quality varies widely from one location in the metropolitan area to the next because of wind patterns and variations in terrain. Second, air monitoring stations produce readings for an area larger than a five-digit zip code. The three-digit zip code covers an area that is large enough to represent most of the territory of a monitoring station but not so large as to encompass areas of widely varying air quality.

Definition of asthma. To capture all emergency and urgent asthma-related hospital admissions, we used the following International Classification of Diseases, Ninth Revision (ICD-9), codes, as recommended by the authors of a study on preventable hospitalizations and access to health care.[28]

(*a*) A primary diagnosis of asthma: ICD-9 493
or
(*b*) A primary diagnosis of pneumonia or acute bron-

Figure 1. Boundaries of 900XX zip code region and locations of PM_{10} and O_3 monitoring stations, central Los Angeles, California



Counter to national trends, we found a surprising 4.7% decrease in daily totals of emergency and urgent asthma admissions from 1991 to 1994.

chitis: ICD-9 466.0, 480-483, 485-487, 518.81, 518.82, or 786.09
and
a secondary diagnosis of asthma: ICD-9 493.

Control group. From OSHPD hospital discharge records, we selected a sample of nonrespiratory admissions during 1991–1994 to use as a control group. We defined the sample by including admissions for which any of the following ICD-9 codes was listed as the principal diagnosis, as in a study by Thurston et al.[4]:

Eye disorders	365, 366.0–366.3
Cerebrovascular diseases	430, 431, 433–436
Gastric and peptic ulcers	531.0–531.3, 532.0–532.3, 533.0–533.3, 534.0–534.3, 535
Acute appendicitis	540
Gallstones	574
Kidney and urethra diseases	590, 599

The control group consisted of 34,564 hospital admissions during 1991–1994 for residents of the 900XX zip code region that met the same limiting criteria as the sample of asthma admissions (urgent and emergency admissions, excluding scheduled admissions and transfers).

PM_{10} and O_3 levels. We obtained hourly PM_{10} readings for 1991–1994 from the South Coast Air Quality Management District's Los Angeles International Airport monitoring station (in zip code 90045). From the California Air Resources Board, we obtained hourly O_3 readings from the two O_3 monitoring stations in the region of interest, located in zip codes 90025 and 90012 (see Figure 1); we averaged the two readings to obtain an overall regional reading for each hour.

From these data, we calculated average daily (24-hour) concentrations for 1991–1994 for $PM_{10}$and O_3 using the method employed by Pope et al.[29]

We used MapInfo® for the Power Mac (Version 4.0.3) to convert monitoring locations in latitude and longitude to zip codes.

We obtained data on daily maximums for both temperature and relative humidity and on average daily wind speed for 1991–1994 from the National Climatic Data Center in Asheville, North Carolina. We included wind speed because of the possible effect of intermittent desert winds—known as Santa Ana conditions—unique to Southern California.

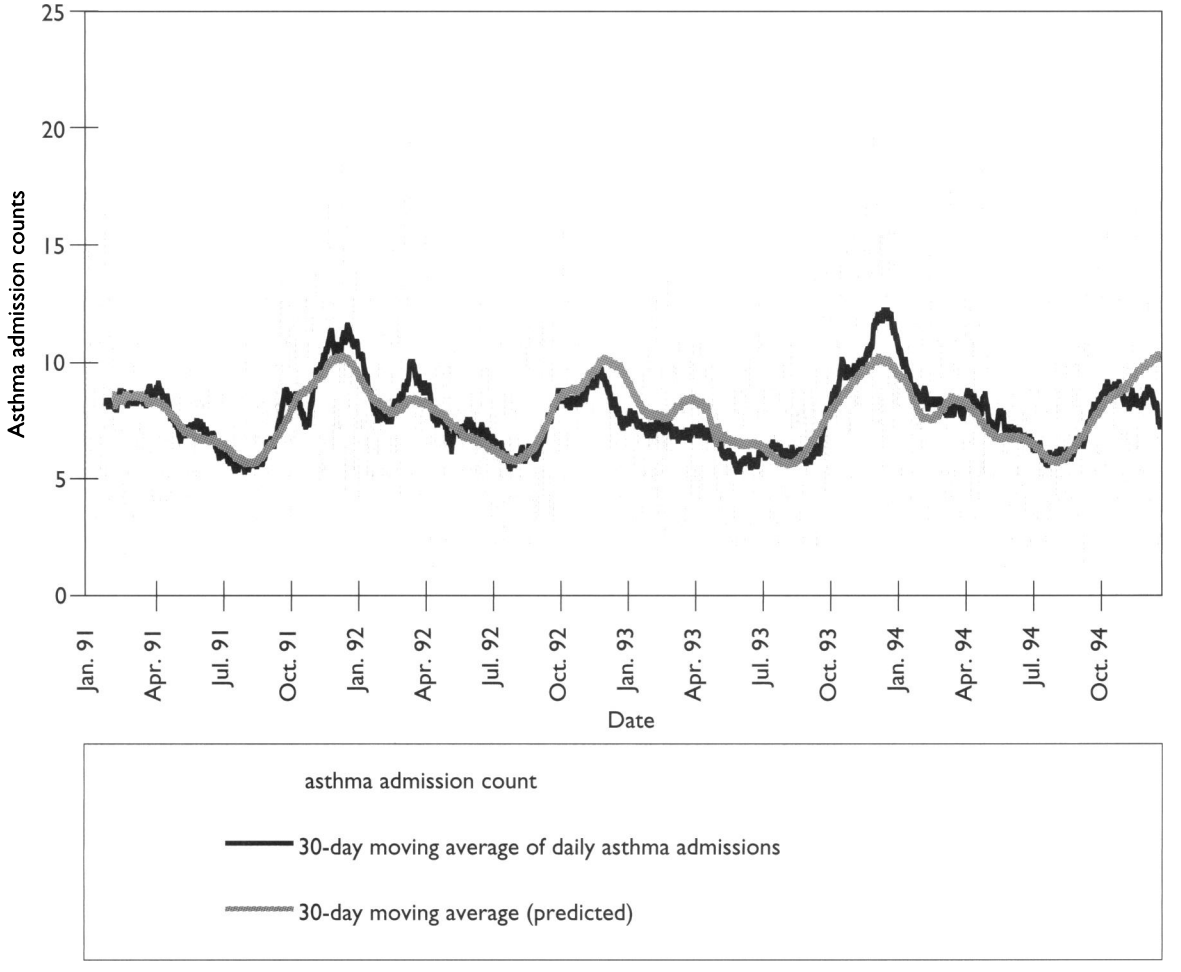
Data analysis. All analyses were conducted using SAS version 6.12.[30]

Insurance status. We first sorted hospital admissions into four groups by insurance status: (*a*) total asthma admissions; (*b*) asthma admissions with MediCal (California's Medicaid program) as the primary source of coverage; (*c*) asthma admissions with a health insurance program other than MediCal as the primary source of coverage; (*d*) asthma admissions of uninsured patients (those with no insurance listed).

Relationship between PM_{10} and O_3 exposure and asthma admissions: bivariate analyses. To determine whether an exposure-response relationship existed, we compared mean admission day pollution levels across quartiles of daily counts of asthma admissions.

Relationship between PM_{10} and O_3 exposure and asthma admissions: multivariate analyses. Because we found substantial multicollinearity ($r > 0.90$) between variables measuring periodic cycles and those measuring pollution and meteorologic conditions, we analyzed differences across insurance groups in daily admission counts in two steps: we first removed periodic cycles from the data and then regressed the remaining variation in daily counts (the regression residuals) on variables measuring pollution exposure, meteorologic conditions, and age and ethnic mix.

Figure 2. Daily counts of asthma-related emergency and urgent hospital admissions, 30-day moving average, and predicted 30-day moving average, 900XX zip code region, Los Angeles, California, selected months, 1991–1994



Removing periodic cycles in the data. To remove seasonal and daily cycles in asthma admissions, we applied a Fourier series in conjunction with Poisson regression to filter the data for these wave effects. We used Poisson regression—which produces more efficient estimators than ordinary least squares regression when the dependent variable consists of small counts[31]—because the sampled asthma admissions averaged fewer than eight per day. Poisson regression assumes that each value of the daily admission counts, y_i, is drawn from a Poisson distribution with expected value $\bar{y}$. This expected value is related to the regression variables according to the specification: $\log(\bar{y}) = \beta'x$ where $\mathbf{x}$ is the matrix of regressors.

Using a technique similar to that employed by Hexter and Goldsmith, we fit a Fourier series to cycles nested within an annual cycle that was evident in the data[32] (see Figure 2). The functional specification started with six harmonic (cosine-sine) pairs in addition to sequential day number and its square for trend. This specification allowed for a maximum of six nested cycles, the shortest of which goes through six complete cycles every year. We also included in the specification day of week indicator variables to control for day-by-day variations in numbers of admissions.

We deleted variables through backward elimination with the constraint that both members of a harmonic pair were removed together. In addition, only variables at the beginning or at the end of a sequence were deleted so that a full sequence of harmonic pairs was always maintained. A total of four harmonic pairs and variables for

We found a significant relationship between PM_{10} exposure and MediCal asthma admissions.

linear trend and day of the week were included as a filter for autocorrelation in the analytic specifications.

After eliminating period cycles in the data, we used ordinary least squares regression to determine to what extent the remaining variation in daily admission counts could be explained by exposure to PM_{10} and O_3. As a dependent variable, we used the regression residuals obtained from removing the periodic cycles in the data. Then, for each insurance group, we constructed four analytical models. The first two models were single-period models that measured exposure as the mean concentration of PM_{10} or O_3 on the day of hospital admission. The third and fourth models were multi-period models that used an unrestricted polynomial distributive lag structure to measure exposure for each pollutant over a number of days, producing a more accurate measure than admission day exposure. To simulate these lag structures, we used weighted moving average exposure variables derived from lagged daily mean concentrations for both PM_{10} and O_3. The second and fourth models included controls for meteorologic conditions (maximum daily temperature and relative humidity and mean daily wind speed averaged over the days for which pollution exposure was measured in each model.)

To estimate the weights used to calculate the moving averages, we fit polynomial lag structures to each pollution measure for its high season—the wet season (November 15 through March 1) for PM_{10} and the dry season (May 1 through August 15) for O_3. We determined the lag length and polynomial degree through a sequential testing procedure (partial F-test) beginning with a lag of 15 and a third degree polynomial.[31] This procedure produced eight-day lag structures—including the day of hospital admission—fit to a first degree polynomial for both PM_{10} and O_3. These structures were similar to those used in a previous study employing distributed lag structures to determine exposure-response effects for asthma.[29] We then standardized the regression coefficients to sum to one and applied these coefficients as weights in calculating moving averages.

Adjusting for ethnic and age mix. Ethnic mix may be a confounding factor given that studies have shown that African American or Latino heritage is a risk factor for asthma independent of income.[16,18,19] The age mix of patients is also important because the pattern of insurance coverage is different for children and adults (for example, MediCal covers a higher percentage of children than of adults).

Instead of controlling for ethnic and age mix in every analysis, we first determined the level of correlation with pollution exposure; if the correlation coefficient was sufficiently large ($r > 0.40$), we included ethnic or age mix as a control variable.

Detection of oversampling. Since the unit of analysis was an asthma-related hospital admission, if some asthmatics had multiple admissions during any one year, their experience would have been more heavily weighted in the analyses. The lack of personal identifiers in the data meant that we had to devise a method for approximating the number of multiple admissions to determine the level of bias. We matched admissions during the wet season in a particular year by age, sex, ethnicity, five-digit zip code of residence, and hospital identification number. We considered those that matched to be multiple admissions of the same patient. We also conducted a sensitivity analysis allowing for the possibility that patients changed age during the wet season in a particular year; admissions with an exact match, except for a difference in one year of age, were considered to represent the same patient.

RESULTS

Table 1 shows descriptive statistics for 1991–1994. Counter to national trends, we found a surprising 4.7% decrease in daily totals of emergency and urgent asthma admissions from 1991 to 1994, although patterns varied across insurance subgroups: while mean daily asthma admissions decreased 28% from 1991 to 1994 for people without health insurance and decreased 13.9% for people with primary coverage other than MediCal, mean daily

Table 1. Mean daily counts of emergency and urgent asthma-related hospital admissions; mean daily counts of control admissions; O_3 and PM_{10} levels; and meteorologic conditions, 900XX zip code region, central Los Angeles, California, 1991–1994

	By year								By season					
	1991		1992		1993		1994		Dry season 1991–1994		Wet season 1991–1994		Total 1991–1994	
	n = 2919 admissions		n = 2788 admissions		n = 2761 admissions		n = 2770 admissions		n = 2717 admissions		n = 3208 admissions		n = 11.240 admissions	
Variable	*Mean*	*SD*	*Mean*	*SD*	*Mean*	*SD*	*Mean*	*SD*	*Mean*	*SD*	*Mean*	*SD*	*Mean*	*SD*
Asthma-related hospital admissions per day														
All insurance categories	8.00	3.32	7.64	3.12	7.56	3.29	7.62	3.07	6.35	2.63	8.74	3.30	7.70	3.20
Uninsured	0.94	0.96	0.78	0.89	0.77	0.87	0.68	0.84	0.74	0.83	0.77	0.89	0.79	0.89
MediCal primary coverage	3.75	2.12	3.62	2.13	3.90	2.21	4.08	2.31	3.08	1.84	4.36	2.12	3.84	2.21
Primary coverage other than MediCal	3.31	1.96	3.24	1.98	2.89	1.94	2.85	1.74	2.53	1.66	3.62	2.10	3.07	1.92
Nonrespiratory (control) admissions per day	21.31	4.97	23.69	5.00	24.47	5.63	25.24	5.29	24.34	5.43	23.33	5.65	23.67	5.43
Mean daily PM_{10} reading (mg/m^3)	43.40	18.95	44.33	16.04	45.60	17.10	46.12	16.07	43.23	12.02	43.50 49.14[a]	19.98 21.08[a]	44.81	17.23
Mean daily O_3 reading (parts per billion)	22.61	11.69	19.52	12.23	17.09	9.84	20.39	9.86	27.87	8.65	7.52	4.34	19.88	11.13
Maximum daily temperature (°C)	20.84	3.67	22.13	3.70	22.00	3.45	21.78	4.15	23.22	2.87	19.36	3.52	21.69	3.78
Maximum daily humidity (%)	87.15	10.91	87.31	12.54	89.38	10.95	87.88	9.89	89.70	4.39	82.45	17.38	87.93	11.14
Mean daily wind speed (mph)	7.16	2.11	7.22	2.81	8.31	1.80	7.44	1.75	8.30	1.37	7.38	2.34	7.53	2.20
Percent of admissions in which patient was ≥ 18 years old	62.94		63.41		61.90		63.11		66.36		61.48		62.84	
Percent of admissions in which patient was African American	47.79		49.03		46.41		45.10		48.47		46.32		47.08	
Percent of admissions in which patient was Latina(o)	32.43		30.84		32.76		32.90		32.21		31.97		32.23	

NOTE: The dry season is defined as May 1 through August 15. The wet season is defined as November 15 through March 1. Data were unavailable for January 1–March 1, 1995, so the 1994 wet season data were for November 15–December 31, 1994, only.

[a]The second set of numbers in this cell represent the means over the period October 1 to February 1.

SD = standard deviation

PM_{10}= small particles of solid matter less than 10 microns in diameter

$\mu g/m^3$ = micrograms per cubic meter

O_3 = ozone

asthma admissions for people with MediCal primary coverage *increased* 8.8%.

Asthma admissions were higher across all insurance categories during the wet season than during the dry season (Table 1).

Control (nonrespiratory) hospitalizations increased by 18.4% from 1991 to 1994 (see Table 1).

PM_{10} levels were highest during the months from October through February, roughly corresponding to the wet season (see Table 1 and Figure 2), and O_3 levels were higher during the dry season than during the wet season.

For the four-year period, mean 24-hour PM_{10} concentrations ranged from 10 to 132 micrograms per cubic meter ($\mu g/m^3$), and O_3 concentrations ranged from 0 to 65 parts per billion (not shown). The PM_{10} monitor malfunctioned for the first two months of 1991 and for approximately 30 days during April 1993; therefore, data are missing for these periods. These omissions could have biased

Table 2. Air pollution levels by quartile of daily counts of asthma admissions, 900XX zip code region, Los Angeles, California, 1991–1994

Quartile	Mean admission day PM_{10} levels ($\mu g/m^3$)		Mean admission day O_3 levels (parts per billion)	
	Dry season	Wet season	Dry season	Wet season
Lowest (≤ 5 admissions per day)	41.61	39.18	27.37	6.82
Second (6 or 7 admissions per day)	42.64	43.61	27.56	7.44
Third (8–10 admissions per day)	43.76	43.58	28.51	7.54
Highest (> 10 admissions per day)	43.55	46.62	29.89	7.87

PM_{10} = small particles of solid matter less than 10 microns in diameter
μ/m^3 = micrograms per cubic meter
O_3 = ozone
Dry season = May 1–August 15
Wet season = November 15–March 1

the annual average PM_{10} levels for these two years; however, these annual averages followed the increasing trend that was evident in the other two years of data (Table 1).

Humidity levels were approximately 7% lower during the dry season than the wet season, while the average temperature was approximately 4°C lower during the wet season.

The age and ethnic composition variables did not show any significant trends across years and seasons except that the percentage of patients who were age 18 years and older was significantly lower during the wet season than during the dry season ($P < 0.05$).

Bivariate analysis of associations between PM_{10} and O_3 exposure and asthma admissions. Table 2 shows the means of daily PM_{10} and O_3 concentrations by quartile of daily counts of asthma admissions. The numbers show a positive linear association during both seasons between daily counts of asthma admissions and both PM_{10} and O_3 concentrations. The strongest association, according to this bivariate analysis, occurred during the wet season, during which PM_{10} averages varied from 39.18 $\mu g/m^3$ for the lowest quartile to 46.62 $\mu g/m^3$ for the highest quartile.

Multivariate analysis of associations between PM_{10} and O_3 exposure and asthma admissions. We did not find any statistically significant associations between asthma admissions and atmospheric levels of either PM_{10} or O_3 during the dry season. However, we did find a significant association between PM_{10} levels (but not O_3) and asthma admissions during the wet season.

Table 3 shows the regression results for the wet seasons from 1991 through 1994. Both the single-period and multi-period models produced significant associations between admission counts and PM_{10} exposure ($P < 0.05$). To illustrate the magnitude of this relationship, we chose a benchmark increase in PM_{10} levels, 50 $\mu g/m^3$, which doubled the average daily concentration of PM_{10}. According to the results from Model IV, such a rise in PM_{10} concentrations averaged over eight days would produce an increase of 21.0% in the daily admission count, from 7.70 to 9.32 per day. The other models produced similar exposure-response effects. The maximum to mean level PM_{10} relative risk ratio for admissions was 1.37 ± 0.18 ($P < 0.05$) (relative risk ratio = 1 + [(maximum daily mean PM_{10} reading x regression coefficient)/seasonal mean daily admission count]).

As expected, no statistically significant associations were found between exposure to these pollutants and nonrespiratory admissions.

Effects of exposure on hospital admissions by insurance status. Table 4 shows the regression results by insurance status corresponding to the overall analyses presented in Table 3. Only the results from Models III and IV are presented since they are more indicative than single-period models of the true exposure-response relationship. (When pollution readings are correlated over a number of days, single-period models will produce regression coefficients for admission-day exposure that will be an overestimate of that day's contribution but an underestimate of the lagged and cumulative effect of exposure over time.) We found a significant association ($P < 0.05$) between PM_{10} exposure and admissions for asthmatics with MediCal coverage.

According to the results from Model IV, a 50 $\mu g/m^3$ rise in PM_{10} concentrations averaged over eight days was associated with a 27.4% increase (4.36 to 5.56) in mean

Table 3. Results of ordinary least squares regression analysis of association between exposure to atmospheric pollution and daily counts of hospital admissions, 900XX zip code region, Los Angeles, California, 1991–1994, for wet season (November 15–March 1)

Emergency and urgent asthma-related admissions
n = *3208*

	Single-period models				*Weighted moving average models*			
	Model I		*Model II*		*Model III*		*Model IV*	
Variable	*ß coefficient*	*SE*	*ß coefficient*	*SE*	*ß coefficient*	*SE*	*ß coefficient*	*SE*
Mean admission day PM_{10} level ($\mu g/m^3$)	0.0202[a]	0.0085	0.0283[a]	0.0127	—		—	
Mean admission day O_3 level (parts per billion)	0.0444	0.0385	0.0037	0.0164	—		—	
8-day weighted average PM_{10} level ($\mu g/m^3$)	—		—		0.0350[b]	0.0131	0.0324[a]	0.0162
8-day weighted average O_3 level (parts per billion)	—		—		0.0359	0.0568	-0.0733	0.0687
Maximum daily temperature (°C)	—		-0.0265	0.0468	—		0.2584[a]	0.0963
Maximum daily humidity (percent)	—		-0.0601	0.0291	—		-0.0048	0.0181
Mean daily wind speed (miles per hour)	—		-0.0370	0.1005	—		0.3938	0.1758

Emergency and urgent admissions for selected nonrespiratory conditions
n = *8584*

	Single-period models				*Weighted moving average models*			
	Model I		*Model II*		*Model III*		*Model IV*	
Variable	*ß coefficient*	*SE*	*ß coefficient*	*SE*	*ß coefficient*	*SE*	*ß coefficient*	*SE*
Mean admission day PM_{10} level ($\mu g/m^3$)	-0.0028	0.0144	-0.0027	0.0163	—		—	
Mean admission day O_3 level (parts per billion)	-0.0217	0.0650	-0.0280	0.0709	—		—	
8-day weighted average PM_{10} level ((g/m^3)	—		—		-0.0129	0.0219	-0.0235	0.0275
8-day weighted average O_3 level (parts per billion)	—		—		-0.8699	0.9501	-0.1134	0.1163
Maximum daily temperature (°C)	—		0.0287	0.0970	—		0.1224	0.0630
Maximum daily humidity (percent)	—		0.0030	0.0185	—		0.0069	0.0306
Mean daily wind speed (miles per hour)	—		0.0310	0.1510	—		0.0288	0.2976

NOTES: Intercept values are not listed since they do not have an intuitive meaning in regressing error residuals on the variables included in the model. All models include controls for serial correlation and trend. The final model specifications include variables representing four harmonic (cosine-sine) pairs, linear trend, and day of the week. The ß coefficients represent the effect of a one unit increase of the independent variable on the natural logarithm of the mean of the dependent variable. Models II and IV include controls for weather conditions, while Models I and III do not.

[a]Significant at $P < 0.05$

[b]Significant at $P < 0.01$

PM_{10}= small particles of solid matter less than 10 microns in diameter

$\mu g/m^3$ = micrograms per cubic meter

O_3 = ozone

SE = standard error

daily MediCal admissions. The maximum to mean level PM_{10} relative risk ratio for admissions was 1.47 ± 0.21 ($P < 0.05$). The single-period models, not shown, also showed significant associations ($P < 0.01$) between PM_{10} and asthma-related emergency and urgent MediCal admissions.

There was no evidence of a significant correlation between either ethnic mix or age mix and any of the pollution variables included in the models. We also placed these variables directly into the models as independent regressors—including as effect modifiers—but found they did not substantially change the size of the mea-

Our results suggest that insurance status is a poor proxy for income level, which might better measure access to primary care.

sured pollution effects (not shown). Moreover, the partial F-tests were insignificant ($P > 0.10$), suggesting that these variables be excluded. We also attempted to split the sample into pediatric (age < 18) and adult cases; however, the daily admission counts for each group became too small to detect variations that were correlated with pollution readings. Testing for any remaining autocorrelation in the data, we found that all Durbin-Watson statistic values were sufficiently close to two to preclude this possibility.

Evidence of oversampling. We calculated that the percentage of sampled patients with single admissions was at least 92.8% (2719 single admissions during wet season [estimated] / 2929 total wet season asthma admissions [estimated]), and the percentage of asthma admissions

Table 4. Results of ordinary least squares regression analysis of association between exposure to atmospheric pollution and daily counts of hospital admissions, by insurance status, 900XX zip code region, Los Angeles, California, 1991–1994, for wet season (November 15–March 1)

	Uninsured n = 282				*MediCal* n = 1,599				*Other insurance* n = 1,327			
	Weighted moving average models				*Weighted moving average models*				*Weighted moving average models*			
	Model III		*Model IV*		*Model III*		*Model IV*		*Model III*		*Model IV*	
Variable	*ß coefficient*	*SE*	*ß coefficient*	*SE*	*ß coefficient*	*SE*	*ß coefficient*	*SE*	*ß coefficient*	*SE*	*ß coefficient*	*SE*
8-day weighted average PM_{10} level ($\mu g/m^3$)	0.0010	0.0038	-0.0007	0.0048	0.0239[b]	0.0087	0.0239[a]	0.0108	0.0109	0.0088	0.0104	0.0109
8-day weighted average O_3 level (parts per billion)	0.0026	0.0165	-0.0031	0.0202	0.0265	0.0375	-0.0246	0.0456	0.0055	0.0379	-0.0491	0.0461
Maximum daily temperature (°C)	—		0.0243	0.0283	—		0.1228[a]	0.0639	—		0.1141	0.0646
Maximum daily humidity (percent)	—		0.0020	0.0053	—		0.0066	0.0120	—		-0.0042	0.0121
Mean daily wind speed (miles per hour)	—		0.0125	0.0516	—		0.2057	0.1168	—		0.1856	0.1180

NOTES: Intercept values are not listed since they do not have an intuitive meaning in regressing error residuals on the variables included in the model. All models include controls for serial correlation and trend. The final model specifications include variables representing four harmonic (cosine-sine) pairs, linear trend, and day of the week. The ß coefficients represent the effect of a one unit increase of the independent variable on the natural logarithm of the mean of the dependent variable. Model IV includes controls for ethnicity and weather conditions, while Model III does not.

[a]Significant at $P < 0.05$

[b]Significant at $P < 0.01$

PM_{10}= small particles of solid matter less than 10 microns in diameter

$\mu g/m^3$ = micrograms per cubic meter

O_3 = ozone

SE = standard error

that were the sole admission for a particular patient was at least 84.8% (2719 single admissions during wet season [estimated] / 3208 total wet season asthma admissions). These percentages probably represent a lower bound on the true value since it was possible for admissions to match across all five categories and still represent different patients. It was also possible that patients were admitted to different hospitals for each admission and were therefore counted as different patients by this method; however, we assumed that the vast majority of hospitalized patients were always admitted to the same institution given referral patterns in inner-city areas that often include only a single hospital.[28] Therefore, we concluded that oversampling was not a major source of bias.

Distribution of asthma admissions among hospitals. If asthma admissions were concentrated in only one or a few institutions, then the study results would be heavily weighted by the experiences of people presenting at those hospitals. Although the experiences of three large public institutions dominated the sample, no single institution was responsible for more than 24% of the asthma-related hospital admissions. Of the 3208 hospital admissions for asthma during the wet seasons for 1991–1994, 91.5% were from the 10 hospitals with at least 100 admissions for asthma. The three hospitals with at least 500 asthma admissions—Children's Hospital of Los Angeles, USC Medical Center, and Martin Luther King Jr.–Drew Medical Center—accounted for more than 59.5% of the study sample. Furthermore, these percentages remained stable from year to year. When we redid the analyses without admissions from these three institutions, the results remained similar to those obtained from the full sample.

DISCUSSION

This is the first study of the effect of insurance status on adverse health outcomes related to pollution exposure. The main finding was a moderately strong association between PM_{10} levels and daily asthma admissions, similar in magnitude to that found in a study conducted in Great Britain.[27] A similar study in Detroit found no association, but the study focused on the elderly, who tend not to be as physically active as younger people and may therefore not be as sensitive to fluctuations in outdoor air quality.[33]

We also found a significant relationship ($P < 0.05$) between PM_{10} exposure and MediCal asthma admissions. Studies have shown that the uninsured have difficulties in accessing the primary care services needed to prevent an impending asthma attack;[19,20] however, we found no relationship between PM_{10} exposure and uninsured admissions. If such a relationship exists, a small sample size for uninsured admissions ($n = 282$ during the wet season) may have prevented us from detecting it.

More probably, our results indicate that insurance status is a poor proxy for income level, which might better measure access to primary care. In particular, the uninsured may represent a broader socioeconomic spectrum than MediCal patients, who are enrolled in a program for which, with a few exceptions, eligibility is restricted to the poor. Future research will need to investigate the relationship between pollution exposure, income level, and asthma exacerbations—perhaps using counts of emergency department visits over wider areas to more definitively measure any associations.

Other factors may explain our results. For example, the increase in mean daily asthma admissions with MediCal primary coverage from 1991 to 1994 corresponds to the overall expansion of MediCal coverage in Los Angeles county. According to the US Bureau of the Census, MediCal covered 16.0% of the under-65 population in 1994, compared with just 12.8% in 1991—a 25% increase in the number of beneficiaries (data from the US Bureau of the Census's *Current Population Survey (March)* for the years 1992–1995, obtained from the University of California, Los Angeles, Center for Health Policy Research). Since PM_{10} levels also rose during this period, the observed positive association between PM_{10} exposure and MediCal admissions might be due to the increase in MediCal enrollment. However, the Fourier series used to control for seasonal and daily cycles in the data also included a variable to control for any secular trend in admissions. Thus, these results should not have been affected by the expansion of MediCal.

Other studies have observed an association between O_3 and asthma morbidity, but they were all conducted in regions with different climates from Southern California's.[4,6] Since the present study only examines the severest responses, those culminating in hospitalization, other less severe responses requiring medical attention remain unmeasured. Studies of emergency department visits, inhaler use, or reports of symptoms have hypothesized a relationship between O_3 exposure and asthma morbidity; however, these studies have had mixed results.[5,34,35]

It is curious that daily counts of asthma-related admissions remained largely stable during this period while nonrespiratory (control) admissions increased by more than 18%. Four years may be too short a period to determine a trend; this difference might be due simply to

The uninsured may represent a broader socioeconomic spectrum than MediCal patients, who are enrolled in a program for which, with a few exceptions, eligibility is restricted to the poor.

random variation in admission counts during this time. Further, the large number of people who gained MediCal coverage may have had increased access to primary care, preventing many potential asthma-related hospital admissions. If the expansion in MediCal enrollment had not occurred, then the trend in asthma admissions might well have mirrored that for nonrespiratory admissions.

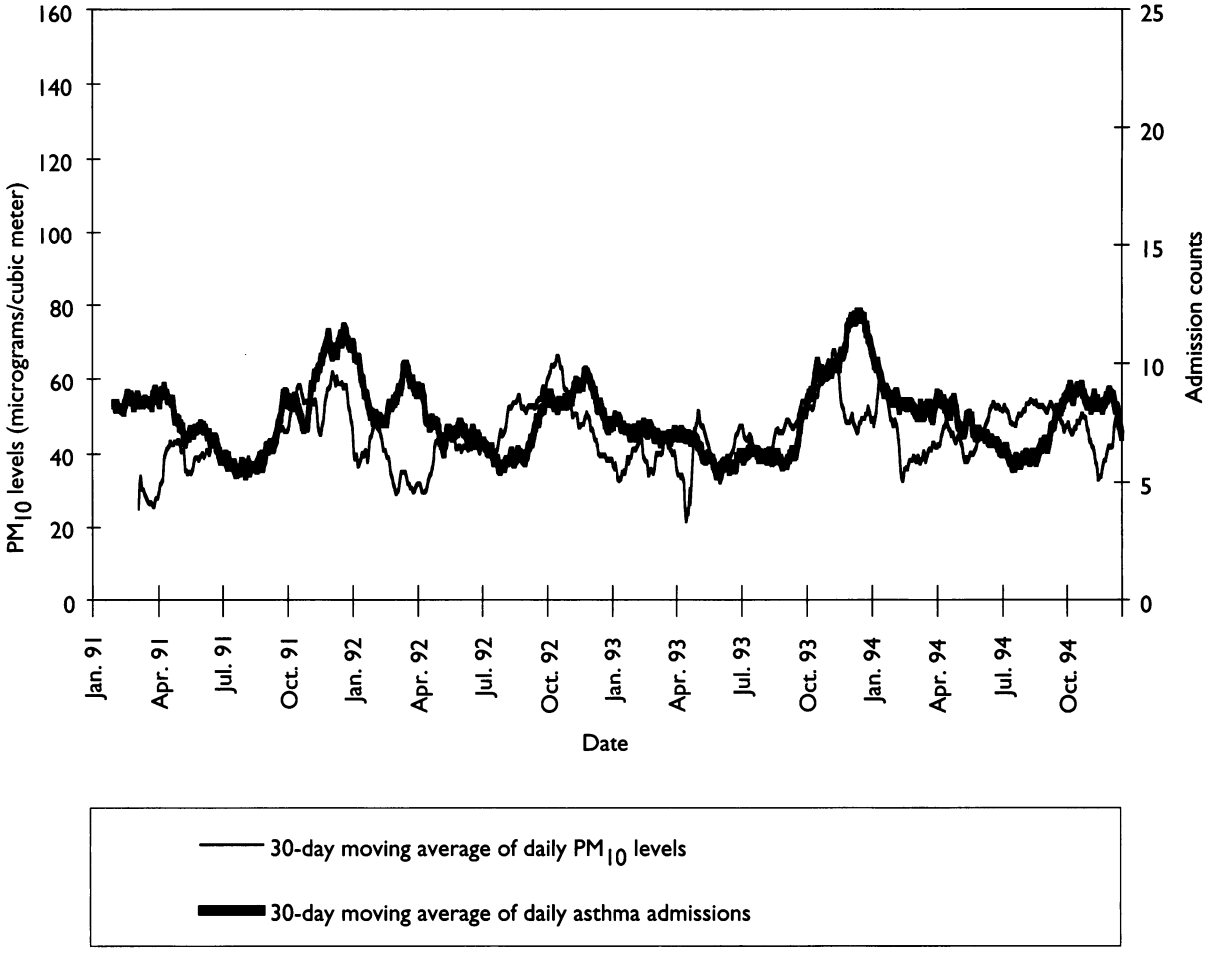
There are a number of limitations to an ecological study of this type. First, this study measured not personal exposure but average exposure over an entire region. There may have been substantial variation in individual exposure in the 900XX zip code area that is not well represented by an average value. Specifically, exposures in Brentwood and Bel Air might have been substantially less than in other areas; however, these two areas together only contributed an average of 0.5% to the overall daily admission counts. Second, this study focused on hospital admissions rather than emergency department visits, which might be a preferable measure of asthma morbidity because of larger sample sizes and greater daily variability. However, given that the state of California only has a mandate to collect data on admissions and not on outpatient visits, data limitations were the deciding factor in our choice of a morbidity measure.

Another concern is that the definition of asthma we used in this study was different from the standard one, which is limited to ICD-9 code 493. Because coding practices are extremely varied with regard to asthma, we felt that the broader definition would capture a larger number of actual asthma admissions.[28] Many exacerbations of asthma manifest themselves as pneumonia or acute bronchitis. Since both of these conditions are more prevalent in the population during the wet season, we reanalyzed the data using the 82.9% of hospital admissions with a primary diagnosis of ICD-9 493 (data not shown). There was little change in the magnitude of the regression coefficients corresponding to pollution exposure; therefore, cases of asthma presenting as pneumonia or acute bronchitis were not driving the results noted during the wet season.

Other studies evaluating the effect of PM_{10} exposure have noted lags of up to one month prior to an asthma episode.[34,36] While we have no disagreement that such a long lag is possible, we feel that the strongest associations should be found within a shorter time and that these studies were hampered by data limitations. Los Angeles made daily PM_{10} readings available beginning in 1991, but many other areas limit readings to every sixth day; therefore, a month-long lag would only incorporate four or five readings from which to evaluate exposure. In one study that did use daily PM_{10} readings, the investigators chose to look at monthly rather than daily fluctuations in admissions.[36] We feel that a daily admission count is a more precise unit of analysis to establish exposure-response relationships; moreover, too many degrees of freedom would be lost if a 30-day lag were used.

A major question is whether the associations observed are causal or merely due to residual confounding from unmeasured factors such as daily pollen counts, incidence of viral infections, prevalence of smoking, concentrations of cockroaches and dust mites, or factors related to socioeconomic status. Ostensibly, the first two factors have seasonal patterns that are eliminated by the Fourier series. Smokers or those exposed to secondhand tobacco smoke are widely known to have more frequent exacerbations of asthma; these exposures could enhance the effects of exposure to atmospheric pollutants. There is some evidence that MediCal/Medicaid beneficiaries include more smokers and people exposed to secondhand smoke than the rest of the population.[37] If true, then the pollution effects noted for the MediCal group might reflect the added sensitivity due to tobacco exposure. In addition, housing conditions that might promote cockroaches and dust

Figure 3. Daily counts of asthma-related emergency and urgent hospital admissions and mean daily PM_{10} levels, 900XX zip code region, Los Angeles, California, for selected months, 1991–1994: 30-day moving averages



mites are closely tied to socioeconomic factors such as poverty, which is also associated with eligibility for MediCal. Nevertheless, residual confounding from these unmeasured factors was probably small given the close tracking between actual and predicted daily asthma admissions, as shown in Figure 2. If the associations between PM_{10} exposure and asthma admissions in this study are causal, then the difference between the observed mean concentration (45 $\mu g/m^3$) and that in the least polluted city in a recent six-city study (18 $\mu g/m^3$) is responsible for 20.1% (100[27 x 0.0324/4.36]) of all asthma admissions in the Los Angeles area.[9] This should be of major concern to public health officials.

Because of variations in weather and demographic patterns, it is important to be careful about making generalizations from these results to the rest of the country. Differences in housing conditions (and thus in concentrations of cockroach eggs and/or dust mites), the magnitude of meteorologic fluctuations, and concentrations of other pollutants could affect the exposure-response relationship.

There has been some recent debate in the literature about whether PM_{10} exposure has an effect on asthma exacerbations. In a 1995 review of the literature, Pope, Bates, and Reizenne suggest that the evidence is sufficient to make an inference of causality between PM_{10} exposure and acute respiratory responses, including asthma; however, another review noted that the statistically significant effects in the published studies were weak and that the criteria for causality were usually not met.[38,39] The results of the present study are not conclusive in this regard, but do show a positive relationship

between PM_{10} exposure and asthma hospitalizations at levels below the current 24-hour NAAQS (National Ambient Air Quality Standard) for PM_{10} of 150 $\mu g/m^3$. Visual inspection of the data also shows a common pattern in daily admission counts and PM_{10} concentrations (Figure 3). These results suggest that a more restrictive standard than 150 $\mu g/m^3$ should be considered.

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