Air Pollution and Daily Hospital Admissions in Metropolitan Los Angeles

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We used daily time–series analysis to evaluate associations between ambient carbon monoxide, nitrogen dioxide, particulate matter ≤ 10 μm in aerodynamic diameter (PM_{10}), or ozone concentrations, and hospital admissions for cardiopulmonary illnesses in metropolitan Los Angeles during 1992–1995. We performed Poisson regressions for the entire patient population and for subgroups defined by season, region, or personal characteristics, allowing for effects of temporal variation, weather, and autocorrelation. CO showed the most consistently significant ($p < 0.05$) relationships to cardiovascular admissions. A wintertime 25th–75th percentile increase in CO (1.1–2.2 ppm) predicted an increase of 4% in cardiovascular admissions. NO_2, and, to a lesser extent, PM_{10} tracked CO and showed similar associations with cardiovascular disease, but O_3 was negatively or nonsignificantly associated. No significant demographic differences were found, although increased cardiovascular effects were suggested in diabetics, in whites and blacks (relative to Hispanics and Asians), and in persons older than 65 years of age. Pulmonary disease admissions associated more with NO_2 and PM_{10} than with CO. Pulmonary effects were generally smaller than cardiovascular effects and were more sensitive to the choice of model. We conclude that in Los Angeles, atmospheric stagnation with high primary ($CO/NO_2/PM_{10}$) pollution, most common in autumn/winter, increases the risk of hospitalization for cardiopulmonary illness. Summer photochemical pollution (high O_3) apparently presents less risk. *Key words*: **air pollutants, carbon monoxide, epidemiology, Los Angeles, morbidity, nitrogen dioxide, ozone, particulate matter.** *Environ Health Perspect* **108:427–434 (2000). [Online 27 March 2000]**
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Time–series analyses of daily mortality or morbidity have shown statistical associations with air pollution in cities throughout the world. Physiologic/toxicologic mechanisms of these phenomena remain unknown, and time–series analyses have not clearly linked specific pollutants with specific health outcomes (*1–3*); thus, their application to pollution-control policy decisions remains controversial (*4*). Combustion-related particulate matter, the only pollutant common to virtually all locations of time–series studies, has been the focus of scientific and regulatory attention (*1,2,4–6*). However, recent studies in a number of North American cities also associate cardiovascular and/or pulmonary disease incidence with pollutant gases such as carbon monoxide, nitrogen dioxide, and/or ozone (*7–12*). Where they are not highly correlated, gas and particulate pollutants appear to have separate statistically and medically significant influences on cardiopulmonary morbidity (*9,11,12*).

The Los Angeles metropolitan area has been studied relatively little by time–series analysis, but is a good candidate for study because of its large diverse population (≈ 14 million); detailed monitoring of air quality and hospital admissions; mild climate, which should limit confounding of pollution effects by weather stresses; and severe but widely variable air pollution (with maximum levels of primary pollutant gases, secondary photochemical oxidant gases, and particulate pollution occurring at somewhat different times and places). Powerful tests of pollution effects should be possible in the entire metropolitan population and in subpopulations defined geographically, demographically, or clinically. We hypothesized that regional and/or seasonal differences in time–series analysis results in the general population and/or in particular subgroups, would allow us to distinguish effects associated with primary pollutants (CO or NO_2), photochemical oxidants (O_3), or particulate matter more clearly than has been possible elsewhere. If so, we could rank these categories of pollution in terms of their public health impact, and thus provide useful guidance for regulatory policymaking and for future research on mechanisms. To test this hypothesis, we analyzed daily admission data for 1992–1995 from the South Coast Air Basin (Los Angeles, Riverside, San Bernardino, and Orange Counties in California, excluding mountain and desert regions of the first three counties) in relation to daily levels of CO, NO_2, O_3, and particulate matter ≤ 10 μm in aerodynamic diameter (PM_{10}).

Methods

Data acquisition and management. After its institutional review board verified confidentiality protection, the California Office of Statewide Health Planning and Development (OSHPD) (Sacramento, CA) provided records of hospital admissions in the metropolitan counties for 1992–1995 (the only years with adequately comparable PM_{10} data). The records included hospital identifier, date, principal and additional diagnoses as *International Classification of Diseases* (ICD; World Health Organization, Geneva) codes, All-Patient-Refined Diagnosis-Related Group (APR-DRG; 3M Inc., Murray, UT)—a broader classification based on Medicare diagnosis-related groups, sex, age, ethnic group, and residence zip code. Daily counts after 21 December 1995 were excluded from analysis because the records for numerous patients not discharged until 1996 were missing, and all 1995 data were excluded from ethnic-group analyses because of changes in OSHPD ethnic classifications. Broad principal-diagnosis categories used in analyses were cardiovascular (APR-DRG 103–144); cerebrovascular (APR-DRG 14–17 and 22); pulmonary (APR-DRG 75–101); and abdominal—a negative control category thought to be unrelated to pollution (APR-DRG 146–207). More-specific principal diagnoses, thought likely to associate with air pollution on the basis of previous epidemiologic or toxicologic evidence, were also analyzed: congestive heart failure (CHF) (APR-DRG 127); myocardial infarction (APR-DRG 111, 115, and 121); cardiac arrhythmia (APR-DRG 138); occlusive stroke (APR-DRG 14); asthma (ICD 493); and chronic obstructive pulmonary disease (COPD) (APR-DRG 88). Analyses excluded patients younger than 30 years of age (with

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exceptions noted in "Results") and prescheduled admissions.

We obtained air pollution and meteorologic data from the South Coast Air Quality Management District (SCAQMD; Diamond Bar, CA) and from the National Weather Service. These data included hourly PM_{10} from six SCAQMD stations with continuous monitors; hourly CO, O_3, NO_2, temperature, and relative humidity from those stations plus others; 24-hr-average PM_{10} measured every sixth day by high-volume samplers at or near each continuous PM_{10} station; and barometric pressure and rainfall at the Los Angeles International Airport. Figure 1 shows monitor locations. Analyses related daily admission counts with 24-hr averages of environmental variables. For O_3, maximum hourly concentrations were also analyzed; they correlated highly with 24-hr averages in all seasons ($r \geq 0.79$) and showed similar relationships to daily morbidity ("Results"). We did not analyze relative humidity because many data were missing or out of range. Stations differed in their continuous PM_{10} monitoring techniques and their relationships of continuous to high-volume sampler data. On the assumption that high-volume data were more comparable throughout the basin, we used season- and station-specific linear regressions to adjust continuous data to conform with high-volume data. We defined seasons to begin 1 January (winter), 1 April (spring), 1 July (summer), and 1 October (autumn).

Geographic differences were investigated across six regions defined by continuous PM_{10} monitoring stations (Figure 1). A region consisted of all zip codes that had a majority of their area closest to its station, except that some western coastal zip codes, which were separated from their closest station (region 1) by mountains, were assigned to region 2 to better represent their air quality. Admitted patients were assigned to regions by their residence zip codes. We excluded the 6.7% with zip codes missing or outside the South Coast Air Basin from regional analyses. We determined pollutant gas concentrations and temperatures for each region by averaging across all monitoring stations within it. Missing air monitoring data (4.4% for PM_{10}, smaller percentages for other variables) were replaced using analysis of variance with maximum likelihood estimation of missing values. For PM_{10}, data from all days in the same season and all stations in the basin were used in estimation; for other variables, only stations in the same region were used.

Statistical analyses. We used BMDP software (SPSS Inc., Chicago) and SAS software (SAS Institute, Inc., Cary, NC) for statistical analyses. Descriptive statistics and correlation patterns were examined regionally and seasonally for admission counts and atmospheric variables. Further descriptive analyses were performed to contrast weather and pollution characteristics between days with unusually high and unusually low observed morbidity relative to predicted values accounting for cyclical and secular trends. Predicted values were from regressions with indicator variables for the day of the week and for weekday holidays, with longer term variation modeled by fitting cubic splines to successive 28-day intervals of data. We then compared weather and pollution statistics between days with high admissions (residual > 85th percentile) and low admissions (residual < 15th percentile), as well as the immediately preceding days.

Time–series analytical approaches included *a*) ordinary least squares (OLS) regression with admission count and atmospheric data filtered by the Shumway 19-day weighted moving average procedure (*13*), with or without an autoregressive component; *b*) regression of log-transformed daily admission counts using polynomial distributed lag models (*14,15*); and *c*) Poisson regression with allowance for overdispersion and autocorrelation, adapted from the analytical strategy of the Particle Epidemiology Evaluation Project (*16*) with modifications. In principle, daily counts are Poisson distributed and require approach *c*; however, given the generally large counts with filtering or smoothing, distributions were reasonably near normal so that other approaches were also feasible (*17*). The different approaches yielded similar conclusions when considering cardiovascular diseases. Polynomial-distributed lag models showed the largest significant effects consistently at lag 0, and effects beyond lag 1 were nearly always nonsignificant. Therefore, we adopted Poisson regression as the primary analytical tool. Predictors of daily admission counts included basis variables of a cubic-spline smooth on time (which accounted for secular trends and seasonal variation); indicator variables for the day of the week and for weekday holidays; indicator variables for hot days (maximum temperature > 85th percentile for entire study period), cold days (minimum temperature < 15th percentile), and rain days (> 0.01 inches at the Los Angeles International Airport); continuous atmospheric variables (one or more pollutant concentrations, barometric pressure, and mean temperature); and an autoregressive term—the residual admission count at lag 1, determined in a preliminary regression including all other predictors. Seasonal variation was more complex for pulmonary diseases than for others, probably because the timing and intensity of winter infectious disease outbreaks varied from year to year. Thus, cubic splines were determined at 28-day intervals when smoothing pulmonary disease counts, and at 4-month intervals otherwise.

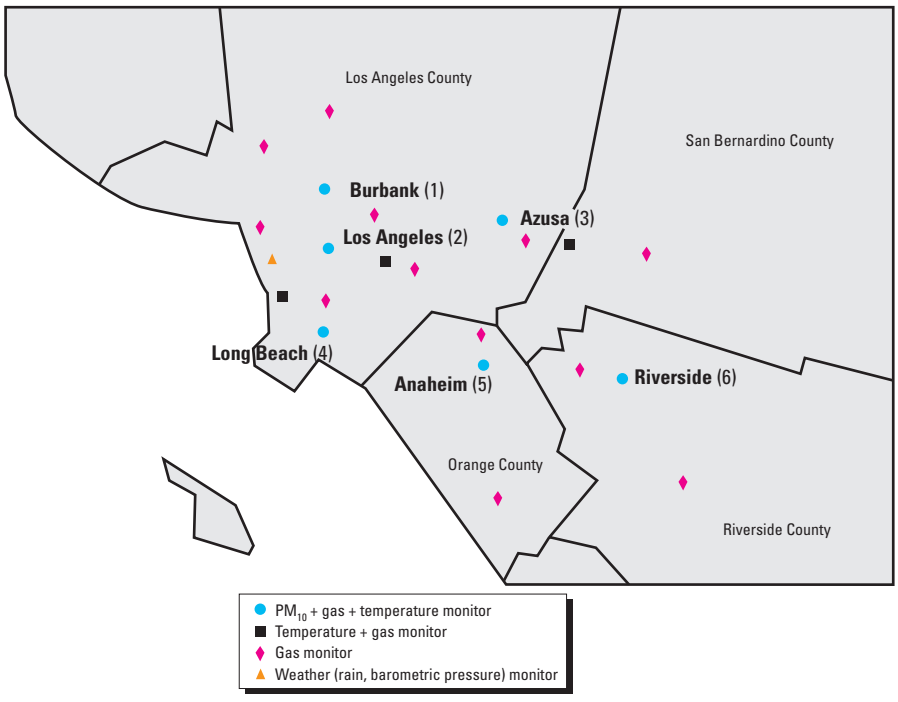

Figure 1. Air monitoring stations that contributed data to the analyses. Cities identified on the map have the continuous PM_{10} monitors that represent the six separate air quality regions analyzed (see text).

Results

Seasonal air quality and hospital admissions. Table 1 presents seasonal pollution, weather, and hospital admission statistics for the entire basin for 1992–1995. Overall means ± SDs

were 1.5 ± 0.8 ppm for CO, 3.4 ± 1.3 parts per hundred million (pphm) for NO_2, 45 ± 18 μg/m^3 for PM_{10}, and 2.4 ± 1.2 pphm for O_3. We determined basinwide means from the six regional values by weighting each region according to its proportion of cardiovascular plus pulmonary admissions (considered to reflect its proportion of the population at risk). These levels reflect a > 80% reduction of CO since the 1960s (*18*), and more modest reductions in the other pollutants. Year-round means and SDs of daily admissions were 428 ± 76 for cardiovascular, 207 ± 54 for pulmonary, 74 ± 14 for cerebrovascular, and 244 ± 39 for abdominal diseases. Seasonal means for abdominal diseases (not tabulated) varied < 3%. All disease categories showed marked variation by day of the week, consistent across seasons. Relative to Monday admissions, Sunday admissions averaged 64% for cardiovascular, 70% for pulmonary, 76% for cerebrovascular, and 67% for abdominal diseases.

Table 2 shows pairwise correlations of basinwide average daily pollutant concentrations, mean temperature, and barometric pressure within each season. NO_2 showed high positive correlations ($r \geq 0.8$) with CO in all seasons, and correlations nearly as high with PM_{10}. O_3 was positively correlated with all three other pollutants only in the spring, and most strongly with PM_{10}. O_3 showed a weaker positive relationship to PM_{10} in the summer and a negative relationship to CO and NO_2 in the winter. Higher mean temperatures were associated with higher pollutant levels in all seasons, with the exception of CO in the autumn. Barometric pressure showed varying relationships with pollutants. Expressing the data as residuals from cubic-spline smoothing brought about no marked changes in these correlations, except that in autumn the positive relationship between O_3 and temperature became nonsignificant. Regional measurements and basinwide averages correlated strongly for every pollutant in every season ($r > 0.7$), except for PM_{10} in the summer ($r = 0.5$–0.6 for some regions). Different regions' measurements of a given pollutant also correlated positively in all seasons. Southern coastal region 4 and eastern inland region 6 contrasted most sharply, with *r*-values between 0.3 and 0.7. In light of this generally similar behavior of air quality in different regions, time–series analyses focused on the entire basin, and regional comparisons were limited to regions 4 and 6.

Figure 2 shows average concentrations by region and season for CO, PM_{10}, and O_3. The mean and variance of CO decreased markedly in the spring and summer in all regions. Autumn and winter CO were highest in the southern coastal region 4. PM_{10} was highest in the summer and autumn, particularly in the eastern inland region 6, but seasonal variation was less for PM_{10} than for CO. O_3 was highest in the spring and summer, particularly in inland regions 3 and 6.

Contrast of atmospheric conditions between days with high and low admission counts. Table 3 summarizes significant ($p < 0.05$) differences in basinwide weather and pollution statistics between days with unusually high and unusually low admission counts (residuals from cubic-spline smoothing) in a particular broad disease category. High-admission days (and/or immediately preceding days) tended to have relatively warm dry weather. Primary pollution (CO and NO_2) was significantly elevated on winter, spring, or autumn days with high cardiovascular admissions; spring and summer days with high pulmonary admissions; and spring and autumn days with high cerebrovascular admissions. Elevated PM_{10} tended to accompany elevated primary pollutants on days with high cardiovascular or pulmonary admissions; PM_{10} also was associated with high abdominal admissions in the spring. O_3 was increased (along with the other pollutants) on days with high pulmonary admissions in spring and summer, the seasons of the highest mean O_3 concentrations (Table 1). By contrast, O_3 was decreased on days with high cardiovascular admissions in the winter, when O_3 was generally low and negatively correlated with the other pollutants.

Table 4 shows mean weather and pollution conditions on days of high and low cardiovascular admissions in the winter and summer for the contrasting southern coastal region 4 and eastern inland region 6. In the summer, pollution (except for CO) and heat were markedly greater in region 6, but there were no clear pollution or temperature differences between high- and low-admission days in either region. In the winter, CO was markedly higher in region 4, and other regional differences were modest. In region 4, winter high-admission days had significantly higher temperature, barometric pressure, CO, NO_2, and PM_{10}, and significantly lower probability of rain, than low-admission days. In region 6, these tendencies were less obvious, but CO and NO_2 were significantly elevated on the days preceding high-admission days. In similar analyses of pulmonary diseases (not tabulated), we found only a few significant associations with high admissions: high same-day PM_{10} in region 4 in the winter,

Table 1. Air[a] and hospital admission statistics for the entire South Coast Air Basin, by season.

Variable (units)	Season	Mean ± SD	Min	Max
CO (ppm)	Winter	1.7 ± 0.8	0.5	5.3
	Spring	1.0 ± 0.3	0.4	2.2
	Summer	1.2 ± 0.4	0.3	2.7
	Autumn	2.1 ± 0.8	0.6	4.3
NO_2 (pphm)	Winter	3.4 ± 1.3	1.1	9.1
	Spring	2.8 ± 0.9	1.1	6.1
	Summer	3.4 ± 1.0	0.7	6.7
	Autumn	4.1 ± 1.4	1.6	8.4
PM_{10} (μg/m^3)	Winter	37 ± 19	5	115
	Spring	42 ± 12	14	83
	Summer	49 ± 10	14	78
	Autumn	54 ± 22	15	132
O_3 (pphm)	Winter	1.4 ± 0.7	0.2	4.4
	Spring	3.2 ± 1.0	0.9	7.0
	Summer	3.3 ± 0.8	0.4	6.3
	Autumn	1.5 ± 0.9	0.1	4.7
Temperature (mean, °C)	Winter	14.8 ± 2.7	8.4	23.2
	Spring	19.2 ± 3.0	9.5	29.2
	Summer	23.9 ± 2.3	19.0	31.2
	Autumn	16.4 ± 3.7	8.7	26.7
Rain (% of days)	Winter	28		
	Spring	4		
	Summer	1		
	Autumn	10		
Cardiovascular adm/day	Winter	450 ± 77	300	607
	Spring	428 ± 76	277	586
	Summer	406 ± 70	239	559
	Autumn	428 ± 76	273	610
Pulmonary adm/day	Winter	241 ± 54	117	574
	Spring	196 ± 34	118	329
	Summer	172 ± 27	107	256
	Autumn	220 ± 63	115	595
Cerebrovascular adm/day	Winter	77 ± 14	44	126
	Spring	74 ± 14	43	116
	Summer	72 ± 14	41	114
	Autumn	75 ± 14	45	117

Abbreviations: adm, admissions; max, maximum; min, minimum; pphm, parts per hundred million.
[a]Pollutant concentrations are averaged across six regions, each weighted according to its proportion of cardiovascular plus pulmonary admissions.

Table 2. Pairwise correlation coefficients (*r*) for atmospheric factors expressed as basinwide 24-hr averages,[a] by season.*

Factor	Season	NO_2	PM_{10}	O_3	T_{mean}	BP[a]
CO	Winter	0.89	0.78	-0.43	0.22	0.43
	Spring	0.92	0.54	0.29	0.38	0.15
	Summer	0.94	0.72	0.03	0.51	-0.17
	Autumn	0.84	0.58	-0.36	-0.08	0.38
NO_2	Winter	–	0.88	-0.23	0.38	0.38
	Spring	–	0.67	0.35	0.53	0.03
	Summer	–	0.80	0.11	0.51	-0.21
	Autumn	–	0.80	-0.00	0.28	0.12
PM_{10}	Winter	–	–	-0.01	0.37	0.39
	Spring	–	–	0.63	0.64	-0.18
	Summer	–	–	0.40	0.44	-0.30
	Autumn	–	–	0.28	0.40	-0.03
O_3	Winter	–	–	–	0.33	-0.11
	Spring	–	–	–	0.57	-0.23
	Summer	–	–	–	0.18	-0.05
	Autumn	–	–	–	0.62	-0.42
T_{mean}	Winter	–	–	–	–	0.13
	Spring	–	–	–	–	-0.34
	Summer	–	–	–	–	-0.20
	Autumn	–	–	–	–	-0.39

Abbreviations: BP, barometric pressure; T, temperature.
[a]BP measured only at the Los Angeles International Airport; other variables averaged from measurements in all six regions, weighted according to regions' proportions of total cardiovascular plus pulmonary admissions.
*$p < 0.05$ for $r > 0.10$; $p < 0.01$ for $r > 0.13$.

high previous-day CO and low previous-day O_3 in region 6 in the winter, and high previous-day NO_2 and O_3 in region 6 in the summer.

Analyses of admission counts in broad disease categories. Table 5 presents the results from single-pollutant autoregressive Poisson models, including all of the time and weather predictors mentioned in "Methods," relating daily average concentrations with same-day hospital admission counts over the entire basin for each broad disease category. Admissions of patients under 30 years of age are not included. To a good approximation, the coefficients represent proportionate increases in admission counts expected from unit increases in pollutant concentrations.

Primary pollution, as represented by CO and NO_2, showed the most consistent associations with cardiovascular-disease admissions; they were significantly related in year-round analyses and in single-season analyses except for the spring. PM_{10} showed a similar pattern of relationships, but was nonsignificant in the summer as well as the spring. The cardiovascular disease/primary pollution relationship was not very sensitive to inclusion or exclusion of weather or other pollutant variables in the models. In the winter, the interquartile range of CO concentrations was 1.1 to 2.2 ppm, and the corresponding predicted increase in cardiovascular admissions was 4.2%, which represented approximately 20 extra admissions/day.

Pulmonary-disease admissions were significantly related to NO_2 or PM_{10} in year-round and winter analyses, and also to NO_2 or CO in autumn. Cerebrovascular-disease admissions were significantly related only to CO or NO_2 only in the spring. Abdominal-disease admissions were significantly related only to NO_2, and only in the year-round analysis.

O_3 showed either negative or nonsignificant positive relationships with cardiovascular, pulmonary, cerebrovascular, and abdominal disease admissions in year-round and single-season analyses. The same was true in OLS regressions of Shumway-filtered data (not tabulated). Alternative analyses intended to give additional weight to high-O_3 conditions, by expressing exposure in terms of daily maxima or in exceedances of an assumed threshold, or by restricting the analysis to the three high-O_3 inland regions, still showed no significant positive associations. In Poisson models excluding mean temperature and barometric pressure, daily mean O_3 showed significant positive associations with pulmonary diseases in the spring and year-round.

Table 6 illustrates the sensitivity of results to the choice of regression procedure and model for the cardiovascular disease/CO relationship in the winter and the pulmonary disease/O_3 relationship in the spring. Across a broad range of models with and without weather and other pollutants as predictors,

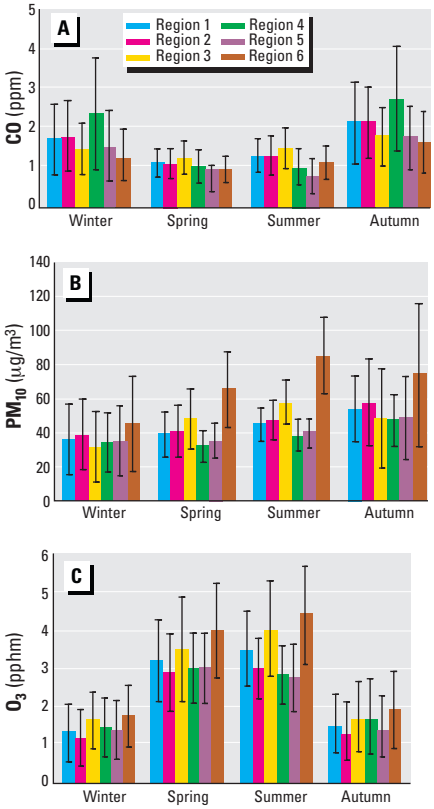

Figure 2. Air quality in six regions (see Figure 1), 1992–1995. Bar = seasonal mean; flag = seasonal SD.

Table 3. Atmospheric variables showing significant ($p < 0.05$) differences between days with high and low hospital admissions in the entire basin, by season.[a]

Disease category	Season	CO	NO_2	PM_{10}	O_3	T_{high}	T_{low}	BP	Rain
Cardiovascular	Winter	+	+	+	–	+		+	–
	Spring	+	+			+	[b]		–
	Summer							+	
	Autumn	+	+	+					
Pulmonary[c]	Spring	+	+	+	+	+			–
	Summer	+	+	+	+	+	+		
	Autumn					+			
Cerebrovascular	Winter					[b]			
	Spring	+	+			[b]	+		
	Autumn	[b]	+			+			–
Abdominal[d]	Spring			+					–

Abbreviations: BP, barometric pressure; T, temperature.
[a]High- and low-admission days (> 85th and < 15th percentiles, respectively) are determined by residuals from regressions accounting for temporal effects (see text). A significant increase in atmospheric variables on high-admission days relative to low-admission days (and/or the immediately preceding days) is indicated by +; a significant decrease by –. [b]The change in atmospheric measurement from the preceding day was significantly more positive on high- than low-admission days, although values measured on high and low days were not significantly different. [c]For pulmonary diseases in winter, high-admission days' increases in NO_2, PM_{10}, and high temperature approached significance ($p < 0.10$). [d]For abdominal diseases in winter, high-admission days' increases in CO approached significance ($p < 0.10$).

Table 4. Atmospheric differences between days of high and low cardiovascular admissions[a] in most contrasting regions/seasons.

Variable (units)	Admissions (n)	Region 4 (Long Beach)		Region 6 (Riverside)	
		Winter	Summer	Winter	Summer
CO (ppm)	Low	2.1	1.0	1.1	1.1
	High	2.9*	1.1	1.3**	1.2
NO_2 (pphm)	Low	3.4	2.8	2.5	3.8
	High	4.4*	2.9	2.9**	4.0
PM_{10} (µg/m^3)	Low	27.6	38.0	39.1	84.6
	High	42.8*	36.9	48.5	83.5
O_3 (pphm)	Low	1.4	2.8	1.9	4.4
	High	1.4	2.9	1.8	4.5
T_{high} (°C)	Low	17.9	25.6	19.2	33.7
	High	20.1*	25.6	19.6	33.9
T_{low} (°C)	Low	10.6	17.7	8.9	17.1
	High	10.4	17.2	9.2	17.0
BP[b] (mbar)	Low	1,016.2	1,012.9	1,017.3	1,012.5
	High	1,018.5*	1,013.0	1,016.4	1,013.5*
Rain[b] (% of days)	Low	38	0	26	0
	High	12*	0	22	2

[a]See Table 3 for definition of high- and low-admission days. [b]Measured at the Los Angeles International Airport, closer to region 4 than region 6. Other measurements made within the indicated region. *Significant ($p < 0.05$) differences. **Although this difference did not reach significance, the difference between days immediately preceding high- and low-admission days was significant ($p < 0.05$).

the estimated winter CO effect was always significant and was reasonably consistent in size. None of the other pollutants' effects was significant when included in a model with CO. The spring O_3 effect on pulmonary admissions was significant when O_3 was the only atmospheric factor in the model, predicting a 1.5% increase in admissions for a 1-pphm increase in daily mean O_3 concentration. However, the O_3 effect was nonsignificant if the model included weather and/or other pollutant variables. Interpretation of these findings is complicated by collinearity and by possibly different characteristics of exposure measurement error for different pollutants (*19*). Nevertheless, it seems clear that in the winter, CO was the analyzed atmospheric factor that was most closely linked with excess cardiovascular morbidity. In the spring, O_3 was the pollutant most closely associated with excess pulmonary morbidity; however, morbidity was still more closely associated with warm temperatures, and all four pollutants tended to rise with temperature, making interpretation difficult.

Because diabetes mellitus is an important risk factor for cardiovascular disease, we reanalyzed cardiovascular admissions separately for diabetics (all of those with ICD code 250 entered among four additional diagnoses in the record; approximately 20% of all patients) and for others, using the autoregressive Poisson model. In year-round analyses, the slope ± SE was 0.039 ± 0.006 for diabetics as compared to 0.031 ± 0.004 for others. Year-round analyses of NO_2 and PM_{10} effects showed similar modest slope increases for diabetics, as did single-season analyses. None of the slope differences between diabetics and others was statistically significant.

Analyses of cardiovascular disease admission counts by age, sex, and ethnicity. Table 7 presents results from single-pollutant autoregressive Poisson models applied to cardiovascular admission counts in three age strata (30–64, 65–74, and ≥ 75 years of age) separately for men and women. Results for O_3 (not tabulated) were never statistically significant. CO effects were near-significant for women 30–64 years of age and sig-nificant in all other age–sex groups in year-round analyses and in one or more seasonal analyses. Effect sizes increased with age similarly in both sexes, but age-related differences were not significant. NO_2 effects showed a similar pattern of significance, but with less suggestion of age dependence. PM_{10} effects were also significant year-round and/or in one season for all groups except men ≥ 75 years of age.

Table 8 presents the results from single-pollutant autoregressive Poisson models applied to cardiovascular admission counts for adults ≥ 30 years of age in four ethnic categories—white (non-Hispanic), black, Hispanic, and other. The other category includes people of Asian-Pacific ancestry (the large majority), Native Americans, and others not classifiable in the first three groups. O_3 effects (not tabulated) were never significant. Regression coefficients, though not significantly different, suggested meaningful ethnic differences in exposure–response relationships. CO, NO_2, and PM_{10} effects were significant in whites in year-round, winter, and autumn analyses. In blacks, CO and NO_2 effects were significant year-round (also in the winter for CO) and were similar to these effects in whites. CO and NO_2 effects in Hispanics were significant year-round but were smaller than these effects in whites and blacks. The remaining (other) category, with a relatively small number of admissions, showed consistently small and nonsignificant regression slopes.

Analyses of admission counts for more specific diagnoses. Table 9 presents results in adults ≥ 30 years of age from single-pollutant autoregressive Poisson models relating basinwide daily average pollutant concentrations with same-day admission counts. Occlusive strokes showed the most consistent positive relationships to pollution: significant associations with O_3 in the summer only; and with CO, NO_2, and PM_{10} year-round and in at least two single-season analyses. Asthma, COPD, and CHF were

Table 5. Poisson regression coefficients (SEs): hospital admissions in broad disease categories in the entire metropolitan area, versus same-day pollution levels.[a]

Pollutant, units	Season	Cardiovascular	Pulmonary	Cerebrovascular	Abdominal
CO (ppm)	All	0.032 (0.003)*	0.007 (0.005)	0.009 (0.007)	0.003 (0.004)
	Winter	0.038 (0.006)*	0.016 (0.009)	-0.008 (0.014)	0.006 (0.008)
	Spring	0.010 (0.015)	0.014 (0.024)	0.107 (0.033)*	-0.007 (0.019)
	Summer	0.035 (0.014)*	0.020 (0.021)	0.030 (0.033)	0.021 (0.018)
	Autumn	0.027 (0.006)*	0.020 (0.008)*	0.008 (0.012)	0.006 (0.007)
NO_2 (pphm)	All	0.014 (0.002)*	0.007 (0.003)*	0.004 (0.004)	0.004 (0.002)*
	Winter	0.016 (0.004)*	0.011 (0.005)*	-0.013 (0.007)	0.002 (0.005)
	Spring	0.001 (0.006)	0.007 (0.010)	0.042 (0.012)*	-0.004 (0.007)
	Summer	0.011 (0.005)*	0.004 (0.008)	0.009 (0.012)	0.008 (0.006)
	Autumn	0.014 (0.003)*	0.012 (0.004)*	0.007 (0.006)	0.007 (0.004)
PM_{10} ($\mu g/m^3$)	All	0.00064 (0.00012)*	0.00057 (0.00018)*	0.00006 (0.00025)	0.00017 (0.00014)
	Winter	0.00095 (0.00024)*	0.00081 (0.00032)*	-0.00021 (0.00052)	0.00013 (0.00030)
	Spring	-0.00031 (0.00037)	0.00010 (0.00061)	0.00126 (0.00083)	0.00039 (0.00047)
	Summer	0.00039 (0.00041)	0.00061 (0.00061)	0.00127 (0.00096)	0.00068 (0.00052)
	Autumn	0.00065 (0.00020)*	0.00078 (0.00029)	-0.00004 (0.00039)	0.00008 (0.00022)
O_3 (pphm)	All	-0.007 (0.003)**	0.008 (0.004)	0.003 (0.005)	0.003 (0.003)
	Winter	-0.021 (0.008)**	-0.006 (0.010)	0.028 (0.016)	-0.012 (0.010)
	Spring	0.003 (0.005)	0.011 (0.008)	0.011 (0.011)	0.000 (0.006)
	Summer	0.001 (0.005)	0.006 (0.007)	0.007 (0.011)	0.011 (0.006)
	Autumn	-0.003 (0.007)	0.009 (0.011)	-0.003 (0.014)	0.000 (0.008)

[a]Regression analyses used 24-hr average measurements of pollutants and same-day admission counts for patients ≥ 30 years of age throughout the South Coast Air Basin. Example interpretation: the coefficient 0.038 relating winter cardiovascular admissions to CO indicates that admissions increase by a factor of $e^{0.038}$, i.e., by 3.9%, with a 1-ppm increase in CO concentration, after allowing for the effects of time and weather on admission rates. *Significant in expected direction, $p < 0.05$. **Significant in the "wrong" direction, $p < 0.05$.

Table 6. Sensitivity of key results to choice of regression procedure and model.

Procedure (additional predictors)	Slope (SE)	Relative risk[a]
Cardiovascular admissions versus CO, winter		
Poisson autoregressive (time)	0.044 (0.005)*	1.050
Poisson autoregressive (time, weather)	0.038 (0.006)*	1.043
Poisson autoregressive (time, weather, O_3)	0.036 (0.007)*	1.040
Poisson autoregressive (all above + PM_{10})	0.033 (0.011)*	1.037
Poisson autoregressive (all above + NO_2)	0.047 (0.013)*	1.053
PDL[b] (time, weather)	0.044 (0.008)*	1.050
OLS autoregressive (time, weather)	19.6 (3.1)*	1.048
Pulmonary admissions versus O_3, spring		
Poisson autoregressive (time)	0.012 (0.004)*	1.015
Poisson autoregressive (time, weather)	0.003 (0.005)	1.003
Poisson autoregressive (time, weather, CO)	0.002 (0.005)	1.002
Poisson autoregressive (all above + PM_{10})	0.008 (0.006)	1.009
Poisson autoregressive (all above + NO_2)	0.008 (0.006)	1.009
PDL[b] (time, weather)	0.003 (0.011)	1.004
OLS autoregressive (time, weather)	1.58 (1.79)	1.010

[a]Predicted relative risk at the 75th percentile concentration of pollutant, versus the 25th percentile. [b]Cubic polynomial distributed lag model, lag 0 slope given, lags 1–3 slopes nonsignificant. *Significant in expected direction, $p < 0.05$.

significantly associated with CO and NO_2 year-round and in one or more single-season analyses. Myocardial infarction was associated with CO and NO_2, and arrhythmias with CO, in year-round analyses only.

We also analyzed asthma admissions for patients 0–29 years of age. In year-round analyses, slopes ± SEs were 0.036 ± 0.016/ppm CO, 0.024 ± 0.008/pphm NO_2, and 0.0011 ± 0.0006/μg/m^3 PM_{10}—all significant ($p < 0.05$) and appreciably larger than the slopes in adults > 30 years of age. O_3 effects were nonsignificant. Most of the admitted patients in this youngest group were children: the mean age was 7.

Relationships of cardiovascular disease admissions to CO or PM_{10} in the two most contrasting regions. Table 10 presents comparative statistics for cardiovascular admissions, CO, and PM_{10} in southern coastal region 4 and eastern inland region 6. The two regions showed reasonably similar daily admission counts and similar positive correlations of daily CO and PM_{10} levels, but markedly different concentration ranges (Figure 2). By OLS regression allowing for autocorrelation, a wintertime 1-ppm rise in CO predicted a 9-μg/m^3 rise in PM_{10} in region 4, but a 25 μg/m^3 rise in region 6. In single-pollutant autoregressive Poisson models, region 4 showed highly significant relationships between PM_{10} and admissions, year-round and in the winter, despite its low PM_{10}. In region 6, despite its high PM_{10}, regression slopes were significantly lower than in region 4, and were not significantly different from zero. Admissions showed a more plausible relationship with CO across the two regions, with highly significant positive slopes in region 4 and modestly lower non-significant slopes in region 6, consistent with its generally lower and less variable CO concentrations.

Table 7. Poisson regression coefficients (SEs): cardiovascular disease admissions in the entire metropolitan area versus same-day pollution levels, by age and sex.[a]

Sex, age in years [count][b]	Pollutant[c]	Year-round coefficient	Significant ($p < 0.05$) positive coefficients for separate seasons
Male 30–64	CO	0.014 (0.007)*	Winter 0.040 (0.013), autumn 0.025 (0.013)
[99 ± 21]	NO_2	0.007 (0.004)	Winter 0.017 (0.008), autumn 0.016 (0.007)
	PM_{10}	0.0003 (0.0003)	Winter 0.0016 (0.0005)
Male 65–74	CO	0.037 (0.009)*	Autumn 0.045 (0.016)
[61 ± 14]	NO_2	0.014 (0.005)*	Autumn 0.024 (0.009)
	PM_{10}	0.0008 (0.0003)*	Autumn 0.0013 (0.0006)
Male 75+	CO	0.040 (0.009)*	Summer 0.068 (0.034), autumn 0.042 (0.015)
[59 ± 13]	NO_2	0.013 (0.005)*	Autumn 0.020 (0.008)
	PM_{10}	0.0003 (0.0003)	None
Female 30–64	CO	0.017 (0.009)	None
[68 ± 15]	NO_2	0.015 (0.004)*	Winter 0.018 (0.008)
	PM_{10}	0.0007 (0.0003)*	None
Female 65–74	CO	0.033 (0.009)*	Winter 0.043 (0.015)
[56 ± 12]	NO_2	0.014 (0.005)*	Winter 0.017 (0.008)
	PM_{10}	0.0002 (0.0003)	Winter 0.0012 (0.0006)
Female 75+	CO	0.040 (0.007)*	Winter 0.047 (0.013), autumn 0.025 (0.011)
[88 ± 17]	NO_2	0.014 (0.004)*	Winter 0.021 (0.008)
	PM_{10}	0.0005 (0.0003)	Winter 0.0012 (0.0005)

[a]These results are for 1992–1994 only. See footnote to Table 5 for explanation of regression procedure and coefficients. [b]Annual mean daily admission count ± SD, for patients ≥ 30 years of age. [c]O_3 results not tabulated; none was significantly positive. *Year-round relationship significant, $p < 0.05$.

Table 8. Poisson regression coefficients (SEs): cardiovascular disease admissions in the entire metropolitan area versus same-day pollution levels, by ethnic category.[a]

Category [count][b]	Pollutant[c]	Year-round coefficient	Significant ($p < 0.05$) positive coefficients for separate seasons
White	CO	0.034 (0.005)*	Winter 0.038 (0.008), autumn 0 .036 (0.008)
[290 ± 53]	NO_2	0.014 (0.003)*	Winter 0.018 (0.005), autumn 0.017 (0.005)
	PM_{10}	0.0006 (0.0002)*	Winter 0.0011 (0.0003), autumn 0.0007 (0.0003)
Black	CO	0.031 (0.010)*	Winter 0.042 (0.017)
[49 ± 11]	NO_2	0.014 (0.006)*	None
	PM_{10}	0.0003 (0.0004)	None
Hispanic	CO	0.019 (0.009)*	None
[63 ± 13]	NO_2	0.010 (0.005)*	None
	PM_{10}	0.0005 (0.0003)	None
Other	CO	0.001 (0.013)	None
[29 ± 8]	NO_2	0.002 (0.007)	None
	PM_{10}	-0.0004 (0.0005)	None

[a]These results are for 1992–1994 only. See footnote to Table 5 for explanation of regression procedure and coefficients. [b]Annual mean daily admission count ± SD, for patients ≥ 30 years of age. [c]O_3 results not tabulated; none was significantly positive. *Year-round relationship significant, $p < 0.05$.

Discussion

Limitations; recommendations for future research. Problems with this and other time–series studies include exposure misclassification, response misclassification, and model misspecification. Exposure misclassification occurs when the monitored environmental factors are not the ones responsible for health effects, when monitoring errors are appreciable and differ by time and location, when monitoring station data poorly represent background air near patients' homes, when personal microenvironments differ from background, or when exposures that precipitate hospital admissions occur away from home. Future expansion of the monitoring program, at least for particulate pollution, should provide better background concentration estimates, allowing more powerful tests for regional differences in effects. New personal monitoring studies, designed to elucidate longitudinal relationships between background and personal exposures, might help to disentangle the effects of particulate pollutants and covarying gases (e.g., CO and NO_2). Small panel studies have suggested that personal particulate exposures track background concentrations closely in healthy older adults and children in The Netherlands (*20,21*), but not in older adults with COPD in Los Angeles (*22*). To our knowledge, no longitudinal studies of personal CO exposure have been reported.

Response misclassification can result from errors in diagnosis or in medical record coding. Reviews suggest that 15–20% of assigned ICD codes are inaccurate (*23,24*). Inaccuracies should increase random errors in specific disease counts and reduce the statistical significance of disease/pollution relationships, but should not introduce bias unless coding inaccuracies covary with pollution. Misdiagnoses are hard to evaluate, but are undoubtedly important, given the complexities of disease processes and the fuzzy boundaries between diagnoses. Wrong diagnoses or codes would likely shift patients to different specific disease counts within the same broad category, and thus should have relatively little effect on broad-category analyses. In any event, we have had only limited success in finding specific pollutant–disease relationships with mechanistic or public-health implications. Future studies focusing on precise diagnoses and accounting for other risk factors (e.g., additional diagnoses and particular demographic characteristics) might be more successful.

Limitations of our primary analytical model include the use of only one pollutant at a time and possibly incomplete accounting for weather and temporal influences. Because estimated CO effects were similar in various single- and multipollutant models that accounted for seasonal and weekly cycles, more complete modeling of weather or temporal effects should not change the conclusions concerning CO. By contrast, estimates of O_3 effects were highly sensitive to inclusion or exclusion of weather and other pollutant variables. If pollutants not in the model affected admissions, the likely result would be to overestimate effects of the modeled pollutant and underestimate total effects of pollution (*12*). Thus, effects we associated with CO might be at least partly due to covarying gases (e.g., oxides of nitrogen) or to particulate substances. Similarly, incomplete accounting for lagged effects would likely result in overestimated effects of very recent exposure, but underestimated cumulative effects of recent and earlier exposures (*14*).

Table 9. Poisson regression coefficients (SEs): hospital admissions in more specific disease categories in the entire metropolitan area, versus same-day pollution levels.[a]

Disease [count][b]	Pollutant	Year-round coefficient	Significant ($p < 0.05$) positive coefficients for separate seasons
Myocardial infarction [47 ± 11]	CO	0.040 (0.009)*	None
	NO_2	0.011 (0.005)*	None
	PM_{10}	0.0006 (0.0003)	None
	O_3	-0.007 (0.007)	None
Congestive heart failure [49 ± 11]	CO	0.025 (0.009)*	Summer 0.074 (0.038)
	NO_2	0.010 (0.005)*	Winter 0.019 (0.009)
	PM_{10}	0.0004 (0.0003)	None
	O_3	-0.001 (0.007)	None
Cardiac arrhythmia [50 ± 10]	CO	0.023 (0.009)*	None
	NO_2	0.006 (0.005)	None
	PM_{10}	0.0002 (0.0003)	None
	O_3	-0.001 (0.007)	None
Occlusive stroke [45 ± 10]	CO	0.044 (0.009)*	Winter 0.036 (0.017), summer 0.091 (0.039), autumn 0.032 (0.015)
	NO_2	0.020 (0.005)*	Winter 0.027 (0.010), autumn 0.021 (.008)
	PM_{10}	0.0013 (0.0003)*	Winter 0.0024 (0.0006), autumn 0.0012 (0.0005)
	O_3	0.007 (0.007)	Summer 0.025 (0.012)
Asthma[c] [38 ± 9]	CO	0.028 (0.010)*	Winter 0.045 (0.017), autumn 0.039 (0.016)
	NO_2	0.014 (0.005)*	Winter 0.028 (0.010), autumn 0.019 (0.008)
	PM_{10}	0.0003 (0.0004)	None
	O_3	-0.001 (0.008)	None
COPD [89 ± 19]	CO	0.019 (0.007)*	Winter 0.035 (0.012), autumn 0.029 (0.011)
	NO_2	0.008 (0.004)*	Autumn 0.016 (0.006)
	PM_{10}	0.0003 (0.0002)	None
	O_3	-0.007 (0.005)	None

[a]See footnote to Table 5 for explanation of regression procedure and coefficients. [b]Annual mean daily admission count ± SD for patients ≥ 30 years of age. [c]See text concerning asthma in patients < 30 years of age. *Year-round relationship significant, $p < 0.05$.

Table 10. Daily cardiovascular (CV) admissions, CO, and PM_{10}: statistics for the two most contrasting regions.

Statistic	Season	Region 4 (Long Beach)	Region 6 (Riverside)
CV admission count interquartile range	All	48–67	43–57
	Winter	52–71	44–59
CO interquartile range (ppm)	All	0.93–2.40	0.81–1.58
	Winter	1.33–3.17	0.74–1.70
PM_{10} interquartile range (μg/m^3)	All	28–45	44–86
	Winter	20–43	24–61
Correlation, CO vs. PM_{10}	All	0.56*	0.55*
	Winter	0.70*	0.75*
Correlation, CO vs. PM_{10} residuals[a]	All	0.63*	0.69*
	Winter	0.72*	0.76*
Regression slope, CV vs. CO[b]	All	0.022 (0.005)*	0.012 (0.008)
	Winter	0.027 (0.008)*	0.017 (0.015)
Regression slope, CV vs. PM_{10}[b]	All	0.0012 (0.0003)*	0.0001 (0.0002)
	Winter	0.0018 (0.0006)*	0.0003 (0.0004)

[a]Observed values minus values predicted by day of the week and cubic-spline smooth of longer term trends. [b]From single-pollutant autoregressive Poisson models including temporal and weather effects. *Significant, $p < 0.05$.

Conclusion

In general, our results from metropolitan Los Angeles appear consistent with reports from elsewhere (*7–12*) that day-to-day increases in urban CO and/or PM_{10} and/or NO_2 are associated with meaningful increases in cardiovascular illnesses. We found only a few equivocally positive relationships between cardiopulmonary morbidity and O_3, in situations when other pollutants and heat stress increased along with O_3. This is surprising, in light of severe O_3 pollution in Los Angeles, obvious acute respiratory effects of O_3 in animal and human exposure studies (*2*), and recent observations of O_3-related hospital admissions in Toronto, Canada, where O_3 levels are lower than in Los Angeles (*12*). O_3 has been linked to mortality in Los Angeles (*13*), although PM_{10} might explain that association (*25*). On the other hand, a recent time–series study of asthma admissions in central and western Los Angeles (*26*) generally supports our findings, showing associations with PM_{10} but not with O_3. The tendency of O_3 concentrations to decrease indoors, where most people spend most of their time (*27*), might attenuate morbidity/O_3 relationships, but would not likely do so in Los Angeles more than in Toronto. In any event, our results suggest that the excess risk of hospitalization in Los Angeles is greater on high-primary-pollution days than on high-O_3 days. The greatest risk of pollution-related hospital admissions apparently occurs on autumn/winter days with weak Santa Ana weather conditions, when air incursion from the desert approximately counterbalances that from the ocean, resulting in maximal atmospheric stagnation.

We could not distinguish clearly among CO-, NO_2- and particle-associated effects. CO showed the strongest statistical relationships with most indices of morbidity even in the regions and seasons with the highest and widest ranging PM_{10}. NO_2 tracked CO closely enough that CO-associated effects might reasonably be attributed to NO_2 and/or another oxide of nitrogen. Weaker statistical relationships of illness to PM_{10} might have resulted from less accurate exposure assessment even if PM_{10} were inherently more toxic (*19*). Too little is known about the relationships between the ambient background and personal exposures to judge which pollutants are most subject to exposure misclassification. Even if PM_{10}/morbidity associations were entirely explainable by CO/morbidity associations, some particulate species closely associated with CO might be the active agent(s). Alternatively, our findings might reflect separate effects of CO and some

component of PM_{10}, as suggested by the Schwartz (*9*) Tucson, Arizona, study. One argument against CO effects per se is that typical ambient background CO concentrations are below normal bloodstream concentrations of metabolically produced CO (*3*). Even on most high-CO days in Los Angeles, inhaling the background concentration should reduce the blood's oxygen-carrying capacity by < 1%. However, CO concentrations near sources (e.g., heavy traffic) exceed background levels and may cause appreciable cardiovascular stress (*9,10*). If both are driven by atmospheric stagnation, these higher microenvironmental concentrations should track background levels. Thus, a low range of monitored background CO does not necessarily rule out an effect of CO on cardiovascular morbidity.

The observed association of all pulmonary diseases with PM_{10} or NO_2 more than CO, and of all cardiovascular diseases with CO more than PM_{10} (Table 5), appears consistent with the well-known properties of CO as a circulatory toxicant without direct effects on the lungs, and of some particulate species as respiratory irritants. The association of occlusive strokes with all four tested pollutants appears consistent with the hypothesis of Seaton et al. (*28*) that urban pollution provokes alveolar inflammation, releasing mediators which increase blood coagulability. A previous finding of increased plasma viscosity during a primary pollution episode in Germany (*29*) also supports that hypothesis. By our data, we cannot test Seaton et al.'s (*28*) attribution of the inflammatory effect to ultrafine particles.

We found possibly meaningful demographic differences in morbidity/pollution relationships, although none of them attained statistical significance. Persons ≥ 65 years of age and diabetics showed somewhat increased cardiovascular disease effects as compared to others without those risk factors, but men did not appear to be more at risk than women of similar age. Persons younger than 30 years of age showed the largest pollution-related effects on asthma. Although air pollution health risks are believed to fall disproportionately on ethnic minorities (*30*), whites usually showed the largest pollution-related effects on cardiovascular disease. Effect sizes in blacks, the minority group generally at greatest risk for cardiovascular disease, were similar to those in whites, whereas effect sizes were generally smaller in Hispanics and undetectable in the other (predominantly Asian) ethnic category. Definitive interpretation would require evaluation of ethnic differences in exposure, susceptibility, and access to hospitals. On average, O_3 exposures in the basin appear higher for whites than for blacks or Asian/Pacific Islanders (*31*). Differences in other exposures apparently have not been studied formally, but the high-primary-pollution regions 2 and 4 have high proportions of minority residents. Thus, smaller effects in some minorities (if real) probably are not explained by less exposure.

The relatively nonspecific pattern of diagnoses suggests that excess patients admitted to hospitals on high-pollution days in metropolitan Los Angeles are individuals with preexisting problems which make them highly vulnerable to any extra stresses on their oxygen delivery systems, including unfavorable changes in the air environment. If so, generalized efforts to preserve cardiopulmonary health should help to prevent (or at least to postpone) pollution-associated illnesses. Our findings suggest that control of primary pollutants is more important to public health than control of O_3, which in any case depends on control of primary pollutants.

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